CATÓLICA
INSTITUTO DE CIÊNCIAS DA SAÚDE

LISBOA · PORTO · VISEU

MESTRADO INTEGRADO EM MEDICINA DENTÁRIA

BIOMEDICINA: BIOLOGIA COMPUTACIONAL E DIAGNÓSTICO SALIVAR

BIOMARCADORES SALIVARES PARA AS DOENÇAS CARDIOVASCULARES EM PACIENTES PORTADORES DE PERIODONTITE CRÓNICA

Dissertação apresentada à Universidade Católica Portuguesa para obtenção do grau de Mestre em Medicina Dentária

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Rafael José Silva Cunha

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Sob a orientação do Professor Doutor Nuno Rosa e co-orientação da Professora Doutora Marlene Barros

Viseu, 2016

"Não faz sentido olhar para trás e pensar: deveria ter feito isto ou aquilo, deveria ter estado lá. Isso não importa. Vamos inventar o amanhã e parar de nos preocupar com o passado."

Steve Jobs

Agradecimentos:

Ao professor Doutor Nuno Rosa, por toda a sua bondade, disponibilidade, paciência, conselhos e sobretudo por todos os conhecimentos transmitidos considerados indispensáveis à conclusão deste trabalho,

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À minha irmã, pelo amor, carinho e apoio incondicional transmitido ao longo destes anos académicos,

À Alexia Oliveira, minha namorada, pelo amor, companheirismo, inestimável apoio e pela enorme compreensão ao longo de toda esta caminhada,

Ao Luís, meu binómio, pela enorme amizade, paciência, ajuda e sobretudo pelos maravilhosos anos de confraternidade vivenciados,

Aos que trabalham no SalivaTec, por se disponibilizarem em todos os momentos para esclarecer qualquer dúvida e do mesmo modo terem proporcionado um celestial meio de trabalho,

Aos meus amigos e colegas de curso, por todos os fantásticos momentos passados durante o meu percurso académico.

Resumo

Introdução: As doenças cardiovasculares são a principal causa de morbilidade e mortalidade em todo o mundo, sendo a aterosclerose a doença cardiovascular mais frequente. Estudos recentes têm evidenciado uma relação entre a periodontite crónica e as doenças cardiovasculares. A plausibilidade biológica para esta associação, baseia-se no facto de os pacientes com periodontite apresentarem níveis aumentados de marcadores inflamatórios, os quais estão associados com a disfunção do endotélio e no desenvolvimento das doenças cardiovasculares. Contudo, os mecanismos subjacentes a esses efeitos ainda não estão totalmente esclarecidos.

Objetivos: Desta forma é objetivo deste trabalho esclarecer quais os mecanismos biológicos e moleculares envolvidos e também avaliar se as proteínas salivares poderão ou não constituir potenciais biomarcadores diferenciadores das duas condições, bem como da associação entre a periodontite crónica e as doenças cardiovasculares.

Material e métodos: Para cumprir estes objetivos, foi feita uma pesquisa bibliográfica com recurso à ferramenta de pesquisa do portal PubMed, usando as palavras-chave, “cardiovascular diseases”, “chronic periodontitis”, “biomarkers”, “proteomics”, “salivar diagnosis”. Posteriormente, foi efetuada a anotação manual da informação relativa aos proteomas de ambas as patologias, com consequente criação de uma base de dados que permitiu atualizar o OralCard. Por último, para ser feito um enriquecimento dos mecanismos envolvidos nas patologias, procedeu-se à ferramenta bioinformática PANTHER.

Resultados: A caracterização funcional do OralOma das patologias em estudo permitiu identificar alterações em determinados processos biológicos e funções moleculares. A pesquisa e anotação do proteoma das doenças cardiovasculares, possibilitou adicionar 25 proteínas à base de dados

OralCard, permitindo pela primeira vez compilar informação relativamente às doenças cardiovasculares nesta base de dados.

Este trabalho permitiu demonstrar que existem diferenças em relação ao tipo e quantificação das proteínas salivares comuns em ambas as patologias. Este conhecimento permitirá aos investigadores nesta área efetuarem estudos no sentido de identificarem biomarcadores diferenciadores das duas condições, bem como da sua associação.

Palavras-chave: Doenças cardiovasculares, periodontite crónica, biomarcadores, proteómica, diagnóstico salivar.

Abstract

Introduction: Cardiovascular diseases are the leading cause of morbidity and mortality in the world, and the atherosclerosis is the most common cardiovascular disease. Recent studies have shown a relationship between chronic periodontitis and cardiovascular diseases. The biological plausibility for this association is based on the fact that the patients with periodontitis presenting increased levels of inflammation markers which are associated with the dysfunction of endothelium and the development of cardiovascular diseases. However, the mechanisms underlying these effects are not yet fully understood.

Objectives: In this way, the aim of this work is clarify the biological and molecular mechanisms involved and to evaluate whether the salivary proteins may or may not be potential biomarkers differentiating the two conditions, as well as the association between chronic periodontitis and cardiovascular disease.

Methods: To achieve these objectives, was made a literature search using the PubMed portal search tool, using the keywords, "cardiovascular diseases", "chronic periodontitis", "biomarkers", "proteomics", "salivary diagnosis". Subsequently, it was performed the manual annotation information concerning proteome of both diseases, with consequent creation of a database which enable update the OralCard. Finally, to be made an enrichment of the mechanisms involved in pathologies, we used the PANTHER bioinformatics tool.

Results: The functional characterization of OralOme of diseases under study enabled the identification of changes in certain biological processes and molecular functions. The establishment of cardiovascular diseases proteoma allowed the addition of 25 new proteins to the OralCard data base. With this, it was possible, for the first time, to compile the information about cardiovascular

diseases to this database. This research showed the existence of differences between the salivary protein quantification of the proteins common to both diseases. This knowledge will allow researchers to endorse studies on this area in order to identify differentiating biomarkers between these two conditions, as well as their association.

***Key words*:** cardiovascular diseases, chronic periodontitis, biomarkers, proteomics, salivary diagnostics.

Nota Prévia

No decorrer desta dissertação irrompem algumas vezes termos na língua inglesa, redigidos em itálico de forma a preservar a imutabilidade universal dos termos usados para descrever conceitos biológicos, entre culturas e línguas diferentes. A classificação de proteínas consoante um sistema de classificação de ontologias implica a sua utilização em língua inglesa, já que a tradução dos mesmos poderia não só alterar o seu significado original, mas também o sentido biológico que lhes são inerentes.

ÍNDICE

Lista de abreviaturas

CP– *Chronic periodontitis*

CRP– *C-reactive protein*

CVD – *Cardiovascular disease*

ICAM-1 – *Intercellular adhesion molecule 1*

IL-1– Interleukin 1

IL-1β– *Interleukin 1 Beta*

IL-6 – *Interleukin 6*

MeSH – *Medical Subject Headings*

MMP-8– *Neutrophil collagenase*

MMP-9– *Matrix metalloproteinase-9*

MMPs– *Matrix metalloproteinases*

NOS – *Nitric oxide synthase*

OMIM– *Online Mendelian Inheritance in Man*

OMS– *Organização Mundial de Saúde*

PANTHER– *Proteín Analysis Through Evolutionary Relationships*

PGE2– *Prostaglandin E2 receptor EP2 subtype*

PMNs– *Polymorphonuclear leukocytes*

TIMP-1 – *Metalloproteinase inhibitor 1*

TNF-α– *Tumor Necrosis Factor alpha*

UniProtKB – *Universal Protein Knowledgebase*

VCAM-1 – *Vascular cell adhesion molecule 1*

ÍNDICE DE FIGURAS

1. INTRODUÇÃO

1.1. DOENÇAS CARDIOVASCULARES

1.1.1. Definição e Epidemiologia

As doenças cardiovasculares são a principal causa de morbilidade e mortalidade em todo o mundo, sendo a aterosclerose a doença cardiovascular mais frequente e também considerada como o principal fator etiológico da mesma [1–3]. A aterosclerose é uma doença progressiva e caracterizada por um acúmulo de partículas lipídicas, elementos fibrosos na parede arterial e pela formação de ateromas, reduzindo desde logo o lúmen arterial, predispondo à trombose coronária, obstrução e resultando em fenómenos isquémicos. Conceitos mais atuais têm introduzido a noção de que, para além do processo trombótico associado, a inflamação e a disfunção endotelial estão também relacionadas com todas as fases da aterosclerose [1,3,4].

Segundo a Organização Mundial de Saúde (OMS), é estimado que as doenças cardiovasculares matem 15,6 milhões de pessoas por ano em todo o mundo, perfazendo um total de 29,6% de todas as mortes. Também na europa é considerada a principal causa de morte (Figura 1), provocando a mesma a 4 milhões de pessoas (quase o dobro das provocadas pelo cancro em todo o continente) [2].

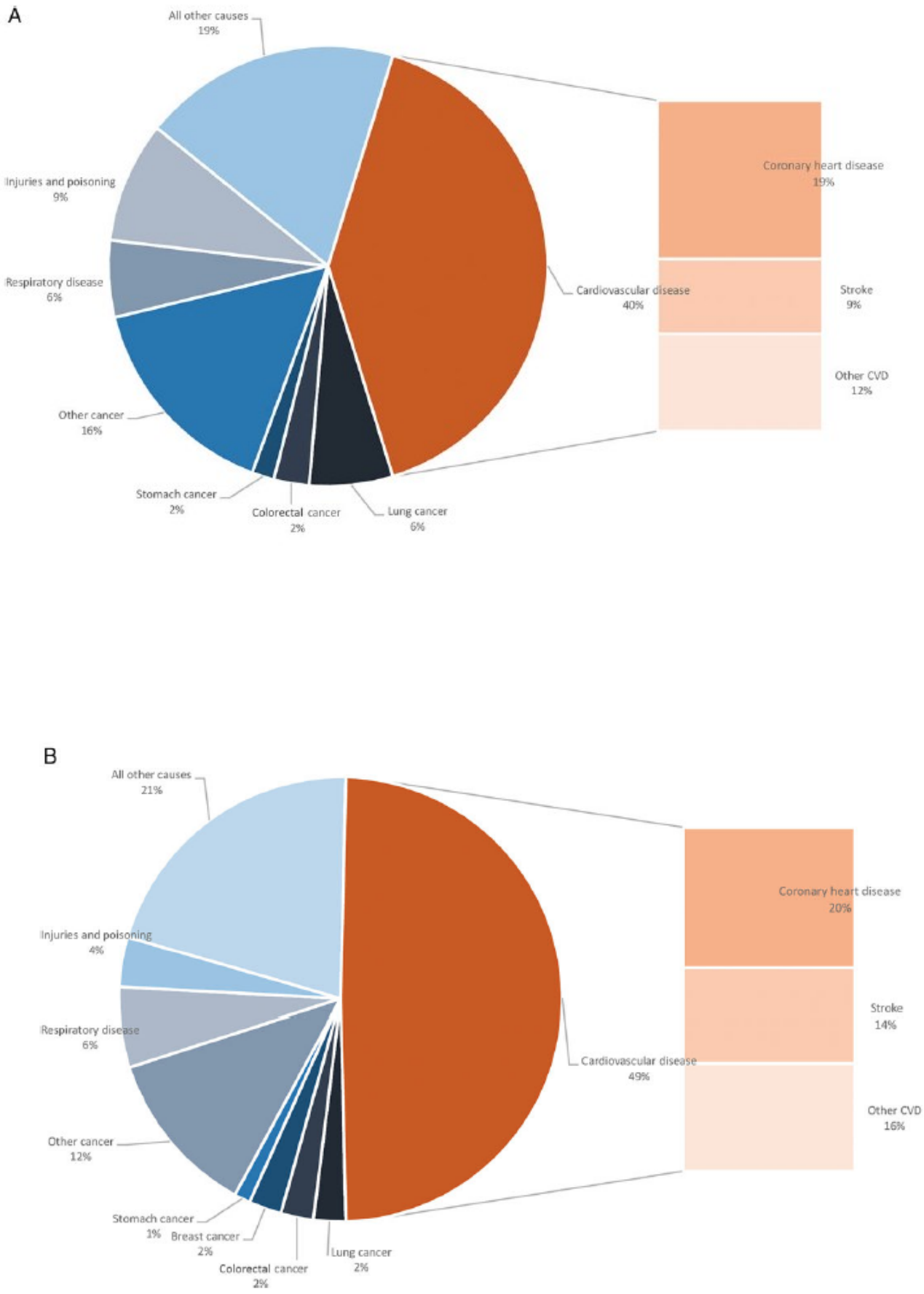


***Figura 1**-Proporção de todas as principais causas de morte na Europa, entre homens (A) e mulheres (B). Sem dados disponíveis em Andorra. Adaptado de Townsend et al (2015)[2].*

1.1.2. Plausibilidade biológica da relação entre a periodontite crónica e as doenças cardiovasculares

Estudos recentes têm evidenciado que para além do processo trombótico associado às doenças cardiovasculares, a inflamação e a disfunção endotelial estão também diretamente relacionadas. Neste sentido, sendo a periodontite caracterizada por ser uma infeção/inflamação crónica que envolve as estruturas anatómicas que rodeiam e suportam os dentes (gengiva, ligamento periodontal e osso alveolar), tem vindo a ser associada com as doenças cardiovasculares [1,5,6]. A plausibilidade biológica para esta relação, baseia-se no facto de os pacientes com periodontite apresentarem níveis aumentados de marcadores inflamatórios (Figura 2), os quais estão associados com a disfunção do endotélio e a ocorrência de episódios cardiovasculares [3].

As citocinas pró-inflamatórias, tais como, *TNF-α*, *IL-1*, *IL-6*, reduzem a expressão endotelial do *NOS* (óxido nítrico sintase). Estas também promovem a expressão da *VCAM-1*, *ICAM-1*, *E-selectina* e *P-selectina*, resultando na ausência de propriedades antiaterogénicas no endotélio. Com efeito, aumentam a migração de leucócitos, na ativação de plaquetas, facilitando a formação e destabilização da placa aterosclerótica [3,7].

O estado de inflamação crónica induzido pela periodontite parece favorecer a formação e o desenvolvimento das doenças cardiovasculares, no entanto são necessários novos estudos que tentem esclarecer completamente os mecanismos biológicos envolvidos nestas patologias[1,3,8].

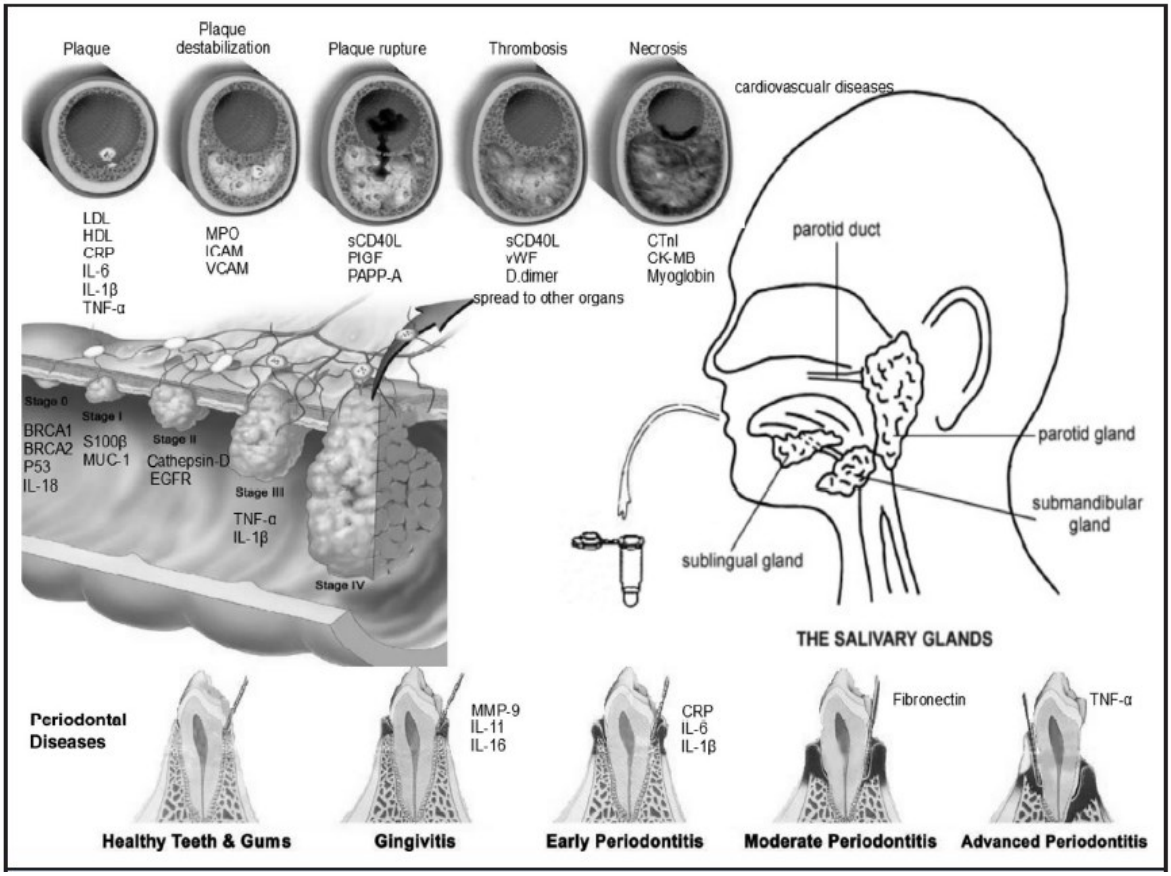


***Figura 2**- Biomarcadores aumentados na saliva durante a inflamação local (doença periodontal) e durante a inflamação sistémica (no desenvolvimento da placa de aterosclerose nas artérias). Adaptado de Pfaffe et al (2011)[9].*

1.1.3. O papel da inflamação no desenvolvimento das doenças cardiovasculares

O desenvolvimento das doenças cardiovasculares começa frequentemente com a ocorrência de mudanças inflamatórias presentes no endotélio, onde as células inflamatórias começam por expressar moléculas de adesão, às quais os leucócitos podem aderir e migrar até à superfície endotelial. Esta adesão acontece graças às interações que ocorrem no endotélio com as selectinas, moléculas de adesão vasculares (*VCAM-1*), intracelulares (*ICAM-1*) e também pelas integrinas presentes na superfície dos leucócitos (Figura 3) [10–12].

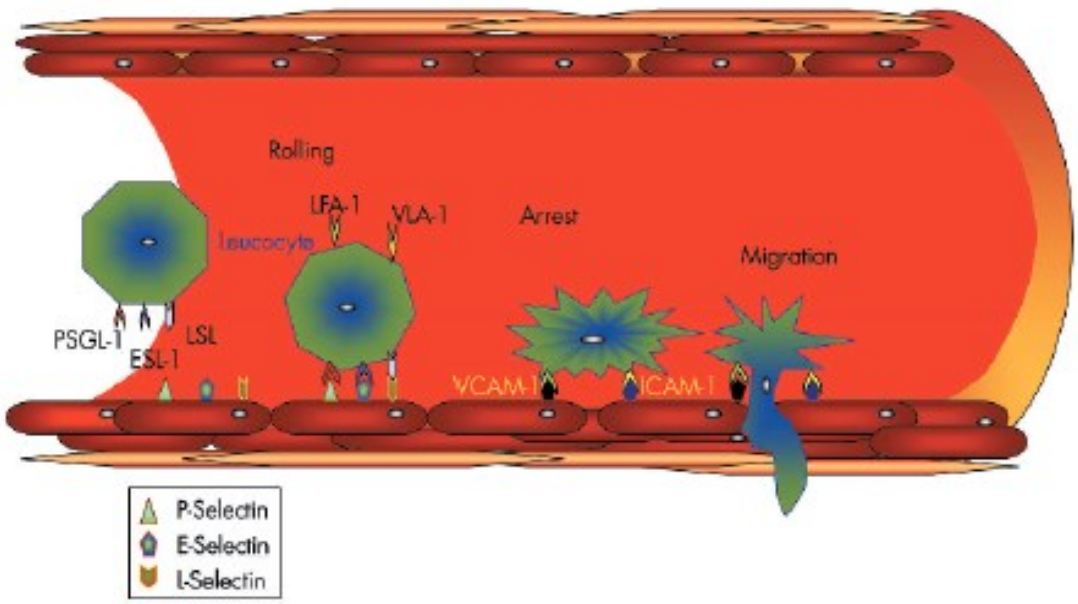


***Figura 3**- A imagem evidencia as Interações que ocorrem entre os leucócitos e as células endoteliais na aterosclerose. Adaptado de Tousoulis et al (2008)[12].*

Os monócitos são atraídos por estas moléculas de adesão e migram até à camada endotelial através de várias quimiocinas pró-inflamatórias [13]. Após a adesão leucocitária, os leucócitos (Linfócitos T, monócitos/macrófagos) acabam por penetrar na camada íntima do endotélio (Figura 4-A)[10,12].

Uma vez dentro da camada íntima, os monócitos continuam a sofrer alterações inflamatórias, transformando-se em macrófagos (Figura 4-B). Estes, são considerados mediadores importantes de inflamação e da resposta imune inata nas lesões ateroscleróticas. Os macrófagos contribuem para a resposta inflamatória local através da produção de citocinas, radicais livres de oxigénio e protéases [10,12].

Estas células são também responsáveis pela captação de lípidos modificados, principalmente o LDL oxidado, pela acumulação de ésteres do colesterol e formação das células espumosas[11–14]. Por outro lado, podem também contribuir para a remodelação da lesão e rotura da placa através da secreção de metaloproteinases da matriz (*MMPs*) e, consequente, evolução da aterosclerose [10,12].

Com a evolução da placa, os Linfócitos T migram até à camada íntima, juntando-se aos macrófagos, onde vão libertar citocinas pró-inflamatórias (que

amplificam a atividade inflamatória) e fatores de crescimento que promovem a migração e proliferação das células musculares lisas (Figura 4-B)[10,14].

Através destes processos inflamatórios, a lesão aterosclerótica vai continuando a progredir cada vez mais até se tornar numa placa complexa [10,12,14]. A secreção das citocinas, vai inibir a produção do colagénio das células musculares lisas e estimulam os macrófagos a expressar enzimas com atividade de colagenase (*MMPs*) (Figura 4-C)[10].

As *MMPs*, são então responsáveis por degradar o colagénio existente na cápsula fibrosa, tornando-a mais fina e friável, ficando assim mais propensa a sofrer a rotura da placa (Figura 4-C)[13,14].

Deste modo, a inflamação é um processo constante em todas as fases no desenvolvimento das doenças cardiovasculares, desde a disfunção do endotélio vascular, à formação e rotura da placa aterosclerótica, responsável pela maior parte das complicações dos eventos cardiovasculares [11–15].

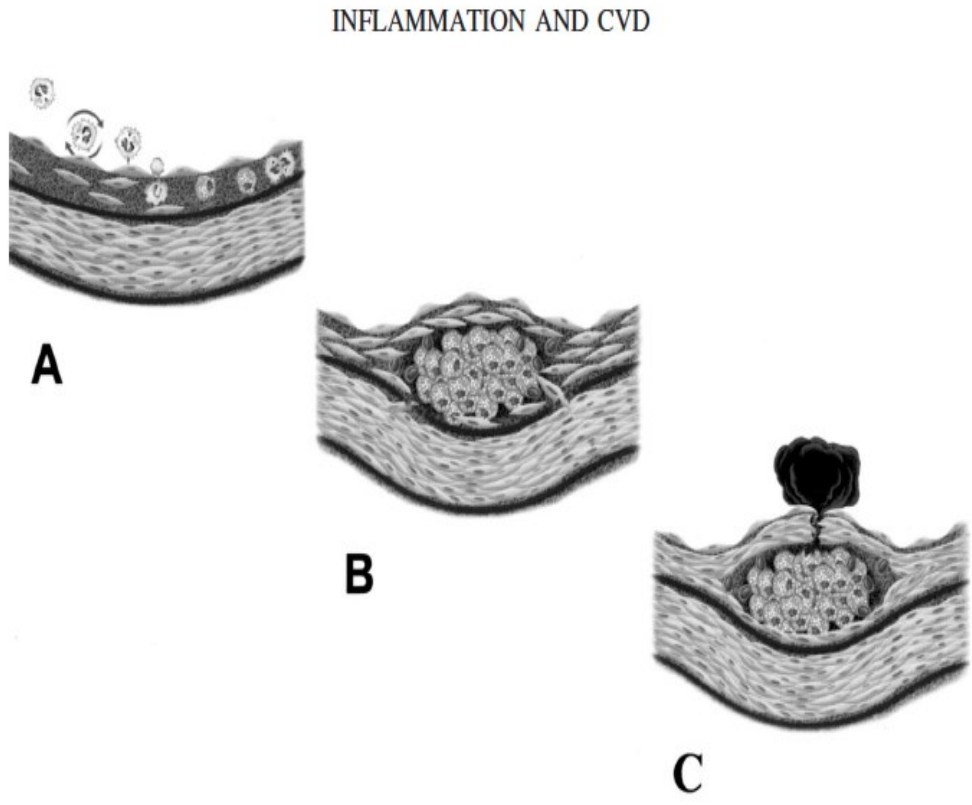


***Figura 4**- Imagem representativa da participação da inflamação em todas as fases da aterosclerose. Adaptado de Libby (2006)[10]*

1.2. SALIVA

A saliva é o único fluído humano que banha de forma contínua a mucosa da cavidade oral, orofaringe e laringe [16]. Este fluído é caracterizado por ter inúmeras funções biológicas, tais como: a actividade antimicrobiana, antiviral, servindo de barreira contra os agentes patogénicos, lubrificação dos tecidos, capacidade de tampão, ingestão de alimentos e degustação, essenciais para a manutenção da saúde oral [17–21]. Além do papel importante na manutenção da homeostasia do sistema da cavidade oral, a saliva é considerada como um meio para a monitorização dos estados de saúde e doença, tanto da cavidade oral como sistémicos [18].

1.2.1. Origem e composição

Em geral, um adulto saudável produz em média, 500-1500 mL de saliva por dia, a uma taxa de aproximadamente 0,5 mL/min, porém, várias condições fisiológicas e patológicas (tais como: sangramento na mucosa oral, esclerose múltipla, epilepsia, fibrose quística) podem modificar a sua produção de forma quantitativa e qualitativamente. Cheirar e provar estimula a produção e secreção da saliva, bem como a mastigação, estados psicológicos e hormonais, drogas, idade, influências hereditárias, higiene oral e exercício físico [9,22].

A secreção da saliva é proveniente de três grandes pares de glândulas salivares: parótidas (20%), submandibulares (65-75%) e sublinguais (7-8%). Estas são responsáveis pela produção de aproximadamente 90% da saliva total, enquanto que as glândulas salivares menores segregam a restante [20,23,24]. As principais glândulas estão localizadas dentro e à volta da boca e garganta, enquanto que as glândulas menores (aproximadamente 600) estão localizadas em toda a extensão da cavidade oral (lábio, língua, palatoglosso e mucosa palatina)[20,23].

É possível designar dois tipos salivares consoante a sua origem: a saliva específica de cada glândula secretora e a saliva total. A avaliação de secreções provenientes de glândulas salivares individuais é normalmente útil para a deteção de patologia numa determinada glândula específica (ex: infeção

e obstrução). Todavia, a saliva total é mais frequentemente analisada para avaliar a existência de doenças sistémicas. A saliva total é um fluído misto, que é derivado não só das glândulas salivares maiores e menores, mas também de origens não-salivares, contendo fluído crevicular gengival, secreções brônquicas e expetoração nasal, soro e derivados de sangue de feridas orais, bactérias e os seus produtos, vírus e fungos, células epiteliais descamadas e restos alimentares [23].

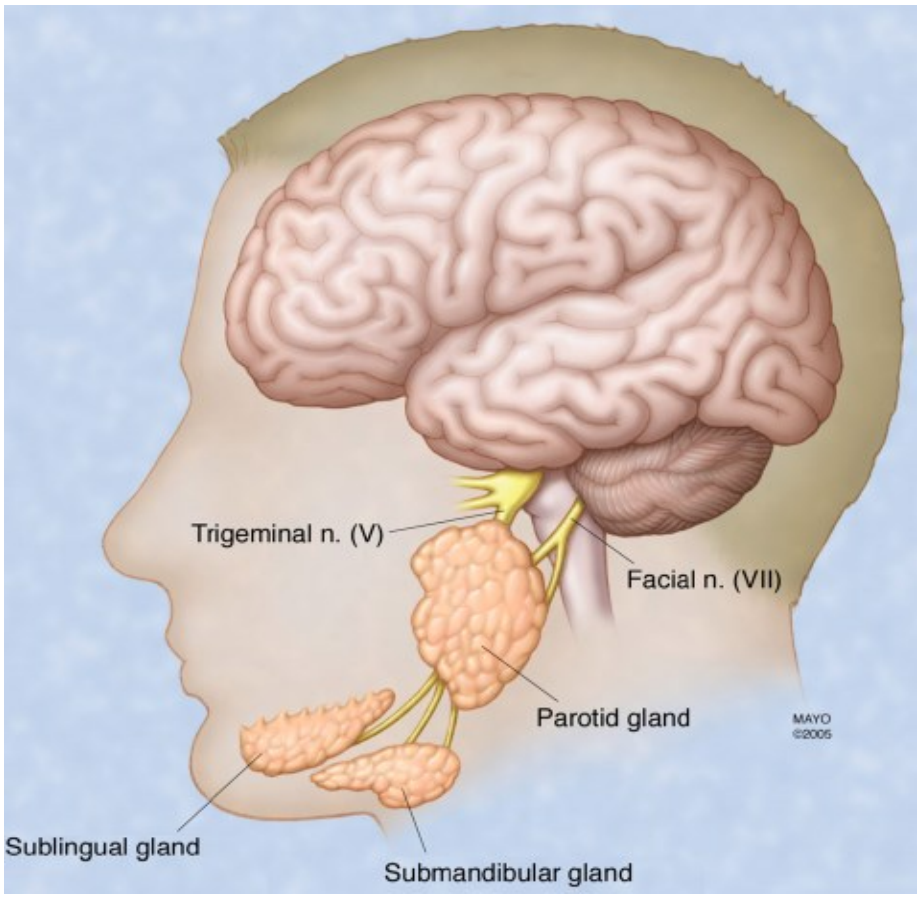


***Figura 5**- Vista lateral da cabeça, mostrando as posições das principais glândulas salivares (parótida, submandibular e sublingual), bem como a sua inervação (nervo trigémio e facial) (adaptado de Yoshizawa et al (2013)[20]).*

No que toca ao fluxo salivar, composição salivar e concentração de cada proteína, estas apresentam variações interindividuais dependendo de local para local no interior da cavidade oral, da altura do dia, da proximidade das refeições, se resulta de uma produção estimulada ou não estimulada. As propriedades do fluxo salivar podem ainda ser afetadas por outros fatores

como a idade, enzimas microbianas orais, atividade das proteases, doenças sistémicas, nutrição, género e o consumo de álcool [9,17,22,24,25].

Assim como o sangue, a saliva é composta por uma enorme variedade de eletrólitos, vitaminas, hormonas, enzimas, anticorpos, fatores de crescimento, constituintes antimicrobianos, citocinas e outras proteínas [22,26,27]. Diversos constituintes podem ser encontrados na saliva em menores quantidades (sódio, magnésio, cloreto), maiores (potássio, cálcio, bicarbonato, fosfato) e em concentrações semelhantes (ácido úrico), comparativamente ao sangue [22]. Os mecanismos pelos quais estas moléculas e substâncias surgem no fluído salivar a partir do sangue são múltiplos, podendo ocorrer por meio de difusão passiva e o transporte ativo (vias intracelulares) e por ultrafiltração (via extracelular), dentro das glândulas salivares ou a partir do fluído crevicular gengival [9,26,27].

1.2.2. A saliva como fluído de diagnóstico

Como referido anteriormente, a saliva tem sido reconhecida como um verdadeiro “espelho” da saúde do corpo, já que integra constituintes que podem ser medidos e usados para avaliar estados de saúde e doença, orais e sistémicos (figura 6) [23].

Como fluído de diagnóstico, a saliva apresenta algumas vantagens distintas comparativamente ao soro, podendo ser recolhida de forma não-invasiva e por indivíduos não especializados. Esta pode ser obtida com equipamentos relativamente simples, fornecendo uma abordagem de custo-benefício promissora para o rastreio de um número elevado de pessoas [9,18,23].

A recolha de forma não-invasiva reduz drasticamente a ansiedade e o desconforto, aumentando a vontade dos indivíduos em efetuarem análises com uma periodicidade mais curta e podendo ser usada em situações clínicas desafiadoras, como a obtenção de amostras de crianças ou pacientes com deficiências, em que a recolha através do sangue poderia ser um ato difícil de executar [9,23]. Complementarmente, é também mais fácil de manusear durante os procedimentos de diagnóstico do que o sangue, uma vez que não coagula, reduzindo desde logo o número de manipulações necessárias.

Além disso, para os profissionais de saúde, é considerado um teste bem mais seguro, existindo um risco mínimo de infeções durante a sua recolha [18,23,26].

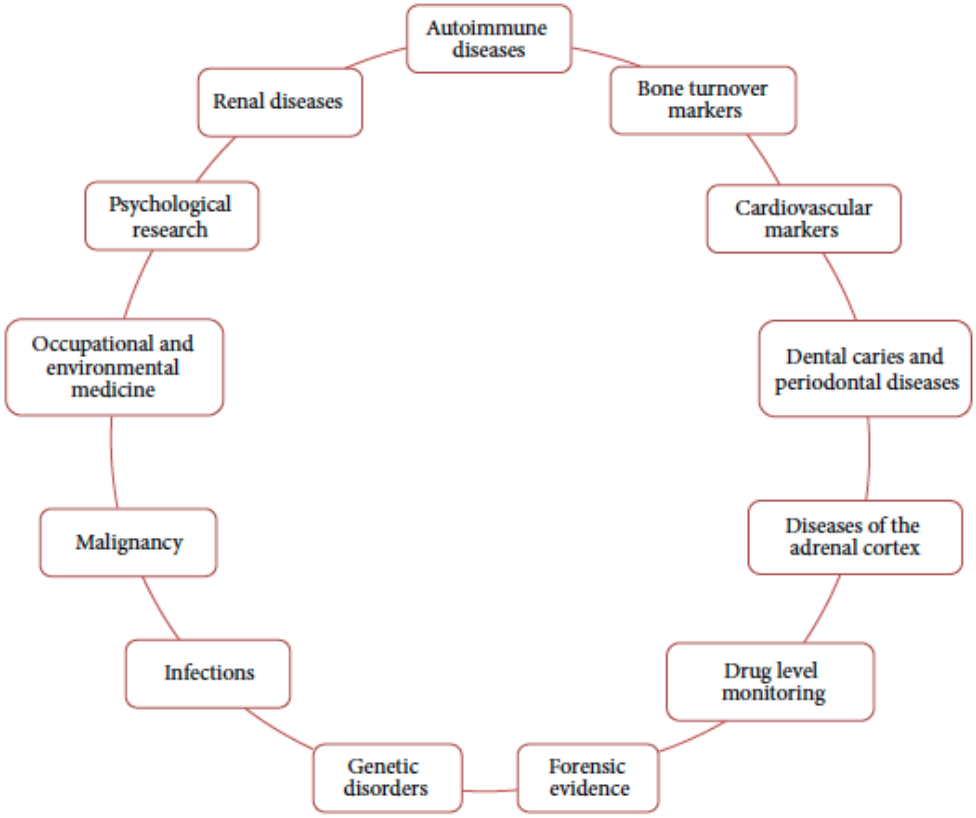


***Figura 6** - Representação esquemática da capacidade de deteção de doenças sistémicas segundo análises salivares. Adaptado de Malathi et al (2014)[27].*

Considerando que as proteínas são os constituintes mais importantes da saliva, uma análise abrangente nos últimos anos tem conduzido à identificação do conteúdo proteómico da saliva, esta considerada essencial para caracterizar as funções das proteínas salivares e revelar potenciais biomarcadores envolvidos em diferentes condições fisiopatológicas [9].

Contudo, a descrição do estado dos proteomas, não é só ser capaz de utilizar essa informação para compreender melhor os sistemas biológicos, mas também para a sua utilização em vertentes clínicas. Uma análise global dos proteomas salivares humanos pode fornecer um espectro abrangente da saúde oral e geral, através de alterações na expressão das proteínas [28].

Em direção a estes objetivos, a identificação da presença de proteínas não é considerada suficiente, ou seja, estes objetivos requerem uma caracterização detalhada da proteína [17]. Com os avanços importantes nas tecnologias usadas na área da proteómica, nomeadamente a espectrometria de massa, foi possível identificar e caracterizar proteínas e péptidos de forma mais precisa, levando a uma ampla gama de aplicações para os ensaios de proteómica e tornando-a ideal para a deteção de biomarcadores [19,28]. Deste modo, os proteomas salivares humanos representam um campo de pesquisa biomédica interessante, tanto para o diagnóstico como para a monitorização de diversas condições patológicas, nomeadamente as doenças cardiovasculares [4,28,29].

1.2.3. Biomarcadores salivares de suscetibilidade às doenças cardiovasculares

Dado o grande impacto que as doenças cardiovasculares têm no mundo inteiro, torna-se fulcral estabelecer-se um diagnóstico correto o mais cedo possível, visto que a ausência de sintomas na fase inicial leva a um diagnóstico tardio [23,30]. Independentemente dos médicos terem uma grande experiência clínica, medidas objetivas e baseadas na evidência são cada vez mais necessárias no momento do diagnóstico [23,30].

Assim, novos estudos de proteómica do fluído salivar humano, abriram novas portas para a descoberta de potenciais biomarcadores de suscetibilidade de um individuo ao desenvolvimento das doenças cardiovasculares [30,31].

O termo biomarcador tem sido usado ao longo dos últimos anos, no sentido lato, para designar uma espécie molecular significativa num fluído (tal como o sangue ou saliva). No sentido restrito, é definido como uma medida farmacológica ou fisiológica utilizada na predição de um evento tóxico; tratando-se de uma molécula específica no organismo, que tem uma determinada característica que a converte num ótimo instrumento de medida para o diagnóstico, monitorização ou efeitos de um determinado tratamento [32,33].

Neste sentido, a identificação de proteínas salivares como biomarcadores nos estágios iniciais das doenças cardiovasculares poderá no

futuro, possibilitar a identificação dos pacientes como suscetíveis, quando um determinado marcador molecular se encontrar presente ou alterado na análise salivar executada.

Desta forma, podem alterar-se estilos de vida e desenhar-se intervenções terapêuticas que conduzem ao aumento das taxas de sobrevivência, diminuição do sofrimento e redução da probabilidade de reincidência [4,29,34].

2. OBJETIVOS

A realização do presente trabalho tem como fundamento o estudo de alterações proteicas salivares em pacientes com periodontite crónica e doenças cardiovasculares, que auxiliem o esclarecimento dos mecanismos biológicos que correlacionam as duas patologias, bem como a identificação de potenciais biomarcadores salivares de doença cardiovascular.

Deste modo, os principais objetivos deste trabalho são:

- Revisão da literatura científica de modo a identificar estudos de proteómica da cavidade oral em periodontite crónica e doenças cardiovasculares;
- Catalogação das proteínas humanas da cavidade oral de pacientes com periodontite crónica e doenças cardiovasculares, anotando manualmente as informações consideradas pertinentes para a interpretação de dados destas patologias no OralCard [35];
- Avaliação das principais diferenças entre o proteoma salivar em indivíduos que apresentem apenas periodontite crónica e indivíduos que apresentem periodontite crónica e doenças cardiovasculares simultaneamente;
- Análise funcional das proteínas salivares em periodontite crónica e doenças cardiovasculares no sentido de esclarecer os mecanismos moleculares de cada uma das patologias;
- Identificação de possíveis biomarcadores diferenciadores das duas condições, bem como da associação entre a periodontite crónica e a patologia cardiovascular.

3. MATERIAIS E MÉTODOS

3.1. PROTEOMA ORAL HUMANO EM PERIODONTITE CRÓNICA E DOENÇAS CARDIOVASCULARES

3.1.1. Seleção dos estudos

Foi realizada uma pesquisa bibliográfica com recurso à ferramenta de pesquisa do portal PubMed, usando como palavras-chave os seguintes termos MeSH (*Medical Subject Headings*): “cardiovascular diseases”, “chronic periodontitis”, “biomarkers”, “proteomics”, “salivar diagnosis”.

De maneira a ser executada uma revisão ordenada fundamentada na correlação entre as proteínas salivares da periodontite crónica e das doenças cardiovasculares, foram aplicados os seguintes filtros de pesquisa: “*Humans*” e “*Clinical trial*”, com a finalidade em serem incluídos, exclusivamente, artigos de índole experimental referentes à espécie *Homo Sapiens*.

Fundamentado na análise dos artigos compilados foi realizado um levantamento de todas as proteínas salivares identificadas em pacientes com periodontite crónica e patologia cardiovascular em conjunto, ou isoladamente , que ainda não tinham sido depositadas na base de dados do proteoma oral do OralCard, com vista à atualização da base de dados alusiva ao Oraloma humano das doenças em estudo.

3.1.2. Anotação manual da informação relativa aos proteomas de ambas as patologias em estudo

A análise detalhada de todos os estudos experimentais selecionados possibilitou consumar a anotação manual dos dados referentes às proteínas identificadas na saliva em pacientes com periodontite crónica e com doenças cardiovasculares. A informação anotada manualmente é essencial para a interpretação funcional dos dados de proteómica recolhidos.

Para se obter uma organização seriada das informações recolhidas, foram construídas tabelas integrando as diversas características das proteínas, tais como: dados de identificação das proteínas, o local da proveniência da

amostra, a relação do estado de saúde e doença, a caracterização do dador da amostra, a caracterização dos métodos de recolha e análise da amostra, bem como da presença de modificações pós-tradução nas proteínas identificadas e se estas já foram ou não propostas pelo autor como biomarcadores (Figura 7).

UniprotKBAC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL
Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro
Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*
Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	Biomarker	Citation (NCBI ID)	Obs.

***Figura 7**- Cabeçalhos que permitem a organização da informação catalogada dos diferentes estudos de proteómica analisados (proteínas identificadas na periodontite crónica e doenças cardiovasculares) numa tabela em Microsoft Excel 2011.*

3.1.2.1. Identificação das proteínas salivares em periodontite crónica e doenças cardiovasculares

No que concerne à identificação das proteínas salivares foram usados quatro campos (Figura 8) para efetuar o registo da informação relativa das proteínas.

UniprotKBAC	Gene name	Name	Organism

***Figura 8**- Cabeçalho da tabela em Microsoft Excel 2011 referente à identificação de cada proteína anotada manualmente.*

Com suporte da pesquisa e seleção na base de dados *online* UniProtKB [36–38] (https://www.uniprot.org/) (Figura 9) é concedido um código de identificação universal (UniProtKB) a cada proteína.

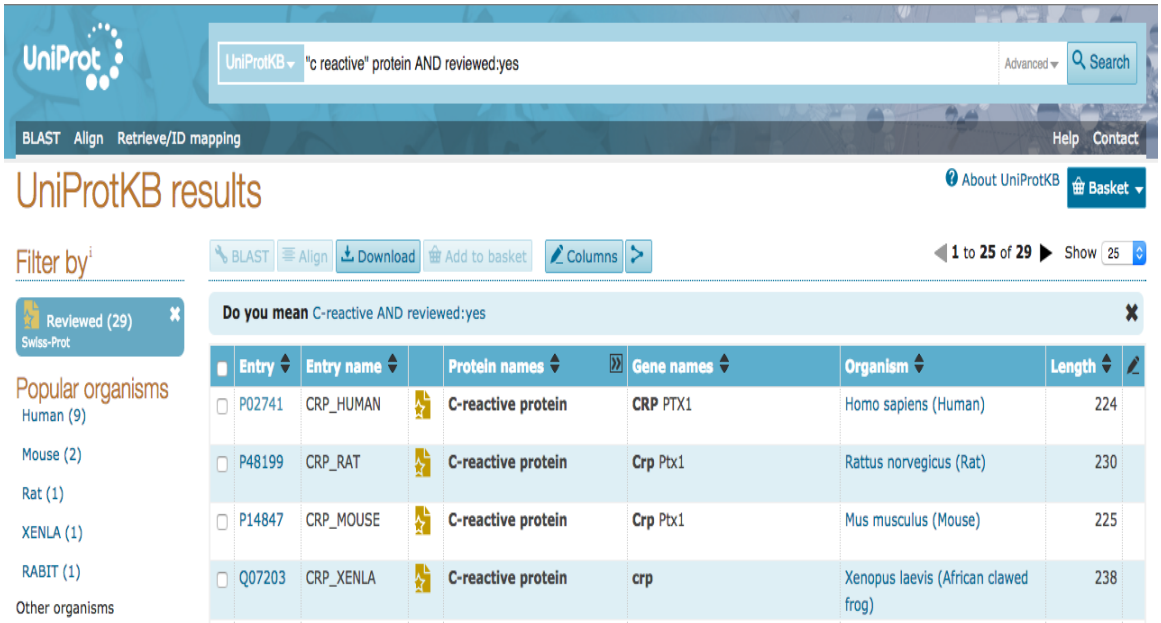


***Figura 9**- Exemplo elucidativo da visualização da ferramenta de pesquisa de proteínas do UniProt.*

Partindo da utilização do site da UniProt, foram anotadas informações imprescindíveis acerca da identificação de cada proteína identificada no decurso da análise bibliográfica, nomeadamente, código de entrada da base de dados, nome do gene associado e a que organismo pertence.

3.1.2.2. Origem das amostras salivares estudadas e respetivo conteúdo proteico

Em conformidade com a proveniência das amostras salivares anotadas das patologias em estudo, as proteínas foram associadas a um dos seguintes grupos: saliva total, fluído crevicular e mucosa oral, (Figura 10).

Whole Saliva	Crevicular Fluid	Mucosa

***Figura 10**- Cabeçalho da base de dados referente à origem das amostras salivares de cada proteína anotada manualmente.*

Com o auxílio a uma ferramenta especializada no cálculo e desenho de diagramas de Venn (http://bioinfogp.cnb.csic.es/tools/venny/), foi possível consumar uma análise dos dados relativos à proveniência das proteínas, descritas na figura 11. Esta ferramenta permite calcular as interseções de diversas listas de elementos (apresentando um máximo de 4 listas), permitindo a identificação dos elementos comuns ou exclusivos entre determinadas listas.

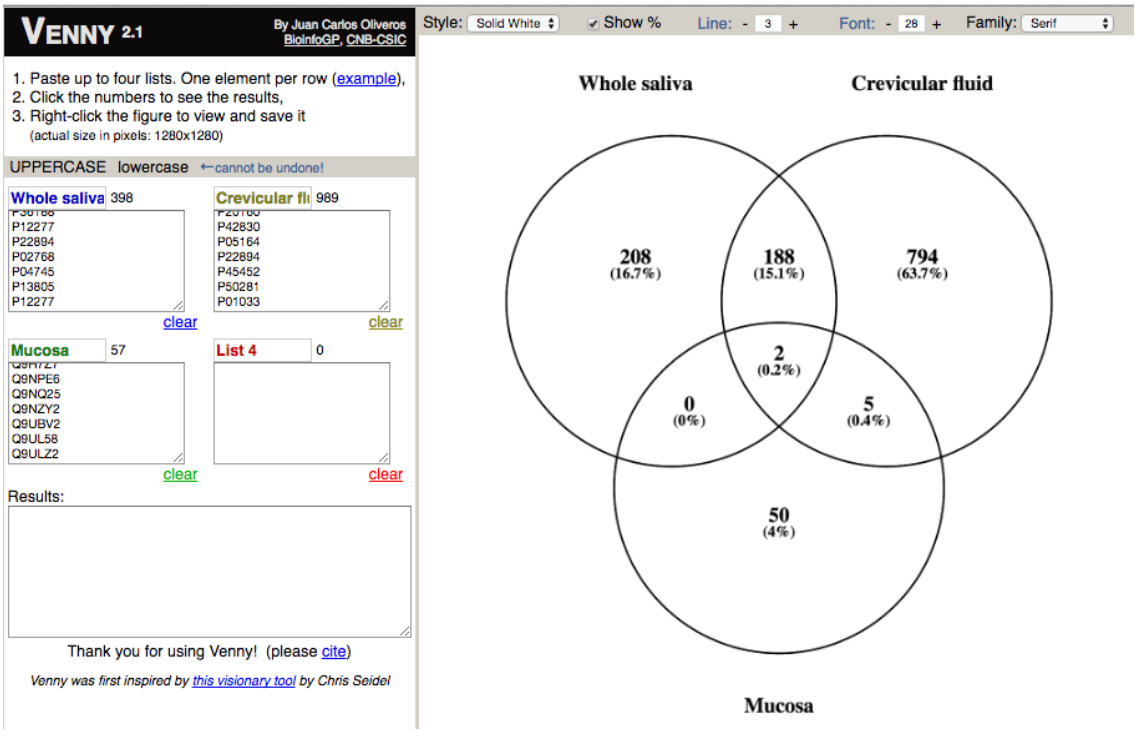


***Figura 11**- Interface da ferramenta Venny na análise da interseção das proteínas de saliva total, fluído crevicular e mucosa oral.*

3.1.2.3. Correlação com os estados de saúde e de doença

A anotação desta informação permite não só deduzir se determinadas proteínas estão presentes no estado de saúde ou doença (Figura 12), mas também de perceber se as mesmas são exclusivas de uma determinada patologia (periodontite crónica ou doenças cardiovasculares).

No sentido de identificar as doenças em estudo foram anotados o código MeSH ID e a sua identificação no *Online Mendelian Inheritance in Man (OMIM ID).*

Health	Disease Name	Disease (MeSH ID)	Regulation

Figura 12*- Cabeçalho da base de dados referente à correlação das proteínas salivares anotadas com os estados de saúde e doença.*

No que concerne à regulação de cada proteína, esta remete-se à variação da sua expressão em doença comparativamente ao controlo saudável. A regulação das proteínas salivares identificadas tanto em periodontite crónica como nas doenças cardiovasculares teve por base a seguinte fórmula:

$$\textit{Fold Change calculation} = \frac{\text{Número de ocorrências na doença}}{\text{Número de ocorrências em saúde}} = \text{Valor}$$

Hipótese a) valor (≥) 1 : Preconiza que a regulação é positiva (+)

Hipótese b) 0 < valor <1: Preconiza que a regulação é negativa (−)

No caso da hipótese b) ocorrer, é necessário efetuar o cálculo do quociente para que seja obtido um valor de regulação negativo:

$$Quociente = \frac{1}{Valor} = \gamma \text{ (ao } \gamma \text{ é } atríbuido\ um\ valor\ negativo\text{: } -\gamma)$$

Desse modo, cada vez que uma proteína apresenta um *Fold Change calculation* de 0,5, indica que esta se encontra diminuída na doença 2 vezes quanto à sua expressão, comparativamente ao estado saudável. Esta transformação dos valores permite identificar de forma mais evidente a variação negativa na quantidade de uma determinada proteína num estado de doença comparativamente ao estado saudável.

3.1.2.4. Caracterização do dador da amostra analisada

A caracterização do dador da amostra analisada, integra a sua faixa etária, o género e os seus hábitos sociais - tendo como exemplo disso, não só a toma de medicamentos (antibacterianos, anti-inflamatórios, antibióticos ou imunossupressores 6 meses antes do estudo, que possam ter efeitos sobre o sistema imunitário e consecutivas alterações a nível periodontal), mas também os hábitos tabágicos ou o consumo de álcool.

Age group	Gender*	Social Habits*

***Figura 13**- Cabeçalho da base de dados referente à caracterização do dador da amostra analisada.*

3.1.2.5. Métodos de amostragem e análise

Os métodos utilizados para a recolha da amostra e posterior análise, foram anotados com a finalidade em caracterizar as metodologias aplicadas na colheita, identificação e análise proteica. Resultante da heterogeneidade de técnicas de amostragem e análise proteómica, esta informação torna-se crucial em estudos deste carácter, aumentando a qualidade da interpretação dos resultados.

Methods of Sampling**	Methods of Analysis***

***Figura 14**- Cabeçalho da base de dados referente aos métodos de amostragem e análise das proteínas salivares em periodontite crónica e doenças cardiovasculares.*

3.1.2.6. Caracterização do estudo

Com o intuito em caracterizar o estudo, foi anotado o tipo de estudo (proteómico ou não proteómico) e o NCBI ID - código alusivo do PubMed (https://www.ncbi.nlm.nih.gov/pubmed) relacionado com o mesmo (figura 15).

***Figura 15**- Cabeçalho da base de dados referente à informação dos estudos onde foram identificadas as proteínas salivares em periodontite crónica e doenças cardiovasculares.*

3.1.2.7. Outras informações

Foram também registadas informações sobre o estudo no que diz respeito à presença de modificações pós-tradução nas proteínas identificadas e se estas foram já propostas ou não como biomarcadores das patologias (critério este estabelecido pelo autor do estudo).

***Figura 16**- Cabeçalho da base de dados referente a outras informações necessárias para caracterizar as proteínas salivares em periodontite crónica e doenças cardiovasculares.*

3.1.3. Caracterização funcional

3.1.3.1. Enriquecimento em processos biológicos e funções moleculares das proteínas em estudo

Após a recolha e organização de todos os dados de proteómica existentes relativamente às patologias em estudo, realizou-se uma análise funcional de acordo com as suas ontologias génicas (Figura 18). O objetivo desta análise, foi efetuar não só um enriquecimento dos processos biológicos em que as proteínas intervêm, bem como das funções moleculares que estas desempenham. Esta classificação foi executada com recurso à ferramenta PANTHER (**P**roteín **AN**alysis **TH**rough **E**volutionary **R**elationships), que permite classificar genes e proteínas segundo as suas funções, obter informações acerca dos processos biológicos, funções moleculares, componentes celulares e vias de sinalização [39–41].

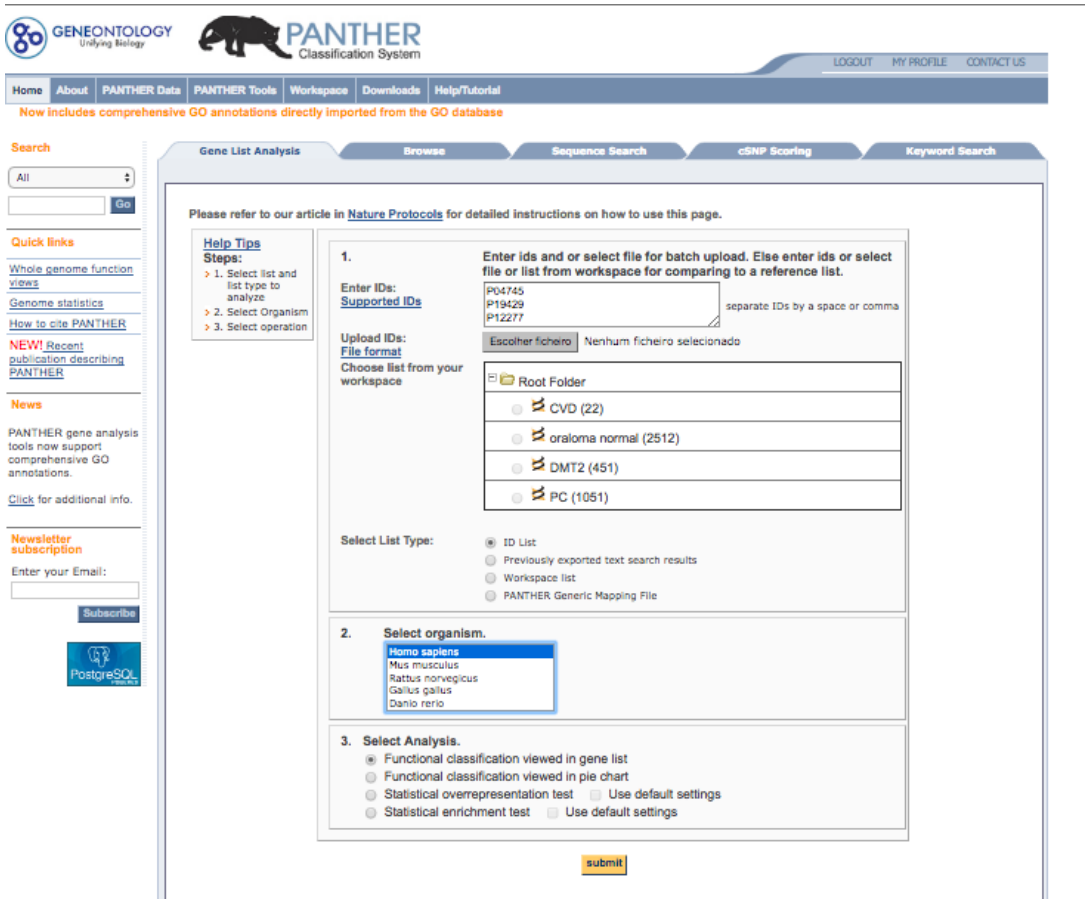


***Figura 17**- Interface do sistema de análise em grupo da ferramenta de classificação PANTHER. As opções selecionadas remetem-se às definições utilizadas para analisar os conjuntos de proteínas anotadas.*

- Para a realização desta caracterização, faz-se o *input* de dados no PANTHER com os respetivos códigos UniProt KB das proteínas em estudo, neste caso as identificadas em periodontite crónica e doenças cardiovasculares;
- Após essa inserção, escolhe-se o organismo adequado (*Homo sapiens*) e o tipo de análise desejada, neste será "*Functional classification viewed in gene list*";
- Posteriormente à submissão dos dados é apresentada uma tabela com uma série de informações pertencentes às proteínas introduzidas. Nesta interface é possível escolher o tipo de classificação pretendida (Figura 18), sendo que *MF Pie Chart* possibilita a classificação segundo a função molecular, *BP Pie Chart*a classificação segundo os processos biológicos, *CC Pie Chart* segundo os componentes celulares, *PC Pie Chart* segundo a classe proteica e a *Pathway Pie Chart* segundo as vias de sinalização.

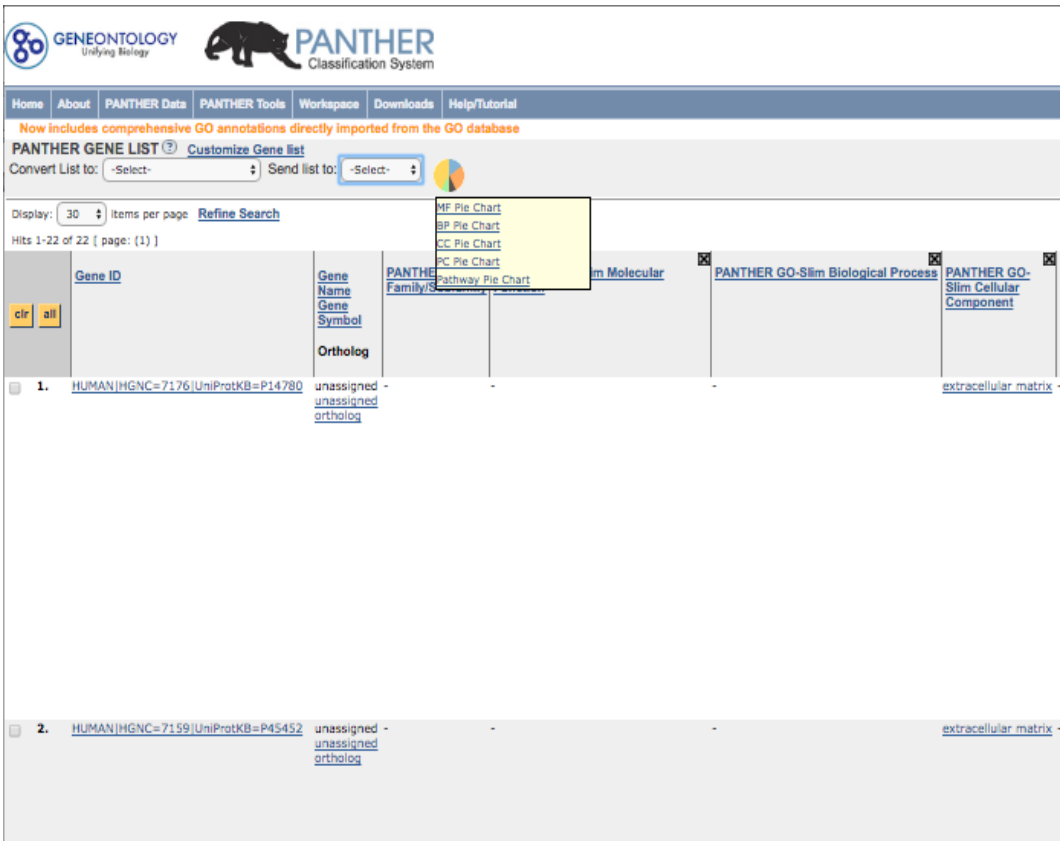


***Figura 18**- Seleção do tipo de classificação por ontologia pretendida, interface da ferramenta PANTHER.*

Neste campo foram apenas utilizadas as seguintes classificações; *MF Pie Chart* (funções moleculares) e *BP Pie Chart* (processos biológicos).

3.1.4. Comparação com o OralOma normal

Adicionalmente à análise funcional dos dados para cada uma das doenças em estudo, foi executada uma comparação destas com uma lista de referência, esta designada por OralOma normal. Esta lista corresponde a todas as proteínas identificadas até ao momento em dadores saudáveis (segundo a base de dados do OralCard [35,42]).

O propósito desta comparação foi determinar estatisticamente (os valores obtidos com os valores esperados) se as diferentes ontologias das proteínas salivares em periodontite crónica e doenças cardiovasculares, resultantes da classificação obtida anteriormente com recurso à ferramenta PANTHER, se encontravam acima ou abaixo dos valores esperados comparativamente ao OralOma normal.

De forma a quantificar a diferença entre os valores observados e os valores esperados de cada item, efetuou-se o cálculo da diferença fracional, esta definida pela seguinte fórmula:

$$Df = \frac{(x^1 - x^2)}{(x^2)}$$

Df - Diferença fracional

x^1 - Relativo ao valor observado

x^2 - Relativo ao valor esperado

Depois do cálculo da diferença fracional recorreu-se à aplicação de um teste binominal, descrito por *Cho* e *Champbell TIGS* em 2000 [43], de forma a obter a significância estatística dos resultados obtidos, expressa segundo um nível de significância (*p-value*). Quanto menor for o *p-value,* mais adverte que o número observado é estatisticamente significativo e potencialmente interessante, significando que as discrepâncias encontradas são grandes o suficiente para garantir que não foram atribuídas de uma forma aleatória.

4. RESULTADOS E DISCUSSÃO

4.1. ORALOMA HUMANO EM ESTUDOS DE PERIODONTITE CRÓNICA E DOENÇAS CARDIOVASCULARES

Como fruto da compilação dos proteomas orais da periodontite crónica e doenças cardiovasculares, a caracterização funcional do OralOma Humano das mesmas torna-se fundamental para a compreensão dos seus mecanismos moleculares, possibilitando à posteriori a identificação de potenciais proteínas biomarcadoras de suscetibilidade às doenças cardiovasculares.

4.1.1. Composição

A identificação das proteínas salivares presentes na cavidade oral de pacientes com periodontite crónica e doenças cardiovasculares foi conseguida através da análise de resultados de estudos experimentais de proteómica. No total foram analisados 40 artigos, com datas de publicação compreendidas entre os anos (2009-2015).

Com os resultados obtidos desta análise foi possível atualizar a base de dados do OralCard com 49 proteínas no que diz respeito a pacientes com periodontite crónica e 25 proteínas em pacientes com doenças cardiovasculares (esta é a primeira base de dados criada que reúne a informação relativa às proteínas presentes e alteradas nas doenças cardiovasculares). Esta constante atualização torna-se imprescindível, não só pela constante publicação de novos estudos experimentais de proteómica salivar que poderão abranger dados de proteínas com perfis de expressão alterados, ainda não identificados, ou confirmar resultados experimentais obtidos por outros autores com estudos experimentais similares.

A informação que caracteriza as patologias em estudo, está compilada na base de dados do OralCard, permitindo desde logo aos seus utilizadores a sua análise de forma simplificada e sistemática.

As tabelas 1 e 2indicam as proteínas orais identificadas em periodontite crónica e doenças cardiovasculares, resultando dos estudos experimentais analisados no decorrer deste trabalho.

Tabela 1 – Proteínas salivares identificadas em periodontite crónica

UniprotKB AC	Nome	Saúde/Doença	Regulação	Citação
P05120	PAI-2	Saúde e Periodontite crónica	↑	*Toyman U et al (2015)*[44]
P00750	t-PA	Saúde e Periodontite crónica	↑	*Toyman U et al (2015)*
P01584	IL-1b	Saúde e Periodontite crónica	↑	*Toyman U et al (2015)*
P08254	MMP-3	Saúde e Periodontite crónica	↑	*Toyman U et al (2015)*
Q5QEX9	Interleukin-17	Saúde e Periodontite crónica	2,3	*Ozçaka O et al (2011)*[45]
Q14116	Interleukin-18	Saúde e Periodontite crónica	1,59	*Ozçaka O et al (2011)*
P49913	Il-37	Saúde e Periodontite crónica	10,22	*Davidopoulou S et al (2013)*[46]
Q9HC84	Mucin-5B	Saúde e Periodontite crónica	1,55	*Acquier AB et al (2015)*[47]
P04745	Alpha-amylase 1	Saúde e Periodontite crónica	1,55	*Acquier AB et al (2015)*
P01033	Metalloproteinase inhibitor 1	Saúde e Periodontite crónica	-1,44	*Fenol A et al (2014)*[48]
P01583	Interleukin-1 alpha	Periodontite crónica	6,24	*Reis C et al (2014)*[49]
P01584	Interleukin-1 beta	Periodontite crónica	57	*Reis C et al (2014)*
P05231	Interleukin-6	Periodontite crónica		*Reis C et al (2014)*
P22301	Interleukin-10	Periodontite crónica	13	*Reis C et al (2014)*
P01375	Tumor necrosis factor	Periodontite crónica	6	*Reis C et al (2014)*
P26022	Pentraxin-related protein PTX3	Saúde e Periodontite crónica	10,66	*Fujita Y et al (2012)*[50]

P01584	Interleukin-1 beta	Saúde e Periodontite crónica	24,65	*Fujita Y et al (2012)*
P05231	Interleukin-6	Saúde e Periodontite crónica	4,64	*Fujita Y et al (2012)*
P10145	Interleukin-8	Saúde e Periodontite crónica	2	*Fujita Y et al (2012)*
P22301	Interleukin-10	Saúde e Periodontite crónica	2,48	*Fujita Y et al (2012)*
P01375	Tumor necrosis factor	Saúde e Periodontite crónica	1,94	*Fujita Y et al (2012)*
P01137	Transforminggrowth factor beta-1	Saúde e Periodontite crónica	-2,14	*Fujita Y et al (2012)*
P10145	Interleukin-8	Saúde e Periodontite crónica		*Erdemir EO et al (2015)*[51]
Q16552	Interleukin-17A	Saúde e Periodontite crónica		*Erdemir EO et al (2015)*
P05362	Intercellular adhesion molecule 1	Saúde e Periodontite crónica		*Erdemir EO et al (2015)*
P22894	Neutrophil collagenase	Periodontite crónica	1,7	*Nizam N et al (2014)*[52]
P05164	Myeloperoxidae	Periodontite crónica	3,3	*Nizam N et al (2014)*
P08246	Neutrophil elastase	Periodontite crónica	7,25	*Nizam N et al (2014)*
P01033	Metalloproteinse inhibitor 1	Periodontite crónica	-3,33	*Nizam N et al (2014)*
P01375	Tumor necrosis factor	Saúde e Periodontite crónica	↑	*Ozgören O et al (2014)*[51]
P22894	Neutrophil collagenase	Saúde e Periodontite crónica	↑	*Ozgören O et al (2014)*
B3VMW0	Lactoferrin	Periodontite crónica	2,42	*Glimvali P et al (2012)*[53]
Q9NRJ3	C-C motifchemokine 28	Saúde e Periodontite crónica	↑	*Ertugrul AS et al (2013)*[54]
P10145	Interleukin-8	Saúde e Periodontite crónica	↑	*Ertugrul AS et al (2013)*

P01584	Interleukin-1 beta	Saúde e Periodontite crónica	↑	*Ertugrul AS et al (2013)*
P01375	Tumor necrosis factor	Saúde e Periodontite crónica	↑	*Ertugrul AS et al (2013)*
A4D0Y8	Leptin	Saúde e Periodontite crónica	↓	*Karthikeyan BV et al (2007)*[55]
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Nizam N et al (2015)*[56]
P05164	Myeloperoxidase	Periodontite crónica	↓	*Nizam N et al (2015)*
P01033	Metalloproteinase inhibitor 1	Periodontite crónica	↓	*Nizam N et al (2015)*
P08246	Neutrophil elastase	Periodontite crónica	↓	*Nizam N et al (2015)*
P80188	Neutrophil gelatinase-associatedlipocalin	Periodontite crónica	↓	*Nizam N et al (2015)*
Q15722	Leukotriene B4 receptor 1	Saúde e Periodontite crónica	1,61	*Sánchez GA et al (2013)*[57]
O60603	Toll-like receptor 2	Saúde e Periodontite crónica	↑	*Swamminathan V et al (2013)*[58]
O00206	Toll-like receptor 4	Saúde e Periodontite crónica	↑	*Swamminathan V et al (2013)*
Q59E93	Membranealanineaminopeptidasevariant	Periodontite crónica	↑	*Aemaimanan P et al (2009)*[59]
P27487	Dipeptidylpeptidase 4	Periodontite crónica	↑	*Aemaimanan P et al (2009)*
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Buduneli E et al (2011)*[60]
P09237	Matrilysin	Periodontite crónica	2,1	*Buduneli E et al (2011)*
P01033	Metalloproteinase inhibitor 1	Periodontite crónica	1,4	*Buduneli E et al (2011)*
P16152	Carbonyl reductase [NADPH] 1	Periodontite crónica	1,57	*Kosaka T et al (2014)*[61]
P01584	Interleukin-1 beta	Periodontite crónica	1,88	*Kosaka T et al (2014)*
P05231	Interleukin-6	Periodontite crónica	1,95	*Kosaka T et al (2014)*
P01375	Tumor necrosis factor	Periodontite crónica	1,70	*Kosaka T et al (2014)*
P43116	Prostaglandin E2	Periodontite	15	*Kosaka T et al*

	receptor EP2 subtype	crónica		*(2014)*
P16152	Carbonyl reductase [NADPH] 1	Periodontite crónica	1,70	*Kosaka T et al (2014)*
P01584	Interleukin-1 beta	Periodontite crónica		*Cetinkaya B et al (2013)*[62]
P05231	Interleukin-6	Periodontite crónica		*Cetinkaya B et al (2013)*
P22301	Interleukin-10	Periodontite crónica		*Cetinkaya B et al (2013)*
P43116	Prostaglandin E2 receptor EP2 subtype	Periodontite crónica		*Cetinkaya B et al (2013)*
P01375	Tumor necrosis factor	Periodontite crónica		*Cetinkaya B et al (2013)*
P05112	Interleukin-4	Periodontite crónica		*Cetinkaya B et al (2013)*
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Cetinkaya B et al (2013)*
P45452	Collagenase 3	Periodontite crónica		*Cetinkaya B et al (2013)*
Q14116	Interleukin-18	Periodontite crónica		*Cetinkaya B et al (2013)*
O95760	Interleukin-33	Periodontite crónica	-1,09	*Buduneli N et al (2012)*[63]
P10645	Chromogranin-A	Periodontite crónica	↑	*Haririan H et al (2012)*[64]
P04745	Alpha-amylase 1	Periodontite crónica	↓	*Haririan H et al (2012)*
P08246	Neutrophil elastase	Periodontite crónica	↑	*Mäntylä P et al (2012)*[65]
P08311	Cathepsin G	Periodontite crónica	↑	*Mäntylä P et al (2012)*
P22894	Neutrophil collagenase	Periodontite crónica	↓	*Rathnayake N et al (2015)*[66]
P05164	Myeloperoxidase	Periodontite crónica	↓	*Rathnayake N et al (2015)*
P01033	Metalloproteinase inhibitor 1	Periodontite crónica	↓	*Rathnayake N et al (2015)*
P05164	Myeloperoxidase	Periodontite crónica	↑	*Rathnayake N et al (2015)*
P14780	Matrix metalloproteinase-9	Periodontite crónica	↑	*Rathnayake N et al (2015)*
P09237	Matrilysin	Periodontite crónica	↑	*Buduneli E et al (2011)*[60]
P01033	Metalloproteinase inhibitor 1	Periodontite crónica	↑	*Buduneli E et al (2011)*
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Buduneli E et al (2011)*
P01009	Alpha-1-antitrypsin	Periodontite crónica	-1,52	*Hayashi S et al (2015)*[67]

B3VMW0	Lactoferrin	Periodontite crónica	-2,78	*Hayashi S et al (2015)*
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Sorsa T et al (2011)*[68]
P45452	Collagenase 3	Periodontite crónica		*Sorsa T et al (2011)*
P01033	Metalloproteinase inhibitor 1	Periodontite crónica		*Sorsa T et al (2011)*
P05231	Interleukin-6	Periodontite crónica	↑	*Costa PP et al (2011)*[69]
P22894	Neutrophil collagenase	Periodontite crónica	↑	*Costa PP et al (2011)*
P25090	N-formylpeptide receptor 2	Periodontite crónica	↓	*Lütfioğlu M et al (2015)*[70]
P10145	Interleukin-8	Periodontite crónica	↑	*Lütfioğlu M et al (2015)*
P20160	Azurocidin	Periodontite crónica	-1,37	*Leppilahti JM et al (2014)*[71]
P42830	C-X-C motif chemokine 5	Periodontite crónica		*Leppilahti JM et al (2014)*
P05164	Myeloperoxidase	Periodontite crónica	-1,04	*Leppilahti JM et al (2014)*
P22894	Neutrophil collagenase	Periodontite crónica	-1,14	*Leppilahti JM et al (2014)*
P45452	Collagenase 3	Periodontite crónica	-1,28	*Leppilahti JM et al (2014)*
P50281	Matrix metalloproteinase-14	Periodontite crónica	-1,22	*Leppilahti JM et al (2014)*
P01033	Metalloproteinase inhibitor 1	Periodontite crónica		*Leppilahti JM et al (2014)*
P02741	C-reactive protein	Periodontite crónica		*Miller CS et al (2014)*[72]
P05231	Interleukin-6	Periodontite crónica		*Miller CS et al (2014)*
P01584	Interleukin-1 beta	Periodontite crónica		*Miller CS et al (2014)*
P05164	Myeloperoxidase	Periodontite crónica		*Miller CS et al (2014)*
P01375	Tumor necrosis factor	Periodontite crónica		*Miller CS et al (2014)*
P14780	Matrix metalloproteinase-9	Periodontite crónica		*Miller CS et al (2014)*
Q15848	Adiponectin	Periodontite crónica		*Miller CS et al (2014)*
Q99930	sICAM-1	Periodontite crónica		*Miller CS et al (2014)*
P15941	Mucin-1	Periodontite crónica	↑	*Miller CS et al (2014)*
P04745	Alpha-amylase 1	Periodontite	↑	*Miller CS et al*

		crónica		*(2014)*
P05112	Interleukin-4	Saúde e Periodontite crónica	1,28	*Prakasam S et al (2014)*[73]
P22301	Interleukin-10	Saúde e Periodontite crónica	1,84	*Prakasam S et al (2014)*
P05231	Interleukin-6	Saúde e Periodontite crónica	1,89	*Prakasam S et al (2014)*
Q16552	Interleukin-17A	Saúde e Periodontite crónica	↓	*Prakasam S et al (2014)*
P01584	Interleukin-1 beta	Periodontite crónica	1,72	*Yoon AJ et al (2012)*[74]

Tabela 2 – Proteínas salivares identificadas em doenças cardiovasculares e periodontite crónica

UniprotKB AC	Nome	Saúde/Doença	Regulação	Citação
P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	1	*Buduneli E et al* (2011)[60]
P09237	Matrilysin	Doenças cardiovasculares e periodontite crónica	1,9	*Buduneli E et al* (2011)
P01033	Metalloproteinase inhibitor 1	Doenças cardiovasculares e periodontite crónica	1,32	*Buduneli E et al* (2011)
P01584	Interleukin-1beta	Doenças cardiovasculares e periodontite crónica	1,57	*Kosaka T et al* (2014)[61]
P05231	Interleukin-6	Doenças cardiovasculares e periodontite crónica	1,88	*Kosaka T et al* (2014)
P01375	Tumor necrosis factor	Doenças cardiovasculares e periodontite crónica	1,95	*Kosaka T et al* (2014)
P43116	Prostaglandin E2 receptor EP2 subtype	Doenças cardiovasculares e periodontite	15	*Kosaka T et al* (2014)

		crónica		
P16152	Carbonyl reductase [NADPH]1	Doenças cardiovasculares e periodontite crónica	1,70	*Kosaka T et al* (2014)
P08246	Neutrophil elastase	Doenças cardiovasculares e periodontite crónica	↑	*Mäntylä P et al* (2012)[65]
P08311	Cathepsin G	Doenças cardiovasculares e periodontite crónica	↑	*Mäntylä P et al* (2012)
P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	-1,23	*Rathnayake N et al* (2015)[66]
P05164	Myeloperoxidase	Doenças cardiovasculares e periodontite crónica	-1,16	*Rathnayake N et al* (2015)
P01033	Metalloproteinase inhibitor 1	Doenças cardiovasculares e periodontite crónica	-1,1	*Rathnayake N et al* (2015)
P14780	Matrixmetalloproteinase -9	Doenças cardiovasculares e periodontite crónica	-1	*Rathnayake N et al* (2015)
P09237	Matrilysin	Doenças cardiovasculares e periodontite crónica	↑	*Buduneli E et al* (2011)[60]
P01033	Metalloproteinase inhibitor 1	Doenças cardiovasculares e periodontite crónica	↑	*Buduneli E et al* (2011)
P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	↑	*Buduneli E et al* (2011)
P01009	Alpha-1-antitrypsin	Doenças cardiovasculares e periodontite crónica	-1,52	*Hayashy S et Al* (2015)[67]
B3VMW0	Lactoferrin	Doenças cardiovasculares e periodontite crónica	-2,78	*Hayashy S et Al* (2015)

P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	↑	*Sorsa T et al*(2011)[68]
P45452	Collagenase 3	Doenças cardiovasculares e periodontite crónica		*Sorsa T et al* (2011)
P01033	Metalloproteinase inhibitor 1	Doenças cardiovasculares e periodontite crónica		*Sorsa T et al* (2011)
P02741	C-reactive protein	Doenças cardiovasculares e periodontite crónica	1,88	*Labat C et* (2013)[75]
Q15722	Leukotriene B4 receptor 1	Doenças cardiovasculares e periodontite crónica	↑	*Labat C et* (2013)
P43116	Prostaglandin E2 receptor EP2 subtype	Doenças cardiovasculares e periodontite crónica	1,25	*Labat C et* (2013)
P14780	Matrixmetalloproteinase -9	Doenças cardiovasculares e periodontite crónica	1,8	*Labat C et* (2013)
F8VV32	Lysozyme	Doenças cardiovasculares e periodontite crónica	1,36	*Labat C et* (2013)
P02741	C-reactiveprotein	Doenças cardiovasculares e periodontite crónica	↑	*Miller CS et* (2014)[76]
P05231	Interleukin-6	Doenças cardiovasculares e periodontite crónica		*Miller CS et* (2014)
P01584	Interleukin-1beta	Doenças cardiovasculares e periodontite crónica		*Miller CS et* (2014)
P05164	Myeloperoxidase	Doenças cardiovasculares e periodontite crónica	↑	*Miller CS et* (2014)
P01375	Tumor necrosis factor	Doenças		*Miller CS et*

		cardiovasculares e periodontite crónica		(2014)
P14780	Matrixmetalloproteinase -9	Doenças cardiovasculares e periodontite crónica		*Miller CS et* (2014)
Q15848	Adiponectin	Doenças cardiovasculares e periodontite crónica		*Miller CS et* (2014)
Q99930	sICAM-1	Doenças cardiovasculares e periodontite crónica	↑	*Miller CS et* (2014)
P08311	Cathepsin G	Doenças cardiovasculares e periodontite crónica	↑	*Mäntylä P et al* (2012)[65]
P45379	TroponinT	Doenças cardiovasculares e periodontite crónica	5,32	*Mirzaii-Dizgah I et al* (2013)[77]
P12277	Creatinine Kinase B-Type	Doenças cardiovasculares e periodontite crónica	↑	*Mirzaii-Dizgah I et al* (2012)[78]
P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	3	*Ehlers V et al* (2011)[79]
P22894	Neutrophil collagenase	Doenças cardiovasculares e periodontite crónica	1,64	*Furuholm J et al* (2006)[80]
P02768	Serum albumin	Doenças cardiovasculares e periodontite crónica		*Toker A et al* (2013)[81]
P04745	Alpha-amylase 1	Doenças cardiovasculares e periodontite crónica	2	*Shen YS et al* (2012)[82]
P19429	Troponin I, cardiacmuscle	Doenças cardiovasculares e periodontite crónica	82	*Shen YS et al* (2012)
P12277	Creatinine Kinase B-Type	Doenças cardiovasculares	7	*Shen YS et al* (2012)

		e periodontite crónica		

Além das demais proteínas identificadas neste trabalho, foram ainda utilizadas 1212 proteínas já anotadas no OralCard referentes à periodontite crónica, perfazendo um total de1241 proteínas que foram utilizadas nas análises conseguintes (Anexos – tabela A).

4.1.2. Origem das amostras salivares catalogadas

As amostras salivares analisadas nos estudos de proteómica oral podem ser provenientes da secreção específica de uma determinada glândula salivar ou da mistura de fluídos orais presentes na cavidade oral dos indivíduos. Desta forma, as proteínas identificadas nas amostras salivares podem não ser exclusivas de secreções salivares, ou seja, podem decorrer de secreções não-salivares.

Do total das proteínas orais identificadas em periodontite crónica e doenças cardiovasculares, 398 são provenientes de amostras de saliva total, 989 do fluido crevicular e 57 da mucosa oral (Figura 19).

A origem das proteínas orais secretadas pelas glândulas salivares é pertinente, uma vez que pode ser utilizada para apontar a algum tipo de alteração da secreção proteica de uma glândula salivar específica, resultante de modificações sistémicas.

Deste modo, é imperativo conhecer a dinâmica das proteínas salivares a nível molecular, para podermos descobrir marcadores úteis para efetuar um diagnóstico [19].

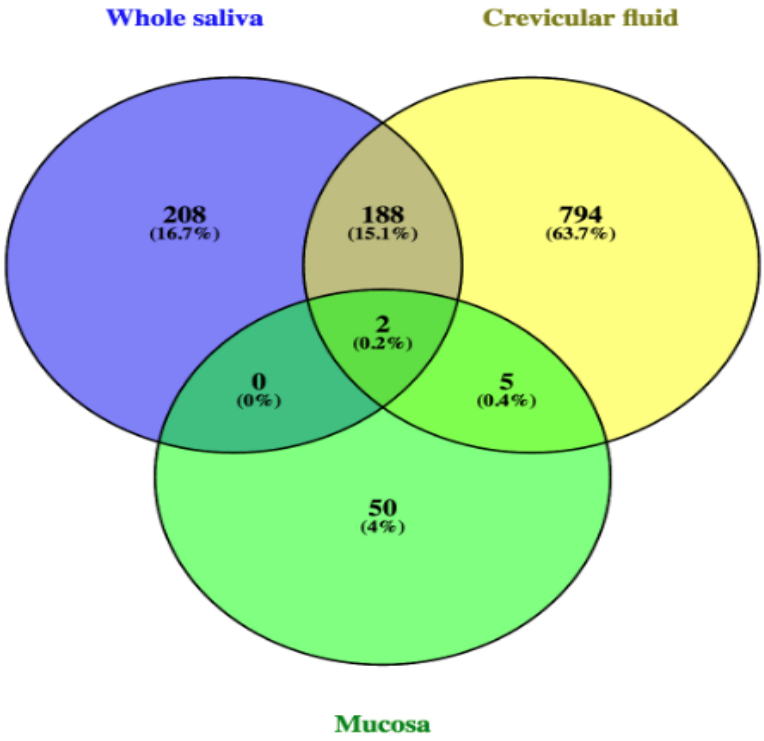


***Figura 19**- Diagrama de Venn representativo da distribuição das proteínas salivares segundo a sua origem. Gráfico obtido com recurso ao programa Venny[83].*

A análise da Figura 19 permite verificar a existência de uma predominância de proteínas identificadas em amostras salivares provenientes da saliva total e do fluído crevicular. O número total de proteínas salivares identificadas anteriormente, é dependente dos métodos usados pelos investigadores, para que seja efetuada a recolha da amostra [17].

A facilidade da recolha das amostras a partir de saliva total e fluído crevicular é relativamente fácil, comparativamente à mucosa, acabando por justificar o número acrescido de proteínas identificadas nos estudos analisados [84,85].

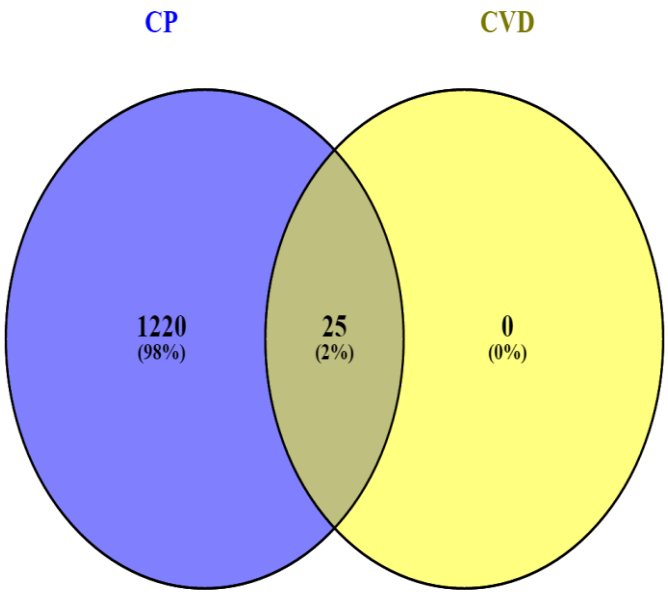
No entanto, o método de recolha a partir do fluído crevicular não é tão simples como o método de recolha de saliva, exigindo mais tempo e mais habilidade técnica do profissional. As análises a partir do fluído crevicular dão origem a menores quantidades de amostra comparativamente à saliva, existindo também a possibilidade destas estarem contaminadas com sangue ou saliva [86].

Além disso, apesar da maior parte das proteínas identificadas nestes estudos ser proveniente do fluido crevicular, verificámos que, das 986 proteínas identificadas no fluído crevicular, 515 foram também já encontradas em amostras de saliva noutros estudos (segundo o OralCard), o que indica que a saliva poderá refletir também este universo.

Desta forma, a identificação dos proteomas salivares identificados em ambas as patologias, através da análise de amostras salivares, parece reforçar a hipótese em ser usada a saliva como fluído de diagnóstico para as doenças cardiovasculares. Porém, é importante padronizarem-se os métodos de recolha da saliva, para que estes sejam utilizados como requisitos de análises de rotina em ambiente clínico [19].

4.1.3. Correlação com os estados de doença

Do conjunto total de proteínas identificadas em estudos relativos a periodontite crónica e doenças cardiovasculares, 1220 foram identificadas em indivíduos com periodontite crónica e 25 foram identificadas em indivíduos com periodontite crónica e doenças cardiovasculares (Figura 20).



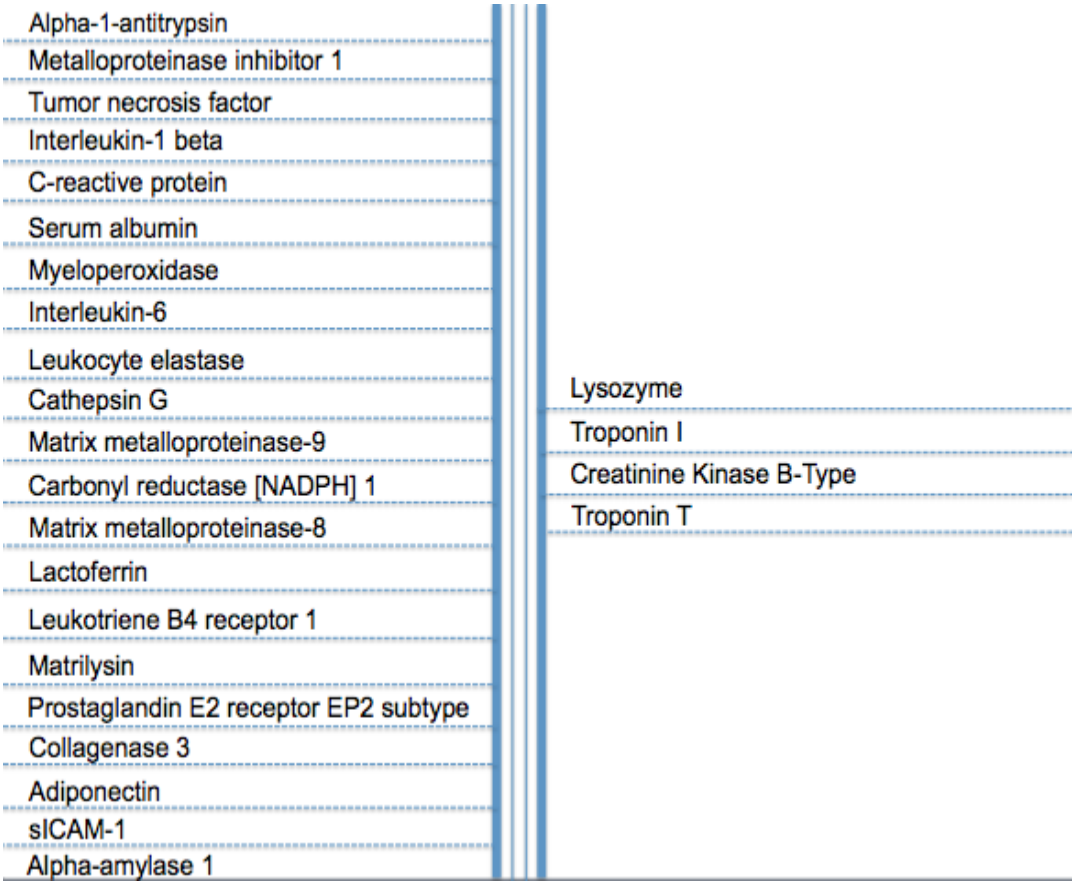


***Figura 20**- Distribuição das proteínas identificadas em relativos a periodontite crónica e doenças cardiovasculares representando quantas proteínas são comuns e exclusivas nas doenças em estudo (em baixo são mencionadas as proteínas comuns em ambas as doenças e as exclusivas das doenças cardiovasculares)(gráfico obtido com recurso ao programa Venny [83]).*

A identificação de proteínas salivares presentes em indivíduos com periodontite crónica e doenças cardiovasculares foi um elemento essencial para a avaliação de potenciais biomarcadores para a patologia cardiovascular. Da figura 20 é possível verificar que algumas das proteínas foram identificadas em ambas as patologias, tratando-se do caso da *Alpha-1-antitrypsin*, *Metalloproteinase inhibitor 1*, *Tumor necrosis factor, Interleukin-1 beta*, *C-reactive proteín*, *serum albumin*, *Myeloperoxidase*, *Interleukin-6*, *Leukocyte elastase*, *Cathepsin G*, *Matrix metalloproteinase-9*, *Carbonyl reductase [NADPH] 1*, *Matrix Metalloproteinase-8*, *Lactoferrin*, *Leukotriene B4 receptor 1*, *Matrilysin*, *Prostaglandin E2 receptor EP2 subtype*, *Collagenase 3*, *Adiponectin*, *sICAM-1 e Alpha-amylase 1, Lysozyme, Troponin I e Creatinine Kinase B-Type e Troponin T.* A circunstância de estas proteínas serem identificadas em ambas as patologias, evidencia de certa forma uma determinada certeza quanto à sua relação causal.

De acordo com os resultados dos estudos experimentais (Tabela 1 e 2), foram calculados os valores de *Fold change calculation,* obtendo dados de regulação quanto à variação da expressão proteica nas doenças em questão, comparativamente ao controlo saudável, conseguindo desde logo uma melhor interpretação das alterações moleculares existentes entre os dois estados.

A proteína *Alpha-1-antitrypsin* é um inibidor endógeno das proteases da serina mais abundante no sangue, caracterizada por ser uma proteína de fase aguda. Os seus níveis apresentaram um aumento de expressão poucas horas após o desenvolvimento de inflamação ou infeção existente, tendo sido já evidenciados em situações de enfarte agudo do miocárdio e inflamação periodontal [67,87]. Esta parece demonstrar propriedades anti-inflamatórias únicas, afetando vários tipos de células e na modulação da inflamação, causada por fatores microbianos e do hospedeiro.

Também a *Lactoferrin,* proteína de ligação do ferro, com atividade bactericida está similarmente aumentada na inflamação periodontal, no entanto não foram detetadas correlações significativas entre os seus níveis de expressão e a aterosclerose [88]. Este resultado pode sugerir que o agravamento da condição periodontal avaliada por esta proteína não é considerado um fator de risco para as doenças cardiovasculares.

Relativamente à *Metalloproteinase inhibitor 1 (TIMP-1),*é um inibidor das *MMPs* que degradam as proteínas da matriz extracelular (provocando a destruição dos tecidos periodontais) e tendo sido já identificada em diversos estudos [48,52,56,60,89], apresentando níveis de concentração mais baixos em pacientes com periodontite crónica comparativamente a indivíduos saudáveis.

Foi também demonstrado que esta leva à inibição da atividade da *MMP-9* durante o período de recuperação do enfarte e na proteção contra a rotura da placa após o enfarte de miocárdio. A *TIMP-1* é amplamente sintetizada por inúmeras células e tecidos e induzida por várias citocinas pró-inflamatórias (*IL-1, IL-6 e TNF-*α). A existência de uma desregulação no equilíbrio entre as *MMPs* e a produção das *TIMP-1* está diretamente correlacionado com diversas condições patológicas como a periodontite, artrite, crescimento tumoral e metástases [48].

TNF-α, IL-6, IL-1β foram também algumas proteínas identificadas com elevada significância na saliva em indivíduos com patologia periodontal. Estas citocinas pró-inflamatórias têm um amplo espectro de atividade biológica, desempenhando um papel importante em muitas doenças inflamatórias nomeadamente na indução, regulação e na destruição do tecido periodontal [90,91]. Estas induzem alterações vasculares e na migração de células, como os neutrófilos para o periodonto. *IL-1β e TNF-α* são considerados mediadores primários das doenças inflamatórias crónicas, caracterizando-se não só pela indução da destruição tecidular, mas também pela perda óssea causada, nomeadamente nas doenças periodontais [49,54].

Estudos recentes sugerem que níveis salivares aumentados destas proteínas na periodontite, estão intimamente relacionados com a disfunção endotelial, já que ao ser produzido um persistente estado de infeção/inflamação crónica, os agentes infeciosos podem contribuir para um aumento da intensidade da aterosclerose na carótida [61].

Destaque também para a *PGE2,*que também se encontra aumentada e associada à deposição de aterosclerose na carótida em mulheres [61]. Este potente mensageiro biolipídico encontrar-se aumentado após ocorrem lesões de inflamação neuronal, desempenhando um papel importante em diversas condições patológicas e fisiológicas no cérebro.

A PGE2 encontra-se regulada positivamente de forma significativa depois de se suceder uma hemorragia intracerebral e contribui para a resposta inflamatória através do seu subtipo prostanóide do receptor 2 (EP2) [92]. A sinalização através deste recetor tem demonstrado efeitos benéficos após um acidente vascular cerebral isquémico transitório (AIT), pelo seu papel na inflamação, acabando por modular a lesão cerebral de forma positiva, pelo que pode representar um novo alvo terapêutico desta condição [92,93].

É de salientar que para além do seu papel importante no cérebro, estes recetores podem também levar ao desenvolvimento de várias doenças cardiovasculares (como a isquemia do miocárdio, cardiomiopatia cardíaca, fibrose cardíaca e aterosclerose), já que podem ser expressos por várias células, como os cardiomiócitos, células endoteliais vasculares e células do músculo liso [94].

A *C-reactive proteín* foi uma proteína identificada muito aumentada na saliva de indivíduos que sofreram enfarte agudo do miocárdio [72]. A *CRP* pertence à família de proteínas *pentraxina* e é sintetizada pelos hepatócitos e por alguns tecidos extra-hepáticos, como o músculo vascular liso, placas ateroscleróticas e tecidos intracardíacos [95]. No caso da existência de uma inflamação crónica de baixa intensidade, a *CRP* pode provocar danos no endotélio vascular, fazendo com que este se torne mais suscetível a fatores pro-aterogénicos, acabando por ocorrer uma infiltração da parede vascular pelas células inflamatórias e deposição de lípidos nas paredes das artérias.

A *CRP* nestas condições induz ainda a ativação das *MMPs* (que causam a destruição do colagénio) em células endoteliais e macrófagos. Todos estes processos acabam por aumentar consideravelmente a probabilidade de remodelação da placa aterosclerótica e consequente destabilização e rotura [95].

A serum albumin é mais uma proteína que tem apresentado níveis salivares significativamente mais elevados em indivíduos que sofreram isquemia do miocárdio, comparativamente a indivíduos saudáveis [81,96]. A isquemia do miocárdio dá origem a uma alteração na estrutura do N-terminal desta proteína, fazendo com que esta possa ser medida por um teste. No entanto, a baixa especificidade e a deteção de alguns falsos-positivos, faz com que sejam necessários novos estudos para validar esta proteína como um biomarcador mais fiável [81].

Myeloperoxidase ,Leukocyte elastase, Cathepsin G, Matrix metalloproteinase-9 e *Matrix Metalloproteinase-8,* apresentaram também níveis salivares mais elevados em pacientes com periodontite crónica, comparativamente com os grupos saudáveis [52,66,68,71,72]. Durante os estados de inflamação os neutrófilos são estimulados a produzir enzimas, tais como as *MMPs*, Leukocyte elastase e a *Myeloperoxidase* [97].

A *Myeloperoxidase* é uma enzima antimicrobiana dos *PMNs* libertados para o ambiente extracelular após a estimulação dos neutrófilos. O seu principal papel é produzir o ácido hipocloroso para matar as bactérias e o aumento da sua concentração no soro tem sido considerado como um

indicador de risco para a inflamação local (destruição do tecido periodontal) e sistémica (aterosclerose) [52,66].

A *MMP-8,*também conhecida como *colagenase 2*, está relacionada com condições inflamatórias e é expressa principalmente por neutrófilos, mas também por células do músculo liso e macrófagos em lesões ateroscleróticas [52,66]. Perante os seus níveis fisiológicos, esta parece exercer propriedades protetoras e anti-inflamatórias na perda de osso alveolar durante a inflamação periodontal.

Por outro lado, quando aumentada, esta proteína está associada à destruição do colagénio nos tecidos periodontais, na apoptose das células endoteliais e na conversão de lesões ateroscleróticas estáveis para lesões não estáveis. Levando desta forma à rotura das placas ateroscleróticas. Também a*MMP-9* tem-se apresentado aumentada em situações de inflamação periodontal e os seus níveis no soro e na saliva parecem evidenciar que poderá ser um bom marcador de doença cardiovascular [52,66].

A *Leukocyte elastase* é uma enzima proteolítica que pode causar a destruição do tecido conjuntivo pela digestão de quase todos os tipos de proteínas da matriz extracelular, bem como das proteínas do plasma e é responsável pela inativação da *TIMP-1* [52,56].

A *Cathepsin G*, uma das principais enzimas microbianas demonstra contribuir para a coagulação sanguínea e crescimento de trombos vasculares [65].

Mäntylä et al. em 2016, verificou que a atividade da *Leukocyte elastase e* da *Cathepsin G* na saliva de pacientes com enfarte agudo do miocárdio e periodontite se encontravam significativamente maiores em comparação com pacientes sem periodontite, sendo, por isso, apontados como potenciais biomarcadores de suscetibilidade à doença [65].

A Carbonyl reductase *[NADPH] 1*é uma enzima monomérica e citosólica pertencente às desidrogenases de cadeia curta. Esta proteína foi também identificada na saliva, apresentando níveis mais elevados em indivíduos com periodontite e aterosclerose, comparativamente a indivíduos saudáveis [61,98]. O facto desta proteína aparecer em maior quantidade poderá levá-la a desempenhar um papel crucial no controlo da diferenciação e regeneração de

células musculares [99]. Por outro lado, também tem demonstrado ter uma função protetora em relação ao stress oxidativo, neurodegeneração e apoptose [100]. No entanto, a função biológica e o seu mecanismo na isquemia ainda não estão totalmente esclarecidos [98].

Leukotriene B4 receptor 1, é um mediador lipídico potente, associado com a imunidade adquirida, conhecido por desempenhar dois papéis distintos na cavidade oral, como mecanismo de defesa do hospedeiro contra as infeções e como mediador inflamatório na doença periodontal [57]. Esta proteína tem apresentado níveis salivares significativamente mais elevados em pacientes com periodontite, comparativamente a indivíduos saudáveis, níveis estes relacionados com a resposta a estímulos inflamatórios e no progresso da doença de forma crónica [57].

*Matrilysin (MMP-7)*é uma proteína pertencente à família das *MMPs* e desempenha um papel importante na defesa e reparação do epitélio. Além da sua deteção na saliva, também parece estar presente nos tecidos gengivais e no fluído crevicular em pacientes com periodontite [60].

Collagenase 3 (MMP-13), foi mais uma proteína identificada na saliva com níveis aumentados em pacientes com periodontite, comparativamente a pacientes saudáveis [71]. Esta proteína é expressa em níveis muito baixos quando estamos perante um miocárdio normal, no entanto, está substancialmente aumentada em pacientes com doenças cardiovasculares [101].

Adiponectin e sICAM-1, foram duas proteínas identificadas com elevada significância na saliva em indivíduos que tiveram enfarte agudo do miocárdio [72]. A *Adiponectin* é um derivado de uma proteína semelhante ao colagénio e tem-lhe sido dada bastante importância, pelo simples facto de que as suas propriedades anti-inflamatórias e antiaterogénicas servem como um fator de proteção para o individuo [102].

SICAM-1 é uma molécula de adesão celular solúvel, que pode representar um marcador importante do surgimento de uma disfunção endotelial precoce, já que envolve a ativação ou o dano nas células, nomeadamente nas plaquetas ou no endotélio vascular. O aumento da sua expressão parece desempenhar um papel fundamental nas fases iniciais da

aterosclerose, pelo que esta tem como função modelar a adesão de leucócitos circulantes nas células endoteliais [103].

Alpha-amylase 1 é uma proteína muito abundante na saliva, que, para além da função na digestão de amido, apresenta funções predominantemente protetoras (atuando na formação da película adquirida nas superfícies dos dentes e no efeito inibidor direto sobre o crescimento de certas bactérias), impedindo, deste modo, algumas das reações inflamatórias destrutivas dos tecidos [47]. Esta proteína é expressa em níveis bastante mais elevados em pacientes com periodontite crónica, comparativamente com os grupos de controlo [47]. Num estudo de *Shen et al* [104], foi demonstrado claramente que os pacientes com enfarte agudo do miocárdio tiveram um aumento significativo desta proteína, comparativamente a indivíduos saudáveis [104].

O facto desta proteína estar aumentada tanto em periodontite crónica, como em doença cardiovascular, significa que esta poderá ser um bom indicador molecular do fundo comum entre as duas patologias.

Da análise da Tabela 2 foi ainda possível verificar que das 25 proteínas identificadas em ambas as patologias, 4 ainda não foram identificadas de forma isolada em periodontite crónica, como é o caso da *Lysozyme, Troponin I e Creatinine Kinase B-Type e Troponin T.*

Lysozyme é uma enzima antimicrobiana, capaz de provocar danos na parede celular dos peptidoglicanos e de alguns microrganismos. A flora bacteriana parece ter um papel importante na sobre-regulação desta enzima, sendo deste modo, considerada como uma proteína de defesa contra as infeções [105,106]. A sua presença na saliva tem vindo a ser associada com a hipertensão e doença arterial coronária [107].

Troponin I é a subunidade inibitória da troponina (proteína do músculo cardíaco), que participa na modelação da interação da actina e miosina [77,108].Num estudo de *Mirzaii-Dizgah et al* [77], foram identificados níveis salivares aumentados em pacientes com enfarte agudo do miocárdio, comparativamente a indivíduos saudáveis.

Creatinine Kinase B-Type é uma isoenzima que desempenha um papel central na transdução de energia, principalmente em tecidos com grandes requisitos de energia (músculo esquelético, coração e cérebro), contribuindo

para a homeostasia da energia miocárdica. Normalmente, esta é sobre-expressa em diversas condições, tal como no desafio metabólico, hipertrofia, hipertensão e hipoxia [110].*Num estudo de Mirzaii-Dizgah et al* [78]*, também os níveis salivares foram mais elevados em pacientes com* enfarte agudo do miocárdio, comparativamente a indivíduos saudáveis.

Troponin T, é uma subunidade de ligação da tropomiosina à troponina, (complexo regulador do filamento fino) que confere cálcio-sensibilidade através da atividade da ATPase actomiosiana no músculo estriado cardíaco [37,108]. Também esta proteína apresentou níveis salivares aumentados em pacientes com enfarte agudo do miocárdio, comparativamente a indivíduos saudáveis [108]. Com base nestes resultados, podemos concluir que após um enfarte agudo do miocárdio, existem aumentos significativos dos níveis salivares de diversas proteínas em ambas as patologias.

De um ponto de vista científico estes resultados tornam estas proteínas bastante interessantes, na medida em que possam ser usadas como biomarcadores de diferenciação entre a periodontite crónica e a patologia cardiovascular. Desta forma, é ambicionado que num futuro próximo possam vir a ser usados biomarcadores salivares como alternativa ao soro, tornando possível efetuar um diagnóstico precoce e monitorização deste tipo de situações clínicas [77].

4.1.4.Caracterização do dador da amostra analisada

De forma a caracterizar com mais acerto os indivíduos da amostra, foram recolhidos os dados relativamente à sua faixa etária, género e hábitos sociais pertinentes. No que concerne à análise da faixa etária, o intervalo de variação de todos os indivíduos incluídos nos estudos analisados, compreendeu-se entre 18 e 84 anos de idade.

Esta grande variabilidade etária poderá levar a alterações genómicas, repercussões nos processos biológicos e em modificações decorrentes do envelhecimento na cavidade oral (na diminuição da capacidade de produção de saliva pelas glândulas salivares, bem como da composição da saliva) [110,111]. Todos estes fatores, poderão consequentemente levar a alterações de expressão de algumas proteínas [110].

Alusivo à análise dos géneros, os estudos experimentais analisados continham na amostra indivíduos do sexo feminino e masculino. Nos estudos analisados, o tabagismo foi o único hábito social referido, visto que todos os outros hábitos eram fatores de exclusão para esses mesmos estudos.

A presença de hábitos como este, poderá ditar um aumento da inflamação e da tensão exercida nas paredes arteriais (alterações biológicas na vasculatura) [83].

O tabagismo é por isso, considerado um fator de risco modificável para as doenças cardiovasculares, desta forma a sua cessação é um alvo importante no controlo e prevenção de complicações cardiovasculares e deverá ser considerado aquando da análise de dados de proteómica relativos a doenças cardiovasculares e outras condições que envolvam um estado inflamatório alterado [83].

4.1.5. Métodos de amostragem e análise

Neste trabalho foram analisados estudos de proteómica em larga escala, dos quais resultou a identificação de números elevados de proteínas orais em periodontite crónica e estudos mais dirigidos, que permitiram a identificação de um número mais reduzido de proteínas orais em doenças cardiovasculares.

Os estudos dirigidos pretenderam analisar proteínas em que existem evidências prévias da sua possível implicação na doença, ou seja, identificam proteínas que poderão desempenhar um papel importante nas patologias cardiovasculares.

Em contrapartida, o facto de o estudo ser dirigido e o reduzido número de proteínas nele identificadas, faz com que seja mais difícil retirarem-se conclusões significativas acerca das suas funções nas doenças cardiovasculares. Posto isto, será aconselhável realizar mais estudos de proteómica oral nesta área, especialmente de proteómica quantificativa, de forma a identificar um maior número de proteínas e se poderem correlacionar as variações de expressão proteica das doenças comparativamente ao Oraloma normal.

A anotação dos métodos de amostragem e análise é relevante aquando da análise dos dados pois a sua heterogeneidade pode influenciar a deteção de proteínas, tornando mais difícil a interpretação dos dados obtidos nos estudos.

4.1.6. Outras informações

Como referido na secção material e métodos, subseção "Outras informações", foram também analisadas informações consideradas relevantes para a análise e interpretação dos dados de proteómica. Neste item de informações foram anotadas as proteínas que os autores propuseram como biomarcadores (Figura 21).

Assim, das 1241 proteínas anotadas em periodontite crónica, 72 foram sugeridas como biomarcadores, tendo já sido 25 quantificadas. Relativamente às doenças cardiovasculares, apesar dos níveis salivares de diversas proteínas refletirem o mesmo que ocorre no soro, estudos adicionais têm de ser realizados para que sejam feitas sugestões como biomarcadores [78].

Dada a incidência e prevalência das doenças cardiovasculares à escala mundial, associada à dificuldade inerente em ser efetuado um diagnóstico precoce, urge a necessidade em serem descobertos biomarcadores que possam avaliar a suscetibilidade individual a este problema [113].

A identificação, quantificação e aplicação de biomarcadores de diagnóstico, que permitam deduzir de forma precoce a suscetibilidade de um determinado indivíduo ao desenvolvimento de patologias cardiovasculares, poderá representar um contributo fundamental para o controlo, a nível mundial desta condição. No entanto, para que uma proteína possa ser considerada como um biomarcador molecular confiável, terá de existir especificidade [114].

A circunstância de estarmos a falar de biomarcadores de suscetibilidade, é que a especificidade pode depender de um conjunto de eventualidades: a proteína pode ser encontrada exclusivamente em indivíduos com doenças cardiovasculares e deixar de ser identificável quando a condição não está presente (em indivíduos saudáveis); pode apresentar alterações na sua quantificação, derivadas dos mecanismos desregulados (processos biológicos e funções moleculares) ocorrentes nas doenças cardiovasculares.

Pode ainda, uma determinada proteína estar presente em indivíduos com ou sem doença (cardiovascular) e seja possível estabelecer-se um intervalo de quantificação preditivo da probabilidade, de que num dado momento, o individuo se encontre em risco de desenvolver novas lesões.

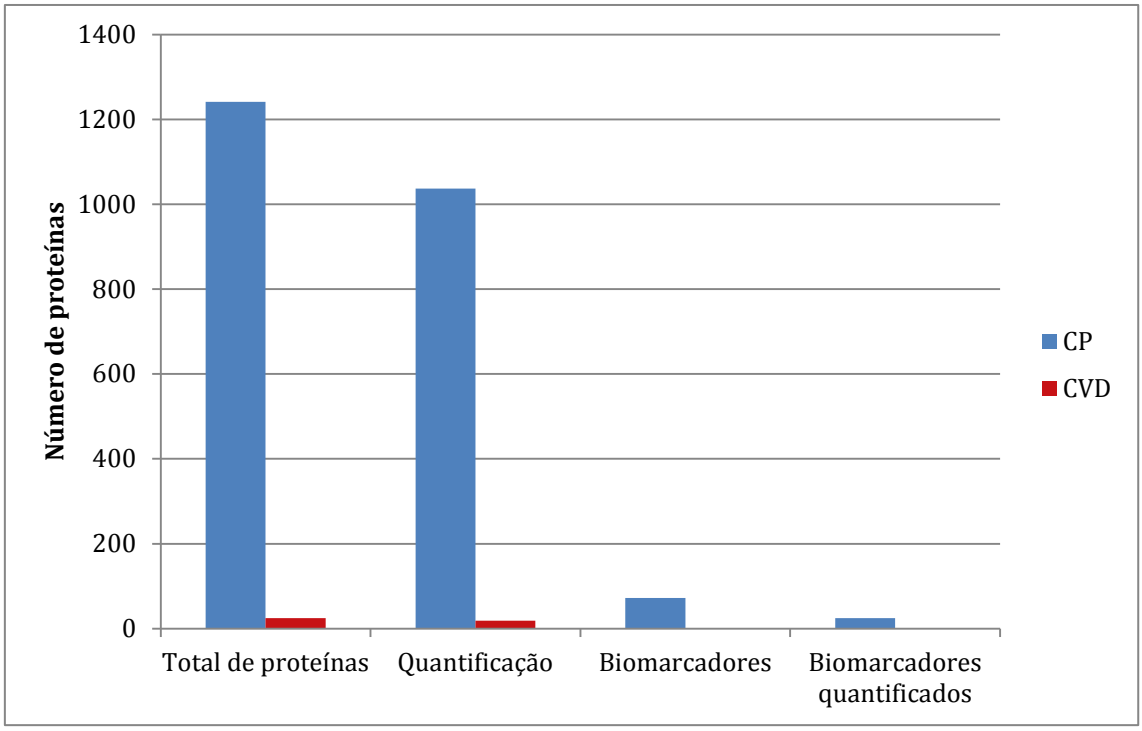


***Figura 21**- Anotação do número de proteínas com potencial de biomarcador em periodontite crónica e doenças cardiovasculares.*

4.2. CARACTERIZAÇÃO FUNCIONAL DO ORALOMA EM PERIODONTITE CRÓNICA E DOENÇAS CARDIOVASCULARES

4.2.1. Processos biológicos das proteínas salivares em periodontite crónica e doenças cardiovasculares

A identificação das proteínas salivares em periodontite crónica e doenças cardiovasculares permitiu a sua catalogação segundo a ontologia "*Biological Process*", recorrendo à ferramenta PANTHER [39,40], de acordo com "Material e métodos – secção Caracterização funcional".

Esta análise permitiu catalogar os processos biológicos em que intervêm as proteínas salivares da periodontite crónica e doenças cardiovasculares (Figura 22).

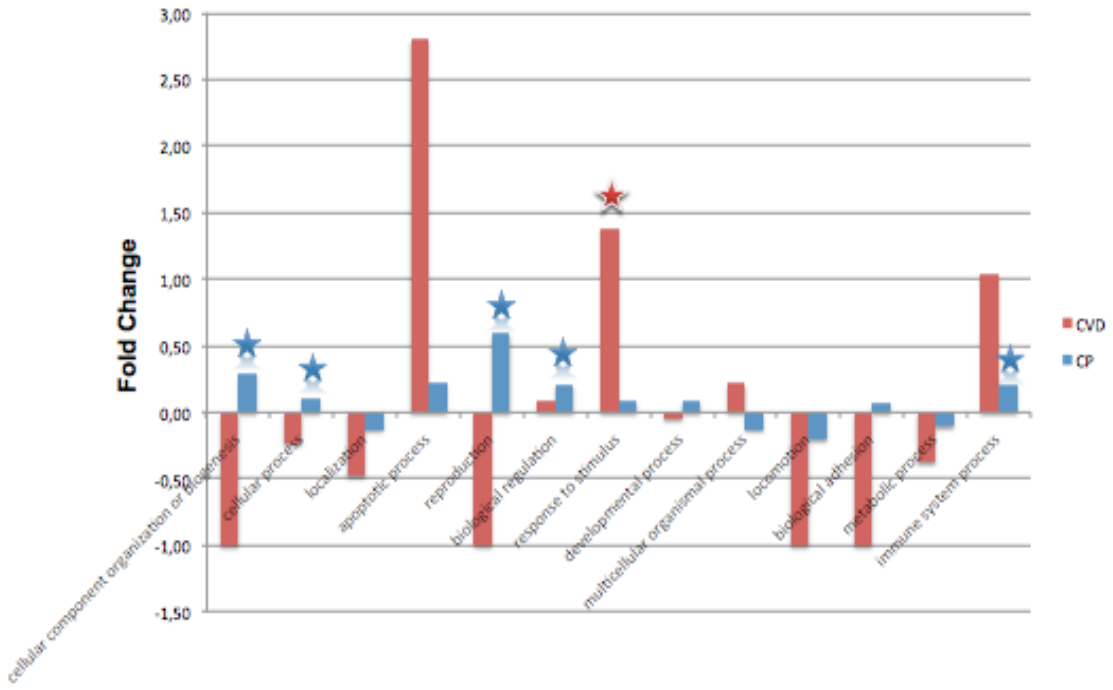


***Figura 22**- Gráfico representativo da distribuição das proteínas salivares em periodontite crónica e doenças cardiovasculares, anotadas na ontologia "Biological Process" do PANTHER [39,40]. É* evidenciada a diferença fraccional entre as proteínas das patologias em e em estudo e o OralOma normal. As estrelas (★) asinalam os processos biológicos que apresentam uma alteração em relação ao OralOma normal considerada estatisticamente significativa (p≤0,05).

A análise da Figura 22 permite concluir que as proteínas salivares em periodontite crónica e doenças cardiovasculares estão envolvidas em treze processos biológicos.

O “*celular component organization or biogenesis*”, “*celular process*”, “*reproduction*”, “*biological regulation*” e o “*immune system process*” estão significativamente aumentados em periodontite crónica com um p-value<0,05; também a “*response to stimulus*” apresentou um aumento com significância estatística, apresentando um p-value<0,05 mas em doenças cardiovasculares.

Dos treze processos biológicos envolvidos nas patologias em estudo, foram selecionados e estudados apenas três (Figura 23), estes considerados os mais importantes pela desregulação apresentada (maior número de proteínas) nas doenças cardiovasculares. O objetivo foi compreender que proteínas salivares comuns em ambas as patologias se encontram mais desreguladas nesses mesmos processos.

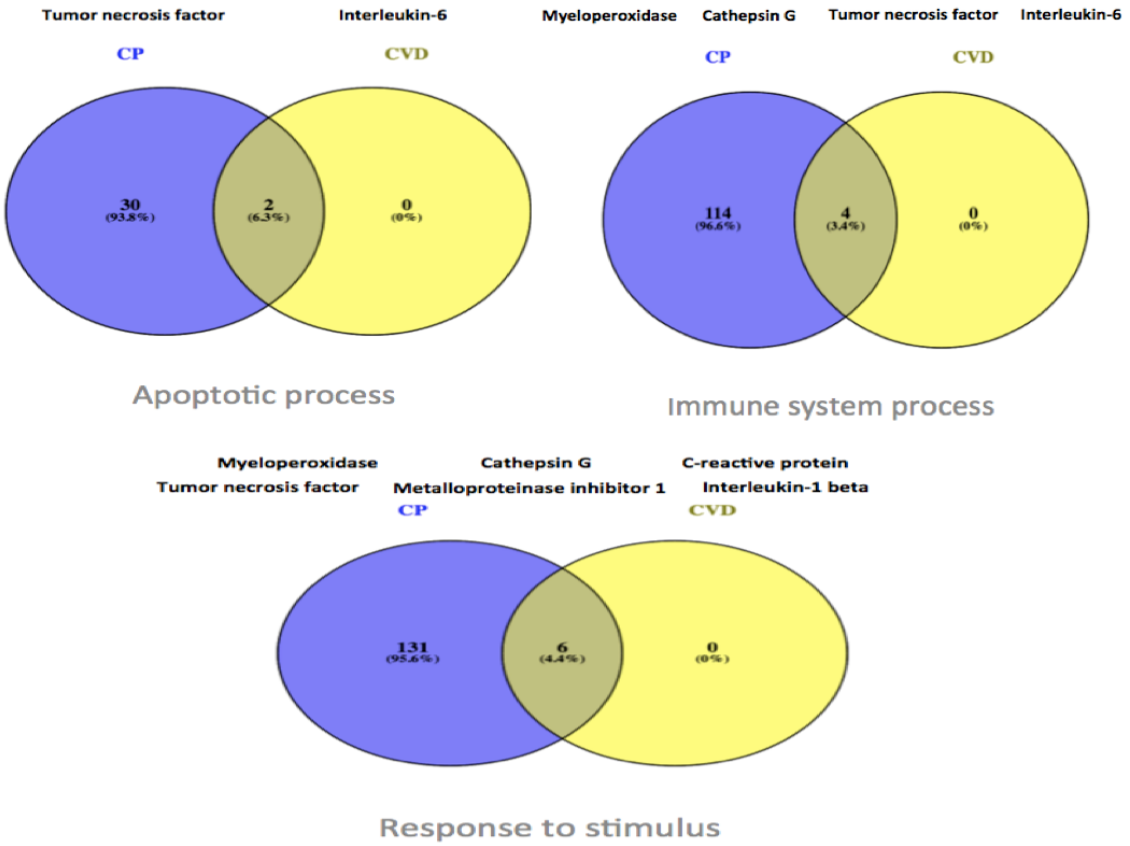


Figura 23*- Distribuição das proteínas salivares comuns identificadas em estudos relativos a periodontite crónica e doenças cardiovasculares, nos processos biológicos mais desregulados (e cima de cada processo biológico são mencionadas as proteínas comuns em ambas as patologias) (gráfico obtido com recurso ao programa Venny [83]).*

Analisando as proteínas envolvidas nos processos de "*response to stimulus"* e "*immune system process",* constatou-se que a maioria delas são citocinas. Estas desempenham um papel importante na comunicação intercelular, no sistema imunitário, na ativação e resolução da inflamação, levando a reações imunes e resultando no aumento da aterosclerose [115].

A presença aumentada de proteínas relacionadas a estes processos biológicos alterados nas doenças cardiovasculares, é consistente com o conhecimento relativamente à presença de inflamação associada à patologia [116].

Um dos processos biológicos mais alterados em doenças cardiovasculares, comparativamente ao Oraloma normal, como é possível verificar na Figura 20, é o processo "*Apoptosis*".

Foi demonstrado que a apoptose poderá estar envolvida na perda aguda e crónica de cardiomiócitos no enfarte do miocárdio, doença cardíaca isquémica, várias formas de cardiomiopatia e no desenvolvimento de insuficiência cardíaca. Conjuntamente, o aparecimento da apoptose no tecido muscular estriado cardíaco (miocárdio), ainda numa fase inicial após a sua lesão e passado um mês, sugerem que esta possa ser um fator importante para a sua remodelação e no subsequente desenvolvimento de insuficiência cardíaca (pela perda dos cardiomiócitos) [116].

É evidente que a apoptose desempenha um papel fulcral na patogénese de várias doenças cardiovasculares e a inibição deste processo biológico poderá ser um alvo extremamente importante para a sua intervenção terapêutica [113,116]. Num estudo de *Kalliolias e Ivashkiv* [117]*,* sugerem que *TNF-α* é cardiotóxico para o miocárdio saudável e potencialmente cardioprotetor quando este falha. A sua cardiotoxicidade está ligada à sua indução após a apoptose dos cardiomiócitos. Para além do seu papel no miocárdio, esta proteína está também implicada na patogénese da aterosclerose, afetando o metabolismo lipídico, na ativação de células endoteliais e consequentemente na indução da inflamação vascular [117].

A identificação destas proteínas no proteoma oral em doenças cardiovasculares, envolvidas neste processo biológico, impulsiona a importância da apoptose no desenvolvimento das doenças cardiovasculares.

No entanto, são necessários mais estudos para compreender os mecanismos moleculares que governam estes processos [116].

4.2.2. Funções moleculares das proteínas salivares em periodontite crónica e doenças cardiovasculares

A identificação das proteínas salivares em periodontite crónica e doenças cardiovasculares permitiu a sua catalogação segundo a ontologia "*Molecular function*", recorrendo à ferramenta PANTHER [39,40], de acordo com "Material e métodos – secção Caracterização funcional".

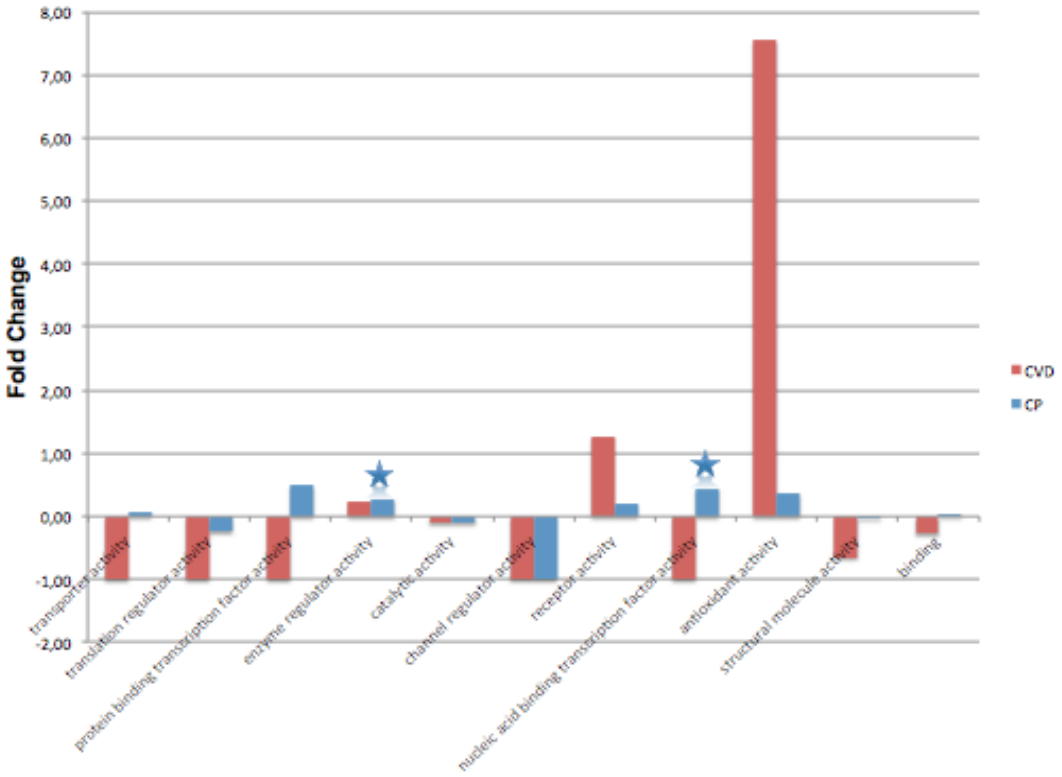


***Figura 24**- Gráfico representativo da distribuição das proteínas salivares em periodontite crónica e doenças cardiovasculares, anotadas na ontologia "Molecular function" do PANTHER [39,40]. É evidenciada a diferença fraccional entre as proteínas das patologias em estudo e o OralOma normal. As estrelas assinalam as funções moleculares que apresentam uma alteração em relação ao OralOma normal considerada estatisticamente* significativa (p≤0,05).

Esta análise permitiu catalogar as funções moleculares desempenhadas pelas proteínas salivares em periodontite crónica e doenças cardiovasculares (Figura 24).

A análise da Figura 24 permite inferir que as proteínas salivares em periodontite crónica e doenças cardiovasculares estão envolvidas em

onzefunções moleculares. As proteínas salivares com“enzyme regulator activity”e “*nucleic acid binding transcription factor activity”* possuem um aumento estatisticamente significativo em periodontite crónica com um p-value<0,05.

Das onze funções moleculares envolvidas nas patologias em estudo, foram selecionadas e estudadas apenas quatro (Figura 25), estas consideradas as mais pertinentes pela desregulação apresentada nas doenças cardiovasculares. O objetivo foi compreender que proteínas salivares comuns em ambas as patologias se encontram mais desreguladas nessas mesmas funções.

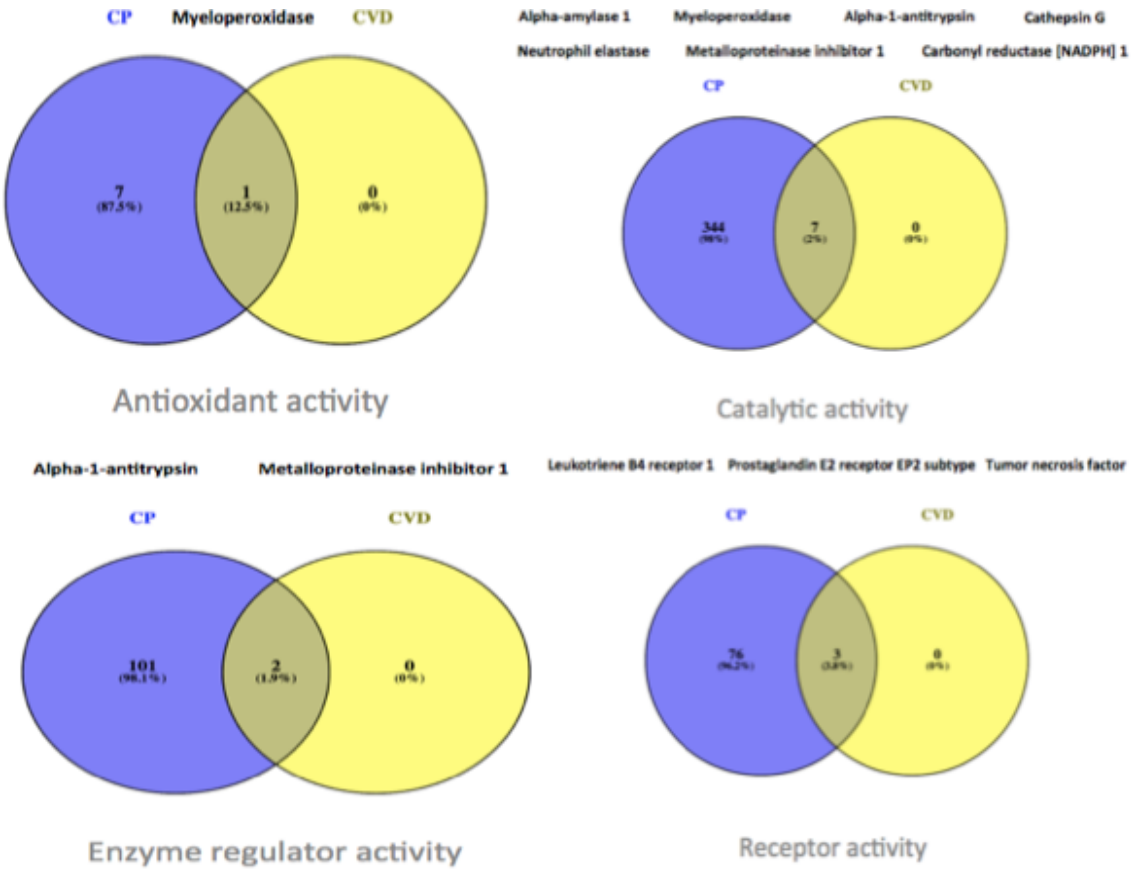


Figura 25*- Distribuição das proteínas salivares comuns identificadas em estudos relativos a periodontite crónica e doenças cardiovasculares, nas funções moleculares mais desreguladas (e cima de cada função molecular são mencionadas as proteínas comuns em ambas as patologias) (gráfico obtido com recurso ao programa Venny [83]).*

O fundamento para o aumento evidente do número de proteínas orais com função antioxidante pode estar ligada ao facto de os pacientes com doenças cardiovasculares apresentarem uma maior propensão em sofrerem stress oxidativo [118].

Sabe-se que concentrações significativas das moléculas envolvidas no stress oxidativo agem na sinalização e desempenham um papel determinante na regulação do tónus vascular, crescimento e proliferação celular, apoptose e resposta inflamatória [118]. Além disso, o stress oxidativo na parede vascular promove aterogénese, tendo sido implicado desta forma na patogénese de várias doenças cardiovasculares, incluindo a aterosclerose [118].

Uma das principais moléculas relacionadas com este processo é a *Myeloperoxidase* (Figura 26), libertada pelos neutrófilos na sequência da resposta inflamatória desencadeada nas placas de aterosclerose [119,120]. Apesar dos resultados obtidos na figura 26 não irem ao encontro do que seria de esperar, ou seja, a presença de um aumento desta proteína em indivíduos com doenças cardiovasculares, devemos ter em consideração que o valor apresentado representa a média da regulação nos diversos estudos, sendo que grande parte dos estudos em que foi identificada apresentou uma regulação aumentada. O facto de não se observar o que seria de esperar, pode dever-se pelo motivo de que os indivíduos neste estudo eram fumadores, o que poderá levar a que esta proteína se encontre contra-regulada [66].

No entanto, pode afirmar-se que, em pacientes com doenças cardiovasculares, ocorre uma sobre-regulação homeostática do sistema enzimático antioxidante em resposta ao aumento dos radicais livres para evitar danos vasculares [121].

É de salientar também o aumento do número de proteínas orais envolvidas na "*receptor activity*" em doenças cardiovasculares. A razão pelo qual acontece, prende-se pelo facto da actividade de receptores como; o *Leukotriene B4 receptor 1* e *Prostaglandin E2 receptor EP2* (Figura 26), estarem envolvidos no recrutamento de células do sistema imunológico e na regulação da inflamação das placas ateroscleróticas [122].

Neste sentido, estes mediadores lipídicos favorecem a destabilização e consequente rotura das placas ateroscleróticas, pelo que poderão representar possíveis alvos terapêuticos para controlar a inflamação ocorrente nessas mesmas placas [122].

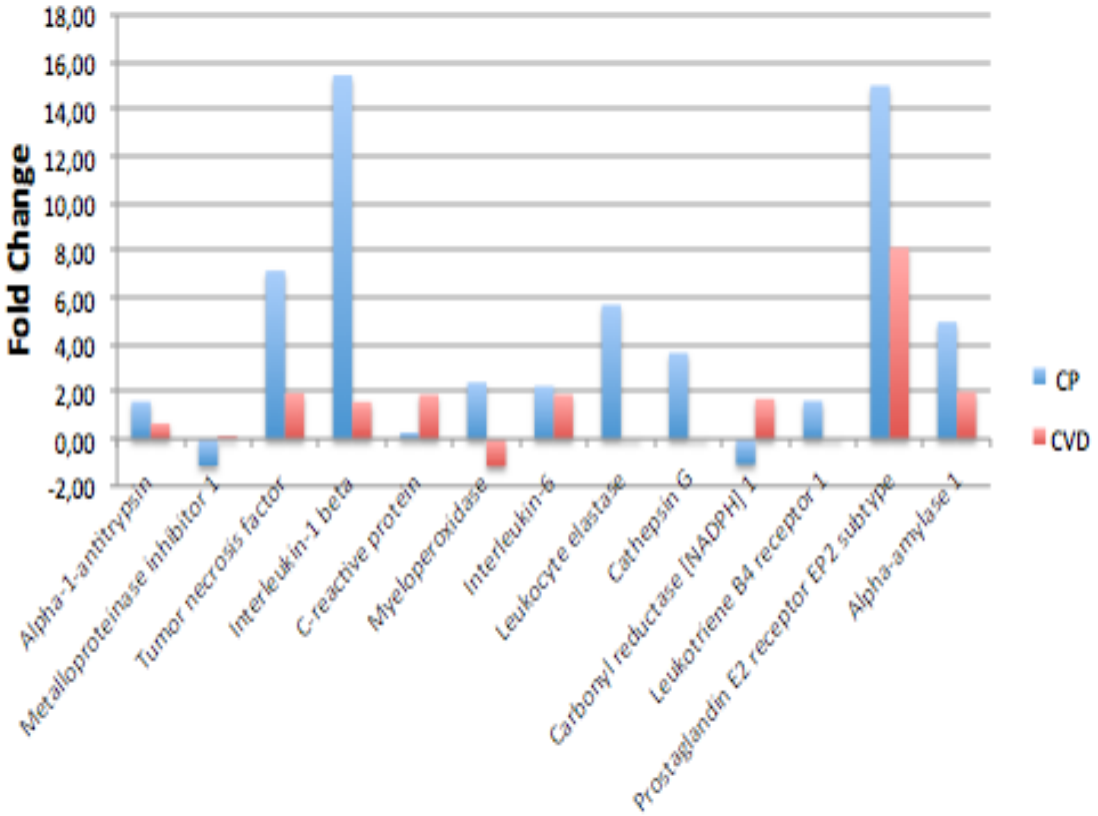


***Figura 26**- Dados de quantificação das proteínas salivares comuns em ambas as patologias, nos processos biológicos e funções moleculares mais desregulados, recorrendo à ferramenta PANTHER.*

5. CONCLUSÃO

Neste trabalho foi efetuada uma análise de resultados de estudos de proteómica oral, com amostras obtidas da cavidade oral de indivíduos com periodontite crónica e doenças cardiovasculares, sendo realizada uma anotação manual de informações consideradas imprescindíveis para a interpretação dos dados catalogados. Esta pesquisa e anotação permitiu a atualização da base de dados do OralCard, compilando pela primeira vez informação respeitante às doenças cardiovasculares nesta base de dados.

A análise de estudos de proteómica no contexto das doenças cardiovasculares permitiu a identificação de um total de 25 proteínas salivares o que, quando comparado com o número de proteínas já identificadas em periodontite crónica (1241), salienta a necessidade da realização de novos estudos de proteómica com a finalidade em identificar um maior número de proteínas relacionadas a estas patologias. Só com este incremento de informação molecular será possível, no futuro, identificar e validar biomarcadores de suscetibilidade às doenças cardiovasculares na saliva.

É necessário realçar que são muitas as variáveis que podem influenciar a comparação entre os estudos selecionados e os próprios resultados relatados. Como exemplos disso tem-se: a falta da padronização de protocolos de recolha de saliva e metodologias usadas para a sua análise, o tipo de origem das amostras (saliva total, fluído crevicular e mucosa), bem como a variação biológica normal das moléculas na saliva (variação diurna, variação intra-sujeito, idade e sexo) e a variação interindividual (dieta, estilos de vida, medicamentos, tabaco, álcool). Deste modo, este tipo de estudos que compilam e integram a informação e a interpretam tendo em conta os parâmetros de variabilidade, são imprescindíveis para o avanço do conhecimento molecular e a identificação de biomarcadores de saúde e doença.

Relativamente à caracterização funcional do Oraloma das doenças cardiovasculares, esta permitiu verificar um número elevado de proteínas orais envolvidas nos processos biológicos e funções moleculares mais desregulados, contribuindo desde logo para o desenvolvimento das doenças cardiovasculares, seja pelo recrutamento de células do sistema imune ou no papel que têm na regulação da inflamação nas placas ateroscleróticas.

Posto isto, pretende-se que a realização deste trabalho possa contribuir para uma melhor compreensão dos mecanismos biológicos e moleculares que levam ao desenvolvimento das doenças cardiovasculares. No entanto serão necessários mais estudos que relacionem as moléculas comuns às duas doenças para que esta relação seja completamente esclarecida.

Por último, o presente trabalho demonstrou que existem diferenças em relação aos dados de quantificação das proteínas salivares comuns em ambas as patologias permitindo a investigadores nesta área efetuarem diversos estudos e identificarem biomarcadores diferenciadores das duas condições, bem como da associação entre a periodontite crónica e as doenças cardiovasculares.

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7. ANEXOS

Tabela A

Lista de proteínas salivares identificadas em periodontite crónica até ao momento e depositadas na base de dados do OralCard (**Tabela A**).

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
A0AVI2	FER1L5	Fer-1-like protein 5	Homo sapiens (Human)						x					x	CP	68055113	1,04	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A0AVT1	UBA6 MOP4 UBE1L2	Ubiquitin-like modifier-activating enzyme 6 (Ubiquitin-activating enzyme 6) (Monocyte protein 4) (MOP-4) (Ubiquitin-activating enzyme E1-like protein 2) (E1-L2)	Homo sapiens (Human)						x					x	CP	68055113	3,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A0FGR8	ESYT2 FAM62B KIAA1228	Extended synaptotagmin-2 (E-Syt2) (Chr2Syt)	Homo sapiens (Human)						x					x	CP	68055113	3,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A0JLQ0	AZGP1	AZGP1 protein (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A0M8Q6	IGLC7	Ig lambda-7 chain C region	Homo sapiens (Human)						x					x	CP	68055113	-2,50	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A0N5G5	V<kappa>3	Rheumatoid factor D5 light chain (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
A0PJH2	ATP5H	ATP5H protein (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,51	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A1E959	ODAM APIN	Odontogenic ameloblast-associated protein (Apin)	Homo sapiens (Human)							x					CP	68055113	,38(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	non-proteomics			24122488
A2KLM6	IGVH	Immunolgoobulin heavy chain (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
A2N2F5	VL4	VL4 protein (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A2RTX5	TARSL2	Probable threonine--tRNA ligase 2, cytoplasmic (EC 6.1.1.3) (Threonyl-tRNA synthetase) (ThrRS) (Threonyl-tRNA synthetase-like protein 2)	Homo sapiens (Human)						x					x	CP	68055113	1,25	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A4D0S4	LAMB4	Laminin subunit beta-4 (Laminin beta-1-related protein)	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A4D1T9	PRSS37 TRYX2	Probable inactive serine protease 37 (Probable inactive trypsin-X2)	Homo sapiens (Human)						x					x	CP	68055113	2,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A5A3E0	POTEF A26C1B	POTE ankyrin domain family member F (ANKRD26-like family C member 1B) (Chimeric POTE-actin protein)	Homo sapiens (Human)						x						CP	68055113	15,00						Proteomics			24098404
P81605	DCD AIDD DSEP	Dermcidin (EC 3.4.-.-) (Preproteolysin) [Cleaved into: Survival-promoting peptide; DCD-1]	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q17R89	ARHGAP44 KIAA0672 RICH2	Rho GTPase-activating protein 44 (NPC-A-10) (Rho-type GTPase-activating protein RICH2) (RhoGAP interacting with CIP4 homologs protein 2) (RICH-2)	Homo sapiens (Human)						x					x	CP	68055113	2,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q71UI9	H2AFV H2AV	Histone H2A.V (H2A.F/Z)	Homo sapiens (Human)						x						CP	68055113	6,00						Proteomics			24098404
A6NGQ3	OBSCN	Obscurin	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A6NIZ0	NDEL1	Nuclear distribution protein nudE-like 1	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A6NJ16	IGHV4OR15-8 VSIG6	Putative V-set and immunoglobulin domain-containing-like protein IGHV4OR15-8 (Immunoglobulin heavy variable 4 orphon 15-8) (Putative V-set and immunoglobulin domain-containing protein 6)	Homo sapiens (Human)						x					x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
A6NL76	ACTA1	Actin, alpha skeletal muscle	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q71UI9	H2AFV H2AV	Histone H2A.V (H2A.F/Z)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			
A7E2Y1	MYH7B KIAA1512	Myosin-7B (Antigen MLAA-21) (Myosin cardiac muscle beta chain) (Myosin heavy chain 7B, cardiac muscle beta isoform) (Slow A MYH14)	Homo sapiens (Human)						x					x	CP	68055113	11,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A7KAX9	ARHGAP32 GRIT KIAA0712 RICS	Rho GTPase-activating protein 32 (Brain-specific Rho GTPase-activating protein) (GAB-associated Cdc42/Rac GTPase-activating protein) (GC-GAP) (GTPase regulator interacting with TrkA) (Rho-type GTPase-activating protein 32) (Rho/Cdc42/Rac GTPase-activating protein RICS) (RhoGAP involved in the beta-catenin-N-cadherin and NMDA receptor signaling) (p200RhoGAP) (p250GAP)	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
A8K2U0	A2ML1 CPAMD9	Alpha-2-macroglobulin-like protein 1 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 9)	Homo sapiens (Human)						x					x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
A8K2U0	A2ML1 CPAMD9	Alpha-2-macroglobulin-like protein 1 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 9)	Homo sapiens (Human)						x					x	CP	68055113	3,68	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8K2U0	A2ML1 CPAMD9	Alpha-2-macroglobulin-like protein 1 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 9)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
A8MQ03	CYSRT1 C9orf169	Cysteine-rich tail protein 1	Homo sapiens (Human)						x					x	CP	68055113	1,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8MT21	KRT15	Keratin, type I cytoskeletal 15	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P68366	TUBA4A TUBA1	Tubulin alpha-4A chain (Alpha-tubulin 1) (Testis-specific alpha-tubulin) (Tubulin H2-alpha) (Tubulin alpha-1 chain)	Homo sapiens (Human)						x					x	CP	68055113	2,38	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8MVG2	SKINTL SKINT1	Putative selection and upkeep of intraepithelial T-cells protein 1 homolog (Skint-1) (Skint-like pseudogene)	Homo sapiens (Human)						x					x	CP	68055113	5,17	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8MX94	GSTP1	Glutathione S-transferase P	Homo sapiens (Human)						x					x	CP	68055113	2,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8MZ87	KLC2	Kinesin light chain 2	Homo sapiens (Human)						x					x	CP	68055113	8,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
A8MZH6	OOSP1	Putative oocyte-secreted protein 1 homolog	Homo sapiens (Human)						x					x	CP	68055113	11,45	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B0YIW2	APOC3	Apolipoprotein C-III (Apolipoprotein C-III variant 1)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B0YJC4	VIM	Vimentin (Vimentin variant 3)	Homo sapiens (Human)						x					x	CP	68055113	2,20						Proteomics			24098404
B0YJC5	VIM	Vimentin (Vimentin variant 4)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B1AH78	RAC2	Ras-related C3 botulinum toxin substrate 2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,22	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B1AJY5	PSMD10	26S proteasome non-ATPase regulatory subunit 10	Homo sapiens (Human)						x					x	CP	68055113	3,94	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B1AK87	CAPZB hCG_41078	Capping protein (Actin filament) muscle Z-line, beta, isoform CRA_a (F-actin-capping protein subunit beta)	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B1AN48	SPRR3	Small proline-rich protein 3 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	5,24	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B2R825	0	Alpha-1,4 glucan phosphorylase (EC 2.4.1.1)	Homo sapiens (Human)						x					x	CP	68055113	3,96	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3EWG3	FAM25A	Protein FAM25A	Homo sapiens (Human)						x					x	CP	68055113	2,88	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KPA5	0	cDNA FLJ31518 fis, clone NT2RI2000064	Homo sapiens (Human)						x					x	CP	68055113	3,12	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P19012	KRT15 KRTB	Keratin, type I cytoskeletal 15 (Cytokeratin-15) (CK-15) (Keratin-15) (K15)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B3KSA0	0	cDNA FLJ35812 fis, clone TESTI2006051, highly similar to Acyl-coenzyme A thioesterase 1 (EC 3.1.2.2)	Homo sapiens (Human)						x					x	CP	68055113	2,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KTE1	0	Actin-related protein 2/3 complex subunit 5	Homo sapiens (Human)						x					x	CP	68055113	5,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KTE3	0	cDNA FLJ38125 fis, clone D6OST2000127, moderately similar to RAS-RELATED PROTEIN RAB-8B	Homo sapiens (Human)						x					x	CP	68055113	3,77	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KUS0	SLC12A4 hCG_2039906	Solute carrier family 12 (Potassium/chloride transporters), member 4, isoform CRA_e (cDNA FLJ40489 fis, clone TESTI2044194, highly similar to Solute carrier family 12 member 4)	Homo sapiens (Human)						x					x	CP	68055113	2,16	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KVK2	GNB1	Guanine nucleotide-binding protein G(I)/G(S)/G(T) subunit beta-1 (cDNA FLJ16663 fis, clone THYMU2005759, highly similar to Guanine nucleotide-binding proteinG(I)/G(S)/G(T) subunit beta 1)	Homo sapiens (Human)						x					x	CP	68055113	-1,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B3KVW7	0	cDNA FLJ41648 fis, clone FEBRA2024150, moderately similar to Rab6-interacting protein 1	Homo sapiens (Human)						x					x	CP	68055113	23,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DDD4	0	cDNA FLJ60408, highly similar to Homo sapiens WD repeat domain 27 (WDR27), mRNA	Homo sapiens (Human)						x					x	CP	68055113	3,78	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9UJU6	DBNL CMAP SH3P7 PP5423	Drebrin-like protein (Cervical SH3P7) (Cervical mucin-associated protein) (Drebrin-F) (HPK1-interacting protein of 55 kDa) (HIP-55) (SH3 domain-containing protein 7)	Homo sapiens (Human)						x					x	CP	68055113	2,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P40926	MDH2	Malate dehydrogenase, mitochondrial (EC 1.1.1.37)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DE78	0	cDNA FLJ52141, highly similar to 14-3-3 protein gamma	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B4DEI3	0	cDNA FLJ57715, highly similar to Voltage-dependent anion-selective channel protein 1	Homo sapiens (Human)						x					x	CP	68055113	3,82	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DF49	0	cDNA FLJ52535, highly similar to Homo sapiens lysosomal-associated membrane protein 2 (LAMP2), transcript variant LAMP2B, mRNA	Homo sapiens (Human)						x					x	CP	68055113	3,30	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DF70	0	cDNA FLJ60461, highly similar to Peroxiredoxin-2 (EC 1.11.1.15)	Homo sapiens (Human)						x					x	CP	68055113	3,56	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DG06	0	cDNA FLJ61229, highly similar to RasGAP-activating-like protein 1	Homo sapiens (Human)						x					x	CP	68055113	3,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DHV9	0	cDNA FLJ59258, highly similar to Grave disease carrier protein	Homo sapiens (Human)						x					x	CP	68055113	37,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B4DIC4	0	cDNA FLJ52195, highly similar to LIM and SH3 domain protein 1	Homo sapiens (Human)						x					x	CP	68055113	2,38	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P39656	DDOST KIAA0115 OST48 OK/SW-cl.45	Dolichyl-diphosphooligosaccharide--protein glycosyltransferase 48 kDa subunit (DDOST 48 kDa subunit) (Oligosaccharyl transferase 48 kDa subunit) (EC 2.4.99.18)	Homo sapiens (Human)						x					x	CP	68055113	4,47	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DKK9	0	cDNA FLJ52967, highly similar to DnaJ homolog subfamily B member 1	Homo sapiens (Human)						x					x	CP	68055113	1,89	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DL87	0	cDNA FLJ52243, highly similar to Heat-shock protein beta-1	Homo sapiens (Human)						x					x	CP	68055113	2,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DL87	0	cDNA FLJ52243, highly similar to Heat-shock protein beta-1	Homo sapiens (Human)						x					x	CP	68055113	1,56						Proteomics			24098404
B4DLI3	0	cDNA FLJ51237, moderately similar to Carcinoembryonic antigen-related cell adhesion molecule 8	Homo sapiens (Human)						x					x	CP	68055113	2,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DLV7	0	cDNA FLJ60299, highly similar to Rab GDP dissociation inhibitor beta	Homo sapiens (Human)						x					x	CP	68055113	3,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DM33	0	cDNA FLJ52068, highly similar to Microtubule-associated protein RP/EB family member 1	Homo sapiens (Human)						x					x	CP	68055113	2,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B4DM79	0	cDNA FLJ53848, highly similar to Inter-alpha-trypsin inhibitor heavy chain H2	Homo sapiens (Human)						x					x	CP	68055113	2,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DM97	0	cDNA FLJ55002, highly similar to Alpha-centractin	Homo sapiens (Human)						x					x	CP	68055113	3,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DNQ3	0	cDNA FLJ60741, highly similar to Receptor-interacting serine/threonine-protein kinase 5 (EC 2.7.11.1)	Homo sapiens (Human)						x					x	CP	68055113	3,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DNW1	0	cDNA FLJ59940, highly similar to Tubulin beta-2C chain	Homo sapiens (Human)						x					x	CP	68055113	4,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DP93	0	cDNA FLJ53437, highly similar to Major vault protein	Homo sapiens (Human)						x					x	CP	68055113	2,99	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DPJ2	0	Annexin	Homo sapiens (Human)						x					x	CP	68055113	3,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DPN0	0	cDNA FLJ51265, moderately similar to Beta-2-glycoprotein 1 (Beta-2-glycoprotein I)	Homo sapiens (Human)						x					x	CP	68055113	1,59	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B4DQ53	0	cDNA FLJ51275	Homo sapiens (Human)						x					x	CP	68055113	3,87	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9BRF8	CPPED1 CSTP1	Serine/threonine-protein phosphatase CPPED1 (EC 3.1.3.16) (Calcineurin-like phosphoesterase domain-containing protein 1) (Complete S-transactivated protein 1)	Homo sapiens (Human)						x					x	CP	68055113	3,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DQ92	0	cDNA FLJ59379, highly similar to Hematopoietic lineage cell-specific protein	Homo sapiens (Human)						x					x	CP	68055113	2,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DQ93	0	cDNA FLJ52996, highly similar to Syntenin-1	Homo sapiens (Human)						x					x	CP	68055113	3,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DR52	0	Histone H2B	Homo sapiens (Human)						x					x	CP	68055113	1,84						Proteomics			24098404
B4DRL2	0	cDNA FLJ60138	Homo sapiens (Human)						x					x	CP	68055113	-1,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DRR0	0	cDNA FLJ53910, highly similar to Keratin, type II cytoskeletal 6A	Homo sapiens (Human)						x					x	CP	68055113	4,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DRW1	0	cDNA FLJ55805, highly similar to Keratin, type II cytoskeletal 4	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B4DSX6	0	cDNA FLJ57427, highly similar to Glycogenin-1 (EC 2.4.1.186)	Homo sapiens (Human)						x					x	CP	68055113	2,07	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B4DUA5	0	cDNA FLJ59430, highly similar to Protein disulfide-isomerase (EC 5.3.4.1)	Homo sapiens (Human)						x					x	CP	68055113	2,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DV10	0	cDNA FLJ59142, highly similar to Epididymal secretory protein E1	Homo sapiens (Human)						x					x	CP	68055113	2,59	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DVE9	0	cDNA FLJ51605, moderately similar to Homo sapiens RNA binding motif protein 18 (RBM18), mRNA	Homo sapiens (Human)						x					x	CP	68055113	4,58	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DW08	0	cDNA FLJ50886, highly similar to Aconitate hydratase, mitochondrial(EC 4.2.1.3)	Homo sapiens (Human)						x					x	CP	68055113	10,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DWP0	0	cDNA FLJ53107, highly similar to Squalene synthetase (EC 2.5.1.21)	Homo sapiens (Human)						x					x	CP	68055113	1,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4DXR3	0	cDNA FLJ58640, highly similar to Calmodulin-binding transcription activator 1	Homo sapiens (Human)						x					x	CP	68055113	14,77	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P25705	ATP5A1 ATP5A ATP5AL2 ATPM	ATP synthase subunit alpha, mitochondrial	Homo sapiens (Human)						x					x	CP	68055113	3,14	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender#	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B4E0J3	0	cDNA FLJ58965, highly similar to Nonspecific lipid-transfer protein (EC 2.3.1.176)	Homo sapiens (Human)						x					x	CP	68055113	2,76	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4E0T0	0	cDNA FLJ51589, highly similar to Neutrophil collagenase (EC 3.4.24.34)	Homo sapiens (Human)						x					x	CP	68055113	3,19	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4E190	0	cDNA FLJ57770, moderately similar to ADP-ribosylation factor 3	Homo sapiens (Human)						x					x	CP	68055113	3,16	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08236	GUSB	Beta-glucuronidase (EC 3.2.1.31) (Beta-G1)	Homo sapiens (Human)						x					x	CP	68055113	3,43	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4E1R7	0	cDNA FLJ58224, highly similar to Calpain-2 catalytic subunit (EC 3.4.22.53)	Homo sapiens (Human)						x					x	CP	68055113	2,53	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4E310	0	cDNA FLJ53133, highly similar to Erythrocyte band 7 integral membrane protein	Homo sapiens (Human)						x					x	CP	68055113	3,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B4E335	0	cDNA FLJ52842, highly similar to Actin, cytoplasmic 1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B7WNR0	ALB	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113	2,74						Proteomics			24098404
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21.-) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kaliocin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B7Z5E7	0	cDNA FLJ51046, highly similar to 60 kDa heat shock protein, mitochondrial	Homo sapiens (Human)						x					x	CP	68055113	2,72	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
B7Z972	0	Protein-L-isoaspartate O-methyltransferase (EC 2.1.1.77)	Homo sapiens (Human)						x					x	CP	68055113	2,67	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B7ZLF8	0	Uncharacterized protein	Homo sapiens (Human)						x					x	CP	68055113	2,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9ULD4	BRPF3 KIAA1286	Bromodomain and PHD finger-containing protein 3	Homo sapiens (Human)						x					x	CP	68055113	2,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B7ZW15	0	Uncharacterized protein	Homo sapiens (Human)						x					x	CP	68055113	3,14	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
B8ZZJ2	ACTG2	Actin, gamma-enteric smooth muscle	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
B9A041	MDH1	Malate dehydrogenase, cytoplasmic	Homo sapiens (Human)						x					x	CP	68055113	2,78	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9IYB4	PDK1	[Pyruvate dehydrogenase (acetyl-transferring)] kinase isozyme 1, mitochondrial (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	4,47	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9J0D1	H2AFV	Histone H2A	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9J0E4	CSTA	Cystatin-A	Homo sapiens (Human)						x					x	CP	68055113	2,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9J386	H2AFV	Histone H2A	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9J3N8	HSPB1	Heat shock protein beta-1	Homo sapiens (Human)						x					x	CP	68055113	1,80						Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
C9J6B6	EIF4G1	Eukaryotic translation initiation factor 4 gamma 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	18,95	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9J6C2	RFTN2	Raftlin-2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,88	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9JCF5	LTF	Lactotransferrin (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JFL5	ACTG2	Actin, gamma-enteric smooth muscle (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JGI3	TYMP	Thymidine phosphorylase (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,16	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9JKR2	ALB hCG_14967	Albumin, isoform CRA_k (Serum albumin)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JM50	KRT19	Keratin, type I cytoskeletal 19 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JTV4	EFHD1	EF-hand domain-containing protein D1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9JTX5	ACTB	Actin, cytoplasmic 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JUM1	ACTB	Actin, cytoplasmic 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
C9JZ81	XRCC5	X-ray repair cross-complementing protein 5 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9JZD1	ARPC3	Actin-related protein 2/3 complex subunit 3 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
C9JZR7	ACTB	Actin, cytoplasmic 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
D1MGQ2	HBA2 hCG_1745306	Alpha-2 globin chain (Delta globin) (HCG1745306, isoform CRA_b)	Homo sapiens (Human)						x					x	CP	68055113	7,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
D3DP16	FGG hCG_28288	Fibrinogen gamma chain, isoform CRA_a	Homo sapiens (Human)						x					x	CP	68055113	1,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
D8RA82	ANXA3	Annexin	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
D8RAZ8	ANXA3	Annexin (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
D8RCA8	ANXA3	Annexin (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,59						Proteomics			24098404
D8RF35	GC	Vitamin D-binding protein	Homo sapiens (Human)						x					x	CP	68055113	2,06						Proteomics			24098404
D8RFG5	ANXA3	Annexin (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
D8RHD5	ALB	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
E7EMB3	CALM2	Calmodulin	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E7EQB2	LTF	Lactotransferrin (Fragment)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E7ER44	LTF	Lactotransferrin	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E7ETZ0	CALM1	Calmodulin	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E7EVS6	ACTB	Actin, cytoplasmic 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
E7EWB4	DPYSL5	Dihydropyrimidinase-related protein 5 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
E9PEW8	HBD	Hemoglobin subunit delta (Fragment)	Homo sapiens (Human)						x						CP	68055113	23,00						Proteomics			24098404
E9PFT6	HBD	Hemoglobin subunit delta	Homo sapiens (Human)						x						CP	68055113	23,00						Proteomics			24098404
P63267	ACTG2 ACTA3 ACTL3 ACTSG	Actin, gamma-enteric smooth muscle (Alpha-actin-3) (Gamma-2-actin) (Smooth muscle gamma-actin)	Homo sapiens (Human)						x						CP	68055113	17,00						Proteomics			24098404
E9PJD2	CMPK1	UMP-CMP kinase	Homo sapiens (Human)						x					x	CP	68055113	1,14	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
E9PK25	CFL1	Cofilin-1	Homo sapiens (Human)						x						CP	68055113	4,00						Proteomics			24098404
E9PLJ3	CFL1	Cofilin-1 (Fragment)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E9PM76	SULT1A2	Sulfotransferase (EC 2.8.2.-)	Homo sapiens (Human)						x					x	CP	68055113	3,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
E9PP50	CFL1	Cofilin-1 (Fragment)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E9PQB7	CFL1	Cofilin-1 (Fragment)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
E9PQZ1	EEF1D	Elongation factor 1-delta	Homo sapiens (Human)						x					x	CP	68055113	2,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
F5GWP8	KRT17	Keratin, type I cytoskeletal 17	Homo sapiens (Human)						x						CP	68055113	7,00						Proteomics			24098404
F5H6H4	CLIP1	CAP-Gly domain-containing linker protein 1	Homo sapiens (Human)						x					x	CP	68055113	10,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
F5H763	NUMA1	Nuclear mitotic apparatus protein 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,93	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
F8VSA6	NEDD8	NEDD8	Homo sapiens (Human)						x					x	CP	68055113	3,07	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
F8WDS9	LANCL1	LanC-like protein 1	Homo sapiens (Human)						x					x	CP	68055113	-1,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O00206	TLR4	Toll-like receptor 4 (hToll) (CD antigen CD284)	Homo sapiens (Human)					x						x	CP	68055113	2,41	40-65	MF		Participants were asked to come to the laboratory at 07:00 am following an overnight fast, during which they were instructed not to eat, drink (except water), chew gum or brush teeth. Whole saliva samples were obtained by expectorating into polypropylene tubes. Patients were advised to rinse his or her mouth several times with water and then to relax for five minutes. Patients were asked to swallow to void the mouth of saliva and asked to lean their head forward over the polypropylene tube and funnel. Patients kept their mouth slightly open to allow saliva to drain into the tube.	ELISA; AST and ALT levels were analyzed on Roche p-800 modular system using the specific kits provided by the manufacturer.	on-proteomics			25345339
O00299	CLIC1 G6 NCC27	Chloride intracellular channel protein 1 (Chloride channel ABP) (Nuclear chloride ion channel 27) (NCC27) (Regulatory nuclear chloride ion channel protein) (hRNCC)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O00299	CLIC1 G6 NCC27	Chloride intracellular channel protein 1 (Chloride channel ABP) (Nuclear chloride ion channel 27) (NCC27) (Regulatory nuclear chloride ion channel protein) (hRNCC)	Homo sapiens (Human)						x					x	CP	68055113					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)					x						x	CP	68055113	1,05	22-62	MF		Unstimulated whole expectorated saliva (5 mL) was collected from each subject between 10 AM and 12 PM according to a modification of the method described by Navazesh [28]. The patients were asked to swallow saliva first, and then allow the saliva to drain passively for five minutes over the lower lip into a sterile tube. The collected saliva was immediately placed on ice prior to freezing at –80°C. The samples were defrosted and analyzed within 6 months of collection.	ELISA	on-proteomics			24236245
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)						x					x	CP	68055113	5,93	42-50	MF		GCF was taken from the mesiobuccal aspect of each site (tooth) for up to 28 teeth per patient. Prior to the collection, supragingival plaque was removed using a sterile instrument. The site was isolated using cotton rolls and dried using a short blast of air directly through the contact (not into the sulcus/pocket). A methylcellulose strip (Pro Flow, Inc., Amityville, NY) was inserted into the sulcus/pocket until light resistance was felt. The strip stayed in position for 30 seconds.	ELISA	on-proteomics			24303954
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)						x					x	CP	68055113	-5,00					Non-proteomics			14742657
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)						x					x	CP	68055113	-3,45					Non-proteomics		x	17355365
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)					x						x	CP	68055113	1,60					Non-proteomics			22126530
O00300	TNFRSF11B OCIF OPG	Tumor necrosis factor receptor superfamily member 11B (Osteoclastogenesis inhibitory factor) (Osteoprotegerin)	Homo sapiens (Human)					x						x	CP	68055113			M/F	All individuals possessed at least 20 teeth and had not received periodontal treatment or antibiotic therapy for medical or dental reasons for 3 mos prior to the investigation. Individuals were excluded if they possessed a history of metabolic bone diseases, autoimmune diseases, unstable diabetes, or post-menopausal osteoporosis. Pregnant or lactating women were excluded from participating in the study.	Unstimulated whole saliva was collected at each study visit via passive drooling into sterile plastic tubes from all participants (Mandel and Wotman, 1976). Samples were placed on ice, supplemented with a proteinase inhibitor combination of 1% aprotinin and 0.5% phenylmethylsulphonylfluoride, and aliquotted prior to storage at -80°C.	Protein biomarker levels were determined by colorimetric-based enzyme-linked immunosorbent assays (ELISAs), fluorescence-based protein microarrays, and radioimmunoassay (RIA), run according to manufacturer protocols. ELISAs (R&D Systems Inc., Minneapolis, MN, USA) were used for measurement of MMP-8 and -9, calprotectin, and osteoprotegerin (OPG). Detection of the cytokines interleukin (IL)-β, IL-2, IL-4, IL-5, IL-6, IL-10, and IL-13, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ was accomplished with a protein microarray (Whatman Inc., Florham Park, NJ, USA).	on-Proteomics			21406610
O00322	UPK1A TSPAN21	Uroplakin-1a (UP1a) (Tetraspanin-21) (Tspan-21) (Uroplakin Ia) (UPIa) (UPKa)	Homo sapiens (Human)							x					CP	68055113	,30(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
O00327	ARNTL BHLHE5 BMAL1 MOP3 PASD3	Aryl hydrocarbon receptor nuclear translocator-like protein 1 (Basic-helix-loop-helix-PAS protein MOP3) (Brain and muscle ARNT-like 1) (Class E basic helix-loop-helix protein 5) (bHLHe5) (Member of PAS protein 3) (PAS domain-containing protein 3) (bHLH-PAS protein JAP3)	Homo sapiens (Human)							x					CP	68055113	,29(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
O00360	0	Putative p150	Homo sapiens (Human)						x					x	CP	68055113	2,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
O00408	PDE2A	cGMP-dependent 3',5'-cyclic phosphodiesterase (EC 3.1.4.17) (Cyclic GMP-stimulated phosphodiesterase) (CGS-PDE) (cGSPDE)	Homo sapiens (Human)						x					x	CP	68055113	1,17	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O00443	PIK3C2A	Phosphatidylinositol 4-phosphate 3-kinase C2 domain-containing subunit alpha (PI3K-C2-alpha) (PtdIns-3-kinase C2 subunit alpha) (EC 2.7.1.154) (Phosphoinositide 3-kinase-C2-alpha)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O00448	0	Mucin MUC5B (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O00462	MANBA MANB1	Beta-mannosidase (EC 3.2.1.25) (Lysosomal beta A mannosidase) (Mannanase) (Mannase)	Homo sapiens (Human)					x						x	CP	68055113	-1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O00562	PITPNM1 DRES9 NIR2 PITPNM	Membrane-associated phosphatidylinositol transfer protein 1 (Drosophila retinal degeneration B homolog) (Phosphatidylinositol transfer protein, membrane-associated 1) (PITPnm 1) (Pyk2 N-terminal domain-interacting receptor 2) (NIR-2)	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O00584	RNASET2 RNASE6PL	Ribonuclease T2 (EC 3.1.27.-) (Ribonuclease 6)	Homo sapiens (Human)					x						x	CP	68055113	-1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O00584	RNASET2 RNASE6PL	Ribonuclease T2 (EC 3.1.27.-) (Ribonuclease 6)	Homo sapiens (Human)						x					x	CP	68055113	8,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O00748	CES2 ICE	Cocaine esterase (EC 3.1.1.84) (Carboxylesterase 2) (CE-2) (hCE-2) (EC 3.1.1.1) (Methylumbelliferyl-acetate deacetylase 2) (EC 3.1.1.56)	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O00763	ACACB ACC2 ACCB	Acetyl-CoA carboxylase 2 (EC 6.4.1.2) (ACC-beta) [Includes: Biotin carboxylase (EC 6.3.4.14)]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O14633	LCE2B LEP10 SPRL1B XP5	Late cornified envelope protein 2B (Late envelope protein 10) (Skin-specific protein Xp5) (Small proline-rich-like epidermal differentiation complex protein 1B)	Homo sapiens (Human)							x					CP	68055113	,27(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
O14684	PTGES MGST1L1 MPGES1 PGES PIG12	Prostaglandin E synthase (EC 5.3.99.3) (Microsomal glutathione S-transferase 1-like 1) (MGST1-L1) (Microsomal prostaglandin E synthase 1) (MPGES-1) (p53-induced gene 12 protein)	Homo sapiens (Human)							x					CP	68055113		35-69			The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451
O14788	TNFSF11 OPGL RANKL TRANCE	Tumor necrosis factor ligand superfamily member 11 (Osteoclast differentiation factor) (ODF) (Osteoprotegerin ligand) (OPGL) (Receptor activator of nuclear factor kappa-B ligand) (RANKL) (TNF-related activation-induced cytokine) (TRANCE) (CD antigen CD254) [Cleaved into: Tumor necrosis factor ligand superfamily member 11, membrane form; Tumor necrosis factor ligand superfamily member 11, soluble form]	Homo sapiens (Human)							x				x	CP	68055113	1,29	22-62	MF		Unstimulated whole expectorated saliva (5 mL) was collected from each subject between 10 AM and 12 PM according to a modification of the method described by Navazesh [26]. The patients were asked to swallow saliva first, and then allow the saliva to drain passively for five minutes over the lower lip into a sterile tube. The collected saliva was immediately placed on ice prior to freezing at –80°C. The samples were defrosted and analyzed within 6 months of collection.	ELISA	on-proteomics			24236245
O14788	TNFSF11 OPGL RANKL TRANCE	Tumor necrosis factor ligand superfamily member 11 (Osteoclast differentiation factor) (ODF) (Osteoprotegerin ligand) (OPGL) (Receptor activator of nuclear factor kappa-B ligand) (RANKL) (TNF-related activation-induced cytokine) (TRANCE) (CD antigen CD254) [Cleaved into: Tumor necrosis factor ligand superfamily member 11, membrane form; Tumor necrosis factor ligand superfamily member 11, soluble form]	Homo sapiens (Human)							x				x	CP	68055113	3,50					Non-proteomics			14742657
O14788	TNFSF11 OPGL RANKL TRANCE	Tumor necrosis factor ligand superfamily member 11 (Osteoclast differentiation factor) (ODF) (Osteoprotegerin ligand) (OPGL) (Receptor activator of nuclear factor kappa-B ligand) (RANKL) (TNF-related activation-induced cytokine) (TRANCE) (CD antigen CD254) [Cleaved into: Tumor necrosis factor ligand superfamily member 11, membrane form; Tumor necrosis factor ligand superfamily member 11, soluble form]	Homo sapiens (Human)							x				x	CP	68055113	-1,60					Non-proteomics			15732858
O14788	TNFSF11 OPGL RANKL TRANCE	Tumor necrosis factor ligand superfamily member 11 (Osteoclast differentiation factor) (ODF) (Osteoprotegerin ligand) (OPGL) (Receptor activator of nuclear factor kappa-B ligand) (RANKL) (TNF-related activation-induced cytokine) (TRANCE) (CD antigen CD254) [Cleaved into: Tumor necrosis factor ligand superfamily member 11, membrane form; Tumor necrosis factor ligand superfamily member 11, soluble form]	Homo sapiens (Human)							x				x	CP	68055113	54,41					Non-proteomics		x	17355365
O14788	TNFSF11 OPGL RANKL TRANCE	Tumor necrosis factor ligand superfamily member 11 (Osteoclast differentiation factor) (ODF) (Osteoprotegerin ligand) (OPGL) (Receptor activator of nuclear factor kappa-B ligand) (RANKL) (TNF-related activation-induced cytokine) (TRANCE) (CD antigen CD254) [Cleaved into: Tumor necrosis factor ligand superfamily member 11, membrane form; Tumor necrosis factor ligand superfamily member 11, soluble form]	Homo sapiens (Human)							x				x	CP	68055113	8,05					Non-proteomics			17321485
O14979	HNRNPDL HNRPDL JKTBP	Heterogeneous nuclear ribonucleoprotein D-like (hnRNP D-like) (hnRNP DL) (AU-rich element RNA binding factor) (JKT41-binding protein) (Protein laAUF1)	Homo sapiens (Human)							x				x	CP	68055113	1,95	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
O15117	FYB SLAP130	FYN-binding protein (Adhesion and degranulation promoting adaptor protein) (ADAP) (FYB-120/130) (p120/p130) (FYN-T-binding protein) (SLAP-130) (SLP-76-associated phosphoprotein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15143	ARPC1B ARC41	Actin-related protein 2/3 complex subunit 1B (Arp2/3 complex 41 kDa subunit) (p41-ARC)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15144	ARPC2 ARC34 PRO2446	Actin-related protein 2/3 complex subunit 2 (Arp2/3 complex 34 kDa subunit) (p34-ARC)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15145	ARPC3 ARC21	Actin-related protein 2/3 complex subunit 3 (Arp2/3 complex 21 kDa subunit) (p21-ARC)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15145	ARPC3 ARC21	Actin-related protein 2/3 complex subunit 3 (Arp2/3 complex 21 kDa subunit) (p21-ARC)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
O15162	PLSCR1	Phospholipid scramblase 1 (PL scramblase 1) (Ca(2+)-dependent phospholipid scramblase 1) (Erythrocyte phospholipid scramblase) (MmTRA1b)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15226	NKRF ITBA4 NRF	NF-kappa-B-repressing factor (NFkB-repressing factor) (Protein ITBA4) (Transcription factor NRF)	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O15226	NKRF ITBA4 NRF	NF-kappa-B-repressing factor (NFkB-repressing factor) (Protein ITBA4) (Transcription factor NRF)	Homo sapiens (Human)						x					x	CP	68055113	10,28	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O15294	OGT	UDP-N-acetylglucosamine--peptide N-acetylglucosaminyltransferase 110 kDa subunit (EC 2.4.1.255) (O-GlcNAc transferase subunit p110) (O-linked N-acetylglucosamine transferase 110 kDa subunit) (OGT)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O15516	CLOCK BHLHE8 KIAA0334	Circadian locomoter output cycles protein kaput (hCLOCK) (EC 2.3.1.48) (Class E basic helix-loop-helix protein 8) (bHLHe8)	Homo sapiens (Human)					x						x	CP	68055113	1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O43293	DAPK3 ZIPK	Death-associated protein kinase 3 (DAP kinase 3) (EC 2.7.11.1) (DAP-like kinase) (Dlk) (MYPT1 kinase) (Zipper-interacting protein kinase) (ZIP-kinase)	Homo sapiens (Human)					x						x	CP	68055113	1,35	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O43399	TPD52L2	Tumor protein D54 (hD54) (Tumor protein D52-like 2)	Homo sapiens (Human)						x					x	CP	68055113	2,36	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O43707	ACTN4	Alpha-actinin-4 (Non-muscle alpha-actinin 4)	Homo sapiens (Human)						x					x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O43707	ACTN4	Alpha-actinin-4 (Non-muscle alpha-actinin 4)	Homo sapiens (Human)						x					x	CP	68055113	2,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O43707	ACTN4	Alpha-actinin-4 (Non-muscle alpha-actinin 4)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O43707	ACTN4	Alpha-actinin-4 (Non-muscle alpha-actinin 4)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
O43790	KRT86 KRTHB6	Keratin, type II cuticular Hb6 (Hair keratin K2.11) (Keratin-86) (K86) (Type II hair keratin Hb6) (Type-II keratin Kb26)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O43852	CALU	Calumenin (Crocalbin) (IEF SSP 9302)	Homo sapiens (Human)					x						x	CP	68055113	-1,34	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O60218	AKR1B10 AKR1B11	Aldo-keto reductase family 1 member B10 (EC 1.1.1.-) (ARL-1) (Aldose reductase-like) (Aldose reductase-related protein) (ARP) (hARP) (Small intestine reductase) (SI reductase)	Homo sapiens (Human)						x					x	CP	68055113	-4,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O60218	AKR1B10 AKR1B11	Aldo-keto reductase family 1 member B10 (EC 1.1.1.-) (ARL-1) (Aldose reductase-like) (Aldose reductase-related protein) (ARP) (hARP) (Small intestine reductase) (SI reductase)	Homo sapiens (Human)						x					x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O60218	AKR1B10 AKR1B11	Aldo-keto reductase family 1 member B10 (EC 1.1.1.-) (ARL-1) (Aldose reductase-like) (Aldose reductase-related protein) (ARP) (hARP) (Small intestine reductase) (SI reductase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
O60234	GMFG	Glia maturation factor gamma (GMF-gamma)	Homo sapiens (Human)						x					x	CP	68055113	1,88	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O60235	TMPRSS11D HAT	Transmembrane protease serine 11D (EC 3.4.21.-) (Airway trypsin-like protease) [Cleaved into: Transmembrane protease serine 11D non-catalytic chain; Transmembrane protease serine 11D catalytic chain]	Homo sapiens (Human)					x						x	CP	68055113	1,32	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O60292	SIPA1L3 KIAA0545 SPAL3	Signal-induced proliferation-associated 1-like protein 3 (SIPA1-like protein 3) (SPA-1-like protein 3)	Homo sapiens (Human)						x					x	CP	68055113	-1,82	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O60437	PPL KIAA0568	Periplakin (190 kDa paraneoplastic pemphigus antigen) (195 kDa cornified envelope precursor protein)	Homo sapiens (Human)						x					x	CP	68055113	4,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O60447	EVI5 NB4S	Ecotropic viral integration site 5 protein homolog (EVI-5) (Neuroblastoma stage 4S gene protein)	Homo sapiens (Human)						x					x	CP	68055113	1,38	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O60814	HIST1H2BK H2BFT HIRIP1	Histone H2B type 1-K (H2B K) (HIRA-interacting protein 1)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
O75037	KIF21B KIAA0449	Kinesin-like protein KIF21B	Homo sapiens (Human)					x						x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75131	CPNE3 CPN3 KIAA0636	Copine-3 (Copine III)	Homo sapiens (Human)						x					x	CP	68055113	4,41	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75152	ZC3H11A KIAA0663 ZC3HDC11A	Zinc finger CCCH domain-containing protein 11A	Homo sapiens (Human)						x					x	CP	68055113	2,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75223	GGCT C7orf24 CRF21	Gamma-glutamylcyclotransferase (EC 2.3.2.4) (Cytochrome c-releasing factor 21)	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75367	H2AFY MACROH2A1	Core histone macro-H2A.1 (Histone macroH2A1) (mH2A1) (Histone H2A.y) (H2A/y) (Medulloblastoma antigen MU-MB-50.205)	Homo sapiens (Human)						x					x	CP	68055113	3,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75368	SH3BGRL	SH3 domain-binding glutamic acid-rich-like protein	Homo sapiens (Human)					x						x	CP	68055113	1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
O75368	SH3BGRL	SH3 domain-binding glutamic acid-rich-like protein	Homo sapiens (Human)						x					x	CP	68055113	1,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75369	FLNB FLN1L FLN3 TABP TAP	Filamin-B (FLN-B) (ABP-278) (ABP-280 homolog) (Actin-binding-like protein) (Beta-filamin) (Filamin homolog 1) (Fh1) (Filamin-3) (Thyroid autoantigen) (Truncated actin-binding protein) (Truncated ABP)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O75382	TRIM3 BERP RNF22 RNF97	Tripartite motif-containing protein 3 (Brain-expressed RING finger protein) (RING finger protein 22) (RING finger protein 97)	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O75533	SF3B1 SAP155	Splicing factor 3B subunit 1 (Pre-mRNA-splicing factor SF3b 155 kDa subunit) (SF3b155) (Spliceosome-associated protein 155) (SAP 155)	Homo sapiens (Human)						x					x	CP	68055113	1,60	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75594	PGLYRP1 PGLYRP PGRP TNFSF3L SBBI68	Peptidoglycan recognition protein 1 (Peptidoglycan recognition protein short) (PGRP-S)	Homo sapiens (Human)					x						x	CP	68055113	1,88	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75636	FCN3 FCNH HAKA1	Ficolin-3 (Collagen/fibrinogen domain-containing lectin 3 p35) (Collagen/fibrinogen domain-containing protein 3) (Hakata antigen)	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75691	UTP20 DRIM	Small subunit processome component 20 homolog (Down-regulated in metastasis protein) (Novel nucleolar protein 73) (NNP73) (Protein Key-1A6)	Homo sapiens (Human)						x					x	CP	68055113	1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O75695	RP2	Protein XRP2	Homo sapiens (Human)						x					x	CP	68055113	3,51	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O75717	WDHD1 AND1	WD repeat and HMG-box DNA-binding protein 1 (Acidic nucleoplasmic DNA-binding protein 1) (And-1)	Homo sapiens (Human)						x					x	CP	68055113	-1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75829	LECT1 CHMI	Leukocyte cell-derived chemotaxin 1 [Cleaved into: Chondrosurfactant protein (CH-SP); Chondromodulin-1 (Chondromodulin-I) (ChM-I)]	Homo sapiens (Human)						x					x	CP	68055113	-1,59	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O75874	IDH1 PICD	Isocitrate dehydrogenase [NADP] cytoplasmic (IDH) (EC 1.1.1.42) (Cytosolic NADP-isocitrate dehydrogenase) (IDP) (NADP(+)-specific ICDH) (Oxalosuccinate decarboxylase)	Homo sapiens (Human)						x					x	CP	68055113	4,07	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O76075	DFFB CAD DFF2 DFF40	DNA fragmentation factor subunit beta (EC 3.-.-.-) (Caspase-activated deoxyribonuclease) (CAD) (Caspase-activated DNase) (Caspase-activated nuclease) (CPAN) (DNA fragmentation factor 40 kDa subunit) (DFF-40)	Homo sapiens (Human)						x					x	CP	68055113	3,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
O94782	USP1	Ubiquitin carboxyl-terminal hydrolase 1 (EC 3.4.19.12) (Deubiquitinating enzyme 1) (hUBP) (Ubiquitin thioesterase 1) (Ubiquitin-specific-processing protease 1)	Homo sapiens (Human)						x					x	CP	68055113	1,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O94822	LTN1 C21orf10 C21orf98 KIAA0714 RNF160	E3 ubiquitin-protein ligase listerin (EC 6.3.2.-) (RING finger protein 160) (Zinc finger protein 294)	Homo sapiens (Human)						x					x	CP	68055113	1,23	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q03001	DST BP230 BP240 BPAG1 DMH DT KIAA0728	Dystonin (230 kDa bullous pemphigoid antigen) (230/240 kDa bullous pemphigoid antigen) (Bullous pemphigoid antigen 1) (BPA) (Bullous pemphigoid antigen) (Dystonia musculorum protein) (Hemidesmosomal plaque protein)	Homo sapiens (Human)						x					x	CP	68055113	1,13	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O94868	FCHSD2 KIAA0769 SH3MD3	F-BAR and double SH3 domains protein 2 (Carom) (SH3 multiple domains protein 3)	Homo sapiens (Human)					x						x	CP	68055113	-1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O95221	OR5F1	Olfactory receptor 5F1 (Olfactory receptor 11-10) (OR11-10) (Olfactory receptor OR11-167)	Homo sapiens (Human)						x					x	CP	68055113	49,45	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O95274	LYPD3 C4.4A UNQ491/PRO1007	Ly6/PLAUR domain-containing protein 3 (GPI-anchored metastasis-associated protein C4.4A homolog) (Matrigel-induced gene C4 protein) (MIG-C4)	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O95359	TACC2	Transforming acidic coiled-coil-containing protein 2 (Anti-Zuai-1) (AZU-1)	Homo sapiens (Human)						x					x	CP	68055113	-1,04	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O95447	LCA5L C21orf13	Lebercilin-like protein (Leber congenital amaurosis 5-like protein)	Homo sapiens (Human)						x					x	CP	68055113	20,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O95498	VNN2	Vascular non-inflammatory molecule 2 (Vanin-2) (EC 3.5.1.92) (Glycosylphosphatidyl inositol-anchored protein GPI-80) (Protein FOAP-4)	Homo sapiens (Human)						x					x	CP	68055113	3,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
O95622	ADCY5	Adenylate cyclase type 5 (EC 4.6.1.1) (ATP pyrophosphate-lyase 5) (Adenylate cyclase type V) (Adenylyl cyclase 5)	Homo sapiens (Human)					x						x	CP	68055113	1,19	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O95625	ZBTB11	Zinc finger and BTB domain-containing protein 11	Homo sapiens (Human)					x						x	CP	68055113	1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
O95678	KRT75 K6HF KB18	Keratin, type II cytoskeletal 75 (Cytokeratin-75) (CK-75) (Keratin-6 hair follicle) (hK6hf) (Keratin-75) (K75) (Type II keratin-K6hf) (Type-II keratin Kb18)	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
O95714	HERC2	E3 ubiquitin-protein ligase HERC2 (EC 6.3.2.-) (HECT domain and RCC1-like domain-containing protein 2)	Homo sapiens (Human)						x					x	CP	68055113	1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
O95932	TGM6 TGM3L	Protein-glutamine gamma-glutamyltransferase 6 (EC 2.3.2.13) (Transglutaminase Y) (TGY) (TGase Y) (Transglutaminase-3-like) (TGase-3-like) (Transglutaminase-6) (TG6) (TGase-6)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
O95996	APC2 APCL	Adenomatous polyposis coli protein 2 (Adenomatous polyposis coli protein-like) (APC-like)	Homo sapiens (Human)						x					x	CP	68055113	-1,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P00338	LDHA PIG19	L-lactate dehydrogenase A chain (LDH-A) (EC 1.1.1.27) (Cell proliferation-inducing gene 19 protein) (LDH muscle subunit) (LDH-M) (Renal carcinoma antigen NY-REN-59)	Homo sapiens (Human)					x						x	CP	68055113	1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00338	LDHA PIG19	L-lactate dehydrogenase A chain (LDH-A) (EC 1.1.1.27) (Cell proliferation-inducing gene 19 protein) (LDH muscle subunit) (LDH-M) (Renal carcinoma antigen NY-REN-59)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00338	LDHA PIG19	L-lactate dehydrogenase A chain (LDH-A) (EC 1.1.1.27) (Cell proliferation-inducing gene 19 protein) (LDH muscle subunit) (LDH-M) (Renal carcinoma antigen NY-REN-59)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00441	SOD1	Superoxide dismutase [Cu-Zn] (EC 1.15.1.1) (Superoxide dismutase 1) (hSod1)	Homo sapiens (Human)						x					x	CP	68055113	1,71	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P00441	SOD1	Superoxide dismutase [Cu-Zn] (EC 1.15.1.1) (Superoxide dismutase 1) (hSod1)	Homo sapiens (Human)					x						x	CP	68055113	2,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at -80ºC until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P00441	SOD1	Superoxide dismutase [Cu-Zn] (EC 1.15.1.1) (Superoxide dismutase 1) (hSod1)	Homo sapiens (Human)						x					x	CP	68055113	+	36	M/F	All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. Since smoking is a risk factor for periodontal disease, the current study did not include smokers.	GCF was collected from the labial side of maxillary incisors without crown and restoration.	Western blot	on-Proteomics		x	22623421
P00450	CP	Ceruloplasmin (EC 1.16.3.1) (Ferroxidase)	Homo sapiens (Human)						x					x	CP	68055113	1,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P00450	CP	Ceruloplasmin (EC 1.16.3.1) (Ferroxidase)	Homo sapiens (Human)					x						x	CP	68055113	1,52	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00450	CP	Ceruloplasmin (EC 1.16.3.1) (Ferroxidase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00450	CP	Ceruloplasmin (EC 1.16.3.1) (Ferroxidase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00491	PNP NP	Purine nucleoside phosphorylase (PNP) (EC 2.4.2.1) (Inosine phosphorylase) (Inosine-guanosine phosphorylase)	Homo sapiens (Human)					x						x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00491	PNP NP	Purine nucleoside phosphorylase (PNP) (EC 2.4.2.1) (Inosine phosphorylase) (Inosine-guanosine phosphorylase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00491	PNP NP	Purine nucleoside phosphorylase (PNP) (EC 2.4.2.1) (Inosine phosphorylase) (Inosine-guanosine phosphorylase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00558	PGK1 PGKA MIG10 OK/SW-cl.110	Phosphoglycerate kinase 1 (EC 2.7.2.3) (Cell migration-inducing gene 10 protein) (Primer recognition protein 2) (PRP 2)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00558	PGK1 PGKA MIG10 OK/SW-cl.110	Phosphoglycerate kinase 1 (EC 2.7.2.3) (Cell migration-inducing gene 10 protein) (Primer recognition protein 2) (PRP 2)	Homo sapiens (Human)						x					x	CP	68055113	3,38	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P00558	PGK1 PGKA MIG10 OK/SW-cl.110	Phosphoglycerate kinase 1 (EC 2.7.2.3) (Cell migration-inducing gene 10 protein) (Primer recognition protein 2) (PRP 2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00558	PGK1 PGKA MIG10 OK/SW-cl.110	Phosphoglycerate kinase 1 (EC 2.7.2.3) (Cell migration-inducing gene 10 protein) (Primer recognition protein 2) (PRP 2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00734	F2	Prothrombin (EC 3.4.21.5) (Coagulation factor II) [Cleaved into: Activation peptide fragment 1; Activation peptide fragment 2; Thrombin light chain; Thrombin heavy chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P00734	F2	Prothrombin (EC 3.4.21.5) (Coagulation factor II) [Cleaved into: Activation peptide fragment 1; Activation peptide fragment 2; Thrombin light chain; Thrombin heavy chain]	Homo sapiens (Human)						x					x	CP	68055113		30-72	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P00736	C1R	Complement C1r subcomponent (EC 3.4.21.41) (Complement component 1 subcomponent r) [Cleaved into: Complement C1r subcomponent heavy chain; Complement C1r subcomponent light chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00738	HP	Haptoglobin (Zonulin) [Cleaved into: Haptoglobin alpha chain; Haptoglobin beta chain]	Homo sapiens (Human)						x					x	CP	68055113	1,45	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P00738	HP	Haptoglobin (Zonulin) [Cleaved into: Haptoglobin alpha chain; Haptoglobin beta chain]	Homo sapiens (Human)					x						x	CP	68055113	1,30	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00738	HP	Haptoglobin (Zonulin) [Cleaved into: Haptoglobin alpha chain; Haptoglobin beta chain]	Homo sapiens (Human)					x						x	CP	68055113	3,64	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Collected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P00738	HP	Haptoglobin (Zonulin) [Cleaved into: Haptoglobin alpha chain; Haptoglobin beta chain]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00738	HP	Haptoglobin (Zonulin) [Cleaved into: Haptoglobin alpha chain; Haptoglobin beta chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00739	HPR	Haptoglobin-related protein	Homo sapiens (Human)						x					x	CP	68055113	-1,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P00739	HPR	Haptoglobin-related protein	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00747	PLG	Plasminogen (EC 3.4.21.7) [Cleaved into: Plasmin heavy chain A; Activation peptide; Angiostatin; Plasmin heavy chain A, short form; Plasmin light chain B]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00751	CFB BF BFD	Complement factor B (EC 3.4.21.47) (C3/C5 convertase) (Glycine-rich beta glycoprotein) (GBG) (PBF2) (Properdin factor B) [Cleaved into: Complement factor B Ba fragment; Complement factor B Bb fragment]	Homo sapiens (Human)					x						x	CP	68055113	1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00751	CFB BF BFD	Complement factor B (EC 3.4.21.47) (C3/C5 convertase) (Glycine-rich beta glycoprotein) (GBG) (PBF2) (Properdin factor B) [Cleaved into: Complement factor B Ba fragment; Complement factor B Bb fragment]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00915	CA1	Carbonic anhydrase 1 (EC 4.2.1.1) (Carbonate dehydratase I) (Carbonic anhydrase B) (CAB) (Carbonic anhydrase I) (CA-I)	Homo sapiens (Human)						x						CP	68055113	+					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P00915	CA1	Carbonic anhydrase 1 (EC 4.2.1.1) (Carbonate dehydratase I) (Carbonic anhydrase B) (CAB) (Carbonic anhydrase I) (CA-I)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P00915	CA1	Carbonic anhydrase 1 (EC 4.2.1.1) (Carbonate dehydratase I) (Carbonic anhydrase B) (CAB) (Carbonic anhydrase I) (CA-I)	Homo sapiens (Human)						x					x	CP	68055113		30-62	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P00918	CA2	Carbonic anhydrase 2 (EC 4.2.1.1) (Carbonate dehydratase II) (Carbonic anhydrase C) (CAC) (Carbonic anhydrase II) (CA-II)	Homo sapiens (Human)						x					x	CP	68055113	6,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01008	SERPINC1 AT3 PRO0309	Antithrombin-III (ATIII) (Serpin C1)	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01008	SERPINC1 AT3 PRO0309	Antithrombin-III (ATIII) (Serpin C1)	Homo sapiens (Human)						x					x	CP	68055113	2,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01008	SERPINC1 AT3 PRO0309	Antithrombin-III (ATIII) (Serpin C1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)						x					x	CP	68055113	2,16						Proteomics			24098404
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)						x					x	CP	68055113	1,88	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)					x						x	CP	68055113	1,50	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)						x					x	CP	68055113	1,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01009	SERPINA1 AAT PI PRO0684 PRO2209	Alpha-1-antitrypsin (Alpha-1 protease inhibitor) (Alpha-1-antiproteinase) (Serpin A1) [Cleaved into: Short peptide from AAT (SPAAT)]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01011	SERPINA3 AACT GIG24 GIG25	Alpha-1-antichymotrypsin (ACT) (Cell growth-inhibiting gene 24/25 protein) (Serpin A3) [Cleaved into: Alpha-1-antichymotrypsin His-Pro-less]	Homo sapiens (Human)					x						x	CP	68055113	1,43	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01019	AGT SERPINA8	Angiotensinogen (Serpin A8) [Cleaved into: Angiotensin-1 (Angiotensin 1-10) (Angiotensin I) (Ang I); Angiotensin-2 (Angiotensin 1-8) (Angiotensin II) (Ang II); Angiotensin-3 (Angiotensin 2-8) (Angiotensin III) (Ang III) (Des-Asp[1-angiotensin II); Angiotensin-4 (Angiotensin 3-8) (Angiotensin IV) (Ang IV); Angiotensin 1-9; Angiotensin 1-7; Angiotensin 1-5; Angiotensin 1-4]	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics		x	22092770
P01019	AGT SERPINA8	Angiotensinogen (Serpin A8) [Cleaved into: Angiotensin-1 (Angiotensin 1-10) (Angiotensin I) (Ang I); Angiotensin-2 (Angiotensin 1-8) (Angiotensin II) (Ang II); Angiotensin-3 (Angiotensin 2-8) (Angiotensin III) (Ang III) (Des-Asp[1-angiotensin II); Angiotensin-4 (Angiotensin 3-8) (Angiotensin IV) (Ang IV); Angiotensin 1-9; Angiotensin 1-7; Angiotensin 1-5; Angiotensin 1-4]	Homo sapiens (Human)						x					x	CP	68055113		30-61	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)						x					x	CP	68055113	1,51	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)					x						x	CP	68055113	1,84	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)						x					x	CP	68055113	2,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01023	A2M CPAMD5 FWP007	Alpha-2-macroglobulin (Alpha-2-M) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 5)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01024	C3 CPAMD1	Complement C3 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 1) [Cleaved into: Complement C3 beta chain; C3-beta-c (C3bc); Complement C3 alpha chain; C3a anaphylatoxin; Acylation stimulating protein (ASP) (C3adesArg); Complement C3b alpha' chain; Complement C3c alpha' chain fragment 1; Complement C3dg fragment; Complement C3g fragment; Complement C3d fragment; Complement C3f fragment; Complement C3c alpha' chain fragment 2]	Homo sapiens (Human)						x					x	CP	68055113	1,44	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01024	C3 CPAMD1	Complement C3 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 1) [Cleaved into: Complement C3 beta chain; C3-beta-c (C3bc); Complement C3 alpha chain; C3a anaphylatoxin; Acylation stimulating protein (ASP) (C3adesArg); Complement C3b alpha' chain; Complement C3c alpha' chain fragment 1; Complement C3dg fragment; Complement C3g fragment; Complement C3d fragment; Complement C3f fragment; Complement C3c alpha' chain fragment 2]	Homo sapiens (Human)						x					x	CP	68055113	1,52	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01024	C3 CPAMD1	Complement C3 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 1) [Cleaved into: Complement C3 beta chain; C3-beta-c (C3bc); Complement C3 alpha chain; C3a anaphylatoxin; Acylation stimulating protein (ASP) (C3adesArg); Complement C3b alpha' chain; Complement C3c alpha' chain fragment 1; Complement C3dg fragment; Complement C3g fragment; Complement C3d fragment; Complement C3f fragment; Complement C3c alpha' chain fragment 2]	Homo sapiens (Human)						x					x	CP	68055113	1,64	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01024	C3 CPAMD1	Complement C3 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 1) [Cleaved into: Complement C3 beta chain; C3-beta-c (C3bc); Complement C3 alpha chain; C3a anaphylatoxin; Acylation stimulating protein (ASP) (C3adesArg); Complement C3b alpha' chain; Complement C3c alpha' chain fragment 1; Complement C3dg fragment; Complement C3g fragment; Complement C3d fragment; Complement C3f fragment; Complement C3c alpha' chain fragment 2]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01024	C3 CPAMD1	Complement C3 (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 1) [Cleaved into: Complement C3 beta chain; C3-beta-c (C3bc); Complement C3 alpha chain; C3a anaphylatoxin; Acylation stimulating protein (ASP) (C3adesArg); Complement C3b alpha' chain; Complement C3c alpha' chain fragment 1; Complement C3dg fragment; Complement C3g fragment; Complement C3d fragment; Complement C3f fragment; Complement C3c alpha' chain fragment 2]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01033	TIMP1 CLGI TIMP	Metalloproteinase inhibitor 1 (Erythroid-potentiating activity) (EPA) (Fibroblast collagenase inhibitor) (Collagenase inhibitor) (Tissue inhibitor of metalloproteinases 1) (TIMP-1)	Homo sapiens (Human)					x						x	CP	68055113	-1,17	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01034	CST3	Cystatin-C (Cystatin-3) (Gamma-trace) (Neuroendocrine basic polypeptide) (Post-gamma-globulin)	Homo sapiens (Human)					x						x	CP	68055113	-1,30	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01036	CST4	Cystatin-S (Cystatin-4) (Cystatin-SA-III) (Salivary acidic protein 1)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01036	CST4	Cystatin-S (Cystatin-4) (Cystatin-SA-III) (Salivary acidic protein 1)	Homo sapiens (Human)					x						x	CP	68055113	-1,29	25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P01037	CST1	Cystatin-SN (Cystain-SA-I) (Cystatin-1) (Salivary cystatin-SA-1)	Homo sapiens (Human)						x					x	CP	68055113	-7,20			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01037	CST1	Cystatin-SN (Cystain-SA-I) (Cystatin-1) (Salivary cystatin-SA-1)	Homo sapiens (Human)					x						x	CP	68055113	-1,29	25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P01037	CST1	Cystatin-SN (Cystain-SA-I) (Cystatin-1) (Salivary cystatin-SA-1)	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01037	CST1	Cystatin-SN (Cystain-SA-I) (Cystatin-1) (Salivary cystatin-SA-1)	Homo sapiens (Human)						x					x	CP	68055113	1,57	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01040	CSTA STF1 STFA	Cystatin-A (Cystatin-AS) (Stefin-A) [Cleaved into: Cystatin-A, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	-2,20			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01040	CSTA STF1 STFA	Cystatin-A (Cystatin-AS) (Stefin-A) [Cleaved into: Cystatin-A, N-terminally processed]	Homo sapiens (Human)					x						x	CP	68055113	1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01042	KNG1 BDK KNG	Kininogen-1 (Alpha-2-thiol proteinase inhibitor) (Fitzgerald factor) (High molecular weight kininogen) (HMWK) (Williams-Fitzgerald-Flaujeac factor) [Cleaved into: Kininogen-1 heavy chain; T-kinin (Ile-Ser-Bradykinin); Bradykinin (Kallidin I); Lysyl-bradykinin (Kallidin II); Kininogen-1 light chain; Low molecular weight growth-promoting factor]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01258	CALCA CALC1	Calcitonin [Cleaved into: Calcitonin; Katacalcin (Calcitonin carboxyl-terminal peptide) (CCP) (PDN-21)]	Homo sapiens (Human)						x					x	CP	68055113	1,67	32-41	MF	non-smokers	Particpants were instructed to refrain from eating, drinking, chewing gum and brushing their teeth in the morning of the saliva sample collection. Unstimulated whole saliva samples were obtained by expectorating into polypropylene tubes	Saliva samples were studied using BRAHMS ProCT original kit in the device	on-proteomics		x	25703733
P01375	TNF TNFA TNFSF2	Tumor necrosis factor (Cachectin) (TNF-alpha) (Tumor necrosis factor ligand superfamily member 2) (TNF-a) [Cleaved into: Tumor necrosis factor, membrane form (N-terminal fragment) (NTF); Intracellular domain 1 (ICD1); Intracellular domain 2 (ICD2); C-domain 1; C-domain 2; Tumor necrosis factor, soluble form]	Homo sapiens (Human)						x					x	CP	68055113	1,05	30-40	MF	Participants had not a history of using alcohol, tobacco, anti-inflammatory drugs and antibiotics during previous 3 months or periodontal treatments in last 4 months. They had more than 20 teeth, no sustained systemic diseases and the women were not also pregnant or lactating	To avoid changes in antioxidants, all samples were collected in a given time (11-12 am) in the morning. The participants (cases and controls) washed her/him mouth with water before sampling; then discharge his/her unstimulated saliva in a sterile tube using 5 ml sterile tubes	TNF-α level were measured by enzyme-linked immunosorbent assay and were compared with the control group	on-proteomics			24554882
P01375	TNF TNFA TNFSF2	Tumor necrosis factor (Cachectin) (TNF-alpha) (Tumor necrosis factor ligand superfamily member 2) (TNF-a) [Cleaved into: Tumor necrosis factor, membrane form (N-terminal fragment) (NTF); Intracellular domain 1 (ICD1); Intracellular domain 2 (ICD2); C-domain 1; C-domain 2; Tumor necrosis factor, soluble form]	Homo sapiens (Human)						x					x	CP	68055113	2,13		MF	non-smoking	Three milliliter of unstimulated saliva was taken	salivary TNF-α determined by using ELISA technique (Quantikine Human total TNF-A immunoassay kit).	on-proteomics		x	25024542
P01375	TNF TNFA TNFSF2	Tumor necrosis factor (Cachectin) (TNF-alpha) (Tumor necrosis factor ligand superfamily member 2) (TNF-a) [Cleaved into: Tumor necrosis factor, membrane form (N-terminal fragment) (NTF); Intracellular domain 1 (ICD1); Intracellular domain 2 (ICD2); C-domain 1; C-domain 2; Tumor necrosis factor, soluble form]	Homo sapiens (Human)						x					x	CP	68055113	20,19		MF	Diabetic, non-smoking	Three milliliter of unstimulated saliva was taken	salivary TNF-α determined by using ELISA technique (Quantikine Human total TNF-A immunoassay kit).	on-proteomics		x	25024542
P01375	TNF TNFA TNFSF2	Tumor necrosis factor (Cachectin) (TNF-alpha) (Tumor necrosis factor ligand superfamily member 2) (TNF-a) [Cleaved into: Tumor necrosis factor, membrane form (N-terminal fragment) (NTF); Intracellular domain 1 (ICD1); Intracellular domain 2 (ICD2); C-domain 1; C-domain 2; Tumor necrosis factor, soluble form]	Homo sapiens (Human)						x					x	CP	68055113	6,03		MF	Smoking	Three milliliter of unstimulated saliva was taken	salivary TNF-α determined by using ELISA technique (Quantikine Human total TNF-A immunoassay kit).	on-proteomics		x	25024542

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01375	TNF TNFA TNFSF2	Tumor necrosis factor (Cachectin) (TNF-alpha) (Tumor necrosis factor ligand superfamily member 2) (TNF-a) [Cleaved into: Tumor necrosis factor, membrane form (N-terminal fragment) (NTF); Intracellular domain 1 (ICD1); Intracellular domain 2 (ICD2); C-domain 1; C-domain 2; Tumor necrosis factor, soluble form]	Homo sapiens (Human)					x							CP	68055113	.06 (VS DMT2)		MF	non-smoking	Three milliliter of unstimulated saliva was taken	salivary TNF-α determined by using ELISA technique (Quantikine Human total TNF-A immunoassay kit).	on-proteomics		x	25024542
P01579	IFNG	Interferon gamma (IFN-gamma) (Immune interferon)	Homo sapiens (Human)					x						x	CP	68055113	1,45		MF		Prior initial periodontal treatment period, about 5 mL of unstimulated whole saliva was collected from each subject into a 50 mL sterile plastic centrifuge tube (Greiner Bio-one1, Frickenhausen, Germany) before breakfast intake and any dental hygiene procedure. No antiseptic mouth rinse was used before collection. Immediately after collection, whole saliva was clarified by centrifugation for 5-min at 800 g (IEC1 Centra CL2 Centrifuge, Thermo Electron Corporation, Milford, MA, USA). The supernatants were collected and aliquoted into 500 mL volumes and frozen at 75 8C for until processed.	ELISA	on-proteomics			25285903
P01584	IL1B IL1F2	Interleukin-1 beta (IL-1 beta) (Catabolin)	Homo sapiens (Human)						x					x	CP	68055113	4,08	42-50	MF		GCF was taken from the mesiobuccal aspect of each site (tooth) for up to 28 teeth per patient. Prior to the collection, supragingival plaque was removed using a sterile instrument. The site was isolated using cotton rolls and dried using a short blast of air directly through the contact (not into the sulcus/pocket). A methylcellulose strip (Pro Flow, Inc., Amityville, NY) was inserted into the sulcus/pocket until light resistance was felt. The strip stayed in position for 30 seconds.	ELISA	on-proteomics			24303954
P01584	IL1B IL1F2	Interleukin-1 beta (IL-1 beta) (Catabolin)	Homo sapiens (Human)					x						x	CP	68055113	3,3	62-75	MF		At the beginning of the oral examination, the subjects chewed a piece of paraffin for 5 min and at least 2 ml of stimulated whole saliva was collected. The samples were stored at −70 °C until analyses.	IL-1β was measured by flow cytometry -based Luminex technology, with commercially available kits (Milliplex Map Kit MPXHCYTO-60k, Millipore, Billerica, MA, USA) with a detection limit of 0.4 pg/ml.	on-proteomics			24460823
P01584	IL1B IL1F2	Interleukin-1 beta (IL-1 beta) (Catabolin)	Homo sapiens (Human)						x					x	CP	68055113							Non-Proteomics			12892443
P01584	IL1B IL1F2	Interleukin-1 beta (IL-1 beta) (Catabolin)	Homo sapiens (Human)					x						x	CP	68055113			M/F	All individuals possessed at least 20 teeth and had not received periodontal treatment or antibiotic therapy for medical or dental reasons for 3 mos prior to the investigation. Individuals were excluded if they possessed a history of metabolic bone diseases, autoimmune diseases, unstable diabetes, or post-menopausal osteoporosis. Pregnant or lactating women were excluded from participating in the study.	Unstimulated whole saliva was collected at each study visit via passive drooling into sterile plastic tubes from all participants (Mandel and Wotman, 1976). Samples were placed on ice, supplemented with a proteinase inhibitor combination of 1% aprotinin and 0.5% phenylmethylsulphonylfluoride, and aliquotted prior to storage at -80°C.	Protein biomarker levels were determined by colorimetric-based enzyme-linked immunosorbent assays (ELISAs), fluorescence-based protein microarrays, and radioimmunoassay (RIA), run according to manufacturer protocols. ELISAs (R&D Systems Inc., Minneapolis, MN, USA) were used for measurement of MMP-8 and -9, calprotectin, and osteoprotegerin (OPG). Detection of the cytokines interleukin (IL)-β, IL-2, IL-4, IL-5, IL-6, IL-10, and IL-13, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ was accomplished with a protein microarray (Whatman Inc., Florham Park, NJ, USA).	on-Proteomics			21406610
P01584	IL1B IL1F2	Interleukin-1 beta (IL-1 beta) (Catabolin)	Homo sapiens (Human)							x					CP	68055113		35-68			The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451
P01591	JCHAIN IGCJ IGJ	Immunoglobulin J chain (Joining chain of multimeric IgA and IgM)	Homo sapiens (Human)					x						x	CP	68055113	-1,24	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01591	JCHAIN IGCJ IGJ	Immunoglobulin J chain (Joining chain of multimeric IgA and IgM)	Homo sapiens (Human)						x					x	CP	68055113	3,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01591	JCHAIN IGCJ IGJ	Immunoglobulin J chain (Joining chain of multimeric IgA and IgM)	Homo sapiens (Human)					x							CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P01591	JCHAIN IGCJ IGJ	Immunoglobulin J chain (Joining chain of multimeric IgA and IgM)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01593	0	Ig kappa chain V-I region AG	Homo sapiens (Human)						x						CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01593	0	Ig kappa chain V-I region AG	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01593	0	Ig kappa chain V-I region AG	Homo sapiens (Human)						x					x	CP	68055113		30-66	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P01602	IGKV1-5	Ig heavy chain V-I region 5 (Ig kappa chain V-I region HK102) (Immunoglobulin kappa variable 1-5) (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01609	0	Ig kappa chain V-I region Scw	Homo sapiens (Human)						x					x	CP	68055113	-1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01613	0	Ig kappa chain V-I region Ni	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01617	0	Ig kappa chain V-II region TEW	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01620	0	Ig kappa chain V-III region SIE	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01622	0	Ig kappa chain V-III region Ti	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01622	0	Ig kappa chain V-III region Ti	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01623	0	Ig kappa chain V-III region WOL	Homo sapiens (Human)						x					x	CP	68055113	3,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01625	0	Ig kappa chain V-IV region Len	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01625	0	Ig kappa chain V-IV region Len	Homo sapiens (Human)						x					x	CP	68055113	2,05	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01700	0	Ig lambda chain V-I region HA	Homo sapiens (Human)					x						x	CP	68055113	-1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01703	0	Ig lambda chain V-I region NEWM	Homo sapiens (Human)						x					x	CP	68055113	3,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01714	0	Ig lambda chain V-III region SH	Homo sapiens (Human)					x						x	CP	68055113	-1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01717	0	Ig lambda chain V-IV region Hil	Homo sapiens (Human)					x						x	CP	68055113	1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01743	0	Ig heavy chain V-I region HG3	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01763	0	Ig heavy chain V-III region WEA	Homo sapiens (Human)					x						x	CP	68055113	-1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01764	IGHV3-23	Ig heavy chain V-III region 23 (Ig heavy chain V-III region VH26) (Immunoglobulin heavy variable 3-23)	Homo sapiens (Human)					x						x	CP	68055113	1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01764	IGHV3-23	Ig heavy chain V-III region 23 (Ig heavy chain V-III region VH26) (Immunoglobulin heavy variable 3-23)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P01764	IGHV3-23	Ig heavy chain V-III region 23 (Ig heavy chain V-III region VH26) (Immunoglobulin heavy variable 3-23)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01766	0	Ig heavy chain V-III region BRO	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01767	0	Ig heavy chain V-III region BUT	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01780	0	Ig heavy chain V-III region JON	Homo sapiens (Human)					x						x	CP	68055113	-1,11	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01781	0	Ig heavy chain V-III region GAL	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01824	0	Ig heavy chain V-II region WAH	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01825	0	Ig heavy chain V-II region NEWM	Homo sapiens (Human)					x						x	CP	68055113	-1,17	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)						x					x	CP	68055113	1,4	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)						x					x	CP	68055113	-50,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)					x						x	CP	68055113	-1,25	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)						x					x	CP	68055113	3,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)						x						CP	68055113	+					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01833	PIGR	Polymeric immunoglobulin receptor (PIgR) (Poly-Ig receptor) (Hepatocellular carcinoma-associated protein TB6) [Cleaved into: Secretory component]	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC, Q-TOF	Proteomics			20215060
P01834	IGKC	Ig kappa chain C region	Homo sapiens (Human)					x						x	CP	68055113	-1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01834	IGKC	Ig kappa chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01834	IGKC	Ig kappa chain C region	Homo sapiens (Human)						x					x	CP	68055113	1,34	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P0CG04	IGLC1	Ig lambda-1 chain C regions	Homo sapiens (Human)						x					x	CP	68055113	1,57	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01857	IGHG1	Ig gamma-1 chain C region	Homo sapiens (Human)					x						x	CP	68055113	1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01857	IGHG1	Ig gamma-1 chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01857	IGHG1	Ig gamma-1 chain C region	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P01857	IGHG1	Ig gamma-1 chain C region	Homo sapiens (Human)						x					x	CP	68055113	1,85	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01859	IGHG2	Ig gamma-2 chain C region	Homo sapiens (Human)					x						x	CP	68055113	1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01859	IGHG2	Ig gamma-2 chain C region	Homo sapiens (Human)						x					x	CP	68055113	2,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P01859	IGHG2	Ig gamma-2 chain C region	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01859	IGHG2	Ig gamma-2 chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01859	IGHG2	Ig gamma-2 chain C region	Homo sapiens (Human)						x					x	CP	68055113	1,63	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01860	IGHG3	Ig gamma-3 chain C region (HDC) (Heavy chain disease protein)	Homo sapiens (Human)					x						x	CP	68055113	1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01860	IGHG3	Ig gamma-3 chain C region (HDC) (Heavy chain disease protein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01861	IGHG4	Ig gamma-4 chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01861	IGHG4	Ig gamma-4 chain C region	Homo sapiens (Human)						x					x	CP	68055113	2,34	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P01871	IGHM	Ig mu chain C region	Homo sapiens (Human)					x						x	CP	68055113	-1,30	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01871	IGHM	Ig mu chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01876	IGHA1	Ig alpha-1 chain C region	Homo sapiens (Human)					x						x	CP	68055113	-1,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01876	IGHA1	Ig alpha-1 chain C region	Homo sapiens (Human)						x					x	CP	68055113	4,54	25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P01876	IGHA1	Ig alpha-1 chain C region	Homo sapiens (Human)					x						x	CP	68055113					unstimulated	2-DE + MALDI-TOF-TOF MS	Proteomics		x	22165124
P01876	IGHA1	Ig alpha-1 chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01877	IGHA2	Ig alpha-2 chain C region	Homo sapiens (Human)					x						x	CP	68055113	-1,19	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P01877	IGHA2	Ig alpha-2 chain C region	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P01877	IGHA2	Ig alpha-2 chain C region	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02042	HBD	Hemoglobin subunit delta (Delta-globin) (Hemoglobin delta chain)	Homo sapiens (Human)						x						CP	68055113	26,00						Proteomics			24098404
P02042	HBD	Hemoglobin subunit delta (Delta-globin) (Hemoglobin delta chain)	Homo sapiens (Human)						x					x	CP	68055113	6,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02042	HBD	Hemoglobin subunit delta (Delta-globin) (Hemoglobin delta chain)	Homo sapiens (Human)						x					x	CP	68055113	60,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02042	HBD	Hemoglobin subunit delta (Delta-globin) (Hemoglobin delta chain)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02042	HBD	Hemoglobin subunit delta (Delta-globin) (Hemoglobin delta chain)	Homo sapiens (Human)						x					x	CP	68055113		30-64	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics		x	23190455
P02462	COL4A1	Collagen alpha-1(IV) chain [Cleaved into: Arresten]	Homo sapiens (Human)						x					x	CP	68055113	-1,15	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02462	COL4A1	Collagen alpha-1(IV) chain [Cleaved into: Arresten]	Homo sapiens (Human)						x					x	CP	68055113	-1,11	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)						x					x	CP	68055113	1,04	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)					x						x	CP	68055113	2,55	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)						x					x	CP	68055113	4,44	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02533	KRT14	Keratin, type I cytoskeletal 14 (Cytokeratin-14) (CK-14) (Keratin-14) (K14)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02538	KRT6A K6A KRT6D	Keratin, type II cytoskeletal 6A (Cytokeratin-6A) (CK-6A) (Cytokeratin-6D) (CK-6D) (Keratin-6A) (K6A) (Type-II keratin Kb6) (allergen Hom s 5)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P02538	KRT6A K6A KRT6D	Keratin, type II cytoskeletal 6A (Cytokeratin-6A) (CK-6A) (Cytokeratin-6D) (CK-6D) (Keratin-6A) (K6A) (Type-II keratin Kb6) (allergen Hom s 5)	Homo sapiens (Human)						x					x	CP	68055113	1,11	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02538	KRT6A K6A KRT6D	Keratin, type II cytoskeletal 6A (Cytokeratin-6A) (CK-6A) (Cytokeratin-6D) (CK-6D) (Keratin-6A) (K6A) (Type-II keratin Kb6) (allergen Hom s 5)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113	1,36						Proteomics			24098404
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113	1,25	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113	1,76	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113		36	M/F	All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. Since smoking is a risk factor for periodontal disease, the current study did not include smokers.	GCF was collected from the labial side of maxillary incisors without crown and restoration.	Western blot	on-Proteomics		x	22623421
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02647	APOA1	Apolipoprotein A-I (Apo-AI) (ApoA-I) (Apolipoprotein A1) [Cleaved into: Proapolipoprotein A-I (ProapoA-I); Truncated apolipoprotein A-I (Apolipoprotein A-I(1-242))]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02649	APOE	Apolipoprotein E (Apo-E)	Homo sapiens (Human)						x						CP	68055113	8,00						Proteomics			24098404
P02652	APOA2	Apolipoprotein A-II (Apo-AII) (ApoA-II) (Apolipoprotein A2) [Cleaved into: Proapolipoprotein A-II (ProapoA-II); Truncated apolipoprotein A-II (Apolipoprotein A-II(1-76))]	Homo sapiens (Human)						x					x	CP	68055113	3,05						Proteomics			24098404
P02652	APOA2	Apolipoprotein A-II (Apo-AII) (ApoA-II) (Apolipoprotein A2) [Cleaved into: Proapolipoprotein A-II (ProapoA-II); Truncated apolipoprotein A-II (Apolipoprotein A-II(1-76))]	Homo sapiens (Human)						x					x	CP	68055113	1,53	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02652	APOA2	Apolipoprotein A-II (Apo-AII) (ApoA-II) (Apolipoprotein A2) [Cleaved into: Proapolipoprotein A-II (ProapoA-II); Truncated apolipoprotein A-II (Apolipoprotein A-II(1-76))]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02652	APOA2	Apolipoprotein A-II (Apo-AII) (ApoA-II) (Apolipoprotein A2) [Cleaved into: Proapolipoprotein A-II (ProapoA-II); Truncated apolipoprotein A-II (Apolipoprotein A-II(1-76))]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02654	APOC1	Apolipoprotein C-I (Apo-CI) (ApoC-I) (Apolipoprotein C1) [Cleaved into: Truncated apolipoprotein C-I]	Homo sapiens (Human)						x						CP	68055113	13,00						Proteomics			24098404
P02655	APOC2 APC2	Apolipoprotein C-II (Apo-CII) (ApoC-II) (Apolipoprotein C2) [Cleaved into: Proapolipoprotein C-II (ProapoC-II)]	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P02656	APOC3	Apolipoprotein C-III (Apo-CIII) (ApoC-III) (Apolipoprotein C3)	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
P02656	APOC3	Apolipoprotein C-III (Apo-CIII) (ApoC-III) (Apolipoprotein C3)	Homo sapiens (Human)						x					x	CP	68055113	1,58	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02671	FGA	Fibrinogen alpha chain [Cleaved into: Fibrinopeptide A; Fibrinogen alpha chain]	Homo sapiens (Human)						x					x	CP	68055113	1,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02671	FGA	Fibrinogen alpha chain [Cleaved into: Fibrinopeptide A; Fibrinogen alpha chain]	Homo sapiens (Human)					x						x	CP	68055113	1,69	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02671	FGA	Fibrinogen alpha chain [Cleaved into: Fibrinopeptide A; Fibrinogen alpha chain]	Homo sapiens (Human)						x					x	CP	68055113	2,20				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02671	FGA	Fibrinogen alpha chain [Cleaved into: Fibrinopeptide A; Fibrinogen alpha chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02675	FGB	Fibrinogen beta chain [Cleaved into: Fibrinopeptide B; Fibrinogen beta chain]	Homo sapiens (Human)					x						x	CP	68055113	1,39	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02675	FGB	Fibrinogen beta chain [Cleaved into: Fibrinopeptide B; Fibrinogen beta chain]	Homo sapiens (Human)						x					x	CP	68055113	1,47	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02675	FGB	Fibrinogen beta chain [Cleaved into: Fibrinopeptide B; Fibrinogen beta chain]	Homo sapiens (Human)						x					x	CP	68055113	4,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02675	FGB	Fibrinogen beta chain [Cleaved into: Fibrinopeptide B; Fibrinogen beta chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02675	FGB	Fibrinogen beta chain [Cleaved into: Fibrinopeptide B; Fibrinogen beta chain]	Homo sapiens (Human)					x						x	CP	68055113					unstimulated	2-DE + MALDI-TOF-TOF MS	Proteomics			22165124
P02679	FGG PRO2061	Fibrinogen gamma chain	Homo sapiens (Human)					x						x	CP	68055113	1,53	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02679	FGG PRO2061	Fibrinogen gamma chain	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02679	FGG PRO2061	Fibrinogen gamma chain	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02741	CRP PTX1	C-reactive protein [Cleaved into: C-reactive protein(1-205)]	Homo sapiens (Human)						x					x	CP	68055113	-1,19	42-50	MF		GCF was taken from the mesiobuccal aspect of each site (tooth) for up to 28 teeth per patient. Prior to the collection, supragingival plaque was removed using a sterile instrument. The site was isolated using cotton rolls and dried using a short blast of air directly through the contact (not into the sulcus/pocket). A methylcellulose strip (Pro Flow, Inc., Amityville, NY) was inserted into the sulcus/pocket until light resistance was felt. The strip stayed in position for 30 seconds.	ELISA	on-proteomics			24303954
P02741	CRP PTX1	C-reactive protein [Cleaved into: C-reactive protein(1-205)]	Homo sapiens (Human)					x						x	CP	68055113	1,72		MF	Lack of asystemic disease, not taking antibiotics over the last month, no intra oral lesions, not doing scaling and root planning not doing periodontal surgery at least within the last 6 months, taking no medication that would affect the periodontal tissues and not being an active smoker.	Firstly, by using spitting method, unstimulated saliva sample was collected from each subject.	ELISA	on-proteomics			24551806

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02743	APCS PTX2	Serum amyloid P-component (SAP) (9.5S alpha-1-glycoprotein) [Cleaved into: Serum amyloid P-component(1-203)]	Homo sapiens (Human)						x					x	CP	68055113	1,65	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02749	APOH B2G1	Beta-2-glycoprotein 1 (APC inhibitor) (Activated protein C-binding protein) (Anticardiolipin cofactor) (Apolipoprotein H) (Apo-H) (Beta-2-glycoprotein I) (B2GPI) (Beta(2)GPI)	Homo sapiens (Human)					x						x	CP	68055113	1,32	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02750	LRG1 LRG	Leucine-rich alpha-2-glycoprotein (LRG)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02750	LRG1 LRG	Leucine-rich alpha-2-glycoprotein (LRG)	Homo sapiens (Human)						x					x	CP	68055113	1,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02751	FN1 FN	Fibronectin (FN) (Cold-insoluble globulin) (CIG) [Cleaved into: Anastellin; Ugl-Y1; Ugl-Y2; Ugl-Y3]	Homo sapiens (Human)					x						x	CP	68055113	1,42	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02751	FN1 FN	Fibronectin (FN) (Cold-insoluble globulin) (CIG) [Cleaved into: Anastellin; Ugl-Y1; Ugl-Y2; Ugl-Y3]	Homo sapiens (Human)						x					x	CP	68055113	2,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02751	FN1 FN	Fibronectin (FN) (Cold-insoluble globulin) (CIG) [Cleaved into: Anastellin; Ugl-Y1; Ugl-Y2; Ugl-Y3]	Homo sapiens (Human)						x					x	CP	68055113						immunoblotting and analyzed by NIH image software	on-Proteomics		x	12416766
P02751	FN1 FN	Fibronectin (FN) (Cold-insoluble globulin) (CIG) [Cleaved into: Anastellin; Ugl-Y1; Ugl-Y2; Ugl-Y3]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02753	RBP4 PRO2222	Retinol-binding protein 4 (Plasma retinol-binding protein) (PRBP) (RBP) [Cleaved into: Plasma retinol-binding protein(1-182); Plasma retinol-binding protein(1-181); Plasma retinol-binding protein(1-179); Plasma retinol-binding protein(1-176)]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02760	AMBP HCP ITIL	Protein AMBP (Cleaved into: Alpha-1-microglobulin (Protein HC) (Alpha-1 microglycoprotein) (Complex forming glycoprotein heterogeneous in charge); Inter-alpha-trypsin inhibitor light chain (ITI-LC) (Bikunin) (EDC1) (HI-30) (Uronic-acid-rich protein); Trypstatin]	Homo sapiens (Human)						x					x	CP	68055113	1,44	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02763	ORM1 AGP1	Alpha-1-acid glycoprotein 1 (AGP 1) (Orosomucoid-1) (OMD 1)	Homo sapiens (Human)						x					x	CP	68055113	-1,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02763	ORM1 AGP1	Alpha-1-acid glycoprotein 1 (AGP 1) (Orosomucoid-1) (OMD 1)	Homo sapiens (Human)					x						x	CP	68055113	1,31	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02763	ORM1 AGP1	Alpha-1-acid glycoprotein 1 (AGP 1) (Orosomucoid-1) (OMD 1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02765	AHSG FETUA PRO2743	Alpha-2-HS-glycoprotein (Alpha-2-Z-globulin) (Ba-alpha-2-glycoprotein) (Fetuin-A) [Cleaved into: Alpha-2-HS-glycoprotein chain A; Alpha-2-HS-glycoprotein chain B]	Homo sapiens (Human)					x						x	CP	68055113	1,68	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02765	AHSG FETUA PRO2743	Alpha-2-HS-glycoprotein (Alpha-2-Z-globulin) (Ba-alpha-2-glycoprotein) (Fetuin-A) [Cleaved into: Alpha-2-HS-glycoprotein chain A; Alpha-2-HS-glycoprotein chain B]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02766	TTR PALB	Transthyretin (ATTR) (Prealbumin) (TBPA)	Homo sapiens (Human)						x						CP	68055113	23,00						Proteomics			24098404
P02766	TTR PALB	Transthyretin (ATTR) (Prealbumin) (TBPA)	Homo sapiens (Human)					x						x	CP	68055113	1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02766	TTR PALB	Transthyretin (ATTR) (Prealbumin) (TBPA)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02766	TTR PALB	Transthyretin (ATTR) (Prealbumin) (TBPA)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)					x						x	CP	68055113	1,42	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113	1,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)					x						x	CP	68055113	2,17	25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)					x						x	CP	68055113					unstimulated	2-DE + MALDI-TOF-TOF MS	Proteomics			22165124
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)					x						x	CP	68055113		37-52	M/F	Obese	Whole saliva samples were collected in the morning, at least 2 h after taking breakfast. The patients were asked to rinse their mouth and drink a glass of water, to encourage salivation (Schipper et al. 2007a,b). Using the Saliva-check kit BUFFER (GC France), the total stimulated saliva was collected. The subjects were asked to chew a piece of parafilm to activate salivation. At 30 s, a timer was triggered for a period of 5 min. The subjects spat the whole saliva into a sterile cup. The volume of saliva (in ml) was noted to allow calculation of the flow rate of saliva (Data S1). The saliva was then transferred using a pipette into a sterile centrifuge tube. No protease inhibitors were added during saliva sampling and treatment. Mucins, cells and food debris were eliminated by centrifugation at 10,000 g for 15 min. at 4°C. The clear supernatant of saliva was carefully collected in 15 ml tubes and immediately stored at 80°C prior to proteomic analysis.	SELDI-TOF)	Proteomics			22780105
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO(	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113	2,4	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)						x						CP	68055113	9,00						Proteomics			24098404
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)					x						x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)						x					x	CP	68055113	1,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02774	GC	Vitamin D-binding protein (DBP) (VDB) (Gc-globulin) (Group-specific component)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02786	TFRC	Transferrin receptor protein 1 (TR) (TIR) (TfR1) (Trfr) (T9) (p90) (CD antigen CD71) [Cleaved into: Transferrin receptor protein 1, serum form (sTfR)]	Homo sapiens (Human)						x					x	CP	68055113	1,29	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)						x					x	CP	68055113	1,07	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)					x						x	CP	68055113	1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)						x					x	CP	68055113	1,34	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)					x						x	CP	68055113					unstimulated	2-DE + MALDI-TOF-TOF MS	Proteomics		x	22165124
P02787	TF PRO1400	Serotransferrin (Transferrin) (Beta-1 metal-binding globulin) (Siderophilin)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21 .) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kalicin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21 .) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kalicin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)						x					x	CP	68055113	1,65	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21 .) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kalicin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)					x						x	CP	68055113	1,88	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21 .) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kalicin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02788	LTF GIG12 LF	Lactotransferrin (Lactoferrin) (EC 3.4.21 .) (Growth-inhibiting protein 12) (Talalactoferrin) [Cleaved into: Lactoferricin-H (Lfcin-H); Kalicin-1; Lactoferroxin-A; Lactoferroxin-B; Lactoferroxin-C]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02790	HPX	Hemopexin (Beta-1B-glycoprotein)	Homo sapiens (Human)					x						x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P02790	HPX	Hemopexin (Beta-1B-glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113	1,90	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P02790	HPX	Hemopexin (Beta-1B-glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02790	HPX	Hemopexin (Beta-1B-glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P02812	PRB2	Basic salivary proline-rich protein 2 (Salivary proline-rich protein) (Con1 glycoprotein) [Cleaved into: Basic proline-rich peptide IB-1; Basic proline-rich peptide P-E (IB-9); Basic proline-rich peptide IB-7; Basic proline-rich peptide IB-8c (Basic peptide P-F); Basic proline-rich peptide IB-4]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P02814	SMR3B PBII PRL3 PROL3	Submaxillary gland androgen-regulated protein 3B (Proline-rich peptide P-B) (Proline-rich protein 3) [Cleaved into: Peptide P-A; Peptide D1A]	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P02814	SMR3B PBII PRL3 PROL3	Submaxillary gland androgen-regulated protein 3B (Proline-rich peptide P-B) (Proline-rich protein 3) [Cleaved into: Peptide P-A; Peptide D1A]	Homo sapiens (Human)						x					x	CP	68055113	4,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P02814	SMR3B PBII PRL3 PROL3	Submaxillary gland androgen-regulated protein 3B (Proline-rich peptide P-B) (Proline-rich protein 3) (Cleaved into: Peptide P-A; Peptide D1A)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P03973	SLPI WAP4 WFDC4	Antileukoproteinase (ALP) (BLPI) (HUSI-1) (Mucus proteinase inhibitor) (MPI) (Protease inhibitor WAP4) (Secretory leukocyte protease inhibitor) (Seminal proteinase inhibitor) (WAP four-disulfide core domain protein 4)	Homo sapiens (Human)					x						x	CP	68055113	1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P03973	SLPI WAP4 WFDC4	Antileukoproteinase (ALP) (BLPI) (HUSI-1) (Mucus proteinase inhibitor) (MPI) (Protease inhibitor WAP4) (Secretory leukocyte protease inhibitor) (Seminal proteinase inhibitor) (WAP four-disulfide core domain protein 4)	Homo sapiens (Human)						x					x	CP	68055113	3,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P03973	SLPI WAP4 WFDC4	Antileukoproteinase (ALP) (BLPI) (HUSI-1) (Mucus proteinase inhibitor) (MPI) (Protease inhibitor WAP4) (Secretory leukocyte protease inhibitor) (Seminal proteinase inhibitor) (WAP four-disulfide core domain protein 4)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04004	VTN	Vitronectin (VN) (S-protein) (Serum-spreading factor) (V75) [Cleaved into: Vitronectin V65 subunit; Vitronectin V10 subunit; Somatomedin-B]	Homo sapiens (Human)						x					x	CP	68055113	1,72	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04004	VTN	Vitronectin (VN) (S-protein) (Serum-spreading factor) (V75) [Cleaved into: Vitronectin V65 subunit; Vitronectin V10 subunit; Somatomedin-B]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04035	HMGCR	3-hydroxy-3-methylglutaryl-coenzyme A reductase (HMG-CoA reductase) (EC 1.1.1.34)	Homo sapiens (Human)					x						x	CP	68055113	1,34	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04040	CAT	Catalase (EC 1.11.1.6)	Homo sapiens (Human)					x						x	CP	68055113	1,94	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04040	CAT	Catalase (EC 1.11.1.6)	Homo sapiens (Human)						x					x	CP	68055113	2,38	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04040	CAT	Catalase (EC 1.11.1.6)	Homo sapiens (Human)						x					x	CP	68055113	2,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04040	CAT	Catalase (EC 1.11.1.6)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04075	ALDOA ALDA	Fructose-bisphosphate aldolase A (EC 4.1.2.13) (Lung cancer antigen NY-LU-1) (Muscle-type aldolase)	Homo sapiens (Human)					x						x	CP	68055113	1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04075	ALDOA ALDA	Fructose-bisphosphate aldolase A (EC 4.1.2.13) (Lung cancer antigen NY-LU-1) (Muscle-type aldolase)	Homo sapiens (Human)						x					x	CP	68055113	3,16	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04075	ALDOA ALDA	Fructose-bisphosphate aldolase A (EC 4.1.2.13) (Lung cancer antigen NY-LU-1) (Muscle-type aldolase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04075	ALDOA ALDA	Fructose-bisphosphate aldolase A (EC 4.1.2.13) (Lung cancer antigen NY-LU-1) (Muscle-type aldolase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)						x					x	CP	68055113	1,78						Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)						x					x	CP	68055113	1,42	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)						x					x	CP	68055113	-5,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)					x						x	CP	68055113	-1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)						x					x	CP	68055113	2,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04080	CSTB CST6 STFB	Cystatin-B (CPI-B) (Liver thiol proteinase inhibitor) (Stefin-B)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04083	ANXA1 ANX1 LPC1	Annexin A1 (Annexin I) (Annexin-1) (Calpactin II) (Calpactin-2) (Chromobindin-9) (Lipocortin I) (Phospholipase A2 inhibitory protein) (p35)	Homo sapiens (Human)						x					x	CP	68055113	-1,35						Proteomics			24098404
P04083	ANXA1 ANX1 LPC1	Annexin A1 (Annexin I) (Annexin-1) (Calpactin II) (Calpactin-2) (Chromobindin-9) (Lipocortin I) (Phospholipase A2 inhibitory protein) (p35)	Homo sapiens (Human)						x					x	CP	68055113	1,41	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P04083	ANXA1 ANX1 LPC1	Annexin A1 (Annexin I) (Annexin-1) (Calpactin II) (Calpactin-2) (Chromobindin-9) (Lipocortin I) (Phospholipase A2 inhibitory protein) (p35)	Homo sapiens (Human)						x					x	CP	68055113	4,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04083	ANXA1 ANX1 LPC1	Annexin A1 (Annexin I) (Annexin-1) (Calpactin II) (Calpactin-2) (Chromobindin-9) (Lipocortin I) (Phospholipase A2 inhibitory protein) (p35)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04083	ANXA1 ANX1 LPC1	Annexin A1 (Annexin I) (Annexin-1) (Calpactin II) (Calpactin-2) (Chromobindin-9) (Lipocortin I) (Phospholipase A2 inhibitory protein) (p35)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04114	APOB	Apolipoprotein B-100 (Apo B-100) [Cleaved into: Apolipoprotein B-48 (Apo B-48)]	Homo sapiens (Human)						x					x	CP	68055113	1,44	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P04114	APOB	Apolipoprotein B-100 (Apo B-100) [Cleaved into: Apolipoprotein B-48 (Apo B-48)]	Homo sapiens (Human)						x					x	CP	68055113	5,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04114	APOB	Apolipoprotein B-100 (Apo B-100) [Cleaved into: Apolipoprotein B-48 (Apo B-48)]	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04114	APOB	Apolipoprotein B-100 (Apo B-100) [Cleaved into: Apolipoprotein B-48 (Apo B-48)]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04196	HRG	Histidine-rich glycoprotein (Histidine-proline-rich glycoprotein) (HPRG)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04207	0	Ig kappa chain V-III region CLL (Rheumatoid factor)	Homo sapiens (Human)					x						x	CP	68055113	1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04211	0	Ig lambda chain V region 4A	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P04211	0	Ig lambda chain V region 4A	Homo sapiens (Human)						x					x	CP	68055113	2,63	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04217	A1BG	Alpha-1B-glycoprotein (Alpha-1-B glycoprotein)	Homo sapiens (Human)					x						x	CP	68055113	1,48	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04217	A1BG	Alpha-1B-glycoprotein (Alpha-1-B glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04217	A1BG	Alpha-1B-glycoprotein (Alpha-1-B glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04220	0	Ig mu heavy chain disease protein (BOT)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04259	KRT6B K6B KRTL1	Keratin, type II cytoskeletal 6B (Cytokeratin-6B) (CK-6B) (Keratin-6B) (K6B) (Type-II keratin Kb10)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P04259	KRT6B K6B KRTL1	Keratin, type II cytoskeletal 6B (Cytokeratin-6B) (CK-6B) (Keratin-6B) (K6B) (Type-II keratin Kb10)	Homo sapiens (Human)						x					x	CP	68055113	5,54	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04259	KRT6B K6B KRTL1	Keratin, type II cytoskeletal 6B (Cytokeratin-6B) (CK-6B) (Keratin-6B) (K6B) (Type-II keratin Kb10)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04259	KRT6B K6B KRTL1	Keratin, type II cytoskeletal 6B (Cytokeratin-6B) (CK-6B) (Keratin-6B) (K6B) (Type-II keratin Kb10)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)						x					x	CP	68055113	1,2	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)					x						x	CP	68055113	-1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)						x					x	CP	68055113	2,28	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)						x					x	CP	68055113	103,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)					x							CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P04264	KRT1 KRTA	Keratin, type II cytoskeletal 1 (67 kDa cytokeratin) (Cytokeratin-1) (CK-1) (Hair alpha protein) (Keratin-1) (K1) (Type-II keratin Kb1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04406	GAPDH GAPD CDABP0047 OK/SW-cl.12	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) (EC 1.2.1.12) (Peptidyl-cysteine S-nitrosylase GAPDH) (EC 2.6.99.-)	Homo sapiens (Human)						x					x	CP	68055113	2,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P04406	GAPDH GAPD CDABP0047 OK/SW-cl.12	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) (EC 1.2.1.12) (Peptidyl-cysteine S-nitrosylase GAPDH) (EC 2.6.99.-)	Homo sapiens (Human)					x						x	CP	68055113	1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04406	GAPDH GAPD CDABP0047 OK/SW-cl.12	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) (EC 1.2.1.12) (Peptidyl-cysteine S-nitrosylase GAPDH) (EC 2.6.99.-)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P04406	GAPDH GAPD CDABP0047 OK/SW-cl.12	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) (EC 1.2.1.12) (Peptidyl-cysteine S-nitrosylase GAPDH) (EC 2.6.99.-)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04406	GAPDH GAPD CDABP0047 OK/SW-cl.12	Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) (EC 1.2.1.12) (Peptidyl-cysteine S-nitrosylase GAPDH) (EC 2.6.99.-)	Homo sapiens (Human)						x					x	CP	68055113		30-63	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P04430	0	Ig kappa chain V-I region BAN	Homo sapiens (Human)					x						x	CP	68055113	-1,25	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04433	0	Ig kappa chain V-III region VG (Fragment)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04434	0	Ig kappa chain V-III region VH (Fragment)	Homo sapiens (Human)					x						x	CP	68055113	1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04632	CAPNS1 CAPN4 CAPNS	Calpain small subunit 1 (CSS1) (Calcium-activated neutral proteinase small subunit) (CANP small subunit) (Calcium-dependent protease small subunit) (CDPS) (Calcium-dependent protease small subunit 1) (Calpain regulatory subunit)	Homo sapiens (Human)						x					x	CP	68055113	2,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113	1,62	25-60	MF	Exclusion criteria included subjects with history of any systemic diseases or conditions, subjects with history of any salivary gland diseases, subjects with any apparent oral infections (i.e. herpes or candida) or injuries or bleeding in the oral cavity unrelated to	Subjects were instructed not to brush their teeth, or eat or drink one hour before the time of saliva collection. Unstimulated whole saliva was collected between 11am and 12 Noon to avoid diurnal variation with patients seated with instructions to allow saliva to accumulate in the floor of the mouth and to spit without stimulation into the sterile container. The collected samples were immediately taken for biochemical analysis.	Amylase estimation was done by Kinetic method. 25µl of saliva sample and 1000µl of the reagent (ready to use liquid kit) was mixed and incubated at 37oC. After 15sec, it was measured and absorbance was taken at 405nm.	on-proteomics			25478449
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113	-1,17	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113	1,13	25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113	20,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04745	AMY1A AMY1; AMY1B AMY1; AMY1C AMY1	Alpha-amylase 1 (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 1) (Salivary alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04792	HSPB1 HSP27 HSP28	Heat shock protein beta-1 (HspB1) (28 kDa heat shock protein) (Estrogen-regulated 24 kDa protein) (Heat shock 27 kDa protein) (HSP 27) (Stress-responsive protein 27) (SRP27)	Homo sapiens (Human)						x						CP	68055113	8,00						Proteomics			24098404
P04792	HSPB1 HSP27 HSP28	Heat shock protein beta-1 (HspB1) (28 kDa heat shock protein) (Estrogen-regulated 24 kDa protein) (Heat shock 27 kDa protein) (HSP 27) (Stress-responsive protein 27) (SRP27)	Homo sapiens (Human)						x					x	CP	68055113	-5,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P04792	HSPB1 HSP27 HSP28	Heat shock protein beta-1 (HspB1) (28 kDa heat shock protein) (Estrogen-regulated 24 kDa protein) (Heat shock 27 kDa protein) (HSP 27) (Stress-responsive protein 27) (SRP27)	Homo sapiens (Human)					x						x	CP	68055113	1,11	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P04792	HSPB1 HSP27 HSP28	Heat shock protein beta-1 (HspB1) (28 kDa heat shock protein) (Estrogen-regulated 24 kDa protein) (Heat shock 27 kDa protein) (HSP 27) (Stress-responsive protein 27) (SRP27)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P04839	CYBB NOX2	Cytochrome b-245 heavy chain (EC 1.-.-.-) (CGD91-phox) (Cytochrome b(558) subunit beta) (Cytochrome b558 subunit beta) (Heme-binding membrane glycoprotein gp91phox) (NADPH oxidase 2) (Neutrophil cytochrome b 91 kDa polypeptide) (Superoxide-generating NADPH oxidase heavy chain subunit) (gp91-1) (gp91-phox) (p22 phagocyte B-cytochrome)	Homo sapiens (Human)						x					x	CP	68055113	3,21	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P04908	HIST1H2AB H2AFM; HIST1H2AE H2AFA	Histone H2A type 1-B/E (Histone H2A.2) (Histone H2A/a) (Histone H2A/m)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P05089	ARG1	Arginase-1 (EC 3.5.3.1) (Liver-type arginase) (Type I arginase)	Homo sapiens (Human)						x					x	CP	68055113	4,28	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05089	ARG1	Arginase-1 (EC 3.5.3.1) (Liver-type arginase) (Type I arginase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P05090	APOD	Apolipoprotein D (Apo-D) (ApoD)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113	1,26						Proteomics			24098404
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113	2,34	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)					x						x	CP	68055113	1,32	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)					x						x	CP	68055113	2,31	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics		x	20149214
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113	7,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113			M/F	All individuals possessed at least 20 teeth and had not received periodontal treatment or antibiotic therapy for medical or dental reasons for 3 mos prior to the investigation. Individuals were excluded if they possessed a history of metabolic bone diseases, autoimmune diseases, unstable diabetes, or post-menopausal osteoporosis. Pregnant or lactating women were excluded from participating in the study.	Unstimulated whole saliva was collected at each study visit via passive drooling into sterile plastic tubes from all participants (Mandel and Wotman, 1976). Samples were placed on ice, supplemented with a proteinase inhibitor combination of 1% aprotinin and 0.5% phenylmethylsulphonylfluoride, and aliquotted prior to storage at -80°C.	Protein biomarker levels were determined by colorimetric-based enzyme-linked immunosorbent assays (ELISAs), fluorescence-based protein microarrays, and radioimmunoassay (RIA), run according to manufacturer protocols. ELISAs (R&D Systems Inc., Minneapolis, MN, USA) were used for measurement of MMP-8 and -9, calprotectin, and osteoprotegerin (OPG). Detection of the cytokines interleukin (IL)-β, IL-2, IL-4, IL-5, IL-6, IL-10, and IL-13, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ was accomplished with a protein microarray (Whatman Inc., Florham Park, NJ, USA).	on-Proteomics			21406610
P05109	S100A8 CAGA CFAG MRP8	Protein S100-A8 (Calgranulin-A) (Calprotectin L1L subunit) (Cystic fibrosis antigen) (CFAG) (Leukocyte L1 complex light chain) (Migration inhibitory factor-related protein 8) (MRP-8) (p8) (S100 calcium-binding protein A8) (Urinary stone protein band A)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P05141	SLC25A5 ANT2	ADP/ATP translocase 2 (ADP,ATP carrier protein 2) (ADP,ATP carrier protein, fibroblast isoform) (Adenine nucleotide translocator 2) (ANT 2) (Solute carrier family 25 member 5) [Cleaved into: ADP/ATP translocase 2, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	5,93	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05155	SERPING1 C1IN C1NH	Plasma protease C1 inhibitor (C1 Inh) (C1Inh) (C1 esterase inhibitor) (C1-inhibitng factor) (Serpin G1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P05155	SERPING1 C1IN C1NH	Plasma protease C1 inhibitor (C1 Inh) (C1Inh) (C1 esterase inhibitor) (C1-inhibitng factor) (Serpin G1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)						x					x	CP	68055113	1,80						Proteomics			24098404
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)						x					x	CP	68055113	2,12	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)					x						x	CP	68055113	2,92	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)						x					x	CP	68055113	3,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P05164	MPO	Myeloperoxidase (MPO) (EC 1.11.2.2) [Cleaved into: Myeloperoxidase; 89 kDa myeloperoxidase; 84 kDa myeloperoxidase; Myeloperoxidase light chain; Myeloperoxidase heavy chain]	Homo sapiens (Human)					x						x	CP	68055113		28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at -80ºC until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P05231	IL6 IFNB2	Interleukin-6 (IL-6) (B-cell stimulatory factor 2) (BSF-2) (CTL differentiation factor) (CDF) (Hybridoma growth factor) (Interferon beta-2) (IFN-beta-2)	Homo sapiens (Human)					x						x	CP	68055113	3,75	39-51	MF		for UWS collection, all individuals (N = 88) were comfortably seated on a chair and requested to expectorate (without swallowing) into a gauged measuring cylinder for 5 continuous minutes. UWS flow rate (UWSFR) was measured and recorded in milliliters per minute. Immediately after collection, UWS samples were placed on ice and aliquoted before freezing at -80 C.	ELISA	on-proteomics			24171502
P05231	IL6 IFNB2	Interleukin-6 (IL-6) (B-cell stimulatory factor 2) (BSF-2) (CTL differentiation factor) (CDF) (Hybridoma growth factor) (Interferon beta-2) (IFN-beta-2)	Homo sapiens (Human)					x						x	CP	68055113	1,19	42-78	MF		Saliva samples were collected in the morning between 6 am and 12 pm, using previously described methods. Participants were asked to not eat, drink, or perform any kind of oral hygiene procedures before saliva collection. Just before saliva collection, a cup of water was given to the participant for rinsing. Five minutes after rinsing, participants were asked to spit into a 50-mL sterile plastic tube kept in ice. A maximum of 8 mL saliva was collected within 30 minutes.	ELISA	on-proteomics			24147842
P05231	IL6 IFNB2	Interleukin-6 (IL-6) (B-cell stimulatory factor 2) (BSF-2) (CTL differentiation factor) (CDF) (Hybridoma growth factor) (Interferon beta-2) (IFN-beta-2)	Homo sapiens (Human)					x						x	CP	68055113	+	35-65	M/F	Non-smoker, no intake of antibiotics in the last 6 months, no pregnancy or lactation	Non-stimulated whole expectorated saliva; Subjects refrained from eating, drinking, and oral hygiene.	ELISA	on-Proteomics			20192865
P05546	SERPIND1 HCF2	Heparin cofactor 2 (Heparin cofactor II) (HC-II) (Protease inhibitor leuserpin-2) (HLS2) (Serpin D1)	Homo sapiens (Human)						x					x	CP	68055113	2,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05787	KRT8 CYK8	Keratin, type II cytoskeletal 8 (Cytokeratin-8) (CK-8) (Keratin-8) (K8) (Type-II keratin Kb8)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P05787	KRT8 CYK8	Keratin, type II cytoskeletal 8 (Cytokeratin-8) (CK-8) (Keratin-8) (K8) (Type-II keratin Kb8)	Homo sapiens (Human)					x						x	CP	68055113	-1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06310	0	Ig kappa chain V-II region RPMI 6410	Homo sapiens (Human)						x					x	CP	68055113	1,97	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06312	IGKV4-1	Ig kappa chain V-IV region (Fragment)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P06314	0	Ig kappa chain V-IV region B17	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06331	0	Ig heavy chain V-II region ARH-77	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06396	GSN	Gelsolin (AGEL) (Actin-depolymerizing factor) (ADF) (Brevin)	Homo sapiens (Human)						x					x	CP	68055113	1,25	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P06396	GSN	Gelsolin (AGEL) (Actin-depolymerizing factor) (ADF) (Brevin)	Homo sapiens (Human)					x						x	CP	68055113	1,60	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P06396	GSN	Gelsolin (AGEL) (Actin-depolymerizing factor) (ADF) (Brevin)	Homo sapiens (Human)						x					x	CP	68055113	2,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06396	GSN	Gelsolin (AGEL) (Actin-depolymerizing factor) (ADF) (Brevin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06396	GSN	Gelsolin (AGEL) (Actin-depolymerizing factor) (ADF) (Brevin)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P06681	C2	Complement C2 (EC 3.4.21.43) (C3/C5 convertase) [Cleaved into: Complement C2b fragment; Complement C2a fragment]	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)						x					x	CP	68055113	1,46						Proteomics			24098404
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)						x					x	CP	68055113	2,41	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)					x						x	CP	68055113	1,27	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)					x						x	CP	68055113	1,99	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics		x	20149214
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)						x					x	CP	68055113	3,65	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)						x						CP	68055113	43,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P06702	S100A9 CAGB CFAG MRP14	Protein S100-A9 (Calgranulin-B) (Calprotectin L1H subunit) (Leukocyte L1 complex heavy chain) (Migration inhibitory factor-related protein 14) (MRP-14) (p14) (S100 calcium-binding protein A9)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P06703	S100A6 CACY	Protein S100-A6 (Calcyclin) (Growth factor-inducible protein 2A9) (MLN 4) (Prolactin receptor-associated protein) (PRA) (S100 calcium-binding protein A6)	Homo sapiens (Human)					x						x	CP	68055113	1,64	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P06703	S100A6 CACY	Protein S100-A6 (Calcyclin) (Growth factor-inducible protein 2A9) (MLN 4) (Prolactin receptor-associated protein) (PRA) (S100 calcium-binding protein A6)	Homo sapiens (Human)						x					x	CP	68055113	2,76	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06703	S100A6 CACY	Protein S100-A6 (Calcyclin) (Growth factor-inducible protein 2A9) (MLN 4) (Prolactin receptor-associated protein) (PRA) (S100 calcium-binding protein A6)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06727	APOA4	Apolipoprotein A-IV (Apo-AIV) (ApoA-IV) (Apolipoprotein A4)	Homo sapiens (Human)						x						CP	68055113	20,00						Proteomics			24098404
P06731	CEACAM5 CEA	Carcinoembryonic antigen-related cell adhesion molecule 5 (Carcinoembryonic antigen) (CEA) (Meconium antigen 100) (CD antigen CD66e)	Homo sapiens (Human)					x						x	CP	68055113	1,56	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06733	ENO1 ENO1L1 MBPB1 MPB1	Alpha-enolase (EC 4.2.1.11) (2-phospho-D-glycerate hydro-lyase) (C-myc promoter-binding protein) (Enolase 1) (MBP-1) (MPB-1) (Non-neural enolase) (NNE) (Phosphopyruvate hydratase) (Plasminogen-binding protein)	Homo sapiens (Human)					x						x	CP	68055113	1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P06733	ENO1 ENO1L1 MBPB1 MPB1	Alpha-enolase (EC 4.2.1.11) (2-phospho-D-glycerate hydro-lyase) (C-myc promoter-binding protein) (Enolase 1) (MBP-1) (MPB-1) (Non-neural enolase) (NNE) (Phosphopyruvate hydratase) (Plasminogen-binding protein)	Homo sapiens (Human)						x					x	CP	68055113	2,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06733	ENO1 ENO1L1 MBPB1 MPB1	Alpha-enolase (EC 4.2.1.11) (2-phospho-D-glycerate hydro-lyase) (C-myc promoter-binding protein) (Enolase 1) (MBP-1) (MPB-1) (Non-neural enolase) (NNE) (Phosphopyruvate hydratase) (Plasminogen-binding protein)	Homo sapiens (Human)						x					x	CP	68055113						isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06733	ENO1 ENO1L1 MBPB1 MPB1	Alpha-enolase (EC 4.2.1.11) (2-phospho-D-glycerate hydro-lyase) (C-myc promoter-binding protein) (Enolase 1) (MBP-1) (MPB-1) (Non-neural enolase) (NNE) (Phosphopyruvate hydratase) (Plasminogen-binding protein)	Homo sapiens (Human)						x					x	CP	68055113							Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P06737	PYGL	Glycogen phosphorylase, liver form (EC 2.4.1.1)	Homo sapiens (Human)						x					x	CP	68055113	3,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06744	GPI	Glucose-6-phosphate isomerase (GPI) (EC 5.3.1.9) (Autocrine motility factor) (AMF) (Neuroleukin) (NLK) (Phosphoglucose isomerase) (PGI) (Phosphohexose isomerase) (PHI) (Sperm antigen 36) (SA-36)	Homo sapiens (Human)					x						x	CP	68055113	1,32	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06744	GPI	Glucose-6-phosphate isomerase (GPI) (EC 5.3.1.9) (Autocrine motility factor) (AMF) (Neuroleukin) (NLK) (Phosphoglucose isomerase) (PGI) (Phosphohexose isomerase) (PHI) (Sperm antigen 36) (SA-36)	Homo sapiens (Human)						x					x	CP	68055113	4,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06744	GPI	Glucose-6-phosphate isomerase (GPI) (EC 5.3.1.9) (Autocrine motility factor) (AMF) (Neuroleukin) (NLK) (Phosphoglucose isomerase) (PGI) (Phosphohexose isomerase) (PHI) (Sperm antigen 36) (SA-36)	Homo sapiens (Human)						x					x	CP	68055113						isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P06744	GPI	Glucose-6-phosphate isomerase (GPI) (EC 5.3.1.9) (Autocrine motility factor) (AMF) (Neuroleukin) (NLK) (Phosphoglucose isomerase) (PGI) (Phosphohexose isomerase) (PHI) (Sperm antigen 36) (SA-36)	Homo sapiens (Human)						x					x	CP	68055113							Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P06748	NPM1 NPM	Nucleophosmin (NPM) (Nucleolar phosphoprotein B23) (Nucleolar protein NO38) (Numatrin)	Homo sapiens (Human)						x					x	CP	68055113	2,87	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06753	TPM3	Tropomyosin alpha-3 chain (Gamma-tropomyosin) (Tropomyosin-3) (Tropomyosin-5) (hTM5)	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P06870	KLK1	Kallikrein-1 (EC 3.4.21.35) (Kidney/pancreas/salivary gland kallikrein) (Tissue kallikrein)	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P06870	KLK1	Kallikrein-1 (EC 3.4.21.35) (Kidney/pancreas/salivary gland kallikrein) (Tissue kallikrein)	Homo sapiens (Human)						x					x	CP	68055113	7,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P06887	0	Ig lambda chain V-I region MEM	Homo sapiens (Human)						x					x	CP	68055113	1,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P06899	HIST1H2BJ H2BFR	Histone H2B type 1-J (Histone H2B.1) (Histone H2B.r) (H2B/r)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P07108	DBI	Acyl-CoA-binding protein (ACBP) (Diazepam-binding inhibitor) (DBI) (Endozepine) (EP)	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07108	DBI	Acyl-CoA-binding protein (ACBP) (Diazepam-binding inhibitor) (DBI) (Endozepine) (EP)	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07108	DBI	Acyl-CoA-binding protein (ACBP) (Diazepam-binding inhibitor) (DBI) (Endozepine) (EP)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P07195	LDHB	L-lactate dehydrogenase B chain (LDH-B) (EC 1.1.1.27) (LDH heart subunit) (LDH-H) (Renal carcinoma antigen NY-REN-46)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P07196	NEFL NF68 NFL	Neurofilament light polypeptide (NF-L) (68 kDa neurofilament protein) (Neurofilament triplet L protein)	Homo sapiens (Human)							x					CP	68055113	.32(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P07197	NEFM NEF3 NFM	Neurofilament medium polypeptide (NF-M) (160 kDa neurofilament protein) (Neurofilament 3) (Neurofilament triplet M protein)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P07237	P4HB ERBA2L PDI PDIA1 PO4DB	Protein disulfide-isomerase (PDI) (EC 5.3.4.1) (Cellular thyroid hormone-binding protein) (Prolyl 4-hydroxylase subunit beta) (p55)	Homo sapiens (Human)					x						x	CP	68055113	1,34	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07237	P4HB ERBA2L PDI PDIA1 PO4DB	Protein disulfide-isomerase (PDI) (EC 5.3.4.1) (Cellular thyroid hormone-binding protein) (Prolyl 4-hydroxylase subunit beta) (p55)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P07339	CTSD CPSD	Cathepsin D (EC 3.4.23.5) [Cleaved into: Cathepsin D light chain; Cathepsin D heavy chain]	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07339	CTSD CPSD	Cathepsin D (EC 3.4.23.5) [Cleaved into: Cathepsin D light chain; Cathepsin D heavy chain]	Homo sapiens (Human)						x					x	CP	68055113	3,01	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07355	ANXA2 ANX2 ANX2L4 CAL1H LPC2D	Annexin A2 (Annexin II) (Annexin-2) (Calpactin I heavy chain) (Calpactin-1 heavy chain) (Chromobindin-8) (Lipocortin II) (Placental anticoagulant protein IV) (PAP-IV) (Protein I) (p36)	Homo sapiens (Human)						x					x	CP	68055113	3,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07355	ANXA2 ANX2 ANX2L4 CAL1H LPC2D	Annexin A2 (Annexin II) (Annexin-2) (Calpactin I heavy chain) (Calpactin-1 heavy chain) (Chromobindin-8) (Lipocortin II) (Placental anticoagulant protein IV) (PAP-IV) (Protein I) (p36)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P07476	IVL	Involucrin	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07477	PRSS1 TRP1 TRY1 TRYP1	Trypsin-1 (EC 3.4.21.4) (Beta-trypsin) (Cationic trypsinogen) (Serine protease 1) (Trypsin I) [Cleaved into: Alpha-trypsin chain 1; Alpha-trypsin chain 2]	Homo sapiens (Human)						x					x	CP	68055113	1,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P07477	PRSS1 TRP1 TRY1 TRYP1	Trypsin-1 (EC 3.4.21.4) (Beta-trypsin) (Cationic trypsinogen) (Serine protease 1) (Trypsin I) [Cleaved into: Alpha-trypsin chain 1; Alpha-trypsin chain 2]	Homo sapiens (Human)						x					x	CP	68055113	1,07	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07686	HEXB HCC7	Beta-hexosaminidase subunit beta (EC 3.2.1.52) (Beta-N-acetylhexosaminidase subunit beta) (Hexosaminidase subunit B) (Cervical cancer proto-oncogene 7 protein) (HCC-7) (N-acetyl-beta-glucosaminidase subunit beta) [Cleaved into: Beta-hexosaminidase subunit beta chain B; Beta-hexosaminidase subunit beta chain A]	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07711	CTSL CTSL1	Cathepsin L1 (EC 3.4.22.15) (Cathepsin L) (Major excreted protein) (MEP) [Cleaved into: Cathepsin L1 heavy chain; Cathepsin L1 light chain]	Homo sapiens (Human)					x						x	CP	68055113	-1,36	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)						x						CP	68055113	4,00						Proteomics			24098404
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)						x					x	CP	68055113	-1,09	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)					x						x	CP	68055113	1,67	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)						x					x	CP	68055113	1,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P07737	PFN1	Profilin-1 (Epididymis tissue protein Li 184a) (Profilin I)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P07858	CTSB CPSB	Cathepsin B (EC 3.4.22.1) (APP secretase) (APPS) (Cathepsin B1) [Cleaved into: Cathepsin B light chain; Cathepsin B heavy chain]	Homo sapiens (Human)					x						x	CP	68055113	-1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07858	CTSB CPSB		Homo sapiens (Human)						x					x	CP	68055113			MF		The concentrations of active elastase and cathepsin B were determined using peptide substrates.	SLPI and alpha1-proteinase inhibitor (alpha1PI) concentrations were determined using enzyme-linked immunosorbent assays (ELISAs). The molecular forms of SLPI were examined by immunoblotting.	on-Proteomics			16953825
P07900	HSP90AA1 HSP90A HSPC1 HSPCA	Heat shock protein HSP 90-alpha (Heat shock 86 kDa) (HSP 86) (HSP86) (Renal carcinoma antigen NY-REN-38)	Homo sapiens (Human)						x					x	CP	68055113	2,61	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P07900	HSP90AA1 HSP90A HSPC1 HSPCA	Heat shock protein HSP 90-alpha (Heat shock 86 kDa) (HSP 86) (HSP86) (Renal carcinoma antigen NY-REN-38)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P07900	HSP90AA1 HSP90A HSPC1 HSPCA	Heat shock protein HSP 90-alpha (Heat shock 86 kDa) (HSP 86) (HSP86) (Renal carcinoma antigen NY-REN-38)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P07951	TPM2 TMSB	Tropomyosin beta chain (Beta-tropomyosin) (Tropomyosin-2)	Homo sapiens (Human)					x						x	CP	68055113	1,69	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P07951	TPM2 TMSB	Tropomyosin beta chain (Beta-tropomyosin) (Tropomyosin-2)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P07996	THBS1 TSP TSP1	Thrombospondin-1	Homo sapiens (Human)						x					x	CP	68055113	-2,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P0DMV9	HSPA1B	Heat shock 70 kDa protein 1A/1B (Heat shock 70 kDa protein 1/2) (HSP70-1/HSP70-2) (HSP70.1/HSP70.2)	Homo sapiens (Human)					x						x	CP	68055113	1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P0DMV9	HSPA1B	Heat shock 70 kDa protein 1A/1B (Heat shock 70 kDa protein 1/2) (HSP70-1/HSP70-2) (HSP70.1/HSP70.2)	Homo sapiens (Human)						x					x	CP	68055113	2,54	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P0DMV9	HSPA1B	Heat shock 70 kDa protein 1A/1B (Heat shock 70 kDa protein 1/2) (HSP70-1/HSP70-2) (HSP70.1/HSP70.2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P0DMV9	HSPA1B	Heat shock 70 kDa protein 1A/1B (Heat shock 70 kDa protein 1/2) (HSP70-1/HSP70-2) (HSP70.1/HSP70.2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08118	MSMB PRSP	Beta-microseminoprotein (Immunoglobulin-binding factor) (IGBF) (PN44) (Prostate secreted seminal plasma protein) (Prostate secretory protein of 94 amino acids) (PSP-94) (PSP94) (Seminal plasma beta-inhibin)	Homo sapiens (Human)						x					x	CP	68055113	2,22	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08123	COL1A2	Collagen alpha-2(I) chain	Homo sapiens (Human)						x					x	CP	68055113	-1,10	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P08236	GUSB		Homo sapiens (Human)						x					x	CP	68055113			MF		GCF was collected from eight posterior sites in each quadrant, and periodontal parameters were recorded	GCF was assayed for IL-8 by ELISA and BG by a fluorometric assay.	on-Proteomics			16911568
P08238	HSP90AB1 HSP90B HSPC2 HSPCB	Heat shock protein HSP 90-beta (HSP 90) (Heat shock 84 kDa) (HSP 84) (HSP84)	Homo sapiens (Human)						x					x	CP	68055113	3,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08246	ELANE ELA2	Leukocyte elastase	Homo sapiens (Human)						x					x	CP	68055113	1,71	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P08246	ELANE ELA2	Neutrophil elastase (EC 3.4.21.37) (Bone marrow serine protease) (Elastase-2) (Human leukocyte elastase) (HLE) (Medullasin) (PMN elastase)	Homo sapiens (Human)						x					x	CP	68055113	2,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08246	ELANE ELA2	Neutrophil elastase (EC 3.4.21.37) (Bone marrow serine protease) (Elastase-2) (Human leukocyte elastase) (HLE) (Medullasin) (PMN elastase)	Homo sapiens (Human)					x						x	CP	68055113	6,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at -80°C until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P08246	ELANE ELA2	Neutrophil elastase (EC 3.4.21.37) (Bone marrow serine protease) (Elastase-2) (Human leukocyte elastase) (HLE) (Medullasin) (PMN elastase)	Homo sapiens (Human)						x					x	CP	68055113	11,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P08246	ELANE ELA2	Neutrophil elastase (EC 3.4.21.37) (Bone marrow serine protease) (Elastase-2) (Human leukocyte elastase) (HLE) (Medullasin) (PMN elastase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08311	CTSG	Cathepsin G	Homo sapiens (Human)						x					x	CP	68055113	2,27						Proteomics			24098404
P08311	CTSG	Cathepsin G (CG) (EC 3.4.21.20)	Homo sapiens (Human)						x					x	CP	68055113	5,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08311	CTSG	Cathepsin G (CG) (EC 3.4.21.20)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P08473	MME EPN	Membrane metallo-endopeptidase	Homo sapiens (Human)							x					CP	68055113	35(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm]) or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P08473	MME EPN	Neprilysin (EC 3.4.24.11) (Atriopeptidase) (Common acute lymphocytic leukemia antigen) (CALLA) (Enkephalinase) (Neutral endopeptidase 24.11) (NEP) (Neutral endopeptidase) (Skin fibroblast elastase) (SFE) (CD antigen CD10)	Homo sapiens (Human)						x					x	CP	68055113	11,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08575	PTPRC CD45	Receptor-type tyrosine-protein phosphatase C (EC 3.1.3.48) (Leukocyte common antigen) (L-CA) (T200) (CD antigen CD45)	Homo sapiens (Human)							x					CP	68055113		35-68			The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451
P08603	CFH HF HF1 HF2	Complement factor H (H factor 1)	Homo sapiens (Human)						x					x	CP	68055113	1,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08603	CFH HF HF1 HF2	Complement factor H (H factor 1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P08603	CFH HF HF1 HF2	Complement factor H (H factor 1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08670	VIM	Vimentin	Homo sapiens (Human)						x					x	CP	68055113	2,15	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08670	VIM	Vimentin	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08670	VIM	Vimentin	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P08670	VIM	Vimentin	Homo sapiens (Human)							x					CP	68055113	29(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm]) or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P08727	KRT19	Keratin, type I cytoskeletal 19	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P08727	KRT19	Keratin, type I cytoskeletal 19 (Cytokeratin-19) (CK-19) (Keratin-19) (K19)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08729	KRT7 SCL	Keratin, type II cytoskeletal 7	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P08729	KRT7 SCL	Keratin, type II cytoskeletal 7	Homo sapiens (Human)						x					x	CP	68055113	1,31	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P08779	KRT16 KRT16A	Keratin, type I cytoskeletal 16	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P08779	KRT16 KRT16A	Keratin, type I cytoskeletal 16	Homo sapiens (Human)						x					x	CP	68055113	-1,14	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P08779	KRT16 KRT16A	Keratin, type I cytoskeletal 16 (Cytokeratin-16) (CK-16) (Keratin-16) (K16)	Homo sapiens (Human)						x					x	CP	68055113	3,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P08779	KRT16 KRT16A	Keratin, type I cytoskeletal 16 (Cytokeratin-16) (CK-16) (Keratin-16) (K16)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P08779	KRT16 KRT16A	Keratin, type I cytoskeletal 16 (Cytokeratin-16) (CK-16) (Keratin-16) (K16)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P08F94	PKHD1 FCYT TIGM1	Polycystic kidney and hepatic disease 1 (autosomal recessive)-like 1	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CF and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P09211	GSTP1 FAEES3 GST3	Glutathione S-transferase P (EC 2.5.1.18) (GST class-pi) (GSTP1-1)	Homo sapiens (Human)						x					x	CP	68055113	-24,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P09211	GSTP1 FAEES3 GST3	Glutathione S-transferase P (EC 2.5.1.18) (GST class-pi) (GSTP1-1)	Homo sapiens (Human)					x						x	CP	68055113	-1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P09211	GSTP1 FAEES3 GST3	Glutathione S-transferase P (EC 2.5.1.18) (GST class-pi) (GSTP1-1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P09228	CST2	Cystatin-SA (Cystatin-2) (Cystatin-S5)	Homo sapiens (Human)					x						x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P09228	CST2	Cystatin-SA (Cystatin-2) (Cystatin-S5)	Homo sapiens (Human)						x					x	CP	68055113	1,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P09228	CST2	Cystatin-SA (Cystatin-2) (Cystatin-S5)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P09382	LGALS1	Galectin-1 (Gal-1) (14 kDa laminin-binding protein) (HLBP14) (14 kDa lectin) (Beta-galactoside-binding lectin L-14-I) (Galaptin) (HBL) (HPL) (Lactose-binding lectin 1) (Lectin galactoside-binding soluble 1) (Putative MAPK-activating protein PM12) (S-Lac lectin 1)	Homo sapiens (Human)						x					x	CP	68055113	2,57	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P09960	LTA4H LTA4	Leukotriene A-4 hydrolase (LTA-4 hydrolase) (EC 3.3.2.6) (Leukotriene A(4) hydrolase)	Homo sapiens (Human)					x						x	CP	68055113	1,50	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P09960	LTA4H LTA4	Leukotriene A-4 hydrolase (LTA-4 hydrolase) (EC 3.3.2.6) (Leukotriene A(4) hydrolase)	Homo sapiens (Human)						x					x	CP	68055113	3,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from culusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P09960	LTA4H LTA4	Leukotriene A-4 hydrolase (LTA-4 hydrolase) (EC 3.3.2.6) (Leukotriene A(4) hydrolase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P09972	ALDOC ALDC	Fructose-bisphosphate aldolase C (EC 4.1.2.13) (Brain-type aldolase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P0C0L4	C4A CO4 CPAMD2	Complement C4-A (Acidic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 2) [Cleaved into: Complement C4 beta chain; Complement C4-A alpha chain; C4a anaphylatoxin; C4b-A; C4d-A; Complement C4 gamma chain]	Homo sapiens (Human)						x					x	CP	68055113	2,71	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P0C0L4	C4A CO4 CPAMD2	Complement C4-A (Acidic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 2) [Cleaved into: Complement C4 beta chain; Complement C4-A alpha chain; C4a anaphylatoxin; C4b-A; C4d-A; Complement C4 gamma chain]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P0C0L4	C4A CO4 CPAMD2	Complement C4-A (Acidic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 2) [Cleaved into: Complement C4 beta chain; Complement C4-A alpha chain; C4a anaphylatoxin; C4b-A; C4d-A; Complement C4 gamma chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P0C0L5	C4B CO4 CPAMD3; C4B_2	Complement C4-B (Basic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 3) [Cleaved into: Complement C4 beta chain; Complement C4-B alpha chain; C4a anaphylatoxin; C4b-B; C4d-B; Complement C4 gamma chain]	Homo sapiens (Human)					x						x	CP	68055113	1,40	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P0C0L5	C4B CO4 CPAMD3; C4B_2	Complement C4-B (Basic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 3) [Cleaved into: Complement C4 beta chain; Complement C4-B alpha chain; C4a anaphylatoxin; C4b-B; C4d-B; Complement C4 gamma chain]	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P0C0L5	C4B CO4 CPAMD3; C4B_2	Complement C4-B (Basic complement C4) (C3 and PZP-like alpha-2-macroglobulin domain-containing protein 3) [Cleaved into: Complement C4 beta chain; Complement C4-B alpha chain; C4a anaphylatoxin; C4b-B; C4d-B; Complement C4 gamma chain]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P0C0S5	H2AFZ H2AZ	Histone H2A.Z	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
P0C0S8	HIST1H2AG H2AFP; HIST1H2AI H2AFC; HIS	Histone H2A type 1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P0C0S8	HIST1H2AG H2AFP; HIST1H2AI H2AFC; HIS	Histone H2A type 1	Homo sapiens (Human)						x					x	CP	68055113	1,92	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P0C221	CCDC175 C14orf38	Coiled-coil domain-containing protein 175	Homo sapiens (Human)					x						x	CP	68055113	1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P0CG04	IGLC1	Ig lambda-1 chain C regions	Homo sapiens (Human)					x						x	CP	68055113	-1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P0CG05	IGLC2	Ig lambda-2 chain C regions	Homo sapiens (Human)					x						x	CP	68055113	1,31	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P0CG05	IGLC2	Ig lambda-2 chain C regions	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P0CG06	IGLC3	Ig lambda-3 chain C regions	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P0CG29	GSTT2	Glutathione S-transferase theta 2	Homo sapiens (Human)							x					CP	68055113	,27(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P0CG38	POTEI	POTE ankyrin domain family member I	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
P0CG39	POTEJ	POTE ankyrin domain family member J	Homo sapiens (Human)						x						CP	68055113	7,00						Proteomics			24098404
P0CG48	UBC	Polyubiquitin-C [Cleaved into: Ubiquitin]	Homo sapiens (Human)					x						x	CP	68055113	1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P10145	CXCL8 IL8	Interleukin-8	Homo sapiens (Human)					x						x	CP	68055113	-1,21	42-78	MF		Saliva samples were collected in the morning between 6 am and 12 pm, using previously described methods. Participants were asked to not eat, drink, or perform any kind of oral hygiene procedures before saliva collection. Just before saliva collection, a cup of water was given to the participant for rinsing. Five minutes after rinsing, participants were asked to spit into a 50-mL sterile plastic tube kept in ice. A maximum of 8 mL saliva was collected within 30 minutes.	ELISA	on-proteomics			24147842
P10147	CCL3 G0S19-1 MIP1A SCYA3	C-C motif chemokine 3	Homo sapiens (Human)					x						x	CP	68055113	18,00						Non-proteomics		x	22126530
P10412	HIST1H1E H1F4	Histone H1.4	Homo sapiens (Human)						x					x	CP	68055113	2,11						Proteomics			24098404
P10412	HIST1H1E H1F4	Histone H1.4 (Histone H1b) (Histone H1s-4)	Homo sapiens (Human)						x					x	CP	68055113	2,26	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P10599	TXN TRDX TRX TRX1	Thioredoxin (Trx) (ATL-derived factor) (ADF) (Surface-associated sulphydryl protein) (SASP)	Homo sapiens (Human)					x						x	CP	68055113	-1,31	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P10599	TXN TRDX TRX TRX1	Thioredoxin (Trx) (ATL-derived factor) (ADF) (Surface-associated sulphydryl protein) (SASP)	Homo sapiens (Human)						x					x	CP	68055113	2,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P10599	TXN TRDX TRX TRX1	Thioredoxin (Trx) (ATL-derived factor) (ADF) (Surface-associated sulphydryl protein) (SASP)	Homo sapiens (Human)					x							CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P10599	TXN TRDX TRX TRX1	Thioredoxin (Trx) (ATL-derived factor) (ADF) (Surface-associated sulphydryl protein) (SASP)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P10620	MGST1 GST12 MGST	Microsomal glutathione S-transferase 1	Homo sapiens (Human)							x					CP	68055113	,26(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	sn-proteomics			24122488
P10909	CLU APOJ CLI KUB1 AAG4	Clusterin (Aging-associated gene 4 protein) (Apolipoprotein J) (Apo-J) (Complement cytolysis inhibitor) (CLI) (Complement-associated protein SP-40,40) (Ku70-binding protein 1) (NA1/NA2) (Testosterone-repressed prostate message 2) (TRPM-2) [Cleaved into: Clusterin beta chain (ApoJalpha) (Complement cytolysis inhibitor a chain); Clusterin alpha chain (ApoJbeta) (Complement cytolysis inhibitor b chain)]	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics		x	22092770
P11021	HSPA5 GRP78	78 kDa glucose-regulated protein (GRP-78) (Endoplasmic reticulum lumenal Ca(2+)-binding protein grp78) (Heat shock 70 kDa protein 5) (Immunoglobulin heavy chain-binding protein) (BiP)	Homo sapiens (Human)					x						x	CP	68055113	1,25	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P11021	HSPA5 GRP78	78 kDa glucose-regulated protein (GRP-78) (Endoplasmic reticulum lumenal Ca(2+)-binding protein grp78) (Heat shock 70 kDa protein 5) (Immunoglobulin heavy chain-binding protein) (BiP)	Homo sapiens (Human)						x					x	CP	68055113	3,30	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P11142	HSPA8 HSC70 HSP73 HSPA10	Heat shock cognate 71 kDa protein (Heat shock 70 kDa protein 8)	Homo sapiens (Human)					x						x	CP	68055113	1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P11142	HSPA8 HSC70 HSP73 HSPA10	Heat shock cognate 71 kDa protein (Heat shock 70 kDa protein 8)	Homo sapiens (Human)						x					x	CP	68055113	2,57	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P11142	HSPA8 HSC70 HSP73 HSPA10	Heat shock cognate 71 kDa protein (Heat shock 70 kDa protein 8)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P11142	HSPA8 HSC70 HSP73 HSPA10	Heat shock cognate 71 kDa protein (Heat shock 70 kDa protein 8)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P11387	TOP1	DNA topoisomerase 1 (EC 5.99.1.2) (DNA topoisomerase I)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P11413	G6PD	Glucose-6-phosphate 1-dehydrogenase (G6PD) (EC 1.1.1.49)	Homo sapiens (Human)						x					x	CP	68055113	4,65	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P11413	G6PD	Glucose-6-phosphate 1-dehydrogenase (G6PD) (EC 1.1.1.49)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P11766	ADH5 ADHX FDH	Alcohol dehydrogenase class-3 (EC 1.1.1.1) (Alcohol dehydrogenase 5) (Alcohol dehydrogenase class chi chain) (Alcohol dehydrogenase class-III) (Glutathione-dependent formaldehyde dehydrogenase) (FALDH) (FDH) (GSH-FDH) (EC 1.1.1.-) (S-(hydroxymethyl)glutathione dehydrogenase) (EC 1.1.1.284)	Homo sapiens (Human)						x					x	CP	68055113	2,41	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P11836	MS4A1 CD20	Membrane-spanning 4-domains, subfamily A, member 1	Homo sapiens (Human)							x					CP	68055113	32(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P12035	KRT3	Keratin, type II cytoskeletal 3	Homo sapiens (Human)						x					x	CP	68055113						Proteomics			24098404
P12035	KRT3	Keratin, type II cytoskeletal 3	Homo sapiens (Human)						x					x	CP	68055113	1,3	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P12107	COL11A1 COLL6	Collagen alpha-1(XI) chain	Homo sapiens (Human)						x					x	CP	68055113	1,75	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P12107	COL11A1 COLL6	Collagen alpha-1(XI) chain	Homo sapiens (Human)						x					x	CP	68055113	1,75	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P12109	COL6A1	Collagen alpha-1(VI) chain	Homo sapiens (Human)					x						x	CP	68055113	-1,53	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P12273	PIP GCDFP15 GPIP4	Prolactin-inducible protein (Gross cystic disease fluid protein 15) (GCDFP-15) (Prolactin-induced protein) (Secretory actin-binding protein) (SABP) (gp17)	Homo sapiens (Human)						x					x	CP	68055113	-9,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P12273	PIP GCDFP15 GPIP4	Prolactin-inducible protein (Gross cystic disease fluid protein 15) (GCDFP-15) (Prolactin-induced protein) (Secretory actin-binding protein) (SABP) (gp17)	Homo sapiens (Human)					x						x	CP	68055113	-1,36	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a paraffin bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P12273	PIP GCDFP15 GPIP4	Prolactin-inducible protein (Gross cystic disease fluid protein 15) (GCDFP-15) (Prolactin-induced protein) (Secretory actin-binding protein) (SABP) (gp17)	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P12273	PIP GCDFP15 GPIP4	Prolactin-inducible protein (Gross cystic disease fluid protein 15) (GCDFP-15) (Prolactin-induced protein) (Secretory actin-binding protein) (SABP) (gp17)	Homo sapiens (Human)						x					x	CP	68055113	1,95	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P12273	PIP GCDFP15 GPIP4	Prolactin-inducible protein (Gross cystic disease fluid protein 15) (GCDFP-15) (Prolactin-induced protein) (Secretory actin-binding protein) (SABP) (gp17)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
P12429	ANXA3 ANX3	Annexin A3	Homo sapiens (Human)						x						CP	68055113	5,00					Proteomics			24098404
P12429	ANXA3 ANX3	Annexin A3 (35-alpha calcimedin) (Annexin III) (Annexin-3) (Inositol 1,2-cyclic phosphate 2-phosphohydrolase) (Lipocortin III) (Placental anticoagulant protein III) (PAP-III)	Homo sapiens (Human)						x					x	CP	68055113	2,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P12429	ANXA3 ANX3	Annexin A3 (35-alpha calcimedin) (Annexin III) (Annexin-3) (Inositol 1,2-cyclic phosphate 2-phosphohydrolase) (Lipocortin III) (Placental anticoagulant protein III) (PAP-III)	Homo sapiens (Human)						x					x	CP	68055113	5,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P12814	ACTN1	Alpha-actinin-1 (Alpha-actinin cytoskeletal isoform) (F-actin cross-linking protein) (Non-muscle alpha-actinin-1)	Homo sapiens (Human)					x						x	CP	68055113	2,12	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P12814	ACTN1	Alpha-actinin-1 (Alpha-actinin cytoskeletal isoform) (F-actin cross-linking protein) (Non-muscle alpha-actinin-1)	Homo sapiens (Human)						x					x	CP	68055113	2,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P12814	ACTN1	Alpha-actinin-1 (Alpha-actinin cytoskeletal isoform) (F-actin cross-linking protein) (Non-muscle alpha-actinin-1)	Homo sapiens (Human)						x					x	CP	68055113	8,80				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P12814	ACTN1	Alpha-actinin-1 (Alpha-actinin cytoskeletal isoform) (F-actin cross-linking protein) (Non-muscle alpha-actinin-1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P12830	CDH1 CDHE UVO	Cadherin-1 (CAM 120/80) (Epithelial cadherin) (E-cadherin) (Uvomorulin) (CD antigen CD324) [Cleaved into: E-Cad/CTF1; E-Cad/CTF2; E-Cad/CTF3]	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P12955	PEPD PRD	Xaa-Pro dipeptidase (X-Pro dipeptidase) (EC 3.4.13.9) (Imidodipeptidase) (Peptidase D) (Proline dipeptidase) (Prolidase)	Homo sapiens (Human)					x						x	CP	68055113	-1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P12956	XRCC6 G22P1	ATP-dependent DNA helicase 2 subunit 1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13010	XRCC5 G22P2	ATP-dependent DNA helicase 2 subunit 2	Homo sapiens (Human)						x					x	CP	68055113	-2,00	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13473	LAMP2	Lysosome-associated membrane glycoprotein 2 (LAMP-2) (Lysosome-associated membrane protein 2) (CD107 antigen-like family member B) (CD antigen CD107b)	Homo sapiens (Human)					x						x	CP	68055113	1,53	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P13489	RNH1 PRI RNH	Ribonuclease inhibitor (Placental ribonuclease inhibitor) (Placental RNase inhibitor) (Ribonuclease/angiogenin inhibitor 1) (RAI)	Homo sapiens (Human)						x					x	CP	68055113	2,09	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13489	RNH1 PRI RNH	Ribonuclease inhibitor (Placental ribonuclease inhibitor) (Placental RNase inhibitor) (Ribonuclease/angiogenin inhibitor 1) (RAI)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13500	CCL2 MCP1 SCYA2	C-C motif chemokine 2	Homo sapiens (Human)					x						x	CP	68055113	1,80						Non-proteomics		x	23375122
P13500	CCL2 MCP1 SCYA2	C-C motif chemokine 2	Homo sapiens (Human)						x					x	CP	68055113	2,00						Non-proteomics		x	23375122
P13639	EEF2 EF2	Elongation factor 2	Homo sapiens (Human)						x					x	CP	68055113	-1,59	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13639	EEF2 EF2	Elongation factor 2 (EF-2)	Homo sapiens (Human)						x					x	CP	68055113	3,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13639	EEF2 EF2	Elongation factor 2 (EF-2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P13639	EEF2 EF2	Elongation factor 2 (EF-2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13645	KRT10 KPP	Keratin, type I cytoskeletal 10	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P13645	KRT10 KPP	Keratin, type I cytoskeletal 10	Homo sapiens (Human)						x					x	CP	68055113	-1,27	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P13645	KRT10 KPP	Keratin, type I cytoskeletal 10 (Cytokeratin-10) (CK-10) (Keratin-10) (K10)	Homo sapiens (Human)						x					x	CP	68055113	2,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13645	KRT10 KPP	Keratin, type I cytoskeletal 10 (Cytokeratin-10) (CK-10) (Keratin-10) (K10)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P13645	KRT10 KPP	Keratin, type I cytoskeletal 10 (Cytokeratin-10) (CK-10) (Keratin-10) (K10)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13646	KRT13	Keratin, type I cytoskeletal 13	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P13646	KRT13	Keratin, type I cytoskeletal 13	Homo sapiens (Human)						x					x	CP	68055113	1,42	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13646	KRT13	Keratin, type I cytoskeletal 13 (Cytokeratin-13) (CK-13) (Keratin-13) (K13)	Homo sapiens (Human)						x					x	CP	68055113	3,45	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13646	KRT13	Keratin, type I cytoskeletal 13 (Cytokeratin-13) (CK-13) (Keratin-13) (K13)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P13646	KRT13	Keratin, type I cytoskeletal 13 (Cytokeratin-13) (CK-13) (Keratin-13) (K13)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13647	KRT5	Keratin, type II cytoskeletal 5	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P13647	KRT5	Keratin, type II cytoskeletal 5	Homo sapiens (Human)						x					x	CP	68055113	1,09	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13647	KRT5	Keratin, type II cytoskeletal 5 (58 kDa cytokeratin) (Cytokeratin-5) (CK-5) (Keratin-5) (K5) (Type-II keratin Kb5)	Homo sapiens (Human)						x					x	CP	68055113	3,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13647	KRT5	Keratin, type II cytoskeletal 5 (58 kDa cytokeratin) (Cytokeratin-5) (CK-5) (Keratin-5) (K5) (Type-II keratin Kb5)	Homo sapiens (Human)						x					x	CP	68055113	12,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P13647	KRT5	Keratin, type II cytoskeletal 5 (58 kDa cytokeratin) (Cytokeratin-5) (CK-5) (Keratin-5) (K5) (Type-II keratin Kb5)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13674	P4HA1 P4HA	Prolyl 4-hydroxylase subunit alpha-1 (4-PH alpha-1) (EC 1.14.11.2) (Procollagen-proline,2-oxoglutarate-4-dioxygenase subunit alpha-1)	Homo sapiens (Human)						x					x	CP	68055113	9,98	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13725	OSM		Homo sapiens (Human)						x					x	CP	68055113	726,00	25-50	MF		60 subjects (age 25-50 years) were enrolled into three groups (n=20 per group), group I (healthy), group II (G) and group III (CP). Group III subjects were followed for 6-8 weeks after the initial periodontal therapy (SRP) as the group IV (after periodontal therapy).	Clinical parameters were assessed as gingival index (GI), probing depth (PD), clinical attachment level (CAL), and radiographic evidence of bone loss. GCF and serum levels of OSM were measured by using Enzyme Linked Immunosorbent Assay (ELISA).	on-Proteomics		x	21404969
P13796	LCP1 PLS2	L-Plastin	Homo sapiens						x					x	CP	68055113	3,07	33-62	MF		After being selected for the study, the subjects were recalled for gingival crevicular fluid sampling with periopaper collection strips (OraFlow Inc., Smithtown, NY, USA). In the generalized AP (n = 20) and CP (n = 21) groups, gingival crevicular fluid samples were collected from the mesiobuccal aspect of a single-rooted tooth with ≥ 6 mm probing depth. In the healthy group (n = 20), gingival crevicular fluid samples were collected from the mesiobuccal aspect of singlerooted teeth exhibiting probing pocket depth up to 3 mm without BOP.	L-plastin levels in gingival crevicular fluid, saliva and serum were measured using ELISA. Statistical analysis was performed using nonparametric methods.	on-proteomics			25040533
P13796	LCP1 PLS2	Plastin-2	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P13796	LCP1 PLS2	Plastin-2	Homo sapiens (Human)						x					x	CP	68055113	1,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P13796	LCP1 PLS2	Plastin-2 (L-plastin) (LC64P) (Lymphocyte cytosolic protein 1) (LCP-1)	Homo sapiens (Human)					x						x	CP	68055113	2,18	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P13796	LCP1 PLS2	Plastin-2 (L-plastin) (LC64P) (Lymphocyte cytosolic protein 1) (LCP-1)	Homo sapiens (Human)						x					x	CP	68055113	2,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at −80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P13796	LCP1 PLS2	Plastin-2 (L-plastin) (LC64P) (Lymphocyte cytosolic protein 1) (LCP-1)	Homo sapiens (Human)						x					x	CP	68055113	13,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P13796	LCP1 PLS2	Plastin-2 (L-plastin) (LC64P) (Lymphocyte cytosolic protein 1) (LCP-1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P13798	APEH D3F15S2 D3S48E DNF15S2	L-Plastin	Homo sapiens					x						x	CP	68055113	1,55	33-62	MF		Briefly, the subjects were asked to rinse their mouth with tap water, following which they expectorated whole saliva into sterile 50-mL tubes for 5 min. All saliva samples were placed on ice and supplemented with EDTA-free Protease Inhibitor Cocktail (Roche Applied Science), before centrifuging at 10,000 g for 15 min at 4°C (24). The resulting supernatants were immediately aliquoted and frozen at 80°C until required for further analysis.	L-plastin levels in gingival crevicular fluid, saliva and serum were measured using ELISA. Statistical analysis was performed using nonparametric methods.	on-proteomics			25040533
P13987	CD59 MIC11 MIN1 MIN2 MIN3 MSK21	CD59 glycoprotein (1F5 antigen) (20 kDa homologous restriction factor) (HRF-20) (HRF20) (MAC-inhibitory protein) (MAC-IP) (MEM43 antigen) (Membrane attack complex inhibition factor) (MACIF) (Membrane inhibitor of reactive lysis) (MIRL) (Protectin) (CD antigen CD59)	Homo sapiens (Human)					x						x	CP	68055113	1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P13987	CD59 MIC11 MIN1 MIN2 MIN3 MSK21	CD59 glycoprotein (1F5 antigen) (20 kDa homologous restriction factor) (HRF-20) (HRF20) (MAC-inhibitory protein) (MAC-IP) (MEM43 antigen) (Membrane attack complex inhibition factor) (MACIF) (Membrane inhibitor of reactive lysis) (MIRL) (Protectin) (CD antigen CD59)	Homo sapiens (Human)						x					x	CP	68055113	3,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at −80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P14174	MIF GLIF MMIF	Macrophage migration inhibitory factor (MIF) (EC 5.3.2.1) (Glycosylation-inhibiting factor) (GIF) (L-dopachrome isomerase) (L-dopachrome tautomerase) (EC 5.3.3.12) (Phenylpyruvate tautomerase)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P14210	HGF HPTA	Hepatocyte growth factor	Homo sapiens (Human)					x						x	CP	68055113	1,27	30-40	MF	non-smokers	Approximately 2mL of unstimulatedwhole saliva was collected in a sterile container using the spit-outmethod.The saliva was collected5minutes after rinsing the mouth thoroughly with distilled water. The saliva samples were kept on ice for an hour.The supernatant (middle 1/3) was collected by centrifugation at 3800 rpm for 10min and stored at −70◦C until analysis.	ELISA kit (human HGF immunoassay, Quantikine; R and D Systems Inc., Minneapolis, MN).	on-proteomics		x	25389376
P14210	HGF HPTA	Hepatocyte growth factor	Homo sapiens (Human)					x						x	CP	68055113	1,57	30-40	MF	smokers	Approximately 2mL of unstimulatedwhole saliva was collected in a sterile container using the spit-outmethod.The saliva was collected5minutes after rinsing the mouth thoroughly with distilled water. The saliva samples were kept on ice for an hour.The supernatant (middle 1/3) was collected by centrifugation at 3800 rpm for 10min and stored at −70◦C until analysis.	ELISA kit (human HGF immunoassay, Quantikine; R and D Systems Inc., Minneapolis, MN).	on-proteomics		x	25389376
P14210	HGF HPTA	Hepatocyte growth factor	Homo sapiens (Human)						x					x	CP	68055113	1,52	30-40	MF	non-smokers	For GCF sampling, teeth numbers 3, 9, 19, and 25 were chosen for both the healthy and periodontitis groups. If one of the participants was missing one of these teeth, then the nearest tooth was used for sampling. Prior to GCF sampling, supraigingival plaque was removed from the interproximal surfaces using a sterile curette, and the tooth was gently dried using an air syringe. The area was carefully isolated to prevent samples from being contaminated by saliva. Care was taken to avoid mechanical injury of the gingival tissues.TheGCF samples were collected by placing a microcapillary pipette at the entrance of the gingival sulcus, gently touching the gingival margin	ELISA kit (human HGF immunoassay, Quantikine; R and D Systems Inc., Minneapolis, MN).	on-proteomics		x	25389376
P14210	HGF HPTA	Hepatocyte growth factor	Homo sapiens (Human)						x					x	CP	68055113	1,81	30-40	MF	smokers	For GCF sampling, teeth numbers 3, 9, 19, and 25 were chosen for both the healthy and periodontitis groups. If one of the participants was missing one of these teeth, then the nearest tooth was used for sampling. Prior to GCF sampling, supraigingival plaque was removed from the interproximal surfaces using a sterile curette, and the tooth was gently dried using an air syringe. The area was carefully isolated to prevent samples from being contaminated by saliva. Care was taken to avoid mechanical injury of the gingival tissues.TheGCF samples were collected by placing a microcapillary pipette at the entrance of the gingival sulcus, gently touching the gingival margin	ELISA kit (human HGF immunoassay, Quantikine; R and D Systems Inc., Minneapolis, MN).	on-proteomics		x	25389376
P14314	PRKCSH G19P1	Glucosidase 2 subunit beta (80K-H protein) (Glucosidase II subunit beta) (Protein kinase C substrate 60.1 kDa protein heavy chain) (PKCSH)	Homo sapiens (Human)					x						x	CP	68055113	1,18	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P14550	AKR1A1 ALDR1 ALR	Alcohol dehydrogenase [NADP(+)] (EC 1.1.1.2) (Aldehyde reductase) (Aldo-keto reductase family 1 member A1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P14618	PKM OIP3 PK2 PK3 PKM2	Pyruvate kinase isozymes M1/M2	Homo sapiens (Human)						x					x	CP	68055113	3,15	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P14618	PKM OIP3 PK2 PK3 PKM2	Pyruvate kinase isozymes M1/M2 (EC 2.7.1.40) (Cytosolic thyroid hormone-binding protein) (CTHBP) (Opa-interacting protein 3) (OIP-3) (Pyruvate kinase 2/3) (Pyruvate kinase muscle isozyme) (Thyroid hormone-binding protein 1) (THBP1) (Tumor M2-PK) (p58)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P14618	PKM OIP3 PK2 PK3 PKM2	Pyruvate kinase isozymes M1/M2 (EC 2.7.1.40) (Cytosolic thyroid hormone-binding protein) (CTHBP) (Opa-interacting protein 3) (OIP-3) (Pyruvate kinase 2/3) (Pyruvate kinase muscle isozyme) (Thyroid hormone-binding protein 1) (THBP1) (Tumor M2-PK) (p58)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P14625	HSP90B1 GRP94 TRA1	Endoplasmin (94 kDa glucose-regulated protein) (GRP-94) (Heat shock protein 90 kDa beta member 1) (Tumor rejection antigen 1) (gp96 homolog)	Homo sapiens (Human)						x					x	CP	68055113	3,30	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P14780	MMP9 CLG4B	Matrix metalloproteinase-9 (MMP-9) (EC 3.4.24.35) (92 kDa gelatinase) (92 kDa type IV collagenase) (Gelatinase B) (GELB) [Cleaved into: 67 kDa matrix metalloproteinase-9; 82 kDa matrix metalloproteinase-9]	Homo sapiens (Human)					x						x	CP	68055113	1,52	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics		x	23790309
P14780	MMP9 CLG4B	Matrix metalloproteinase-9 (MMP-9) (EC 3.4.24.35) (92 kDa gelatinase) (92 kDa type IV collagenase) (Gelatinase B) (GELB) [Cleaved into: 67 kDa matrix metalloproteinase-9; 82 kDa matrix metalloproteinase-9]	Homo sapiens (Human)						x					x	CP	68055113	2,41	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P14780	MMP9 CLG4B	Matrix metalloproteinase-9 (MMP-9) (EC 3.4.24.35) (92 kDa gelatinase) (92 kDa type IV collagenase) (Gelatinase B) (GELB) [Cleaved into: 67 kDa matrix metalloproteinase-9; 82 kDa matrix metalloproteinase-9]	Homo sapiens (Human)						x					x	CP	68055113	6,70					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P14780	MMP9 CLG4B	Matrix metalloproteinase-9 (MMP-9) (EC 3.4.24.35) (92 kDa gelatinase) (92 kDa type IV collagenase) (Gelatinase B) (GELB) [Cleaved into: 67 kDa matrix metalloproteinase-9; 82 kDa matrix metalloproteinase-9]	Homo sapiens (Human)						x					x	CP	68055113			M/F	All individuals possessed at least 20 teeth and had not received periodontal treatment or antibiotic therapy for medical or dental reasons for 3 mos prior to the investigation. Individuals were excluded if they possessed a history of metabolic bone diseases, autoimmune diseases, unstable diabetes, or post-menopausal osteoporosis. Pregnant or lactating women were excluded from participating in the study.	Unstimulated whole saliva was collected at each study visit via passive drooling into sterile plastic tubes from all participants (Mandel and Wotman, 1976). Samples were placed on ice, supplemented with a proteinase inhibitor combination of 1% aprotinin and 0.5% phenylmethylsulphonylfluoride, and aliquotted prior to storage at -80°C.	Protein biomarker levels were determined by colorimetric-based enzyme-linked immunosorbent assays (ELISAs), fluorescence-based protein microarrays, and radioimmunoassay (RIA), run according to manufacturer protocols. ELISAs (R&D Systems Inc., Minneapolis, MN, USA) were used for measurement of MMP-8 and -9, calprotectin, and osteoprotegerin (OPG). Detection of the cytokines interleukin (IL)-β, IL-2, IL-4, IL-5, IL-6, IL-10, and IL-13, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ was accomplished with a protein microarray (Whattman Inc., Florham Park, NJ, USA).	on-Proteomics			21406610
P14780	MMP9 CLG4B	Matrix metalloproteinase-9 (MMP-9) (EC 3.4.24.35) (92 kDa gelatinase) (92 kDa type IV collagenase) (Gelatinase B) (GELB) [Cleaved into: 67 kDa matrix metalloproteinase-9; 82 kDa matrix metalloproteinase-9]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P14780	MMP9 CLG4B	MMP9	Homo sapiens (Human)						x					x	CP	68055113	1,11	42-50	MF		GCF was taken from the mesiobuccal aspect of each site (tooth) for up to 28 teeth per patient. Prior to the collection, supragingival plaque was removed using a sterile instrument. The site was isolated using cotton rolls and dried using a short blast of air directly through the contact (not into the sulcus/pocket). A methylcellulose strip (Pro Flow, Inc., Amityville, NY) was inserted into the sulcus/pocket until light resistance was felt. The strip stayed in position for 30 seconds.	ELISA	on-proteomics			24303954
P14923	JUP CTNNG DP3	Junction plakoglobin (Catenin gamma) (Desmoplakin III) (Desmoplakin-3)	Homo sapiens (Human)						x					x	CP	68055113	3,63	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P14923	JUP CTNNG DP3	Junction plakoglobin (Catenin gamma) (Desmoplakin III) (Desmoplakin-3)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P15104	GLUL GLNS	Glutamine synthetase (GS) (EC 6.3.1.2) (Glutamate decarboxylase) (EC 4.1.1.15) (Glutamate--ammonia ligase)	Homo sapiens (Human)						x					x	CP	68055113	2,43	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P15104	GLUL GLNS	Glutamine synthetase (GS) (EC 6.3.1.2) (Glutamate decarboxylase) (EC 4.1.1.15) (Glutamate--ammonia ligase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P15104	GLUL GLNS	Glutamine synthetase (GS) (EC 6.3.1.2) (Glutamate decarboxylase) (EC 4.1.1.15) (Glutamate--ammonia ligase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P15153	RAC2	Ras-related C3 botulinum toxin substrate 2 (GX) (Small G protein) (p21-Rac2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P15311	EZR VIL2	Ezrin (Cytovillin) (Villin-2) (p81)	Homo sapiens (Human)					x						x	CP	68055113	1,12	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P15311	EZR VIL2	Ezrin (Cytovillin) (Villin-2) (p81)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P15328	FOLR1 FOLR	Folate receptor alpha (FR-alpha) (Adult folate-binding protein) (FBP) (Folate receptor 1) (Folate receptor, adult) (KB cells FBP) (Ovarian tumor-associated antigen MOv18)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P15515	HTN1 HIS1	Histatin-1 (Histidine-rich protein 1) (Post-PB protein) (PPB) [Cleaved into: His1-(31-57)-peptide (His1 31/57) (His1-(12-38)-peptide) (His1 12/38) (Histatin-2)]	Homo sapiens (Human)					x						x	CP	68055113	-2,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P15516	HTN3 HIS2	Histatin-3	Homo sapiens (Human)					x						x	CP	68055113	1,09	20-45	MF	Subject exclusion criteria: 1. Patients with any systemic disorder like diabetes mellitus, hypertension etc. 2. Patients with a known disorder of the salivary gland. 3. Presence of any disorder that might alter the immune system. 4. Patients on any medication that might alter the salivary flow rate or composition. E.g. Beta blockers. 5. Patients who had been administered antibiotics in the past 6 months. 6. Patients consuming tobacco in any form were excluded from the study as the relation of tobacco and histatin levels is not ascertained till date.	Participants were asked to refrain from drinking or eating half an hour before collection of saliva sample. Both test and control subjects reported to the hospital between 10.00 am and 2.00 pm. One millilitre (ml) of unstimulated pooled whole saliva was collected by asking the patient to spit in a sterile container. The patients were told to avoid spitting forcefully in order to avoiding any possible contamination. The saliva samples were centrifuged at 3000 × g for 20 minutes, and the clear supernatant was stored at – 70°C until ssay was performed.	ELISA	on-proteomics			24455076
P15516	HTN3 HIS2	Histatin-3	Homo sapiens (Human)						x					x	CP	68055113	1,04	20-45	MF	Subject exclusion criteria: 1. Patients with any systemic disorder like diabetes mellitus, hypertension etc. 2. Patients with a known disorder of the salivary gland. 3. Presence of any disorder that might alter the immune system. 4. Patients on any medication that might alter the salivary flow rate or composition. E.g. Beta blockers. 5. Patients who had been administered antibiotics in the past 6 months. 6. Patients consuming tobacco in any form were excluded from the study as the relation of tobacco and histatin levels is not ascertained till date.	Multiple test sites were dried and isolated with cotton rolls to prevent any contamination from saliva and blood. Prior to GCF sampling, supragingival calculus was removed using sterile curette. A standard volume of 2 µl was collected extracrevicularly using a calibrated, volumetric, microcapillary pipette measuring 1 to5 µl with a plunger for 5 to 30 minutes.	ELISA	on-proteomics			24455076
P15924	DSP	Desmoplakin (DP) (250/210 kDa paraneoplastic pemphigus antigen)	Homo sapiens (Human)						x					x	CP	68055113	5,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P15924	DSP	Desmoplakin (DP) (250/210 kDa paraneoplastic pemphigus antigen)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P16050	ALOX15 LOG15	Arachidonate 15-lipoxygenase	Homo sapiens (Human)						x					x	CP	68055113	-1,06	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P16104	H2AFX H2AX	Histone H2A.x	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P16104	H2AFX H2AX	Histone H2AX (H2a/x) (Histone H2A.X)	Homo sapiens (Human)						x					x	CP	68055113	3,22	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P16152	CBR1 CBR CRN SDR21C1	Carbonyl reductase [NADPH] 1 (EC 1.1.1.184) (15-hydroxyprostaglandin dehydrogenase [NADP(+)]) (EC 1.1.1.197) (NADPH-dependent carbonyl reductase 1) (Prostaglandin 9-ketoreductase) (Prostaglandin-E(2) 9-reductase) (EC 1.1.1.189)	Homo sapiens (Human)						x					x	CP	68055113	-6,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P16152	CBR1 CBR CRN SDR21C1	Carbonyl reductase [NADPH] 1 (EC 1.1.1.184) (15-hydroxyprostaglandin dehydrogenase [NADP(+)]) (EC 1.1.1.197) (NADPH-dependent carbonyl reductase 1) (Prostaglandin 9-ketoreductase) (Prostaglandin-E(2) 9-reductase) (EC 1.1.1.189)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P16284	PECAM1	Platelet/endothelial cell adhesion molecule 1	Homo sapiens (Human)							x					CP	68055113	31(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P16333	NCK1 NCK	Cytoplasmic protein NCK1	Homo sapiens (Human)						x					x	CP	68055113	2,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P16401	HIST1H1B H1F5	Histone H1.5 (Histone H1a) (Histone H1b) (Histone H1s-3)	Homo sapiens (Human)						x					x	CP	68055113	2,56	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P16402	HIST1H1D H1F3	Histone H1.3	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P16402	HIST1H1D H1F3	Histone H1.3 (Histone H1c) (Histone H1s-2)	Homo sapiens (Human)						x					x	CP	68055113	2,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P16403	HIST1H1C H1F2	Histone H1.2	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P16403	HIST1H1C H1F2	Histone H1.2 (Histone H1c) (Histone H1d) (Histone H1s-1)	Homo sapiens (Human)						x					x	CP	68055113	1,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P17096	HMGA1 HMGIY	High mobility group protein HMG-I/HMG-Y (HMG-I(Y)) (High mobility group AT-hook protein 1) (High mobility group protein A1) (High mobility group protein R)	Homo sapiens (Human)						x					x	CP	68055113	4,97	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P17174	GOT1	Aspartate aminotransferase	Homo sapiens (Human)							x				x	CP	68055113	5,77	40-65	MF		Participants were asked to come to the laboratory at 07:00 am following an overnight fast, during which they were instructed not to eat, drink (except water), chew gum or brush teeth. Whole saliva samples were obtained by expectorating into polypropylene tubes. Patients were advised to rinse his or her mouth several times with water and then to relax for five minutes. Patients were asked to swallow to void the mouth of saliva and asked to lean their head forward over the polypropylene tube and funnel. Patients kept their mouth slightly open to allow saliva to drain into the tube.	ELISA: AST and ALT levels were analyzed on Roche p-800 modular system using the specific kits provided by the manufacturer.	on-proteomics			25345339
P17174	GOT1	Aspartate aminotransferase, cytoplasmic (cAspAT) (EC 2.6.1.1) (EC 2.6.1.3) (Cysteine aminotransferase, cytoplasmic) (Cysteine transaminase, cytoplasmic) (cCAT) (Glutamate oxaloacetate transaminase 1) (Transaminase A)	Homo sapiens (Human)							x				x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P17213	BPI	Bactericidal permeability-increasing protein (BPI) (CAP 57)	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P17643	TYRP1 CAS2 TYRP TYRRP	Tyrosinase-related protein 1	Homo sapiens (Human)							x					CP	68055113	46(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P17661	DES	Desmin	Homo sapiens (Human)						x						CP	68055113	9,00						Proteomics			24098404
P17752	TPH1 TPH TPRH TRPH	Tryptophan 5-hydroxylase 1 (EC 1.14.16.4) (Tryptophan 5-monooxygenase 1)	Homo sapiens (Human)						x					x	CP	68055113	4,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P17931	LGALS3 MAC2	Galectin-3 (Gal-3) (35 kDa lectin) (Carbohydrate-binding protein 35) (CBP 35) (Galactose-specific lectin 3) (Galactoside-binding protein) (GALBP) (IgE-binding protein) (L-31) (Laminin-binding protein) (Lectin L-29) (Mac-2 antigen)	Homo sapiens (Human)					x						x	CP	68055113	1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P17931	LGALS3 MAC2	Galectin-3 (Gal-3) (35 kDa lectin) (Carbohydrate-binding protein 35) (CBP 35) (Galactose-specific lectin 3) (Galactoside-binding protein) (GALBP) (IgE-binding protein) (L-31) (Laminin-binding protein) (Lectin L-29) (Mac-2 antigen)	Homo sapiens (Human)						x					x	CP	68055113	2,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zypperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P18065	IGFBP2 BP2 IBP2	Insulin-like growth factor-binding protein 2 (IBP-2) (IGF-binding protein 2) (IGFBP-2)	Homo sapiens (Human)					x						x	CP	68055113	-1,47	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P18136	0	Ig kappa chain V-III region HIC	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P18206	VCL	Vinculin (Metavinculin) (MV)	Homo sapiens (Human)						x					x	CP	68055113	3,12	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zypperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P18206	VCL	Vinculin (Metavinculin) (MV)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P18510	IL1RN IL1F3 IL1RA	Interleukin-1 receptor antagonist protein (IL-1RN) (IL-1ra) (IRAP) (ICIL-1RA) (IL1 inhibitor) (Anakinra)	Homo sapiens (Human)					x						x	CP	68055113	-1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P18510	IL1RN IL1F3 IL1RA	Interleukin-1 receptor antagonist protein (IL-1RN) (IL-1ra) (IRAP) (ICIL-1RA) (IL1 inhibitor) (Anakinra)	Homo sapiens (Human)						x					x	CP	68055113	2,36	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zypperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P18510	IL1RN IL1F3 IL1RA	Interleukin-1 receptor antagonist protein (IL-1RN) (IL-1ra) (IRAP) (ICIL-1RA) (IL1 inhibitor) (Anakinra)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P18669	PGAM1 PGAMA CDABP0006	Phosphoglycerate mutase 1 (EC 3.1.3.13) (EC 5.4.2.1) (EC 5.4.2.4) (BPG-dependent PGAM 1) (Phosphoglycerate mutase isozyme B) (PGAM-B)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P18669	PGAM1 PGAMA CDABP0006	Phosphoglycerate mutase 1 (EC 3.1.3.13) (EC 5.4.2.1) (EC 5.4.2.4) (BPG-dependent PGAM 1) (Phosphoglycerate mutase isozyme B) (PGAM-B)	Homo sapiens (Human)					x						x	CP	68055113	1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P19012	KRT15 KRTB	Keratin, type I cytoskeletal 15	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P19013	KRT4 CYK4	Keratin, type II cytoskeletal 4	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P19013	KRT4 CYK4	Keratin, type II cytoskeletal 4	Homo sapiens (Human)						x					x	CP	68055113	1,01	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P19013	KRT4 CYK4	Keratin, type II cytoskeletal 4 (Cytokeratin-4) (CK-4) (Keratin-4) (K4) (Type-II keratin Kb4)	Homo sapiens (Human)						x					x	CP	68055113	3,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zypperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P19013	KRT4 CYK4	Keratin, type II cytoskeletal 4 (Cytokeratin-4) (CK-4) (Keratin-4) (K4) (Type-II keratin Kb4)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P19021	PAM	Peptidyl-glycine alpha-amidating monooxygenase (PAM) [Includes: Peptidylglycine alpha-hydroxylating monooxygenase (PHM) (EC 1.14.17.3); Peptidyl-alpha-hydroxyglycine alpha-amidating lyase (EC 4.3.2.5) (Peptidylamidoglycolate lyase) (PAL)]	Homo sapiens (Human)					x						x	CP	68055113	-1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P19823	ITIH2 IGHEP2	Inter-alpha-trypsin inhibitor heavy chain H2 (ITI heavy chain H2) (ITI-HC2) (Inter-alpha-inhibitor heavy chain 2) (Inter-alpha-trypsin inhibitor complex component II) (Serum-derived hyaluronan-associated protein) (SHAP)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P19823	ITIH2 IGHEP2	Inter-alpha-trypsin inhibitor heavy chain H2 (ITI heavy chain H2) (ITI-HC2) (Inter-alpha-inhibitor heavy chain 2) (Inter-alpha-trypsin inhibitor complex component II) (Serum-derived hyaluronan-associated protein) (SHAP)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P19827	ITIH1 IGHEP1	Inter-alpha-trypsin inhibitor heavy chain H1 (ITI heavy chain H1) (ITI-HC1) (Inter-alpha-inhibitor heavy chain 1) (Inter-alpha-trypsin inhibitor complex component III) (Serum-derived hyaluronan-associated protein) (SHAP)	Homo sapiens (Human)						x					x	CP	68055113						isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P19827	ITIH1 IGHEP1	Inter-alpha-trypsin inhibitor heavy chain H1 (ITI heavy chain H1) (ITI-HC1) (Inter-alpha-inhibitor heavy chain 1) (Inter-alpha-trypsin inhibitor complex component III) (Serum-derived hyaluronan-associated protein) (SHAP)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P19878	NCF2 NOXA2 P67PHOX	Neutrophil cytosol factor 2 (NCF-2) (67 kDa neutrophil oxidase factor) (NADPH oxidase activator 2) (Neutrophil NADPH oxidase factor 2) (p67-phox)	Homo sapiens (Human)						x					x	CP	68055113	2,58	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P19961	AMY2B	Alpha-amylase 2B (EC 3.2.1.1) (1,4-alpha-D-glucan glucanohydrolase 2B) (Carcinoid alpha-amylase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P19971	TYMP ECGF1	Thymidine phosphorylase (TP) (EC 2.4.2.4) (Gliostatin) (Platelet-derived endothelial cell growth factor) (PD-ECGF) (TdRPase)	Homo sapiens (Human)						x					x	CP	68055113	-3,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P19971	TYMP ECGF1	Thymidine phosphorylase (TP) (EC 2.4.2.4) (Gliostatin) (Platelet-derived endothelial cell growth factor) (PD-ECGF) (TdRPase)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics		x	22092770
P19971	TYMP ECGF1	Thymidine phosphorylase (TP) (EC 2.4.2.4) (Gliostatin) (Platelet-derived endothelial cell growth factor) (PD-ECGF) (TdRPase)	Homo sapiens (Human)						x					x	CP	68055113		30-73	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
P20061	TCN1 TC1	Transcobalamin-1 (TC-1) (Transcobalamin I) (TC I) (TCI)	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P20061	TCN1 TC1	Transcobalamin-1 (TC-1) (Transcobalamin I) (TC I) (TCI)	Homo sapiens (Human)						x					x	CP	68055113	2,98	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P20160	AZU1	Azurocidin (Cationic antimicrobial protein CAP37) (Heparin-binding protein) (HBP)	Homo sapiens (Human)						x					x	CP	68055113	5,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P20160	AZU1	Azurocidin (Cationic antimicrobial protein CAP37) (Heparin-binding protein) (HBP)	Homo sapiens (Human)						x					x	CP	68055113	7,30				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics		x	21794177
P20671	HIST1H2AD H2AFG	Histone H2A type 1-D	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P20815	CYP3A5	Cytochrome P450, family 3, subfamily A, polypeptide 5	Homo sapiens (Human)							x					CP	68055113	,47(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P20848	SERPINA2 ARGS ATR PIL SERPINA2P	Putative alpha-1-antitrypsin-related protein (Serpin A2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P20929	NEB	Nebulin	Homo sapiens (Human)						x					x	CP	68055113	-1,30	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P20930	FLG	Filaggrin	Homo sapiens (Human)							x					CP	68055113	,36(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P21128	ENDOU	Poly(U)-specific endoribonuclease (EC 3.1.-.-) (Placental protein 11) (PP11) (Protein endoU) (Uridylate-specific endoribonuclease)	Homo sapiens (Human)					x						x	CP	68055113	1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P21333	FLNA FLN FLN1	Filamin-A	Homo sapiens (Human)						x					x	CP	68055113	-1,16	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P21333	FLNA FLN FLN1	Filamin-A (FLN-A) (Actin-binding protein 280) (ABP-280) (Alpha-filamin) (Endothelial actin-binding protein) (Filamin-1) (Non-muscle filamin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P21333	FLNA FLN FLN1	Filamin-A (FLN-A) (Actin-binding protein 280) (ABP-280) (Alpha-filamin) (Endothelial actin-binding protein) (Filamin-1) (Non-muscle filamin)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P21439	ABCB4 MDR3 PGY3	Multidrug resistance protein 3	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P21817	RYR1 RYDR	Ryanodine receptor 1	Homo sapiens (Human)						x					x	CP	68055113	-1,59	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P21817	RYR1 RYDR	Ryanodine receptor 1 (RYR-1) (RyR1) (Skeletal muscle calcium release channel) (Skeletal muscle ryanodine receptor) (Skeletal muscle-type ryanodine receptor) (Type 1 ryanodine receptor)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P22079	LPO SAPX	Lactoperoxidase (LPO) (EC 1.11.1.7) (Salivary peroxidase) (SPO)	Homo sapiens (Human)					x						x	CP	68055113	-1,60	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P22079	LPO SAPX	Lactoperoxidase (LPO) (EC 1.11.1.7) (Salivary peroxidase) (SPO)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P22314	UBA1 A1S9T UBE1	Ubiquitin-like modifier-activating enzyme 1 (Protein A1S9) (Ubiquitin-activating enzyme E1)	Homo sapiens (Human)						x					x	CP	68055113	3,09	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four presected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P22314	UBA1 A1S9T UBE1	Ubiquitin-like modifier-activating enzyme 1 (Protein A1S9) (Ubiquitin-activating enzyme E1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P22392	NME2 NM23B	Nucleoside diphosphate kinase B (NDK B) (NDP kinase B) (EC 2.7.4.6) (C-myc purine-binding transcription factor PUF) (Histidine protein kinase NDKB) (EC 2.7.13.3) (nm23-H2)	Homo sapiens (Human)						x					x	CP	68055113	-3,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P22392	NME2 NM23B	Nucleoside diphosphate kinase B (NDK B) (NDP kinase B) (EC 2.7.4.6) (C-myc purine-binding transcription factor PUF) (Histidine protein kinase NDKB) (EC 2.7.13.3) (nm23-H2)	Homo sapiens (Human)					x						x	CP	68055113	1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P22392	NME2 NM23B	Nucleoside diphosphate kinase B (NDK B) (NDP kinase B) (EC 2.7.4.6) (C-myc purine-binding transcription factor PUF) (Histidine protein kinase NDKB) (EC 2.7.13.3) (nm23-H2)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P22528	SPRR1B	Cornifin-B (14.9 kDa pancornulin) (Small proline-rich protein IB) (SPR-IB)	Homo sapiens (Human)						x					x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P22528	SPRR1B	Cornifin-B (14.9 kDa pancornulin) (Small proline-rich protein IB) (SPR-IB)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P22531	SPRR2E	Small proline-rich protein 2E (SPR-2E) (Small proline-rich protein II) (SPR-II)	Homo sapiens (Human)						x						CP	68055113	+					Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P22626	HNRNPA2B1 HNRPA2B1	Heterogeneous nuclear ribonucleoproteins A2/B1 (hnRNP A2/B1)	Homo sapiens (Human)					x						x	CP	68055113	-1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P22894	MMP8 CLG1	MMP8	Homo sapiens (Human)					x						x	CP	68055113	3,6	39-51	MF		for UWS collection, all individuals (N = 88) were comfortably seated on a chair and requested to expectorate (without swallowing) into a gauged measuring cylinder for 5 continuous minutes. UWS flow rate (UWSFR) was measured and recorded in milliliters per minute. Immediately after collection, UWS samples were placed on ice and aliquoted before freezing at -80 C.	ELISA	on-proteomics			24171502
P22894	MMP8 CLG1	MMP8	Homo sapiens (Human)						x					x	CP	68055113	1,17	42-50	MF		GCF was taken from the mesiobuccal aspect of each site (tooth) for up to 28 teeth per patient. Prior to the collection, supragingival plaque was removed using a sterile instrument. The site was isolated using cotton rols and dried using a short blast of air directly through the contact (not into the sulcus/pocket). A methylcellulose strip (Pro Flow, Inc., Amityville, NY) was inserted into the sulcus/pocket until light resistance was felt. The strip stayed in position for 30 seconds.	ELISA	on-proteomics			24303954
P22894	MMP8 CLG1	MMP8	Homo sapiens (Human)					x						x	CP	68055113	18,15	62-75	MF		At the beginning of the oral examination, the subjects chewed a piece of paraffin for 5 min and at least 2 ml of stimulated whole saliva was collected. The samples were stored at −70 °C until analyses.	MMP-8 was measured with a time-resolved immunofluorometric assay (IFMA) as described in (Gursoy et al. 2010) with a detection limit of 0.08 ng/ml.	on-proteomics			24460823
P22894	MMP8 CLG1	MMP8	Homo sapiens (Human)					x						x	CP	68055113	1,82	30-55	MF		Around 3 ml of unstimulated and whole expectorated saliva was collected	ELISA	on-proteomics			25098434
P22894	MMP8 CLG1	Neutrophil collagenase (EC 3.4.24.34) (Matrix metalloproteinase-8) (MMP-8) (PMNL collagenase) (PMNL-CL)	Homo sapiens (Human)					x						x	CP	68055113	1,82	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P22894	MMP8 CLG1	Neutrophil collagenase (EC 3.4.24.34) (Matrix metalloproteinase-8) (MMP-8) (PMNL collagenase) (PMNL-CL)	Homo sapiens (Human)					x						x	CP	68055113			M/F	All individuals possessed at least 20 teeth and had not received periodontal treatment or antibiotic therapy for medical or dental reasons for 3 mos prior to the investigation. Individuals were excluded if they possessed a history of metabolic bone diseases, autoimmune diseases, unstable diabetes, or post-menopausal osteoporosis. Pregnant or lactating women were excluded from participating in the study.	Unstimulated whole saliva was collected at each study visit via passive drooling into sterile plastic tubes from all participants (Mandel and Wotman, 1976). Samples were placed on ice, supplemented with a proteinase inhibitor combination of 1% aprotinin and 0.5% phenylmethylsulphonylfluoride, and aliquotted prior to storage at -80°C.	Protein biomarker levels were determined by colorimetric-based enzyme-linked immunosorbent assays (ELISAs), fluorescence-based protein microarrays, and radioimmunoassay (RIA), run according to manufacturer protocols. ELISAs (R&D Systems Inc., Minneapolis, MN, USA) were used for measurement of MMP-8 and -9, calprotectin, and osteoprotegerin (OPG). Detection of the cytokines interleukin (IL)-β, IL-2, IL-4, IL-5, IL-6, IL-10, and IL-13, tumor necrosis factor (TNF)-α, and interferon (IFN)-γ was accomplished with a protein microarray (Whatman Inc., Florham Park, NJ, USA).	on-Proteomics			21406610

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P22894	MMP8 CLG1	Neutrophil collagenase (EC 3.4.24.34) (Matrix metalloproteinase-8) (MMP-8) (PMNL collagenase) (PMNL-CL)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P23083	0	Ig heavy chain V-I region V35	Homo sapiens (Human)					x						x	CP	68055113	1,71	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P23246	SFPQ PSF	Splicing factor, proline- and glutamine-rich (100 kDa DNA-pairing protein) (hPOMp100) (DNA-binding p52/p100 complex, 100 kDa subunit) (Polypyrimidine tract-binding protein-associated-splicing factor) (PSF) (PTB-associated-splicing factor)	Homo sapiens (Human)						x					x	CP	68055113	3,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P23280	CA6	Carbonic anhydrase 6 (EC 4.2.1.1) (Carbonate dehydratase VI) (Carbonic anhydrase VI) (CA-VI) (Salivary carbonic anhydrase) (Secreted carbonic anhydrase)	Homo sapiens (Human)					x						x	CP	68055113	-1,71	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P23280	CA6	Carbonic anhydrase 6 (EC 4.2.1.1) (Carbonate dehydratase VI) (Carbonic anhydrase VI) (CA-VI) (Salivary carbonic anhydrase) (Secreted carbonic anhydrase)	Homo sapiens (Human)						x					x	CP	68055113	^-1,4	40-60	M/F	Patients were excluded if they fit into any of the following categories: a history of systemic antibiotic usage over the previous 4 months; periodontal treatment within the past 6 months; were current smokers; pregnant, and had a cerebrovascular or cardiovascular event within the past 12 months.	Volunteers were advised to refrain from eating, drinking, smoking and performing any oral hygiene activity for at least 1 hour before saliva collection. During the saliva collection, volunteers were advised not to talk or move their tongue in the mouth. Saliva collection was usually done in the morning between 9 a.m. and 11 a.m. [24] by spitting into an ice-chilled tube which contained a complete protease inhibitor cocktail at a final concentration recommended by the manufacturer (Roche Ltd, Basel, Switzerland) [20]. Samples were mixed thoroughly before being kept in an ice box and transported to the laboratory for processing. The saliva samples were then centrifuged at 14,000 g for 20 minutes at 4 °C in order to remove any debris, cells and food particles present.	2-DE	Proteomics			22606001
P23280	CA6	Carbonic anhydrase 6 (EC 4.2.1.1) (Carbonate dehydratase VI) (Carbonic anhydrase VI) (CA-VI) (Salivary carbonic anhydrase) (Secreted carbonic anhydrase)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P23280	CA6	Carbonic anhydrase 6 (EC 4.2.1.1) (Carbonate dehydratase VI) (Carbonic anhydrase VI) (CA-VI) (Salivary carbonic anhydrase) (Secreted carbonic anhydrase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P23284	PPIB CYPB	Peptidyl-prolyl cis-trans isomerase B (PPIase B) (EC 5.2.1.8) (CYP-S1) (Cyclophilin B) (Rotamase B) (S-cyclophilin) (SCYLP)	Homo sapiens (Human)						x					x	CP	68055113	1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P23284	PPIB CYPB	Peptidyl-prolyl cis-trans isomerase B (PPIase B) (EC 5.2.1.8) (CYP-S1) (Cyclophilin B) (Rotamase B) (S-cyclophilin) (SCYLP)	Homo sapiens (Human)						x					x	CP	68055113	4,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P23527	HIST1H2BO H2BFH H2BFN	Histone H2B type 1-O	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P23528	CFL1 CFL	Cofilin-1	Homo sapiens (Human)						x						CP	68055113	4,00						Proteomics			24098404
P23528	CFL1 CFL	Cofilin-1 (18 kDa phosphoprotein) (p18) (Cofilin, non-muscle isoform)	Homo sapiens (Human)						x					x	CP	68055113	1,72	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P23528	CFL1 CFL	Cofilin-1 (18 kDa phosphoprotein) (p18) (Cofilin, non-muscle isoform)	Homo sapiens (Human)						x					x	CP	68055113	1,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P23528	CFL1 CFL	Cofilin-1 (18 kDa phosphoprotein) (p18) (Cofilin, non-muscle isoform)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P23528	CFL1 CFL	Cofilin-1 (18 kDa phosphoprotein) (p18) (Cofilin, non-muscle isoform)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P24158	PRTN3 MBN	Myeloblastin	Homo sapiens (Human)						x					x	CP	68055113	1,17	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P24158	PRTN3 MBN	Myeloblastin (EC 3.4.21.76) (AGP7) (C-ANCA antigen) (Leukocyte proteinase 3) (PR-3) (PR3) (Neutrophil proteinase 4) (NP-4) (P29) (Wegener autoantigen)	Homo sapiens (Human)						x					x	CP	68055113	1,50	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P24158	PRTN3 MBN	Myeloblastin (EC 3.4.21.76) (AGP7) (C-ANCA antigen) (Leukocyte proteinase 3) (PR-3) (PR3) (Neutrophil proteinase 4) (NP-4) (P29) (Wegener autoantigen)	Homo sapiens (Human)					x						x	CP	68055113	3,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube, when necessary. Saliva samples were frozen immediately at -80ºC until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P24158	PRTN3 MBN	Myeloblastin (EC 3.4.21.76) (AGP7) (C-ANCA antigen) (Leukocyte proteinase 3) (PR-3) (PR3) (Neutrophil proteinase 4) (NP-4) (P29) (Wegener autoantigen)	Homo sapiens (Human)						x					x	CP	68055113	7,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P24158	PRTN3 MBN	Myeloblastin (EC 3.4.21.76) (AGP7) (C-ANCA antigen) (Leukocyte proteinase 3) (PR-3) (PR3) (Neutrophil proteinase 4) (NP-4) (P29) (Wegener autoantigen)	Homo sapiens (Human)						x						CP	68055113	8,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P24158	PRTN3 MBN	Myeloblastin (EC 3.4.21.76) (AGP7) (C-ANCA antigen) (Leukocyte proteinase 3) (PR-3) (PR3) (Neutrophil proteinase 4) (NP-4) (P29) (Wegener autoantigen)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P24298	GPT AAT1 GPT1	Alanine aminotransferase 1	Homo sapiens (Human)					x						x	CP	68055113	5,80	40-65	MF		Participants were asked to come to the laboratory at 07:00 am following an overnight fast, during which they were instructed not to eat, drink (except water), chew gum or brush teeth. Whole saliva samples were obtained by expectorating into polypropylene tubes. Patients were advised to rinse his or her mouth several times with water and then to relax for five minutes. Patients were asked to swallow to void the mouth of saliva and asked to lean their head forward over the polypropylene tube and funnel. Patients kept their mouth slightly open to allow saliva to drain into the tube.	ELISA; AST and ALT levels were analyzed on Roche p-800 modular system using the specific kits provided by the manufacturer.	on-proteomics			25345339
P24534	EEF1B2 EEF1B EF1B	Elongation factor 1-beta (EF-1-beta)	Homo sapiens (Human)						x					x	CP	68055113	2,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P24666	ACP1	Low molecular weight phosphotyrosine protein phosphatase (LMW-PTP) (LMW-PTPase) (EC 3.1.3.48) (Adipocyte acid phosphatase) (Low molecular weight cytosolic acid phosphatase) (EC 3.1.3.2) (Red cell acid phosphatase 1)	Homo sapiens (Human)						x					x	CP	68055113	6,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P24928	POLR2A POLR2	DNA-directed RNA polymerase II subunit RPB1 (RNA polymerase II subunit B1) (EC 2.7.7.6) (DNA-directed RNA polymerase II subunit A) (DNA-directed RNA polymerase III largest subunit) (RNA-directed RNA polymerase II subunit RPB1) (EC 2.7.7.48)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P25054	APC DP2.5	Adenomatous polyposis coli protein (Protein APC) (Deleted in polyposis 2.5)	Homo sapiens (Human)						x					x	CP	68055113	3,51	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P25311	AZGP1 ZAG ZNGP1	Zinc-alpha-2-glycoprotein (Zn-alpha-2-GP) (Zn-alpha-2-glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113	-8,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P25311	AZGP1 ZAG ZNGP1	Zinc-alpha-2-glycoprotein (Zn-alpha-2-GP) (Zn-alpha-2-glycoprotein)	Homo sapiens (Human)					x						x	CP	68055113	-1,24	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P25311	AZGP1 ZAG ZNGP1	Zinc-alpha-2-glycoprotein (Zn-alpha-2-GP) (Zn-alpha-2-glycoprotein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P25786	PSMA1 HC2 NU PROS30 PSC2	Proteasome subunit alpha type-1 (EC 3.4.25.1) (30 kDa prosomal protein) (PROS-30) (Macropain subunit C2) (Multicatalytic endopeptidase complex subunit C2) (Proteasome component C2) (Proteasome nu chain)	Homo sapiens (Human)						x					x	CP	68055113	3,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P25815	S100P S100E	Protein S100-P	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P25815	S100P S100E	Protein S100-P (Migration-inducing gene 9 protein) (MIG9) (Protein S100-E) (S100 calcium-binding protein P)	Homo sapiens (Human)					x						x	CP	68055113	2,39	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P25815	S100P S100E	Protein S100-P (Migration-inducing gene 9 protein) (MIG9) (Protein S100-E) (S100 calcium-binding protein P)	Homo sapiens (Human)						x					x	CP	68055113	2,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P25815	S100P S100E	Protein S100-P (Migration-inducing gene 9 protein) (MIG9) (Protein S100-E) (S100 calcium-binding protein P)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P25940	COL5A3	Collagen alpha-3(V) chain	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P26038	MSN	Moesin (Membrane-organizing extension spike protein)	Homo sapiens (Human)					x						x	CP	68055113	1,83	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P26038	MSN	Moesin (Membrane-organizing extension spike protein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P26038	MSN	Moesin (Membrane-organizing extension spike protein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P26447	S100A4 CAPL MTS1	Protein S100-A4 (Calvasculin) (Metastasin) (Placental calcium-binding protein) (Protein Mts1) (S100 calcium-binding protein A4)	Homo sapiens (Human)						x					x	CP	68055113	3,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P27169	PON1 PON	Serum paraoxonase/arylesterase 1 (PON 1) (EC 3.1.1.2) (EC 3.1.1.81) (EC 3.1.8.1) (Aromatic esterase 1) (A-esterase 1) (K-45) (Serum aryldialkylphosphatase 1)	Homo sapiens (Human)						x					x	CP	68055113	4,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P27169	PON1 PON	Serum paraoxonase/arylesterase 1 (PON 1) (EC 3.1.1.2) (EC 3.1.1.81) (EC 3.1.8.1) (Aromatic esterase 1) (A-esterase 1) (K-45) (Serum aryldialkylphosphatase 1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P27348	YWHAQ	14-3-3 protein theta	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P27348	YWHAQ	14-3-3 protein theta (14-3-3 protein T-cell) (14-3-3 protein tau) (Protein HS1)	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P27348	YWHAQ	14-3-3 protein theta (14-3-3 protein T-cell) (14-3-3 protein tau) (Protein HS1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P27482	CALML3	Calmodulin-like protein 3 (CaM-like protein) (CLP) (Calmodulin-related protein NB-1)	Homo sapiens (Human)					x						x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P27797	CALR CRTC	Calreticulin (CRP55) (Calregulin) (Endoplasmic reticulum resident protein 60) (ERp60) (HACBP) (grp60)	Homo sapiens (Human)					x						x	CP	68055113	1,88	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P27797	CALR CRTC	Calreticulin (CRP55) (Calregulin) (Endoplasmic reticulum resident protein 60) (ERp60) (HACBP) (grp60)	Homo sapiens (Human)						x					x	CP	68055113	2,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P27797	CALR CRTC	Calreticulin (CRP55) (Calregulin) (Endoplasmic reticulum resident protein 60) (ERp60) (HACBP) (grp60)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P28066	PSMA5	Proteasome subunit alpha type-5 (EC 3.4.25.1) (Macropain zeta chain) (Multicatalytic endopeptidase complex zeta chain) (Proteasome zeta chain)	Homo sapiens (Human)						x					x	CP	68055113	-1,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P28325	CST5	Cystatin-D (Cystatin-5)	Homo sapiens (Human)					x						x	CP	68055113	-1,36	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P28325	CST5	Cystatin-D (Cystatin-5)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P28347	TEAD1 TCF13 TEF1	Transcriptional enhancer factor TEF-1 (NTEF-1) (Protein GT-IIC) (TEA domain family member 1) (TEAD-1) (Transcription factor 13) (TCF-13)	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P28799	GRN	Progranulin	Homo sapiens (Human)					x						x	CP	68055113	1,07		MF	Exclusion criteria were systemic disease, use of medication in the last 6 months, pregnancy, smoking, and periodontal therapy within the last 6 months.	The saliva samples were collected according to the unstimulated saliva collection procedure.	ELISA	on-proteomics			25164155
P29034	S100A2 S100L	Protein S100-A2 (CAN19) (Protein S-100L) (S100 calcium-binding protein A2)	Homo sapiens (Human)						x					x	CP	68055113	-3,20				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P29350	PTPN6 HCP PTP1C	Tyrosine-protein phosphatase non-receptor type 6 (EC 3.1.3.48) (Hematopoietic cell protein-tyrosine phosphatase) (Protein-tyrosine phosphatase 1C) (PTP-1C) (Protein-tyrosine phosphatase SHP-1) (SH-PTP1)	Homo sapiens (Human)						x					x	CP	68055113	3,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P29400	COL4A5	Collagen alpha-5(IV) chain	Homo sapiens (Human)					x						x	CP	68055113	-1,79	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P29400	COL4A5	Collagen alpha-5(IV) chain	Homo sapiens (Human)						x					x	CP	68055113	1,14	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P29401	TKT	Transketolase (TK) (EC 2.2.1.1)	Homo sapiens (Human)					x						x	CP	68055113	1,50	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P29401	TKT	Transketolase (TK) (EC 2.2.1.1)	Homo sapiens (Human)					x						x	CP	68055113	5,37	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a paraffin bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P29401	TKT	Transketolase (TK) (EC 2.2.1.1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P29401	TKT	Transketolase (TK) (EC 2.2.1.1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P29508	SERPINB3 SCCA SCCA1	Serpin B3 (Protein T4-A) (Squamous cell carcinoma antigen 1) (SCCA-1)	Homo sapiens (Human)						x					x	CP	68055113	-4,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P29508	SERPINB3 SCCA SCCA1	Serpin B3 (Protein T4-A) (Squamous cell carcinoma antigen 1) (SCCA-1)	Homo sapiens (Human)					x						x	CP	68055113	-1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P29508	SERPINB3 SCCA SCCA1	Serpin B3 (Protein T4-A) (Squamous cell carcinoma antigen 1) (SCCA-1)	Homo sapiens (Human)						x					x	CP	68055113	6,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P29508	SERPINB3 SCCA SCCA1	Serpin B3 (Protein T4-A) (Squamous cell carcinoma antigen 1) (SCCA-1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P30040	ERP29 C12orf8 ERP28	Endoplasmic reticulum resident protein 29 (ERp29) (Endoplasmic reticulum resident protein 28) (ERp28) (Endoplasmic reticulum resident protein 31) (ERp31)	Homo sapiens (Human)						x					x	CP	68055113	1,98	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P30041	PRDX6 AOP2 KIAA0106	Peroxiredoxin-6 (EC 1.11.1.15) (1-Cys peroxiredoxin) (1-Cys PRX) (24 kDa protein) (Acidic calcium-independent phospholipase A2) (aiPLA2) (EC 3.1.1.-) (Antioxidant protein 2) (Liver 2D page spot 40) (Non-selenium glutathione peroxidase) (NSGPx) (EC 1.11.1.9) (Red blood cells page spot 12)	Homo sapiens (Human)					x						x	CP	68055113	1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P30041	PRDX6 AOP2 KIAA0106	Peroxiredoxin-6 (EC 1.11.1.15) (1-Cys peroxiredoxin) (1-Cys PRX) (24 kDa protein) (Acidic calcium-independent phospholipase A2) (aiPLA2) (EC 3.1.1.-) (Antioxidant protein 2) (Liver 2D page spot 40) (Non-selenium glutathione peroxidase) (NSGPx) (EC 1.11.1.9) (Red blood cells page spot 12)	Homo sapiens (Human)						x					x	CP	68055113	3,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P30041	PRDX6 AOP2 KIAA0106	Peroxiredoxin-6 (EC 1.11.1.15) (1-Cys peroxiredoxin) (1-Cys PRX) (24 kDa protein) (Acidic calcium-independent phospholipase A2) (aiPLA2) (EC 3.1.1.-) (Antioxidant protein 2) (Liver 2D page spot 40) (Non-selenium glutathione peroxidase) (NSGPx) (EC 1.11.1.9) (Red blood cells page spot 12)	Homo sapiens (Human)						x					x	CP	68055113						isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P30044	PRDX5 ACR1 SBBI10	Peroxiredoxin-5, mitochondrial (EC 1.11.1.15) (Alu corepressor 1) (Antioxidant enzyme B166) (AOEB166) (Liver tissue 2D-page spot 71B) (PLP) (Peroxiredoxin V) (Prx-V) (Peroxisomal antioxidant enzyme) (TPx type VI) (Thioredoxin peroxidase PMP20) (Thioredoxin reductase)	Homo sapiens (Human)						x					x	CP	68055113							Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P30048	PRDX3 AOP1	Thioredoxin-dependent peroxide reductase	Homo sapiens (Human)						x					x	CP	68055113	1,69	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P30086	PEBP1 PBP PEBP	Phosphatidylethanolamine-binding protein 1 (PEBP-1) (HCNPpp) (Neuropolypeptide h3) (Prostatic-binding protein) (Raf kinase inhibitor protein) (RKIP) [Cleaved into: Hippocampal cholinergic neurostimulating peptide (HCNP)]	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P30086	PEBP1 PBP PEBP	Phosphatidylethanolamine-binding protein 1 (PEBP-1) (HCNPpp) (Neuropolypeptide h3) (Prostatic-binding protein) (Raf kinase inhibitor protein) (RKIP) [Cleaved into: Hippocampal cholinergic neurostimulating peptide (HCNP)]	Homo sapiens (Human)						x					x	CP	68055113	1,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P30086	PEBP1 PBP PEBP	Phosphatidylethanolamine-binding protein 1 (PEBP-1) (HCNPpp) (Neuropolypeptide h3) (Prostatic-binding protein) (Raf kinase inhibitor protein) (RKIP) [Cleaved into: Hippocampal cholinergic neurostimulating peptide (HCNP)]	Homo sapiens (Human)						x					x	CP	68055113	-						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P30086	PEBP1 PBP PEBP	Phosphatidylethanolamine-binding protein 1 (PEBP-1) (HCNPpp) (Neuropolypeptide h3) (Prostatic-binding protein) (Raf kinase inhibitor protein) (RKIP) [Cleaved into: Hippocampal cholinergic neurostimulating peptide (HCNP)]	Homo sapiens (Human)						x					x	CP	68055113						isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P30101	PDIA3 ERP57 ERP60 GRP58	Protein disulfide-isomerase A3 (EC 5.3.4.1) (58 kDa glucose-regulated protein) (58 kDa microsomal protein) (p58) (Disulfide isomerase ER-60) (Endoplasmic reticulum resident protein 57) (ER protein 57) (ERp57) (Endoplasmic reticulum resident protein 60) (ER protein 60) (ERp60)	Homo sapiens (Human)						x					x	CP	68055113	1,59	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P30101	PDIA3 ERP57 ERP60 GRP58	Protein disulfide-isomerase A3 (EC 5.3.4.1) (58 kDa glucose-regulated protein) (58 kDa microsomal protein) (p58) (Disulfide isomerase ER-60) (Endoplasmic reticulum resident protein 57) (ER protein 57) (ERp57) (Endoplasmic reticulum resident protein 60) (ER protein 60) (ERp60)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P30520	ADSS ADSS2	Adenylosuccinate synthetase isozyme 2 (AMPSase 2) (AdSS 2) (EC 6.3.4.4) (Adenylosuccinate synthetase, acidic isozyme) (Adenylosuccinate synthetase, liver isozyme) (L-type adenylosuccinate synthetase) (IMP--aspartate ligase 2)	Homo sapiens (Human)						x					x	CP	68055113	2,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P30622	CLIP1 CYLN1 RSN	CAP-Gly domain-containing linker protein 1 (Cytoplasmic linker protein 1) (Cytoplasmic linker protein 170 alpha-2) (CLIP-170) (Reed-Sternberg intermediate filament-associated protein) (Restin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P30740	SERPINB1 ELANH2 MNEI PI2	Leukocyte elastase inhibitor	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P30740	SERPINB1 ELANH2 MNEI PI2	Leukocyte elastase inhibitor (LEI) (Monocyte/neutrophil elastase inhibitor) (EI) (M/NEI) (Peptidase inhibitor 2) (PI-2) (Serpin B1)	Homo sapiens (Human)					x						x	CP	68055113	1,19	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4 °C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P30740	SERPINB1 ELANH2 MNEI PI2	Leukocyte elastase inhibitor (LEI) (Monocyte/neutrophil elastase inhibitor) (EI) (M/NEI) (Peptidase inhibitor 2) (PI-2) (Serpin B1)	Homo sapiens (Human)						x					x	CP	68055113	3,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P30740	SERPINB1 ELANH2 MNEI PI2	Leukocyte elastase inhibitor (LEI) (Monocyte/neutrophil elastase inhibitor) (EI) (M/NEI) (Peptidase inhibitor 2) (PI-2) (Serpin B1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P30838	ALDH3A1 ALDH3	Aldehyde dehydrogenase, dimeric NADP-preferring (EC 1.2.1.5) (ALDHIII) (Aldehyde dehydrogenase 3) (Aldehyde dehydrogenase family 3 member A1)	Homo sapiens (Human)					x						x	CP	68055113	-1,18	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4 °C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P30838	ALDH3A1 ALDH3	Aldehyde dehydrogenase, dimeric NADP-preferring (EC 1.2.1.5) (ALDHIII) (Aldehyde dehydrogenase 3) (Aldehyde dehydrogenase family 3 member A1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P31025	LCN1 VEGP	Lipocalin-1 (Tear lipocalin) (Tlc) (Tear prealbumin) (TP) (Von Ebner gland protein) (VEG protein)	Homo sapiens (Human)					x							CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P31025	LCN1 VEGP	Lipocalin-1 (Tear lipocalin) (Tlc) (Tear prealbumin) (TP) (Von Ebner gland protein) (VEG protein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P31146	CORO1A CORO1	Coronin-1A	Homo sapiens (Human)						x					x	CP	68055113	3,06						Proteomics			24098404
P31146	CORO1A CORO1	Coronin-1A (Coronin-like protein A) (Clipin-A) (Coronin-like protein p57) (Tryptophan aspartate-containing coat protein) (TACO)	Homo sapiens (Human)						x					x	CP	68055113	2,64	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P31146	CORO1A CORO1	Coronin-1A (Coronin-like protein A) (Clipin-A) (Coronin-like protein p57) (Tryptophan aspartate-containing coat protein) (TACO)	Homo sapiens (Human)						x					x	CP	68055113	8,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P31146	CORO1A CORO1	Coronin-1A (Coronin-like protein A) (Clipin-A) (Coronin-like protein p57) (Tryptophan aspartate-containing coat protein) (TACO)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P31151	S100A7 PSOR1 S100A7C	Protein S100-A7	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P31151	S100A7 PSOR1 S100A7C	Protein S100-A7 (Psoriasin) (S100 calcium-binding protein A7)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P31513	FMO3	Dimethylaniline monooxygenase [N-oxide-forming] 3	Homo sapiens (Human)						x					x	CP	68055113	-3,85	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P31751	AKT2	RAC-beta serine/threonine-protein kinase (EC 2.7.11.1) (Protein kinase Akt-2) (Protein kinase B beta) (PKB beta) (RAC-PK-beta)	Homo sapiens (Human)					x						x	CP	68055113	1,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4 °C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P31943	HNRNPH1 HNRPH HNRPH1	Heterogeneous nuclear ribonucleoprotein H (hnRNP H) [Cleaved into: Heterogeneous nuclear ribonucleoprotein H, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	2,87	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P31944	CASP14	Caspase-14 (CASP-14) (EC 3.4.22.-) [Cleaved into: Caspase-14 subunit p19; Caspase-14 subunit p10]	Homo sapiens (Human)						x					x	CP	68055113	2,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P31946	YWHAB	14-3-3 protein beta/alpha	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P31946	YWHAB	14-3-3 protein beta/alpha (Protein 1054) (Protein kinase C inhibitor protein 1) (KCIP-1) [Cleaved into: 14-3-3 protein beta/alpha, N-terminally processed]	Homo sapiens (Human)					x						x	CP	68055113	1,70	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C..	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P31946	YWHAB	14-3-3 protein beta/alpha (Protein 1054) (Protein kinase C inhibitor protein 1) (KCIP-1) [Cleaved into: 14-3-3 protein beta/alpha, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	2,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P31946	YWHAB	14-3-3 protein beta/alpha (Protein 1054) (Protein kinase C inhibitor protein 1) (KCIP-1) [Cleaved into: 14-3-3 protein beta/alpha, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P31946	YWHAB	14-3-3 protein beta/alpha (Protein 1054) (Protein kinase C inhibitor protein 1) (KCIP-1) [Cleaved into: 14-3-3 protein beta/alpha, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P31947	SFN HME1	14-3-3 protein sigma	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P31947	SFN HME1	14-3-3 protein sigma (Epithelial cell marker protein 1) (Stratifin)	Homo sapiens (Human)					x						x	CP	68055113	-1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C..	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P31947	SFN HME1	14-3-3 protein sigma (Epithelial cell marker protein 1) (Stratifin)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P31947	SFN HME1	14-3-3 protein sigma (Epithelial cell marker protein 1) (Stratifin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P31947	SFN HME1	14-3-3 protein sigma (Epithelial cell marker protein 1) (Stratifin)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P31949	S100A11 MLN70 S100C	Protein S100-A11	Homo sapiens (Human)						x					x	CP	68055113	2,77						Proteomics			24098404
P31949	S100A11 MLN70 S100C	Protein S100-A11 (Calgizzarin) (Metastatic lymph node gene 70 protein) (MLN 70) (Protein S100-C) (S100 calcium-binding protein A11)	Homo sapiens (Human)					x						x	CP	68055113	1,54	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C..	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P31949	S100A11 MLN70 S100C	Protein S100-A11 (Calgizzarin) (Metastatic lymph node gene 70 protein) (MLN 70) (Protein S100-C) (S100 calcium-binding protein A11)	Homo sapiens (Human)						x					x	CP	68055113	2,52	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P31949	S100A11 MLN70 S100C	Protein S100-A11 (Calgizzarin) (Metastatic lymph node gene 70 protein) (MLN 70) (Protein S100-C) (S100 calcium-binding protein A11)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P32119	PRDX2 NKEFB TDPX1	Peroxiredoxin-2 (EC 1.11.1.15) (Natural killer cell-enhancing factor B) (NKEF-B) (PRP) (Thiol-specific antioxidant protein) (TSA) (Thioredoxin peroxidase 1) (Thioredoxin-dependent peroxide reductase 1)	Homo sapiens (Human)					x						x	CP	68055113	1,19	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C..	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P32119	PRDX2 NKEFB TDPX1	Peroxiredoxin-2 (EC 1.11.1.15) (Natural killer cell-enhancing factor B) (NKEF-B) (PRP) (Thiol-specific antioxidant protein) (TSA) (Thioredoxin peroxidase 1) (Thioredoxin-dependent peroxide reductase 1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P32320	CDA CDD	Cytidine deaminase (EC 3.5.4.5) (Cytidine aminohydrolase)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P32320	CDA CDD	Cytidine deaminase (EC 3.5.4.5) (Cytidine aminohydrolase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P32926	DSG3 CDHF6	Desmoglein-3 (130 kDa pemphigus vulgaris antigen) (PVA) (Cadherin family member 6)	Homo sapiens (Human)					x						x	CP	68055113	-1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P32926	DSG3 CDHF6	Desmoglein-3 (130 kDa pemphigus vulgaris antigen) (PVA) (Cadherin family member 6)	Homo sapiens (Human)						x					x	CP	68055113	3,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P33241	LSP1 WP34	Lymphocyte-specific protein 1 (47 kDa actin-binding protein) (52 kDa phosphoprotein) (pp52) (Lymphocyte-specific antigen WP34)	Homo sapiens (Human)						x					x	CP	68055113	2,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P33778	HIST1H2BB H2BFF	Histone H2B type 1-B	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P34932	HSPA4 APG2	Heat shock 70 kDa protein 4 (HSP70RY) (Heat shock 70-related protein APG-2)	Homo sapiens (Human)						x					x	CP	68055113	2,44	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35030	PRSS3 PRSS4 TRY3 TRY4	Trypsin-3	Homo sapiens (Human)						x					x	CP	68055113	1	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P35052	GPC1	Glypican-1 [Cleaved into: Secreted glypican-1]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35321	SPRR1A	Cornifin-A (19 kDa pancornulin) (SPRK) (Small proline-rich protein IA) (SPR-IA)	Homo sapiens (Human)					x						x	CP	68055113	-1,28	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P35321	SPRR1A	Cornifin-A (19 kDa pancornulin) (SPRK) (Small proline-rich protein IA) (SPR-IA)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35321	SPRR1A	Cornifin-A (19 kDa pancornulin) (SPRK) (Small proline-rich protein IA) (SPR-IA)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P35325	SPRR2B	Small proline-rich protein 2B (SPR-2B)	Homo sapiens (Human)					x						x	CP	68055113	-1,11	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P35325	SPRR2B	Small proline-rich protein 2B (SPR-2B)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35325	SPRR2B	Small proline-rich protein 2B (SPR-2B)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P35326	SPRR2A	Small proline-rich protein 2A (SPR-2A) (2-1)	Homo sapiens (Human)						x					x	CP	68055113	3,60	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35354	PTGS2 COX2	Prostaglandin G/H synthase 2 (EC 1.14.99.1) (Cyclooxygenase-2) (COX-2) (PHS II) (Prostaglandin H2 synthase 2) (PGH synthase 2) (PGHS-2) (Prostaglandin-endoperoxide synthase 2)	Homo sapiens (Human)						x	x				x	CP	68055113						Non-Proteomics		x	12892443
P35354	PTGS2 COX2	Prostaglandin G/H synthase 2 (EC 1.14.99.1) (Cyclooxygenase-2) (COX-2) (PHS II) (Prostaglandin H2 synthase 2) (PGH synthase 2) (PGHS-2) (Prostaglandin-endoperoxide synthase 2)	Homo sapiens (Human)							x					CP	68055113		35-72		The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451
P35354	PTGS2 COX2	Prostaglandin-endoperoxide synthase 2 (prostaglandin G/H synthase and cyclooxygenase)	Homo sapiens (Human)							x					CP	68055113	32(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P35527	KRT9	Keratin, type I cytoskeletal 9	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P35527	KRT9	Keratin, type I cytoskeletal 9 (Cytokeratin-9) (CK-9) (Keratin-9) (K9)	Homo sapiens (Human)						x					x	CP	68055113	1,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35527	KRT9	Keratin, type I cytoskeletal 9 (Cytokeratin-9) (CK-9) (Keratin-9) (K9)	Homo sapiens (Human)						x					x	CP	68055113	33,80			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35527	KRT9	Keratin, type I cytoskeletal 9 (Cytokeratin-9) (CK-9) (Keratin-9) (K9)	Homo sapiens (Human)						x					x	CP	68055113					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P35542	SAA4 CSAA	Serum amyloid A-4 protein	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P35579	MYH9	Myosin-9 (Cellular myosin heavy chain, type A) (Myosin heavy chain 9) (Myosin heavy chain, non-muscle IIa) (Non-muscle myosin heavy chain A) (NMMHC-A) (Non-muscle myosin heavy chain IIa) (NMMHC II-a) (NMMHC-IIA)	Homo sapiens (Human)						x					x	CP	68055113	2,72	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35579	MYH9	Myosin-9 (Cellular myosin heavy chain, type A) (Myosin heavy chain 9) (Myosin heavy chain, non-muscle IIa) (Non-muscle myosin heavy chain A) (NMMHC-A) (Non-muscle myosin heavy chain IIa) (NMMHC II-a) (NMMHC-IIA)	Homo sapiens (Human)						x					x	CP	68055113	18,70			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35579	MYH9	Myosin-9 (Cellular myosin heavy chain, type A) (Myosin heavy chain 9) (Myosin heavy chain, non-muscle IIa) (Non-muscle myosin heavy chain A) (NMMHC-A) (Non-muscle myosin heavy chain IIa) (NMMHC II-a) (NMMHC-IIA)	Homo sapiens (Human)						x					x	CP	68055113					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P35670	ATP7B PWD WC1 WND	Copper-transporting ATPase 2 (EC 3.6.3.4) (Copper pump 2) (Wilson disease-associated protein) [Cleaved into: WND/140 kDa]	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35754	GLRX GRX	Glutaredoxin-1 (Thioltransferase-1) (TTase-1)	Homo sapiens (Human)						x					x	CP	68055113	3,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35908	KRT2 KRT2A KRT2E	Keratin 2	Homo sapiens (Human)							x					CP	68055113	.47(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P35908	KRT2 KRT2A KRT2E	Keratin, type II cytoskeletal 2 epidermal	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P35908	KRT2 KRT2A KRT2E	Keratin, type II cytoskeletal 2 epidermal	Homo sapiens (Human)						x					x	CP	68055113	1,47	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P35908	KRT2 KRT2A KRT2E	Keratin, type II cytoskeletal 2 epidermal (Cytokeratin-2e) (CK-2e) (Epithelial keratin-2e) (Keratin-2 epidermis) (Keratin-2e) (K2e) (Type-II keratin Kb2)	Homo sapiens (Human)						x					x	CP	68055113	1,97	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P35908	KRT2 KRT2A KRT2E	Keratin, type II cytoskeletal 2 epidermal (Cytokeratin-2e) (CK-2e) (Epithelial keratin-2e) (Keratin-2 epidermis) (Keratin-2e) (K2e) (Type-II keratin Kb2)	Homo sapiens (Human)						x					x	CP	68055113	32,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P35908	KRT2 KRT2A KRT2E	Keratin, type II cytoskeletal 2 epidermal (Cytokeratin-2e) (CK-2e) (Epithelial keratin-2e) (Keratin-2 epidermis) (Keratin-2e) (K2e) (Type-II keratin Kb2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P36542	ATP5C1 ATP5C ATP5CL1	ATP synthase subunit gamma, mitochondrial (F-ATPase gamma subunit)	Homo sapiens (Human)						x					x	CP	68055113	3,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P36871	PGM1	Phosphoglucomutase-1	Homo sapiens (Human)						x					x	CP	68055113	-1,45	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P36952	SERPINB5 PI5	Serpin B5 (Maspin) (Peptidase inhibitor 5) (PI-5)	Homo sapiens (Human)					x						x	CP	68055113	1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P36952	SERPINB5 PI5	Serpin B5 (Maspin) (Peptidase inhibitor 5) (PI-5)	Homo sapiens (Human)						x					x	CP	68055113	2,56	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P36955	SERPINF1 PEDF PIG35	Pigment epithelium-derived factor (PEDF) (Cell proliferation-inducing gene 35 protein) (EPC-1) (Serpin F1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P37802	TAGLN2 KIAA0120 CDABP0035	Transgelin-2 (Epididymis tissue protein Li 7e) (SM22-alpha homolog)	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P37837	TALDO1 TAL TALDO TALDOR	Transaldolase (EC 2.2.1.2)	Homo sapiens (Human)					x						x	CP	68055113	1,44	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P37837	TALDO1 TAL TALDO TALDOR	Transaldolase (EC 2.2.1.2)	Homo sapiens (Human)					x						x	CP	68055113	1,65	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P37837	TALDO1 TAL TALDO TALDOR	Transaldolase (EC 2.2.1.2)	Homo sapiens (Human)						x					x	CP	68055113	2,22	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P37837	TALDO1 TAL TALDO TALDOR	Transaldolase (EC 2.2.1.2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P37837	TALDO1 TAL TALDO TALDOR	Transaldolase (EC 2.2.1.2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P38606	ATP6V1A ATP6A1 ATP6V1A1 VPP2	V-type proton ATPase catalytic subunit A (V-ATPase subunit A) (EC 3.6.3.14) (V-ATPase 69 kDa subunit) (Vacuolar ATPase isoform VA68) (Vacuolar proton pump subunit alpha)	Homo sapiens (Human)						x					x	CP	68055113	2,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P38919	EIF4A3 DDX48 KIAA0111	Eukaryotic initiation factor 4A-III (eIF-4A-III) (eIF4A-III) (EC 3.6.4.13) (ATP-dependent RNA helicase DDX48) (ATP-dependent RNA helicase eIF4A-3) (DEAD box protein 48) (Eukaryotic initiation factor 4A-like NUK-34) (Eukaryotic translation initiation factor 4A isoform 3) (Nuclear matrix protein 265) (NMP 265) (hNMP 265) [Cleaved into: Eukaryotic initiation factor 4A-III, N-terminally processed]	Homo sapiens (Human)					x						x	CP	68055113	1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P39060	COL18A1	Collagen alpha-1(XVIII) chain	Homo sapiens (Human)						x					x	CP	68055113	-1,41	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P40121	CAPG AFCP MCP	Macrophage-capping protein (Actin regulatory protein CAP-G)	Homo sapiens (Human)					x						x	CP	68055113	1,72	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P40121	CAPG AFCP MCP	Macrophage-capping protein (Actin regulatory protein CAP-G)	Homo sapiens (Human)						x					x	CP	68055113	1,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P40121	CAPG AFCP MCP	Macrophage-capping protein (Actin regulatory protein CAP-G)	Homo sapiens (Human)						x					x	CP	68055113	2,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P40145	ADCY8	Adenylate cyclase type 8	Homo sapiens (Human)						x					x	CP	68055113	1,11	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P40306	PSMB10 LMP10 MECL1	Proteasome subunit beta type-10 (EC 3.4.25.1) (Low molecular mass protein 10) (Macropain subunit MECI-1) (Multicatalytic endopeptidase complex subunit MECI-1) (Proteasome MECI-1) (Proteasome subunit beta-2i)	Homo sapiens (Human)						x					x	CP	68055113	3,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P40925	MDH1 MDHA	Malate dehydrogenase, cytoplasmic (EC 1.1.1.37) (Cytosolic malate dehydrogenase) (Diiodophenylpyruvate reductase) (EC 1.1.1.96)	Homo sapiens (Human)					x						x	CP	68055113	1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P40926	MDH2	Malate dehydrogenase, mitochondrial (EC 1.1.1.37)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P40939	HADHA HADH	Trifunctional enzyme subunit alpha, mitochondrial (78 kDa gastrin-binding protein) (TP-alpha) [Includes: Long-chain enoyl-CoA hydratase (EC 4.2.1.17); Long chain 3-hydroxyacyl-CoA dehydrogenase (EC 1.1.1.211)]	Homo sapiens (Human)						x					x	CP	68055113	3,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P41159	LEP OB OBS	Leptin	Homo sapiens (Human)					x						x	CP	68055113	-1,42	31-65	MF	We excluded patients with the use of tobacco in any form, alcoholism, pregnancy, presence of any gross pathology and also patients who have received any periodontal therapy in past 12 months or any anti- microbial, anti- inflammatory and immune suppressive therapy in the past 6 months.	Whole unstimulated salivary samples (~2ml) were collected by modified draining method. 4 Patients were asked to expectorate into disposable polypropylene tubes every 30 sec over a period of 5 min.	ELISA	on-proteomics			25537001
P41218	MNDA	Myeloid cell nuclear differentiation antigen	Homo sapiens (Human)						x					x	CP	68055113	3,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P41219	PRPH NEF4 PRPH1	Peripherin	Homo sapiens (Human)						x						CP	68055113	4,00						Proteomics			24098404
P41439	FOLR3	Folate receptor gamma (FR-gamma) (Folate receptor 3)	Homo sapiens (Human)						x					x	CP	68055113	2,11	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P42357	HAL HIS	Histidine ammonia-lyase	Homo sapiens (Human)							x					CP	68055113	,38(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P42694	HELZ DRHC KIAA0054	Probable helicase with zinc finger domain (EC 3.6.4.-) (Down-regulated in human cancers protein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P42858	HTT HD IT15	Huntingtin (Huntington disease protein) (HD protein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P43235	CTSK CTSO CTSO2	Cathepsin K	Homo sapiens (Human)						x						CP	68055113	3,67						Non-proteomics			17321485
P43235	CTSK CTSO CTSO2		Homo sapiens (Human)						x					x	CP	68055113	5,47		MF		GCF/PISF samples were obtained from natural teeth (group T) and dental implants (group I)	Cathepsin-K activity was determined with a commercially available cathepsin-K activity assay kit (BioVision).	on-Proteomics			22010081
P43490	NAMPT PBEF PBEF1	Visfatin	Homo sapiens (Human)					x						x	CP	68055113	1,43	24-62	MF		Unstimulated whole expectorated saliva (5 mL) was collected from each volunteer between 10:00 am and 12:00 pm, according to a modification of the method described by Navazesh.21 The patients were asked to swallow saliva first, and then allow the saliva to drain passively for 5 minutes over the lower lip into a sterile tube. Collected saliva was immediately placed on ice prior to freezing at -80 C.	ELISA	on-proteomics			24228809
P43490	NAMPT PBEF PBEF1	Visfatin	Homo sapiens (Human)					x						x	CP	68055113	2,21		MF	Exclusion criteria were systemic disease, use of medication in the last 6 months, pregnancy, smoking, and periodontal therapy within the last 6 months.	The saliva samples were collected according to the unstimulated saliva collection procedure.	ELISA	on-proteomics			25164155
P43652	AFM ALB2 ALBA	Afamin (Alpha-albumin) (Alpha-Alb)	Homo sapiens (Human)						x					x	CP	68055113	1,85	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P43652	AFM ALB2 ALBA	Afamin (Alpha-albumin) (Alpha-Alb)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P46013	MKI67	Antigen KI-67	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P46013	MKI67	Antigen KI-67	Homo sapiens (Human)						x					x	CP	68055113	1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P46783	RPS10	40S ribosomal protein S10	Homo sapiens (Human)						x					x	CP	68055113	2,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P46939	UTRN DMDL DRP1	Utrophin (Dystrophin-related protein 1) (DRP-1)	Homo sapiens (Human)						x					x	CP	68055113	3,26	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P46940	IQGAP1 KIAA0051	Ras GTPase-activating-like protein IQGAP1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P46940	IQGAP1 KIAA0051	Ras GTPase-activating-like protein IQGAP1 (p195)	Homo sapiens (Human)						x					x	CP	68055113	1,99	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P46940	IQGAP1 KIAA0051	Ras GTPase-activating-like protein IQGAP1 (p195)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P47756	CAPZB	F-actin-capping protein subunit beta (CapZ beta)	Homo sapiens (Human)					x						x	CP	68055113	1,46	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P47756	CAPZB	F-actin-capping protein subunit beta (CapZ beta)	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P47756	CAPZB	F-actin-capping protein subunit beta (CapZ beta)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P48147	PREP PEP	Prolyl endopeptidase (PE) (EC 3.4.21.26) (Post-proline cleaving enzyme)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P48506	GCLC GLCL GLCLC	Glutamate--cysteine ligase catalytic subunit (EC 6.3.2.2) (GCS heavy chain) (Gamma-ECS) (Gamma-glutamylcysteine synthetase)	Homo sapiens (Human)						x					x	CP	68055113	-1,50	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P48594	SERPINB4 PI11 SCCA2	Serpin B4 (Leupin) (Peptidase inhibitor 11) (PI-11) (Squamous cell carcinoma antigen 2) (SCCA-2)	Homo sapiens (Human)					x						x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P48595	SERPINB10 PI10	Serpin B10 (Bomapin) (Peptidase inhibitor 10) (PI-10)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P48668	KRT6C KRT6E	Keratin, type II cytoskeletal 6C	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P48668	KRT6C KRT6E	Keratin, type II cytoskeletal 6C (Cytokeratin-6C) (CK-6C) (Cytokeratin-6E) (CK-6E) (Keratin K6h) (Keratin-6C) (K6C) (Type-II keratin Kb12)	Homo sapiens (Human)						x						CP	68055113	+					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P48735	IDH2	Isocitrate dehydrogenase [NADP], mitochondrial (IDH) (EC 1.1.1.42) (ICD-M) (IDP) (NADP(+)-specific ICDH) (Oxalosuccinate decarboxylase)	Homo sapiens (Human)						x					x	CP	68055113	3,28	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P49411	TUFM	Elongation factor Tu, mitochondrial (EF-Tu) (P43)	Homo sapiens (Human)						x					x	CP	68055113	1,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P49588	AARS	Alanine--tRNA ligase, cytoplasmic (EC 6.1.1.7) (Alanyl-tRNA synthetase) (AlaRS) (Renal carcinoma antigen NY-REN-42)	Homo sapiens (Human)						x					x	CP	68055113	2,56	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P49748	ACADVL VLCAD	Very long-chain specific acyl-CoA dehydrogenase, mitochondrial (VLCAD) (EC 1.3.8.9)	Homo sapiens (Human)						x					x	CP	68055113	2,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P49798	RGS4	Regulator of G-protein signaling 4	Homo sapiens (Human)							x					CP	68055113	,31(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P49913	CAMP CAP18 FALL39 HSD26	Cathelicidin antimicrobial peptide (18 kDa cationic antimicrobial protein) (CAP-18) (hCAP-18) [Cleaved into: Antibacterial protein FALL-39 (FALL-39 peptide antibiotic); Antibacterial protein LL-37]	Homo sapiens (Human)						x					x	CP	68055113	3,16	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P49913	CAMP CAP18 FALL39 HSD26	Cathelicidin antimicrobial peptide (18 kDa cationic antimicrobial protein) (CAP-18) (hCAP-18) [Cleaved into: Antibacterial protein FALL-39 (FALL-39 peptide antibiotic); Antibacterial protein LL-37]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P50395	GDI2 RABGDIB	Rab GDP dissociation inhibitor beta (Rab GDI beta) (Guanosine diphosphate dissociation inhibitor 2) (GDI-2)	Homo sapiens (Human)					x						x	CP	68055113	1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P50395	GDI2 RABGDIB	Rab GDP dissociation inhibitor beta (Rab GDI beta) (Guanosine diphosphate dissociation inhibitor 2) (GDI-2)	Homo sapiens (Human)					x						x	CP	68055113	2,17	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a paraffin bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
P50395	GDI2 RABGDIB	Rab GDP dissociation inhibitor beta (Rab GDI beta) (Guanosine diphosphate dissociation inhibitor 2) (GDI-2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P50552	VASP	Vasodilator-stimulated phosphoprotein (VASP)	Homo sapiens (Human)						x					x	CP	68055113	1,54	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P50748	KNTC1 KIAA0166	Kinetochore-associated protein 1 (Rough deal homolog) (HsROD) (Rod) (hRod)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P50851	LRBA BGL CDC4L LBA	Lipopolysaccharide-responsive and beige-like anchor protein (Beige-like protein) (CDC4-like protein)	Homo sapiens (Human)						x					x	CP	68055113	-3,30				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P51149	RAB7A RAB7	Ras-related protein Rab-7a	Homo sapiens (Human)					x						x	CP	68055113	1,94	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P51149	RAB7A RAB7	Ras-related protein Rab-7a	Homo sapiens (Human)						x					x	CP	68055113	2,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P51149	RAB7A RAB7	Ras-related protein Rab-7a	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P51530	DNA2 DNA2L KIAA0083	DNA replication ATP-dependent helicase/nuclease DNA2 (hDNA2) (DNA replication ATP-dependent helicase-like homolog) [Includes: DNA replication nuclease DNA2 (EC 3.1.-.-); DNA replication ATP-dependent helicase DNA2 (EC 3.6.4.12)]	Homo sapiens (Human)						x					x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P51572	BCAP31 BAP31 DXS1357E	B-cell receptor-associated protein 31 (BCR-associated protein 31) (Bap31) (6C6-AG tumor-associated antigen) (Protein CDM) (p28)	Homo sapiens (Human)						x					x	CP	68055113	5,18	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P51826	AFF3 LAF4	AF4/FMR2 family member 3 (Lymphoid nuclear protein related to AF4) (Protein LAF-4)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52209	PGD PGDH	6-phosphogluconate dehydrogenase, decarboxylating (EC 1.1.1.44)	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P52209	PGD PGDH	6-phosphogluconate dehydrogenase, decarboxylating (EC 1.1.1.44)	Homo sapiens (Human)						x					x	CP	68055113	3,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P52209	PGD PGDH	6-phosphogluconate dehydrogenase, decarboxylating (EC 1.1.1.44)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52209	PGD PGDH	6-phosphogluconate dehydrogenase, decarboxylating (EC 1.1.1.44)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P52272	HNRNPM HNRPM NAGR1	Heterogeneous nuclear ribonucleoprotein M (hnRNP M)	Homo sapiens (Human)						x					x	CP	68055113	2,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P52565	ARHGDIA GDIA1	Rho GDP-dissociation inhibitor 1 (Rho GDI 1) (Rho-GDI alpha)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52565	ARHGDIA GDIA1	Rho GDP-dissociation inhibitor 1 (Rho GDI 1) (Rho-GDI alpha)	Homo sapiens (Human)					x						x	CP	68055113					unstimulated	2-DE + MALDI-TOF-TOF MS	Proteomics			22165124
P52566	ARHGDIB GDIA2 GDID4 RAP1GN1	Rho GDP-dissociation inhibitor 2 (Rho GDI 2) (Ly-GDI) (Rho-GDI beta)	Homo sapiens (Human)					x						x	CP	68055113	1,97	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P52566	ARHGDIB GDIA2 GDID4 RAP1GN1	Rho GDP-dissociation inhibitor 2 (Rho GDI 2) (Ly-GDI) (Rho-GDI beta)	Homo sapiens (Human)						x					x	CP	68055113	7,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52597	HNRNPF HNRPF	Heterogeneous nuclear ribonucleoprotein F (hnRNP F) (Nucleolin-like protein mcs94-1) [Cleaved into: Heterogeneous nuclear ribonucleoprotein F, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	2,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P52630	STAT2	Signal transducer and activator of transcription 2 (p113)	Homo sapiens (Human)						x					x	CP	68055113	1,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P52790	HK3	Hexokinase-3 (EC 2.7.1.1) (Hexokinase type III) (HK III)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52907	CAPZA1	F-actin-capping protein subunit alpha-1 (CapZ alpha-1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P52948	NUP98 ADAR2	Nuclear pore complex protein Nup98-Nup96	Homo sapiens (Human)						x					x	CP	68055113	2,82	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P53355	DAPK1 DAPK	Death-associated protein kinase	Homo sapiens (Human)						x					x	CP	68055113	2,5	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P53634	CTSC CPPI	Dipeptidyl peptidase 1 (EC 3.4.14.1) (Cathepsin C) (Cathepsin J) (Dipeptidyl peptidase I) (DPP-I) (DPPI) (Dipeptidyl transferase) [Cleaved into: Dipeptidyl peptidase 1 exclusion domain chain (Dipeptidyl peptidase I exclusion domain chain); Dipeptidyl peptidase 1 heavy chain (Dipeptidyl peptidase I heavy chain); Dipeptidyl peptidase 1 light chain (Dipeptidyl peptidase I light chain)]	Homo sapiens (Human)					x						x	CP	68055113	-1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacecic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P53634	CTSC CPPI	Dipeptidyl peptidase 1 (EC 3.4.14.1) (Cathepsin C) (Cathepsin J) (Dipeptidyl peptidase I) (DPP-I) (DPPI) (Dipeptidyl transferase) [Cleaved into: Dipeptidyl peptidase 1 exclusion domain chain (Dipeptidyl peptidase I exclusion domain chain); Dipeptidyl peptidase 1 heavy chain (Dipeptidyl peptidase I heavy chain); Dipeptidyl peptidase 1 light chain (Dipeptidyl peptidase I light chain)]	Homo sapiens (Human)						x					x	CP	68055113	5,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at -80°C until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P53804	TTC3 DCRR1 RNF105 TPRD	Tetratricopeptide repeat protein 3	Homo sapiens (Human)						x					x	CP	68055113	1,49	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P54108	CRISP3	Cysteine-rich secretory protein 3 (CRISP-3) (Specific granule protein of 28 kDa) (SGP28)	Homo sapiens (Human)					x						x	CP	68055113	1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P54289	CACNA2D1 CACNL2A CCHL2A MHS3	Voltage-dependent calcium channel subunit alpha-2/delta-1 (Voltage-gated calcium channel subunit alpha-2/delta-1) [Cleaved into: Voltage-dependent calcium channel subunit alpha-2-1; Voltage-dependent calcium channel subunit delta-1]	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P54725	RAD23A	UV excision repair protein RAD23 homolog A (HR23A) (hHR23A)	Homo sapiens (Human)						x					x	CP	68055113	2,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P55072	VCP	Transitional endoplasmic reticulum ATPase (TER ATPase) (EC 3.6.4.6) (15S Mg(2+)-ATPase p97 subunit) (Valosin-containing protein) (VCP)	Homo sapiens (Human)						x					x	CP	68055113	2,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P55072	VCP	Transitional endoplasmic reticulum ATPase (TER ATPase) (EC 3.6.4.6) (15S Mg(2+)-ATPase p97 subunit) (Valosin-containing protein) (VCP)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P55285	CDH6	Cadherin-6	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P55786	NPEPPS PSA	Puromycin-sensitive aminopeptidase (PSA) (EC 3.4.11.14) (Cytosol alanyl aminopeptidase) (AAP-S)	Homo sapiens (Human)					x						x	CP	68055113	-1,25	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P55786	NPEPPS PSA	Puromycin-sensitive aminopeptidase (PSA) (EC 3.4.11.14) (Cytosol alanyl aminopeptidase) (AAP-S)	Homo sapiens (Human)							x				x	CP	68055113	2,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P56199	ITGA1	Integrin alpha-1 (CD49 antigen-like family member A) (Laminin and collagen receptor) (VLA-1) (CD antigen CD49a)	Homo sapiens (Human)							x				x	CP	68055113	5,92	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P56537	EIF6 EIF3A ITGB4BP OK/SW-cl.27	Eukaryotic translation initiation factor 6 (eIF-6) (B(2)GCN homolog) (B4 integrin interactor) (CAB) (p27(BBP))	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P56645	PER3 GIG13	Period circadian clock 3	Homo sapiens (Human)							x					CP	68055113	46(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P57053	H2BFS	Histone H2B type F-S	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P57721	PCBP3	Poly(rC)-binding protein 3 (Alpha-CP3)	Homo sapiens (Human)						x					x	CP	68055113	2,05	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P57789	KCNK10 TREK2	Potassium channel subfamily K member 10 (Outward rectifying potassium channel protein TREK-2) (TREK-2 K(+) channel subunit)	Homo sapiens (Human)						x					x	CP	68055113	1,42	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P58062	SPINK7 ECG2 UNQ745/PRO1474	Serine protease inhibitor Kazal-type 7 (Esophagus cancer-related gene 2 protein) (ECRG-2)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P58107	EPPK1 EPIPL	Epiplakin (450 kDa epidermal antigen)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P58876	HIST1H2BD H2BFB HIRIP2	Histone H2B type 1-D	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P59665	DEFA1 DEF1 DEFA2 MRS; DEFA1B	Neutrophil defensin 1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P59665	DEFA1 DEF1 DEFA2 MRS; DEFA1B	Neutrophil defensin 1	Homo sapiens (Human)						x					x	CP	68055113	1,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P59665	DEFA1 DEF1 DEFA2 MRS; DEFA1B	Neutrophil defensin 1 (Defensin, alpha 1) (HNP-1) (HP-1) (HP1) [Cleaved into: HP 1-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)					x						x	CP	68055113	4,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at –80°C until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P59665	DEFA1 DEF1 DEFA2 MRS; DEFA1B	Neutrophil defensin 1 (Defensin, alpha 1) (HNP-1) (HP-1) (HP1) [Cleaved into: HP 1-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)						x					x	CP	68055113	-	37-52	M/F	Obese	Whole saliva samples were collected in the morning, at least 2 h after taking breakfast. The patients were asked to rinse their mouth and drink a glass of water, to encourage salivation (Schipper et al. 2007a,b). Using the Saliva-check kit BUFFER (GC France), the total stimulated saliva was collected. The subjects were asked to chew a piece of parafilm to activate salivation. At 30 s, a timer was triggered for a period of 5 min. The subjects spat the whole saliva into a sterile cup. The volume of saliva (in ml) was noted to allow calculation of the flow rate of saliva (Data S1). The saliva was then transferred using a pipette into a sterile centrifuge tube. No protease inhibitors were added during saliva sampling and treatment. Mucins, cells and food debris were eliminated by centrifugation at 10,000 g for 15 min. at 4°C. The clear supernatant of saliva was carefully collected in 15 ml tubes and immediately stored at 80°C prior to proteomic analysis.	SELDI-TOF)	Proteomics		x	22780105
P59665	DEFA1 DEF1 DEFA2 MRS; DEFA1B	Neutrophil defensin 1 (Defensin, alpha 1) (HNP-1) (HP-1) (HP1) [Cleaved into: HP 1-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P59666	DEFA3 DEF3	Neutrophil defensin 3	Homo sapiens (Human)						x					x	CP	68055113	1,77						Proteomics			24098404
P59666	DEFA3 DEF3	Neutrophil defensin 3 (Defensin, alpha 3) (HNP-3) (HP-3) (HP3) [Cleaved into: HP 3-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)						x					x	CP	68055113	2,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics		x	23790309
P59666	DEFA3 DEF3	Neutrophil defensin 3 (Defensin, alpha 3) (HNP-3) (HP-3) (HP3) [Cleaved into: HP 3-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)						x					x	CP	68055113	4,00	28-63	M/F		As described recently, whole saliva samples were collected using a sterile glass funnel on weighed 10-mL sterile poly-propylen containers for 10 minutes. No oral stimuli were permitted for 120 minutes prior to collection to exclude any influence of mastication or foodstuffs. The seated patients collected the unstimulated saliva in the bottom of the mouth over the 10-minute period and drained it into a collection tube when necessary. Saliva samples were frozen immediately at –80ºC until analysis, at wich point the samples were thawed and kept on ice.	Western blot	on-Proteomics			23034426
P59666	DEFA3 DEF3	Neutrophil defensin 3 (Defensin, alpha 3) (HNP-3) (HP-3) (HP3) [Cleaved into: HP 3-56; Neutrophil defensin 2 (HNP-2) (HP-2) (HP2)]	Homo sapiens (Human)						x					x	CP	68055113	-	37-52	M/F	Obese	Whole saliva samples were collected in the morning, at least 2 h after taking breakfast. The patients were asked to rinse their mouth and drink a glass of water, to encourage salivation (Schipper et al. 2007a,b). Using the Saliva-check kit BUFFER (GC France), the total stimulated saliva was collected. The subjects were asked to chew a piece of parafilm to activate salivation. At 30 s, a timer was triggered for a period of 5 min. The subjects spat the whole saliva into a sterile cup. The volume of saliva (in ml) was noted to allow calculation of the flow rate of saliva (Data S1). The saliva was then transferred using a pipette into a sterile centrifuge tube. No protease inhibitors were added during saliva sampling and treatment. Mucins, cells and food debris were eliminated by centrifugation at 10,000 g for 15 min. at 4°C. The clear supernatant of saliva was carefully collected in 15 ml tubes and immediately stored at 80°C prior to proteomic analysis.	SELDI-TOF)	Proteomics		x	22780105
P59768	GNG2	Guanine nucleotide-binding protein G(I)/G(S)/G(O) subunit gamma-2 (G gamma-I)	Homo sapiens (Human)						x					x	CP	68055113	14,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P59998	ARPC4 ARC20	Actin-related protein 2/3 complex subunit 4 (Arp2/3 complex 20 kDa subunit) (p20-ARC)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P60022	DEFB1 BD1 HBD1	Defensin, beta 1	Homo sapiens (Human)							x					CP	68055113	,26(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P60174	TPI1 TPI	Triosephosphate isomerase (TIM) (EC 5.3.1.1) (Triose-phosphate isomerase)	Homo sapiens (Human)					x						x	CP	68055113	1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P60174	TPI1 TPI	Triosephosphate isomerase (TIM) (EC 5.3.1.1) (Triose-phosphate isomerase)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P60660	MYL6	Myosin light polypeptide 6 (17 kDa myosin light chain) (LC17) (Myosin light chain 3) (MLC-3) (Myosin light chain alkali 3) (Myosin light chain A3) (Smooth muscle and nonmuscle myosin light chain alkali 6)	Homo sapiens (Human)					x						x	CP	68055113	1,52	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P60660	MYL6	Myosin light polypeptide 6 (17 kDa myosin light chain) (LC17) (Myosin light chain 3) (MLC-3) (Myosin light chain alkali 3) (Myosin light chain A3) (Smooth muscle and nonmuscle myosin light chain alkali 6)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P60709	ACTB	Actin, cytoplasmic 1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P60709	ACTB	Actin, cytoplasmic 1	Homo sapiens (Human)						x					x	CP	68055113	1,62	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P60709	ACTB	Actin, cytoplasmic 1 (Beta-actin) [Cleaved into: Actin, cytoplasmic 1, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P60709	ACTB	Actin, cytoplasmic 1 (Beta-actin) [Cleaved into: Actin, cytoplasmic 1, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P61158	ACTR3 ARP3	Actin-related protein 3 (Actin-like protein 3)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P61158	ACTR3 ARP3	Actin-related protein 3 (Actin-like protein 3)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P61160	ACTR2 ARP2	Actin-related protein 2 (Actin-like protein 2)	Homo sapiens (Human)						x					x	CP	68055113	-1,40				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P61160	ACTR2 ARP2	Actin-related protein 2 (Actin-like protein 2)	Homo sapiens (Human)						x					x	CP	68055113	3,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P61247	RPS3A FTE1 MFTL	40S ribosomal protein S3a (v-fos transformation effector protein) (Fte-1)	Homo sapiens (Human)						x					x	CP	68055113	2,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P61586	RHOA ARH12 ARHA RHO12	Transforming protein RhoA (Rho cDNA clone 12) (h12)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P61626	LYZ LZM	Lysozyme C	Homo sapiens (Human)						x					x	CP	68055113	2,13						Proteomics			24098404
P61626	LYZ LZM	Lysozyme C (EC 3.2.1.17) (1,4-beta-N-acetylmuramidase C)	Homo sapiens (Human)					x						x	CP	68055113	1,18	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P61626	LYZ LZM	Lysozyme C (EC 3.2.1.17) (1,4-beta-N-acetylmuramidase C)	Homo sapiens (Human)						x					x	CP	68055113	2,65	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P61626	LYZ LZM	Lysozyme C (EC 3.2.1.17) (1,4-beta-N-acetylmuramidase C)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P61626	LYZ LZM	Lysozyme C (EC 3.2.1.17) (1,4-beta-N-acetylmuramidase C)	Homo sapiens (Human)						x					x	CP	68055113						Samples were typically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P61626	LYZ LZM	Lysozyme C (EC 3.2.1.17) (1,4-beta-N-acetylmuramidase C)	Homo sapiens (Human)						x					x	CP	68055113		30-68	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics		x	23190455

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P61769	B2M CDABP0092 HDCMA22P	Beta-2-microglobulin [Cleaved into: Beta-2-microglobulin form pI 5.3]	Homo sapiens (Human)					x						x	CP	68055113	-1,40	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P61970	NUTF2 NTF2	Nuclear transport factor 2 (NTF-2) (Placental protein 15) (PP15)	Homo sapiens (Human)					x						x	CP	68055113	1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P61981	YWHAG	14-3-3 protein gamma	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
P61981	YWHAG	14-3-3 protein gamma (Protein kinase C inhibitor protein 1) (KCIP-1) [Cleaved into: 14-3-3 protein gamma, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P62158	CALM1 CALM CAM CAM1; CALM2 CAM2 CA	Calmodulin	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P62195	PSMC5 SUG1	26S protease regulatory subunit 8 (26S proteasome AAA-ATPase subunit RPT6) (Proteasome 26S subunit ATPase 5) (Proteasome subunit p45) (Thyroid hormone receptor-interacting protein 1) (TRIP1) (p45/SUG)	Homo sapiens (Human)					x						x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P62258	YWHAE	14-3-3 protein epsilon (14-3-3E)	Homo sapiens (Human)						x					x	CP	68055113	-5,50				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P62258	YWHAE	14-3-3 protein epsilon (14-3-3E)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P62328	TMSB4X TB4X THYB4 TMSB4	Thymosin beta-4	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P62330	ARF6	ADP-ribosylation factor 6	Homo sapiens (Human)						x					x	CP	68055113	2,19	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P62701	RPS4X CCG2 RPS4 SCAR	40S ribosomal protein S4, X isoform (SCR10) (Single copy abundant mRNA protein)	Homo sapiens (Human)						x					x	CP	68055113	3,43	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P62736	ACTA2 ACTSA ACTVS GIG46	Actin, aortic smooth muscle	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P62736	ACTA2 ACTSA ACTVS GIG46	Actin, aortic smooth muscle	Homo sapiens (Human)						x					x	CP	68055113	1,39	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)						x					x	CP	68055113	2,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)					x						x	CP	68055113	3,29	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P62805	HIST1H4A H4/A H4FA; HIST1H4B H4/I H4FI;	Histone H4	Homo sapiens (Human)						x					x	CP	68055113	1,58	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P62807	HIST1H2BC H2BFL; HIST1H2BE H2BFH; HIS	Histone H2B type 1-C/E/F/G/I	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P62807	HIST1H2BC H2BFL; HIST1H2BE H2BFH; HIS	Histone H2B type 1-C/E/F/G/I	Homo sapiens (Human)						x					x	CP	68055113	1,18	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P62826	RAN ARA24 OK/SW-cl.81	GTP-binding nuclear protein Ran (Androgen receptor-associated protein 24) (GTPase Ran) (Ras-like protein TC4) (Ras-related nuclear protein)	Homo sapiens (Human)					x						x	CP	68055113	1,18	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P62829	RPL23	60S ribosomal protein L23 (60S ribosomal protein L17)	Homo sapiens (Human)						x					x	CP	68055113	3,61	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P62937	PPIA CYPA	Peptidyl-prolyl cis-trans isomerase A (PPIase A) (EC 5.2.1.8) (Cyclophilin A) (Cyclosporin A-binding protein) (Rotamase A)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P62937	PPIA CYPA	Peptidyl-prolyl cis-trans isomerase A (PPIase A) (EC 5.2.1.8) (Cyclophilin A) (Cyclosporin A-binding protein) (Rotamase A)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P62937	PPIA CYPA	Peptidyl-prolyl cis-trans isomerase A (PPIase A) (EC 5.2.1.8) (Cyclophilin A) (Cyclosporin A-binding protein) (Rotamase A) [Cleaved into: Peptidyl-prolyl cis-trans isomerase A, N-terminally processed]	Homo sapiens (Human)					x						x	CP	68055113	1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P62937	PPIA CYPA	Peptidyl-prolyl cis-trans isomerase A (PPIase A) (EC 5.2.1.8) (Cyclophilin A) (Cyclosporin A-binding protein) (Rotamase A) [Cleaved into: Peptidyl-prolyl cis-trans isomerase A, N-terminally processed]	Homo sapiens (Human)						x					x	CP	68055113	2,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P62937	PPIA CYPA	Peptidyl-prolyl cis-trans isomerase A (PPIase A) (EC 5.2.1.8) (Cyclophilin A) (Cyclosporin A-binding protein) (Rotamase A) [Cleaved into: Peptidyl-prolyl cis-trans isomerase A, N-terminally processed]	Homo sapiens (Human)						x					All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	CP	68055113		30-69	M/F	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics		x	23190455
P62979	RPS27A UBA80 UBCEP1	Ubiquitin-40S ribosomal protein S27a (Ubiquitin carboxyl extension protein 80) [Cleaved into: Ubiquitin; 40S ribosomal protein S27a]	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P62993	GRB2 ASH	Growth factor receptor-bound protein 2 (Adapter protein GRB2) (Protein Ash) (SH2/SH3 adapter GRB2)	Homo sapiens (Human)					x						x	CP	68055113	1,96	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P63096	GNAI1	Guanine nucleotide-binding protein G(i) subunit alpha-1 (Adenylate cyclase-inhibiting G alpha protein)	Homo sapiens (Human)						x					x	CP	68055113	4,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P63104	YWHAZ	14-3-3 protein zeta/delta (Protein kinase C inhibitor protein 1) (KCIP-1)	Homo sapiens (Human)					x						x	CP	68055113	1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P63104	YWHAZ	14-3-3 protein zeta/delta (Protein kinase C inhibitor protein 1) (KCIP-1)	Homo sapiens (Human)						x					x	CP	68055113	2,90	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P63104	YWHAZ	14-3-3 protein zeta/delta (Protein kinase C inhibitor protein 1) (KCIP-1)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P63104	YWHAZ	14-3-3 protein zeta/delta (Protein kinase C inhibitor protein 1) (KCIP-1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P63167	DYNLL1 DLC1 DNCL1 DNCLC1 HDLC1	Dynein light chain 1, cytoplasmic (8 kDa dynein light chain) (DLC8) (Dynein light chain LC8-type 1) (Protein inhibitor of neuronal nitric oxide synthase) (PIN)	Homo sapiens (Human)						x					x	CP	68055113	4,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P63220	RPS21	40S ribosomal protein S21	Homo sapiens (Human)						x					x	CP	68055113	2,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P63261	ACTG1 ACTG	Actin, cytoplasmic 2	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P63261	ACTG1 ACTG	Actin, cytoplasmic 2 (Gamma-actin) [Cleaved into: Actin, cytoplasmic 2, N-terminally processed]	Homo sapiens (Human)					x						x	CP	68055113	1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P63267	ACTG2 ACTA3 ACTL3 ACTSG	Actin, gamma-enteric smooth muscle	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P67936	TPM4	Tropomyosin alpha-4 chain	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
P68032	ACTC1 ACTC	Actin, alpha cardiac muscle 1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P68032	ACTC1 ACTC	Actin, alpha cardiac muscle 1 (Alpha-cardiac actin)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P68104	EEF1A1 EEF1A EF1A LENG7	Elongation factor 1-alpha 1 (EF-1-alpha-1) (Elongation factor Tu) (EF-Tu) (Eukaryotic elongation factor 1 A-1) (eEF1A-1) (Leukocyte receptor cluster member 7)	Homo sapiens (Human)						x					x	CP	68055113	2,78	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P68104	EEF1A1 EEF1A EF1A LENG7	Elongation factor 1-alpha 1 (EF-1-alpha-1) (Elongation factor Tu) (EF-Tu) (Eukaryotic elongation factor 1 A-1) (eEF1A-1) (Leukocyte receptor cluster member 7)	Homo sapiens (Human)						x					x	CP	68055113					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P68133	ACTA1 ACTA	Actin, alpha skeletal muscle	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P68133	ACTA1 ACTA	Actin, alpha skeletal muscle (Alpha-actin-1)	Homo sapiens (Human)					x						x	CP	68055113	1,38	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P68133	ACTA1 ACTA	Actin, alpha skeletal muscle (Alpha-actin-1)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P68431	HIST1H3A H3FA; HIST1H3B H3FL; HIST1H3	Histone H3.1	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P68431	HIST1H3A H3FA; HIST1H3B H3FL; HIST1H3	Histone H3.1	Homo sapiens (Human)						x					x	CP	68055113	1,07	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P68871	HBB	Hemoglobin subunit beta	Homo sapiens (Human)						x						CP	68055113	50,00						Proteomics			24098404
P68871	HBB	Hemoglobin subunit beta	Homo sapiens (Human)						x					x	CP	68055113	1,29	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7; Spinorphin]	Homo sapiens (Human)					x						x	CP	68055113	-1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7; Spinorphin]	Homo sapiens (Human)						x					x	CP	68055113	5,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7]	Homo sapiens (Human)						x					x	CP	68055113	81,30			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7]	Homo sapiens (Human)					x							CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P68871	HBB	Hemoglobin subunit beta (Beta-globin) (Hemoglobin beta chain) [Cleaved into: LVV-hemorphin-7]	Homo sapiens (Human)						x					x	CP	68055113		37-52	M/F	Obese	Whole saliva samples were collected in the morning, at least 2 h after taking breakfast. The patients were asked to rinse their mouth and drink a glass of water, to encourage salivation (Schipper et al. 2007a,b). Using the Saliva-check kit BUFFER (GC France), the total stimulated saliva was collected. The subjects were asked to chew a piece of parafilm to activate salivation. At 30 s, a timer was triggered for a period of 5 min. The subjects spat the whole saliva into a sterile cup. The volume of saliva (in ml) was noted to allow calculation of the flow rate of saliva (Data S1). The saliva was then transferred using a pipette into a sterile centrifuge tube. No protease inhibitors were added during saliva sampling and treatment. Mucins, cells and food debris were eliminated by centrifugation at 10,000 g for 15 min. at 4°C. The clear supernatant of saliva was carefully collected in 15 ml tubes and immediately stored at 80°C prior to proteomic analysis.	SELDI-TOF)	Proteomics			22780105

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P68871	HBB	Hemoglobin, beta	Homo sapiens (Human)							x					CP	68055113	,42(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
P69905	HBA1; HBA2	Hemoglobin subunit alpha	Homo sapiens (Human)						x						CP	68055113	31,00						Proteomics			24098404
P69905	HBA1; HBA2	Hemoglobin subunit alpha	Homo sapiens (Human)						x					x	CP	68055113	1,42	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P69905	HBA1; HBA2	Hemoglobin subunit alpha (Alpha-globin) (Hemoglobin alpha chain)	Homo sapiens (Human)						x					x	CP	68055113	7,40	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P69905	HBA1; HBA2	Hemoglobin subunit alpha (Alpha-globin) (Hemoglobin alpha chain)	Homo sapiens (Human)						x					x	CP	68055113	55,70				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P69905	HBA1; HBA2	Hemoglobin subunit alpha (Alpha-globin) (Hemoglobin alpha chain)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
P69905	HBA1; HBA2	Hemoglobin subunit alpha (Alpha-globin) (Hemoglobin alpha chain)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P69905	HBA1; HBA2	Hemoglobin subunit alpha (Alpha-globin) (Hemoglobin alpha chain)	Homo sapiens (Human)						x					x	CP	68055113		37-52	M/F	Obese	Whole saliva samples were collected in the morning, at least 2 h after taking breakfast. The patients were asked to rinse their mouth and drink a glass of water, to encourage salivation (Schipper et al. 2007a,b). Using the Saliva-check kit BUFFER (GC France), the total stimulated saliva was collected. The subjects were asked to chew a piece of parafilm to activate salivation. At 30 s, a timer was triggered for a period of 5 min. The subjects spat the whole saliva into a sterile cup. The volume of saliva (in ml) was noted to allow calculation of the flow rate of saliva (Data S1). The saliva was then transferred using a pipette into a sterile centrifuge tube. No protease inhibitors were added during saliva sampling and treatment. Mucins, cells and food debris were eliminated by centrifugation at 10,000 g for 15 min. at 4°C. The clear supernatant of saliva was carefully collected in 15 ml tubes and immediately stored at 80°C prior to proteomic analysis.	SELDI-TOF)	Proteomics			22780105
P78386	KRT85 KRTHB5	Keratin, type II cuticular Hb5 (Hair keratin K2.12) (Keratin-85) (K85) (Type II hair keratin Hb5) (Type-II keratin Kb25)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P78417	GSTO1 GSTTLP28	Glutathione S-transferase omega-1 (GSTO-1) (EC 2.5.1.18) (Glutathione S-transferase omega 1-1) (GSTO 1-1) (Glutathione-dependent dehydroascorbate reductase) (EC 1.8.5.1) (Monomethylarsonic acid reductase) (MMA(V) reductase) (EC 1.20.4.2) (S-(Phenacyl)glutathione reductase) (SPG-R)	Homo sapiens (Human)						x					x	CP	68055113	-3,00				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P78417	GSTO1 GSTTLP28	Glutathione S-transferase omega-1 (GSTO-1) (EC 2.5.1.18) (Glutathione S-transferase omega 1-1) (GSTO 1-1) (Glutathione-dependent dehydroascorbate reductase) (EC 1.8.5.1) (Monomethylarsonic acid reductase) (MMA(V) reductase) (EC 1.20.4.2) (S-(Phenacyl)glutathione reductase) (SPG-R)	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
P80188	LCN2 HNL NGAL	Neutrophil gelatinase-associated lipocalin (NGAL) (25 kDa alpha-2-microglobulin-related subunit of MMP-9) (Lipocalin-2) (Oncogene 24p3) (Siderocalin LCN2) (p25)	Homo sapiens (Human)						x					x	CP	68055113	1,28	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P80188	LCN2 HNL NGAL	Neutrophil gelatinase-associated lipocalin (NGAL) (25 kDa alpha-2-microglobulin-related subunit of MMP-9) (Lipocalin-2) (Oncogene 24p3) (Siderocalin LCN2) (p25)	Homo sapiens (Human)						x					x	CP	68055113	2,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics	x		23696425
P80188	LCN2 HNL NGAL	Neutrophil gelatinase-associated lipocalin (NGAL) (25 kDa alpha-2-microglobulin-related subunit of MMP-9) (Lipocalin-2) (Oncogene 24p3) (Siderocalin LCN2) (p25)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P80303	NUCB2 NEFA	Nucleobindin-2 (DNA-binding protein NEFA) (Gastric cancer antigen Zg4) (Preproenesfatin) [Cleaved into: Nesfatin-1]	Homo sapiens (Human)						x					x	CP	68055113	-1,42	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P80303	NUCB2 NEFA	Nucleobindin-2 (DNA-binding protein NEFA) (Gastric cancer antigen Zg4) (Preproenesfatin) (Cleaved into: Nesfatin-1]	Homo sapiens (Human)						x					x	CP	68055113	2,71	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P80419	0	Ig heavy chain V-III region GAR	Homo sapiens (Human)						x					x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P80511	S100A12	Protein S100-A12	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P80511	S100A12	Protein S100-A12 (CGRP) (Calcium-binding protein in amniotic fluid 1) (CAAF1) (Calgranulin-C) (CAGC) (Extracellular newly identified RAGE-binding protein) (EN-RAGE) (Migration inhibitory factor-related protein 6) (MRP-6) (p6) (Neutrophil S100 protein) (S100 calcium-binding protein A12) (Cleaved into: Calcitermin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
P80748	0	Ig lambda chain V-III region LOI	Homo sapiens (Human)					x						x	CP	68055113	1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P81605	DCD AIDD DSEP	Dermcidin	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
P81605	DCD AIDD DSEP	Dermcidin (EC 3.4.-.-) (Preproteolysin) [Cleaved into: Survival-promoting peptide; DCD-1]	Homo sapiens (Human)						x					x	CP	68055113	1,39	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P81605	DCD AIDD DSEP	Dermcidin (EC 3.4.-.-) (Preproteolysin) [Cleaved into: Survival-promoting peptide; DCD-1]	Homo sapiens (Human)						x					x	CP	68055113	+	36	M/F	All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. Since smoking is a risk factor for periodontal disease, the current study did not include smokers.	GCF was collected from the labial side of maxillary incisors without crown and restoration.	Western blot	on-Proteomics		x	22623421
P85298	ARHGAP8	Rho GTPase-activating protein 8 (Rho-type GTPase-activating protein 8)	Homo sapiens (Human)					x						x	CP	68055113	1,46	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P98160	HSPG2	Basement membrane-specific heparan sulfate proteoglycan core protein	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P98164	LRP2	Low-density lipoprotein receptor-related protein 2	Homo sapiens (Human)						x					x	CP	68055113	-1,12	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P99999	CYCS CYC	Cytochrome c	Homo sapiens (Human)					x						x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q00610	CLTC CLH17 CLTCL2 KIAA0034	Clathrin heavy chain 1 (Clathrin heavy chain on chromosome 17) (CLH-17)	Homo sapiens (Human)						x					x	CP	68055113	4,34	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q01459	CTBS CTB	Di-N-acetylchitobiase (EC 3.2.1.-)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q01469	FABP5	Fatty acid-binding protein, epidermal (Epidermal-type fatty acid-binding protein) (E-FABP) (Fatty acid-binding protein 5) (Psoriasis-associated fatty acid-binding protein homolog) (PA-FABP)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q01469	FABP5	Fatty acid-binding protein, epidermal (Epidermal-type fatty acid-binding protein) (E-FABP) (Fatty acid-binding protein 5) (Psoriasis-associated fatty acid-binding protein homolog) (PA-FABP)	Homo sapiens (Human)						x					x	CP	68055113	2,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q01469	FABP5	Fatty acid-binding protein, epidermal (Epidermal-type fatty acid-binding protein) (E-FABP) (Fatty acid-binding protein 5) (Psoriasis-associated fatty acid-binding protein homolog) (PA-FABP)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q01469	FABP5	Fatty acid-binding protein, epidermal (Epidermal-type fatty acid-binding protein) (E-FABP) (Fatty acid-binding protein 5) (Psoriasis-associated fatty acid-binding protein homolog) (PA-FABP)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q01518	CAP1 CAP	Adenylyl cyclase-associated protein 1 (CAP 1)	Homo sapiens (Human)					x						x	CP	68055113	1,78	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q01518	CAP1 CAP	Adenylyl cyclase-associated protein 1 (CAP 1)	Homo sapiens (Human)						x					x	CP	68055113	5,30				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q01518	CAP1 CAP	Adenylyl cyclase-associated protein 1 (CAP 1)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q01546	KRT76 KRT2B KRT2P	Keratin, type II cytoskeletal 2 oral	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q01546	KRT76 KRT2B KRT2P	Keratin, type II cytoskeletal 2 oral	Homo sapiens (Human)						x					x	CP	68055113	-1,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q01546	KRT76 KRT2B KRT2P	Keratin, type II cytoskeletal 2 oral (Cytokeratin-2P) (CK-2P) (K2P) (Keratin-76) (K76) (Type-II keratin Kb9)	Homo sapiens (Human)						x					x	CP	68055113	5,18	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q01546	KRT76 KRT2B KRT2P	Keratin, type II cytoskeletal 2 oral (Cytokeratin-2P) (CK-2P) (K2P) (Keratin-76) (K76) (Type-II keratin Kb9)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q01638	IL1RL1 DER4 ST2 T1	Interleukin-1 receptor-like 1 (Protein ST2)	Homo sapiens (Human)						x					x	CP	68055113	9,74	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q01814	ATP2B2 PMCA2	Plasma membrane calcium-transporting ATPase 2 (PMCA2) (EC 3.6.3.8) (Plasma membrane calcium ATPase isoform 2) (Plasma membrane calcium pump isoform 2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q02223	TNFRSF17 BCM BCMA	Tumor necrosis factor receptor superfamily, member 17	Homo sapiens (Human)							x					CP	68055113	,3(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm]) or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q02413	DSG1 CDHF4	Desmoglein-1 (Cadherin family member 4) (Desmosomal glycoprotein 1) (DG1) (DGI) (Pemphigus foliaceus antigen)	Homo sapiens (Human)					x						x	CP	68055113	-1,12	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q02413	DSG1 CDHF4	Desmoglein-1 (Cadherin family member 4) (Desmosomal glycoprotein 1) (DG1) (DGI) (Pemphigus foliaceus antigen)	Homo sapiens (Human)						x					x	CP	68055113	1,93	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q02487	DSC2 CDHF2 DSC3	Desmocollin-2 (Cadherin family member 2) (Desmocollin-3) (Desmosomal glycoprotein II) (Desmosomal glycoprotein III)	Homo sapiens (Human)					x						x	CP	68055113	-1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q02817	MUC2 SMUC	Mucin-2 (MUC-2) (Intestinal mucin-2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q02818	NUCB1 NUC	Nucleobindin-1 (CALNUC)	Homo sapiens (Human)					x						x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q04695	KRT17	Keratin, type I cytoskeletal 17	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q04724	TLE1	Transducin-like enhancer protein 1 (E(Sp1) homolog) (Enhancer of split groucho-like protein 1) (ESG1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q04760	GLO1	Lactoylglutathione lyase (EC 4.4.1.5) (Aldoketomutase) (Glyoxalase I) (Glx I) (Ketone-aldehyde mutase) (Methylglyoxalase) (S-D-lactoylglutathione methylglyoxal lyase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q04917	YWHAH YWHA1	14-3-3 protein eta	Homo sapiens (Human)						x						CP	68055113	3,00						Proteomics			24098404
Q05315	CLC LGALS10 LGALS10A	Galectin-10 (Gal-10) (Charcot-Leyden crystal protein) (CLC) (Eosinophil lysophospholipase) (Lysolecithin acylhydrolase)	Homo sapiens (Human)						x					x	CP	68055113	7,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q05639	EEF1A2 EEF1AL STN	Elongation factor 1-alpha 2 (EF-1-alpha-2) (Eukaryotic elongation factor 1 A-2) (eEF1A-2) (Statin-S1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q05707	COL14A1 UND	Collagen alpha-1(XIV) chain	Homo sapiens (Human)						x					x	CP	68055113	-2,33	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q05707	COL14A1 UND	Collagen alpha-1(XIV) chain	Homo sapiens (Human)						x					x	CP	68055113	-2,33	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q06830	PRDX1 PAGA PAGB TDPX2	Peroxiredoxin-1 (EC 1.11.1.15) (Natural killer cell-enhancing factor A) (NKEF-A) (Proliferation-associated gene protein) (PAG) (Thioredoxin peroxidase 2) (Thioredoxin-dependent peroxide reductase 2)	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q06830	PRDX1 PAGA PAGB TDPX2	Peroxiredoxin-1 (EC 1.11.1.15) (Natural killer cell-enhancing factor A) (NKEF-A) (Proliferation-associated gene protein) (PAG) (Thioredoxin peroxidase 2) (Thioredoxin-dependent peroxide reductase 2)	Homo sapiens (Human)						x					x	CP	68055113	1,76	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q06830	PRDX1 PAGA PAGB TDPX2	Peroxiredoxin-1 (EC 1.11.1.15) (Natural killer cell-enhancing factor A) (NKEF-A) (Proliferation-associated gene protein) (PAG) (Thioredoxin peroxidase 2) (Thioredoxin-dependent peroxide reductase 2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q06830	PRDX1 PAGA PAGB TDPX2	Peroxiredoxin-1 (EC 1.11.1.15) (Natural killer cell-enhancing factor A) (NKEF-A) (Proliferation-associated gene protein) (PAG) (Thioredoxin peroxidase 2) (Thioredoxin-dependent peroxide reductase 2)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q07654	TFF3 ITF TFI	Trefoil factor 3 (Intestinal trefoil factor) (hITF) (Polypeptide P1.B) (hP1.B)	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q07666	KHDRBS1 SAM68	KH domain-containing, RNA-binding, signal transduction-associated protein 1 (GAP-associated tyrosine phosphoprotein p62) (Src-associated in mitosis 68 kDa protein) (Sam68) (p21 Ras GTPase-activating protein-associated p62) (p68)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q08116	RGS1 1R20 BL34 IER1	Regulator of G-protein signaling 1	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q08188	TGM3	Protein-glutamine gamma-glutamyltransferase E (EC 2.3.2.13) (Transglutaminase E) (TG(E)) (TGE) (TGase E) (Transglutaminase-3) (TGase-3) [Cleaved into: Protein-glutamine gamma-glutamyltransferase E 50 kDa catalytic chain; Protein-glutamine gamma-glutamyltransferase E 27 kDa non-catalytic chain]	Homo sapiens (Human)					x						x	CP	68055113	1,30	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q08188	TGM3	Protein-glutamine gamma-glutamyltransferase E (EC 2.3.2.13) (Transglutaminase E) (TG(E)) (TGE) (TGase E) (Transglutaminase-3) (TGase-3) [Cleaved into: Protein-glutamine gamma-glutamyltransferase E 50 kDa catalytic chain; Protein-glutamine gamma-glutamyltransferase E 27 kDa non-catalytic chain]	Homo sapiens (Human)							x				x	CP	68055113	3,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q08188	TGM3	Protein-glutamine gamma-glutamyltransferase E (EC 2.3.2.13) (Transglutaminase E) (TG(E)) (TGE) (TGase E) (Transglutaminase-3) (TGase-3) [Cleaved into: Protein-glutamine gamma-glutamyltransferase E 50 kDa catalytic chain; Protein-glutamine gamma-glutamyltransferase E 27 kDa non-catalytic chain]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q08380	LGALS3BP M2BP	Galectin-3-binding protein (Basement membrane autoantigen p105) (Lectin galactoside-binding soluble 3-binding protein) (Mac-2-binding protein) (MAC2BP) (Mac-2 BP) (Tumor-associated antigen 90K)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q08380	LGALS3BP M2BP	Galectin-3-binding protein (Basement membrane autoantigen p105) (Lectin galactoside-binding soluble 3-binding protein) (Mac-2-binding protein) (MAC2BP) (Mac-2 BP) (Tumor-associated antigen 90K)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q08554	DSC1 CDHF1	Desmocollin 1	Homo sapiens (Human)							x					CP	68055113	,33(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q08AI8	C2orf54	Uncharacterized protein C2orf54	Homo sapiens (Human)							x				x	CP	68055113	34,01	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q09666	AHNAK PM227	Neuroblast differentiation-associated protein AHNAK	Homo sapiens (Human)						x					x	CP	68055113	1,01	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q0EFC9	TC4	TC4 protein	Homo sapiens (Human)						x					x	CP	68055113	3,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q0P5P4	TMSB4X	TMSB4X protein (fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q0VAA2	LRRC74A C14orf166B LRRC74	Uncharacterized protein C14orf166B	Homo sapiens (Human)						x					x	CP	68055113	1,24	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q10588	BST1	ADP-ribosyl cyclase 2 (EC 3.2.2.5) (Bone marrow stromal antigen 1) (BST-1) (Cyclic ADP-ribose hydrolase 2) (cADPr hydrolase 2) (CD antigen CD157)	Homo sapiens (Human)						x					x	CP	68055113	5,47	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q12830	BPTF FAC1 FALZ	Nucleosome-remodeling factor subunit BPTF	Homo sapiens (Human)						x					x	CP	68055113	2,92	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q12913	PTPRJ DEP1	Receptor-type tyrosine-protein phosphatase eta (Protein-tyrosine phosphatase eta) (R-PTP-eta) (EC 3.1.3.48) (Density-enhanced phosphatase 1) (DEP-1) (HPTP eta) (Protein-tyrosine phosphatase receptor type J) (R-PTP-J) (CD antigen CD148)	Homo sapiens (Human)						x					x	CP	68055113	2,50	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q13098	GPS1 COPS1 CSN1	COP9 signalosome complex subunit 1 (SGN1) (Signalosome subunit 1) (G protein pathway suppressor 1) (GPS-1) (JAB1-containing signalosome subunit 1) (Protein MFH)	Homo sapiens (Human)						x					x	CP	68055113	1,81	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q13237	PRKG2 PRKGR2	cGMP-dependent protein kinase 2 (cGK 2) (cGK2) (EC 2.7.11.12) (cGMP-dependent protein kinase II) (cGKII)	Homo sapiens (Human)						x					x	CP	68055113	6,81	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q13296	SCGB2A2 MGB1 UGB2	Mammaglobin-A (Mammaglobin-1) (Secretoglobin family 2A member 2)	Homo sapiens (Human)						x					x	CP	68055113	1,85	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q13423	NNT	NAD(P) transhydrogenase, mitochondrial (EC 1.6.1.2) (Nicotinamide nucleotide transhydrogenase) (Pyridine nucleotide transhydrogenase)	Homo sapiens (Human)					x						x	CP	68055113	1,29	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q13459	MYO9B MYR5	Unconventional myosin-IXb (Unconventional myosin-9b)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q13813	SPTAN1 NEAS SPTA2	Spectrin alpha chain, non-erythrocytic 1 (Alpha-II spectrin) (Fodrin alpha chain) (Spectrin, non-erythroid alpha subunit)	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q13835	PKP1	Plakophilin-1 (Band 6 protein) (B6P)	Homo sapiens (Human)						x						CP	68055113	+					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q13867	BLMH	Bleomycin hydrolase (BH) (BLM hydrolase) (BMH) (EC 3.4.22.40)	Homo sapiens (Human)						x					x	CP	68055113	5,92	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q13885	TUBB2A TUBB2	Tubulin beta-2A chain (Tubulin beta class IIa)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q13972	RASGRF1 CDC25 GNRP GRF1	Ras-specific guanine nucleotide-releasing factor 1	Homo sapiens (Human)						x					x	CP	68055113	-1,22	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q14008	CKAP5 KIAA0097	Cytoskeleton-associated protein 5 (Colonic and hepatic tumor overexpressed gene protein) (Ch-TOG)	Homo sapiens (Human)					x						x	CP	68055113	-1,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q14019	COTL1 CLP	Coactosin-like protein	Homo sapiens (Human)						x					x	CP	68055113	1,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q14116	IL18 IGIF IL1F4	Interleukin 18	Homo sapiens (Human)					x						x	CP	68055113	5,15	40-65	MF		Participants were asked to come to the laboratory at 07:00 am following an overnight fast, during which they were instructed not to eat, drink (except water), chew gum or brush teeth. Whole saliva samples were obtained by expectorating into polypropylene tubes. Patients were advised to rinse his or her mouth several times with water and then to relax for five minutes. Patients were asked to swallow to void the mouth of saliva and asked to lean their head forward over the polypropylene tube and funnel. Patients kept their mouth slightly open to allow saliva to drain into the tube.	ELISA; AST and ALT levels were analyzed on Roche p-800 modular system using the specific kits provided by the manufacturer.	on-proteomics			25345339
Q14134	TRIM29 ATDC	Tripartite motif-containing protein 29 (Ataxia telangiectasia group D-associated protein)	Homo sapiens (Human)						x					x	CP	68055113	4,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from colusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q14264	ERV3-1 ERV3	HERV-R_7q21.2 provirus ancestral Env polyprotein (ERV-3 envelope protein) (ERV3 envelope protein) (ERV3-1 envelope protein) (Envelope polyprotein) (HERV-R envelope protein) (ERV-R envelope protein) [Cleaved into: Surface protein (SU); Transmembrane protein (TM)]	Homo sapiens (Human)						x					x	CP	68055113	-1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q14508	WFDC2 HE4 WAP5	WAP four-disulfide core domain protein 2 (Epididymal secretory protein E4) (Major epididymis-specific protein E4) (Putative protease inhibitor WAP5)	Homo sapiens (Human)						x					x	CP	68055113	-1,41	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q14515	SPARCL1	SPARC-like protein 1 (High endothelial venule protein) (Hevin) (MAST 9)	Homo sapiens (Human)						x					x	CP	68055113	-1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309
Q14517	FAT1 CDHF7 FAT	Protocadherin Fat 1	Homo sapiens (Human)						x					x	CP	68055113	1,38	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q14517	FAT1 CDHF7 FAT	Protocadherin Fat 1 (Cadherin family member 7) (Cadherin-related tumor suppressor homolog) (Protein fat homolog) [Cleaved into: Protocadherin Fat 1, nuclear form]	Homo sapiens (Human)						x					x	CP	68055113	-1,35	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Roselta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q14532	KRT32 HHA2 HKA2 KRTHA2	Keratin, type I cuticular Ha2 (Hair keratin, type I Ha2) (Keratin-32) (K32)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q14563	SEMA3A SEMAD	Semaphorin-3A (Semaphorin III) (Sema III)	Homo sapiens (Human)					x						x	CP	68055113	-1,36	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q14624	ITIH4 IHRP ITIHL1 PK120 PRO1851	Inter-alpha-trypsin inhibitor heavy chain H4 (ITI heavy chain H4) (ITI-HC4) (Inter-alpha-inhibitor heavy chain 4) (Inter-alpha-trypsin inhibitor family heavy chain-related protein) (IHRP) (Plasma kallikrein sensitive glycoprotein 120) (Gp120) (PK-120) [Cleaved into: 70 kDa inter-alpha-trypsin inhibitor heavy chain H4; 35 kDa inter-alpha-trypsin inhibitor heavy chain H4]	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q14624	ITIH4 IHRP ITIHL1 PK120 PRO1851	Inter-alpha-trypsin inhibitor heavy chain H4 (ITI heavy chain H4) (ITI-HC4) (Inter-alpha-inhibitor heavy chain 4) (Inter-alpha-trypsin inhibitor family heavy chain-related protein) (IHRP) (Plasma kallikrein sensitive glycoprotein 120) (Gp120) (PK-120) [Cleaved into: 70 kDa inter-alpha-trypsin inhibitor heavy chain H4; 35 kDa inter-alpha-trypsin inhibitor heavy chain H4]	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q14687	GSE1 KIAA0182	Genetic suppressor element 1	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q14697	GANAB G2AN KIAA0088	Neutral alpha-glucosidase AB (EC 3.2.1.84) (Alpha-glucosidase 2) (Glucosidase II subunit alpha)	Homo sapiens (Human)					x						x	CP	68055113	-1,49	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q14832	GRM3 GPRC1C MGLUR3	Metabotropic glutamate receptor 3 (mGluR3)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q14839	CHD4	Chromodomain-helicase-DNA-binding protein 4 (CHD-4) (EC 3.6.4.12) (ATP-dependent helicase CHD4) (Mi-2 autoantigen 218 kDa protein) (Mi2-beta)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q14974	KPNB1 NTF97	Importin subunit beta-1 (Importin-90) (Karyopherin subunit beta-1) (Nuclear factor p97) (Pore targeting complex 97 kDa subunit) (PTAC97)	Homo sapiens (Human)						x					x	CP	68055113	1,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q14978	NOLC1 KIAA0035 NS5ATP13	Nucleolar and coiled-body phosphoprotein 1 (140 kDa nucleolar phosphoprotein) (Nopp140) (Hepatitis C virus NS5A-transactivated protein 13) (HCV NS5A-transactivated protein 13) (Nucleolar 130 kDa protein) (Nucleolar phosphoprotein p130)	Homo sapiens (Human)					x						x	CP	68055113	1,54	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q14995	NR1D2	Nuclear receptor subfamily 1, group D, member 2	Homo sapiens (Human)							x					CP	68055113	41(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q14999	CUL7 KIAA0076	Cullin-7 (CUL-7)	Homo sapiens (Human)						x					x	CP	68055113	3,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q149N8	SHPRH KIAA2023	E3 ubiquitin-protein ligase SHPRH	Homo sapiens (Human)						x					x	CP	68055113	1,39	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q14CN4	KRT72 K6IRS2 KB35 KRT6 KRT6IRS2	Keratin, type II cytoskeletal 72	Homo sapiens (Human)						x						CP	68055113	6,00						Proteomics			24098404
Q15080	NCF4 SH3PXD4	Neutrophil cytosol factor 4 (NCF-4) (Neutrophil NADPH oxidase factor 4) (SH3 and PX domain-containing protein 4) (p40-phox) (p40phox)	Homo sapiens (Human)						x					x	CP	68055113	3,60	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q15181	PPA1 IOPPP PP	Inorganic pyrophosphatase (EC 3.6.1.1) (Pyrophosphate phospho-hydrolase) (PPase)	Homo sapiens (Human)						x					x	CP	68055113	2,43	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q15185	PTGES3 P23 TEBP	Prostaglandin E synthase 3 (EC 5.3.99.3) (Cytosolic prostaglandin E2 synthase) (cPGES) (Hsp90 co-chaperone) (Progesterone receptor complex p23) (Telomerase-binding protein p23)	Homo sapiens (Human)							x					CP	68055113		35-71			The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q15283	RASA2 GAP1M RASGAP	Ras GTPase-activating protein 2 (GTPase-activating protein 1m) (GAP1m)	Homo sapiens (Human)						x					x	CP	68055113	3,91	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q15303	ERBB4 HER4	Receptor tyrosine-protein kinase erbB-4	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q15399	TLR1 KIAA0012	Toll-like receptor 1 (Toll/interleukin-1 receptor-like protein) (TIL) (CD antigen CD281)	Homo sapiens (Human)						x					x	CP	68055113	4,76	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q15477	SKIV2L DDX13 SKI2W SKIV2 W	Helicase SKI2W (Ski2) (EC 3.6.4.-) (Helicase-like protein) (HLP)	Homo sapiens (Human)					x						x	CP	68055113	-1,22	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q15669	RHOH ARHH TTF	ras homolog family member H	Homo sapiens (Human)							x					CP	68055113	33(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	sn-proteomics			24122488
Q15751	HERC1	Probable E3 ubiquitin-protein ligase HERC1 (EC 6.3.2.-) (HECT domain and RCC1-like domain-containing protein 1) (p532) (p619)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q15771	RAB30	RAB30, member RAS oncogene family	Homo sapiens (Human)							x					CP	68055113	26(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	sn-proteomics			24122488
Q15878	CACNA1E CACH6 CACNL1A6	Voltage-dependent R-type Ca+2 channel subunit α-1E	Homo sapiens (Human)						x					x	CP	68055113	1,28	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q15911	ZFHX3 ATBF1	Zinc finger homeobox protein 3	Homo sapiens (Human)						x					x	CP	68055113	-2,04	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q15942	ZYX	Zyxin (Zyxin-2)	Homo sapiens (Human)						x					x	CP	68055113	2,45	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q16094	0	Calnexin (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,90	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q16206	ENOX2 COVA1	Ecto-NOX disulfide-thiol exchanger 2 (APK1 antigen) (Cytosolic ovarian carcinoma antigen 1) (Tumor-associated hydroquinone oxidase) (tNOX) [Includes: Hydroquinone [NADH] oxidase (EC 1.-.-.-); Protein disulfide-thiol oxidoreductase (EC 1.-.-.-)]	Homo sapiens (Human)					x						x	CP	68055113	-1,66	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q16610	ECM1	Extracellular matrix protein 1 (Secretory component p85)	Homo sapiens (Human)					x						x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q16633	POU2AF1 OBF1	POU class 2 associating factor 1	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q16651	PRSS8	Prostasin (EC 3.4.21.-) (Channel-activating protease 1) (CAP1) (Serine protease 8) [Cleaved into: Prostasin light chain; Prostasin heavy chain]	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q16658	FSCN1 FAN1 HSN SNL	Fascin (55 kDa actin-bundling protein) (Singed-like protein) (p55)	Homo sapiens (Human)						x					x	CP	68055113	2,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q16658	FSCN1 FAN1 HSN SNL	Fascin (55 kDa actin-bundling protein) (Singed-like protein) (p55)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q16777	HIST2H2AC H2AFQ	Histone H2A type 2-C	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q16778	HIST2H2BE H2BFQ	Histone H2B type 2-E	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q16778	HIST2H2BE H2BFQ	Histone H2B type 2-E (Histone H2B-GL105) (Histone H2B.g) (H2B/g)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q16795	NDUFA9 NDUFS2L	NADH dehydrogenase [ubiquinone] 1 alpha subcomplex subunit 9, mitochondrial (Complex I-39kD) (CI-39kD) (NADH-ubiquinone oxidoreductase 39 kDa subunit)	Homo sapiens (Human)						x					x	CP	68055113	11,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q16851	UGP2 UGP1	UTP--glucose-1-phosphate uridylyltransferase (EC 2.7.7.9) (UDP-glucose pyrophosphorylase) (UDPGP) (UGPase)	Homo sapiens (Human)						x					x	CP	68055113	2,34	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q16881	TXNRD1 GRIM12 KDRF	Thioredoxin reductase 1, cytoplasmic (TR) (EC 1.8.1.9) (Gene associated with retinoic and interferon-induced mortality 12 protein) (GRIM-12) (Gene associated with retinoic and IFN-induced mortality 12 protein) (KM-102-derived reductase-like factor) (Thioredoxin reductase TR1)	Homo sapiens (Human)						x					x	CP	68055113	2,45	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q17R60	IMPG1 IPM150 SPACR	Interphotoreceptor matrix proteoglycan 1	Homo sapiens (Human)						x					x	CP	68055113	-1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q17RF5	C4orf26	Chromosome 4 open reading frame 26	Homo sapiens (Human)							x					CP	68055113	,65(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q17RG1	KCTD19	BTB/POZ domain-containing protein KCTD19	Homo sapiens (Human)						x					x	CP	68055113	3,59	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q17RN3	FAM98C	Protein FAM98C	Homo sapiens (Human)						x					x	CP	68055113	2,66	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q17RR3	PNLIPRP3	Pancreatic lipase-related protein 3	Homo sapiens (Human)							x					CP	68055113	,28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q2KHR3	QSER1	Glutamine and serine-rich protein 1	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q2M1Z3	ARHGAP31 CDGAP KIAA1204	Cdc42 GTPase-activating protein	Homo sapiens (Human)						x					x	CP	68055113	2,39	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q2TB90	HKDC1	Putative hexokinase HKDC1 (EC 2.7.1.1) (Hexokinase domain-containing protein 1)	Homo sapiens (Human)					x						x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q3LIC9	Nbla10236	Putative uncharacterized protein Nbla10236 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,32	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q3MIX3	ADCK5	Uncharacterized aarF domain-containing protein kinase 5 (EC 2.7.11.-)	Homo sapiens (Human)						x					x	CP	68055113	2,88	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q3SY84	KRT71 K6IRS1 KB34 KRT6IRS1	Keratin, type II cytoskeletal 71	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
Q3V6T2	CCDC88A APE GRDN KIAA1212	Girdin	Homo sapiens (Human)						x					x	CP	68055113	2,69	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q3ZTR9	BM009	Putative uncharacterized protein BM009	Homo sapiens (Human)						x					x	CP	68055113	5,22	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q49AJ0	FAM135B C8orf32	Protein FAM135B	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q4G0P3	HYDIN HYDIN1 KIAA1864	Hydrocephalus-inducing protein homolog	Homo sapiens (Human)					x						x	CP	68055113	1,36	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
P00751	CFB BF BFD	Complement factor B (Complement factor B preproprotein variant) (Fragment)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q53HE2	0	Triosephosphate isomerase (EC 5.3.1.1) (Fragment)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q53HG6	0	Cysteine-tRNA ligase isoform a variant (Fragment)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q562M3	ACT	Actin-like protein	Homo sapiens (Human)						x					x	CP	68055113	1,69	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q562R1	ACTBL2	Beta-actin-like protein 2	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404

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Q562R1	ACTBL2	Beta-actin-like protein 2 (Kappa-actin)	Homo sapiens (Human)						x					x	CP	68055113	31,42	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q562R1	ACTBL2	Beta-actin-like protein 2 (Kappa-actin)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q56G89	0	Serum albumin	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q58EX7	PLEKHG4 PRTPHN1	Puratrophin-1 (Pleckstrin homology domain-containing family G member 4) (PH domain-containing family G member 4) (Purkinje cell atrophy-associated protein 1)	Homo sapiens (Human)						x					x	CP	68055113	3,63	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q59ES1	0	Leukotriene A4 hydrolase variant (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q59GZ2	0	PLEK protein variant (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,21	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5CZC0	FSIP2	Fibrous sheath-interacting protein 2	Homo sapiens (Human)						x					x	CP	68055113	2,16	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5D862	FLG2 IFPS	Filaggrin family member 2	Homo sapiens (Human)							x					CP	68055113	.36(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q5D862	FLG2 IFPS	Filaggrin-2 (FLG-2) (Intermediate filament-associated and psoriasis-susceptibility protein) (Ifapsoriasin)	Homo sapiens (Human)						x					x	CP	68055113	2,95	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5J875	GUGU	GUGU gamma form	Homo sapiens (Human)						x					x	CP	68055113	1,26	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5JPJ9	DKFZp686D0114	Putative uncharacterized protein DKFZp686D0114	Homo sapiens (Human)						x					x	CP	68055113	3,74	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q5JR07	RHOC	Rho-related GTP-binding protein RhoC (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	3,49	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5JSH3	WDR44	WD repeat-containing protein 44	Homo sapiens (Human)						x					x	CP	68055113	1,23	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5JVS8	VIM	Vimentin (fragment)	Homo sapiens (Human)						x						CP	68055113	15,00						Proteomics			24098404
Q5M9N0	CCDC158	Coiled-coil domain-containing protein 158	Homo sapiens (Human)					x						x	CP	68055113	1,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q5NV90	V2-17	V2-17 protein (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q5QNW6	HIST2H2BF	Histone H2B type 2-F	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q5R372	RABGAP1L HHL KIAA0471	Rab GTPase-activating protein 1-like	Homo sapiens (Human)						x					x	CP	68055113	3,54	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5S007	LRRK2 PARK8	Leucine-rich repeat serine/threonine-protein kinase 2	Homo sapiens (Human)						x					x	CP	68055113	-1,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5SX90	GDI2	Rab GDP dissociation inhibitor beta (Fragment)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q5SYZ4	CRABP2	Cellular retinoic acid-binding protein 2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,95	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5SZK8	FREM2	FRAS1-related extracellular matrix protein 2	Homo sapiens (Human)						x					x	CP	68055113	-2,94	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5T011	SZT2 C1orf84 KIAA0467	Protein SZT2 (Seizure threshold 2 protein homolog)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q5T0Z8	C6orf132	Chromosome 6 open reading frame 132	Homo sapiens (Human)							x					CP	68055113	,26(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q5T123	SH3BGRL3	SH3 domain-binding glutamic acid-rich-like protein 3	Homo sapiens (Human)						x					x	CP	68055113	1,82	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5T1R4	HIVEP3 KBP1 KIAA1555 KRC ZAS3	Transcription factor HIVEP3	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5T3F8	TMEM63B C6orf110	Transmembrane protein 63B	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5T3N0	ANXA1	Annexin A1 (fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q5T3N1	ANXA1	Annexin A1 (fragment)	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q5T619	ZNF648	Zinc finger protein 648	Homo sapiens (Human)						x					x	CP	68055113	9,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5T6L9	ERMARD C6orf70	Transmembrane protein C6orf70	Homo sapiens (Human)						x					x	CP	68055113	1,55	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5T6W2	HNRNPK	Heterogeneous nuclear ribonucleoprotein K (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,71	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5T764	IFIT1B IFIT1L	Interferon-induced protein with tetratricopeptide repeats 1B (Interferon-induced protein with tetratricopeptide repeats 1-like protein)	Homo sapiens (Human)						x					x	CP	68055113	3,82	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5T7W0	ZNF618 KIAA1952	Zinc finger protein 618	Homo sapiens (Human)						x					x	CP	68055113	1,16	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5TB80	CEP162 C6orf84 KIAA1009 QN1	Centrosomal protein of 162 kDa (Cep162) (Protein QN1 homolog)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q5TF21	SOGA3 C6orf174	Protein SOGA3	Homo sapiens (Human)						x					x	CP	68055113	12,37	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5THJ4	VPS13D KIAA0453	Vacuolar protein sorting-associated protein 13D	Homo sapiens (Human)						x					x	CP	68055113	3,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q5THR3	EFCAB6 DJBP KIAA1672	EF-hand calcium-binding domain-containing protein 6	Homo sapiens (Human)						x					x	CP	68055113	-1,96	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5UGI6	SERPING1	Serine/cysteine proteinase inhibitor clade G member 1 splice variant 2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	1,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q5VTT5	MYOM3	Myomesin-3	Homo sapiens (Human)						x					x	CP	68055113	-7,14	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
P06753	TPM3	Tropomyosin 3	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
Q5VU61	TPM3	Tropomyosin 3	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
Q5VU65	NUP210L	Nuclear pore membrane glycoprotein 210-like	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5VUG0	SFMBT2 KIAA1617	Scm-like with four MBT domains protein 2	Homo sapiens (Human)						x					x	CP	68055113	-2,27	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5W041	ARMC3	Armadillo repeat-containing protein	Homo sapiens (Human)						x					x	CP	68055113	4,32	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q5XG99	LYSMD4	LysM and putative peptidoglycan-binding domain-containing protein 4	Homo sapiens (Human)					x						x	CP	68055113	-1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q5XKE5	KRT79 K8L KB38 KRT6L	Keratin, type II cytoskeletal 79	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q658L4	DKFZp666E157 hCG_2044009	Putative uncharacterized protein DKFZp666E157	Homo sapiens (Human)						x					x	CP	68055113	1,13	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q68CQ4	DIEXF C1orf107 DEF	Digestive organ expansion factor homolog	Homo sapiens (Human)						x					x	CP	68055113	1,72	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q68CR1	SEL1L3 KIAA0746	sel-1 suppressor of lin-12-like 3 (C. elegans)	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q6E213	AWAT2 DC4 DGAT2L4 MFAT WS	Acyl-CoA wax alcohol acyltransferase 2 (EC 2.3.1.75) (Acyl-CoA retinol O-fatty-acyltransferase) (ARAT) (Retinol O-fatty-acyltransferase) (EC 2.3.1.76) (Diacylglycerol O-acyltransferase 2-like protein 4) (Diacylglycerol O-acyltransferase candidate 4) (hDC4) (Long-chain-alcohol O-fatty-acyltransferase 2) (Multifunctional O-acyltransferase) (Wax synthase) (hWS)	Homo sapiens (Human)						x					x	CP	68055113	3,66	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6FHJ7	SFRP4 FRPHE	Secreted frizzled-related protein 4	Homo sapiens (Human)							x					CP	68055113	,3(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q6FI13	HIST2H2AA3 H2AFO HIST2H2AA; HIST2H2	Histone H2A type 2-A	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q6I929	pseudoTPMT hCG_1978654	HCG1978654 (Thiopurine methyltransferase processed pseudogene) (EC 2.1.1.67)	Homo sapiens (Human)						x					x	CP	68055113	7,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q6IBS0	TWF2 PTK9L MSTP011	Twinfilin-2 (Aß-related protein) (hA6RP) (Protein tyrosine kinase 9-like) (Twinfilin-1-like protein)	Homo sapiens (Human)						x					x	CP	68055113	3,55	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6IQ22	RAB12	Ras-related protein Rab-12	Homo sapiens (Human)					x						x	CP	68055113	1,60	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6IQ55	TTBK2 KIAA0847	Tau-tubulin kinase 2	Homo sapiens (Human)						x					x	CP	68055113	-1,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q6LCG8	0	Catenin-4 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	13,58	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6MZM9	PRR27 C4orf40	Uncharacterized protein C4orf40	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6MZU6	DKFZp686C15213	Putative uncharacterized protein DKFZp686C15213	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q6N030	DKFZp686I15212	Putative uncharacterized protein DKFZp686I15212	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q6N095	DKFZp686K03196	Putative uncharacterized protein DKFZp686K03196	Homo sapiens (Human)						x					x	CP	68055113	2,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6NS95	IGL@	IGL@ protein	Homo sapiens (Human)						x					x	CP	68055113	1,11	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6NS95	IGL@	IGL@ protein	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q6NSX1	CCDC70	Coiled-coil domain-containing protein 70	Homo sapiens (Human)						x					x	CP	68055113	5,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6NVV3	NIPAL1 NIPA3 NPAL1	Magnesium transporter NIPA3 (NIPA-like protein 1) (Non-imprinted in Prader-Willi/Angelman syndrome region protein 3)	Homo sapiens (Human)					x						x	CP	68055113	-1,31	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6P179	ERAP2 LRAP	Endoplasmic reticulum aminopeptidase 2	Homo sapiens (Human)							x					CP	68055113	,39(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q6P4A8	PLBD1	Phospholipase B-like 1 (EC 3.1.1.-) (LAMA-like protein 1) (Lamina ancestor homolog 1) (Phospholipase B domain-containing protein 1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q6P4F7	ARHGAP11A KIAA0013	Rho GTPase-activating protein 11A	Homo sapiens (Human)						x					x	CP	68055113	-1,01	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q6P5S2	LEG1 C6orf58	UPF0762 protein C6orf58	Homo sapiens (Human)					x						x	CP	68055113	-1,32	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6P5S2	LEG1 C6orf58	UPF0762 protein C6orf58	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8P9A3	ZNF549	Zinc finger protein 549	Homo sapiens (Human)						x					x	CP	68055113	32,56	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6PIF6	MYO7B	Unconventional myosin-VIIb	Homo sapiens (Human)					x						x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6R327	RICTOR KIAA1999	Rapamycin-insensitive companion of mTOR (AVO3 homolog) (hAVO3)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6S8J3	POTEE A26C1A POTE2	ANKRD26-like family C member 1A	Homo sapiens (Human)						x					x	CP	68055113	1,36	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q6S8J3	POTEE A26C1A POTE2	POTE ankyrin domain family member E	Homo sapiens (Human)						x						CP	68055113	15,00						Proteomics			24098404
Q6TBL4	ABCB1	P-glycoprotein 1 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	46,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6UQ28	PLET1 C11orf34	Placenta-expressed transcript 1 protein	Homo sapiens (Human)					x						x	CP	68055113	1,48	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6UWI2	PARM1 UNQ1879/PRO4322	Prostate androgen-regulated mucin-like protein 1	Homo sapiens (Human)							x					CP	68055113	27(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q6UWP8	SBSN UNQ698/PRO1343	Suprabasin	Homo sapiens (Human)						x					x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6UX06	OLFM4 GW112 UNQ362/PRO698	Olfactomedin-4 (OLM4) (Antiapoptotic protein GW112) (G-CSF-stimulated clone 1 protein) (hGC-1) (hOLfD)	Homo sapiens (Human)						x					x	CP	68055113	2,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6UX68	XKR5 XRG5 UNQ2754/PRO6493	XK-related protein 5	Homo sapiens (Human)						x					x	CP	68055113	7,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6UXG3	CD300LG CLM9 TREM4 UNQ422/PRO846	CMRF35-like molecule 9 (CLM-9) (CD300 antigen-like family member G) (Triggering receptor expressed on myeloid cells 4) (TREM-4) (CD antigen CD300g)	Homo sapiens (Human)						x					x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q6UXZ0	TMIGD1 TMIGD UNQ9372/PRO34164	Transmembrane and immunoglobulin domain-containing protein 1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q6XE38	SCGB1D4 UNQ517/PRO812	Secretoglobin family 1D member 4 (IFN-gamma-inducible secretoglobin) (IIS)	Homo sapiens (Human)						x					x	CP	68055113	1,51	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6YI46	TMEM64	Transmembrane protein 64	Homo sapiens (Human)					x						x	CP	68055113	1,51	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZN30	BNC2	Zinc finger protein basonuclin-2	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZNI0	GCNT7 C20orf105	Beta-1,3-galactosyl-O-glycosyl-glycoprotein beta-1,6-N-acetylglucosaminyltransferase 7 (EC 2.4.1.-)	Homo sapiens (Human)					x						x	CP	68055113	1,53	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZNM4	0	cDNA FLJ29005 fis, clone TST07872	Homo sapiens (Human)						x					x	CP	68055113	2,41	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6ZNW9	0	CDNA FLJ26980 fis, clone SLV02741	Homo sapiens (Human)						x					x	CP	68055113	5,93	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6ZRS2	SRCAP KIAA0309	Helicase SRCAP (EC 3.6.4.-) (Domino homolog 2) (Snf2-related CBP activator)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZS10	CLEC17A	C-type lectin domain family 17, member A (Prolectin)	Homo sapiens (Human)					x						x	CP	68055113	-1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZS99	0	cDNA FLJ45706 fis, clone FEBRA2028457, highly similar to Nucleolin	Homo sapiens (Human)						x					x	CP	68055113	4,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6ZSZ6	TSHZ1 SDCCAG33 TSH1	Teashirt homolog 1	Homo sapiens (Human)						x					x	CP	68055113	-1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q6ZUT9	DENND5B	DENN domain-containing protein 5B (Rab6IP1-like protein)	Homo sapiens (Human)						x					x	CP	68055113	23,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6ZVT3	FGD6 KIAA1362 ZFYVE24	FYVE, RhoGEF and PH domain-containing protein 6 (Zinc finger FYVE domain-containing protein 24)	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q6ZVX7	NCCRP1 FBXO50	F-box only protein 50 (NCC receptor protein 1 homolog) (NCCRP-1) (Non-specific cytotoxic cell receptor protein 1 homolog)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q6ZWH5	NEK10	Serine/threonine-protein kinase Nek10 (EC 2.7.11.1) (Never in mitosis A-related kinase 10) (NimA-related protein kinase 10)	Homo sapiens (Human)						x					x	CP	68055113	2,21	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q6ZXV5	TMTC3	Transmembrane and TPR repeat-containing protein 3	Homo sapiens (Human)						x					x	CP	68055113	8,68	22-61	MF		Gingival crevicular fluid was collected from 9·00 am to 12·00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q71DI3	HIST2H3A; HIST2H3C H3F2 H3FM; HIST2H3	Histone H3.2	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q71DI3	HIST2H3A; HIST2H3C H3F2 H3FM; HIST2H3	Histone H3.2 (Histone H3/m) (Histone H3/o)	Homo sapiens (Human)						x					x	CP	68055113	2,36	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q71DI3	HIST2H3A; HIST2H3C H3F2 H3FM; HIST2H3	Histone H3.2 (Histone H3/m) (Histone H3/o)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q71UI9	H2AFV H2AV	Histone H2A.V	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
Q75MP7	MDH2	Putative uncharacterized protein MDH2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q75MY0	ARPC1A	Putative uncharacterized protein ARPC1A (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	4,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q76L83	ASXL2 ASXH2 KIAA1685	Putative Polycomb group protein ASXL2 (Additional sex combs-like protein 2)	Homo sapiens (Human)						x					x	CP	68055113	1,08	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q7KZN9	COX15	Cytochrome c oxidase assembly protein COX15 homolog	Homo sapiens (Human)						x					x	CP	68055113	28,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7L7L0	HIST3H2A	Histone H2A type 3	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q7RTS7	KRT74 K6IRS4 KB37 KRT5C KRT6IRS4	Keratin, type II cytoskeletal 74	Homo sapiens (Human)						x						CP	68055113	2,00						Proteomics			24098404
Q7RTV2	GSTA5	Glutathione S-transferase A5 (EC 2.5.1.18) (GST class-alpha member 5) (Glutathione S-transferase A5-5)	Homo sapiens (Human)						x					x	CP	68055113	11,38	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7Z2Y8	GVINP1 GVIN1 VLIG1	Interferon-induced very large GTPase 1	Homo sapiens (Human)						x					x	CP	68055113	1,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q7Z2Z5	DKFZp686P23184	Putative uncharacterized protein DKFZp686P23184	Homo sapiens (Human)						x					x	CP	68055113	3,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7Z351	DKFZp686N02209	Putative uncharacterized protein DKFZp686N02209	Homo sapiens (Human)						x					x	CP	68055113	1,90	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7Z3Y7	KRT28 KRT25D	Keratin, type I cytoskeletal 28	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q7Z3Y8	KRT27 KRT25C	Keratin, type I cytoskeletal 27	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q7Z3Z0	KRT25 KRT25A	Keratin, type I cytoskeletal 25	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q7Z478	DHX29 DDX29	ATP-dependent RNA helicase DHX29 (EC 3.6.4.13) (DEAH box protein 29) (Nucleic acid helicase DDXx)	Homo sapiens (Human)						x					x	CP	68055113	4,41	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7Z5P9	MUC19	Mucin-19	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q7Z6R9	TFAP2D TFAP2BL1	Transcription factor AP-2-delta (AP2-delta) (Activating enhancer-binding protein 2-delta) (Transcription factor AP-2-beta-like 1)	Homo sapiens (Human)						x					x	CP	68055113	2,59	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q7Z794	KRT77 KRT1B	Keratin, type II cytoskeletal 1b	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q7Z7A1	CNTRL CEP1 CEP110	Centriolin	Homo sapiens (Human)						x					x	CP	68055113	1,09	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics				24738839
Q86T29	ZNF605	Zinc finger protein 605	Homo sapiens (Human)						x					x	CP	68055113	4,06	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics				23696425
Q86TC9	MYPN MYOP	Myopalladin (145 kDa sarcomeric protein)	Homo sapiens (Human)						x					x	CP	68055113	5,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics				23696425
Q86UP0	CDH24 CDH11L UNQ2834/PRO34009	Cadherin-24	Homo sapiens (Human)						x					x	CP	68055113	1,62	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics				24738839
Q86UP2	KTN1 CG1 KIAA0004	Kinectin (CG-1 antigen) (Kinesin receptor)	Homo sapiens (Human)						x					x	CP	68055113	1,53	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics				23696425
Q86UP3	ZFHX4	Zinc finger homeobox protein 4	Homo sapiens (Human)						x					x	CP	68055113	1,14	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics				24738839
Q86UP6	CUZD1 UNQ224/PRO257	CUB and zona pellucida-like domain-containing protein 1 (CUB and ZP domain-containing protein 1) (Transmembrane protein UO-44)	Homo sapiens (Human)						x					x	CP	68055113	12,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics				23696425
Q86UQ4	ABCA13	ATP-binding cassette sub-family A member 13	Homo sapiens (Human)						x					x	CP	68055113	1,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics				24738839
Q86VF7	NRAP	Nebulin-related-anchoring protein (N-RAP)	Homo sapiens (Human)						x					x	CP	68055113	1,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics				23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q86VH2	KIF27	Kinesin-like protein KIF27	Homo sapiens (Human)						x					x	CP	68055113	2,08	46,3	M/F		All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q86VP1	TAX1BP1 T6BP PRO0105	Tax1-binding protein 1 (TRAF6-binding protein)	Homo sapiens (Human)					x						x	CP	68055113	-1,28	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q86W26	NLRP10 NALP10 NOD8 PYNOD	NACHT, LRR and PYD domains-containing protein 10 (Nucleotide-binding oligomerization domain protein 8)	Homo sapiens (Human)						x					x	CP	68055113	1,02	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q86WP2	GPBP1 GPBP SSH6	Vasculin (GC-rich promoter-binding protein 1) (Vascular wall-linked protein)	Homo sapiens (Human)						x					x	CP	68055113	2,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q86XP1	DGKH	Diacylglycerol kinase eta (DAG kinase eta) (EC 2.7.1.107) (Diglyceride kinase eta) (DGK-eta)	Homo sapiens (Human)					x						x	CP	68055113	-1,35	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q86Y46	KRT73 K6IRS3 KB36 KRT6IRS3	Keratin, type II cytoskeletal 73	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q86YH2	ZNF280B SUHW2 ZNF279 ZNF632	Zinc finger protein 280B	Homo sapiens (Human)						x					x	CP	68055113	-1,52	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q86YT5	SLC13A5 NACT	Solute carrier family 13 (sodium-dependent citrate transporter), member 5	Homo sapiens (Human)							x					CP	68055113	,32(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q86YZ3	HRNR S100A18	Hornerin	Homo sapiens (Human)						x					x	CP	68055113	4,50			isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q86Z22	HEL-S-297	Putative uncharacterized protein	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8IUE6	HIST2H2AB	Histone H2A type 2-B	Homo sapiens (Human)						x					x	CP	68055113						Proteomics			24098404
P02768	ALB GIG20 GIG42 PRO0903 PRO1708 PRO	ALB protein	Homo sapiens (Human)						x					x	CP	68055113						Proteomics			24098404
Q8IUL8	CILP2	Cartilage intermediate layer protein 2 (CILP-2) [Cleaved into: Cartilage intermediate layer protein 2 C1; Cartilage intermediate layer protein 2 C2]	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8IVF2	AHNAK2 C14orf78 KIAA2019	AHNK2 HUMAN Protein AHNAK2	Homo sapiens (Human)						x					x	CP	68055113	1,24	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IVF2	AHNAK2 C14orf78 KIAA2019	Protein AHNAK2	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8IVF4	DNAH10 KIAA2017	Dynein heavy chain 10	Homo sapiens (Human)						x					x	CP	68055113	1,29	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IVL0	NAV3 KIAA0938 POMFIL1 STEERIN3	Neuron navigator 3	Homo sapiens (Human)						x					x	CP	68055113	1,17	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IVL1	NAV2 HELAD1 KIAA1419 POMFIL2 RAINB1	Neuron navigator 2	Homo sapiens (Human)						x					x	CP	68055113	-1,96	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IVV2	LOXHD1	Lipoxygenase homology domain-containing protein 1	Homo sapiens (Human)					x						x	CP	68055113	1,55	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q8IWV1	LAX1 LAX	Lymphocyte transmembrane adaptor 1	Homo sapiens (Human)							x					CP	68055113	36(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	tm-proteomics			24122488
Q8IWV7	UBR1	E3 ubiquitin-protein ligase UBR1 (EC 6.3.2.-) (N-recognin-1) (Ubiquitin-protein ligase E3-alpha-1) (Ubiquitin-protein ligase E3-alpha-I)	Homo sapiens (Human)					x						x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8IWY7	TTBK2	Tau-tubulin kinase	Homo sapiens (Human)							x				x	CP	68055113	4,67	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8IWZ8	SUGP1 SF4	SURP and G-patch domain-containing protein 1 (RNA-binding protein RBP) (Splicing factor 4)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8IX01	SUGP2 KIAA0365 SFRS14	SURP and G-patch domain-containing protein 2 (Arginine/serine-rich-splicing factor 14) (Splicing factor, arginine/serine-rich 14)	Homo sapiens (Human)					x						x	CP	68055113	1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8IX19	MCEMP1 C19orf59	Mast cell-expressed membrane protein 1	Homo sapiens (Human)							x				x	CP	68055113	3,11	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8IXL6	FAM20C DMP4	Extracellular serine/threonine protein kinase FAM20C (EC 2.7.11.1) (Dentin matrix protein 4) (DMP-4) (Golgi-enriched fraction casein kinase) (GEF-CK) (Protein FAM20C)	Homo sapiens (Human)					x						x	CP	68055113	-1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8IXQ6	PARP9 BAL BAL1	Poly [ADP-ribose] polymerase 9 (PARP-9) (EC 2.4.2.30) (ADP-ribosyltransferase diphtheria toxin-like 9) (ARTD9) (B aggressive lymphoma protein)	Homo sapiens (Human)							x				x	CP	68055113	1,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8IYT8	ULK2 KIAA0623	Serine/threonine-protein kinase ULK2 (EC 2.7.11.1) (Unc-51-like kinase 2)	Homo sapiens (Human)							x				x	CP	68055113	2,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8IYV9	IZUMO1	Izumo sperm-egg fusion protein 1 (Oocyte binding/fusion factor) (OBF) (Sperm-specific protein izumo)	Homo sapiens (Human)							x				x	CP	68055113	8,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included (GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8IZC6	COL27A1 KIAA1870	Collagen alpha-1(XXVII) chain	Homo sapiens (Human)							x				x	CP	68055113	-1,39	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IZF6	ADGRG4 GPR112	Probable G-protein coupled receptor 112	Homo sapiens (Human)							x				x	CP	68055113	-1,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8IZJ1	UNC5B P53RDL1 UNC5H2 UNQ1883/PRO4	Netrin receptor UNC5B (Protein unc-5 homolog 2) (Protein unc-5 homolog B) (p53-regulated receptor for death and life protein 1)	Homo sapiens (Human)							x				x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q8IZS6	TCTE3 TCTEX1D3	Tctex1 domain-containing protein 3 (T-complex testis-specific protein 3) (T-complex-associated testis-expressed protein 3) (Tcte-3)	Homo sapiens (Human)						x					x	CP	68055113	4,22	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8N130	SLC34A3 NPT2C NPTIIC	Sodium-dependent phosphate transport protein 2C (Sodium-phosphate transport protein 2C) (Na(+)-dependent phosphate cotransporter 2C) (Sodium/inorganic phosphate cotransporter IIC) (Sodium/phosphate cotransporter 2C) (Na(+)/PI cotransporter 2C) (NaPi-2c) (Solute carrier family 34 member 3)	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8N1A0	KRT222 KA21 KRT222P	Keratin-like protein KRT222 (Keratin-222) (Keratin-222 pseudogene)	Homo sapiens (Human)					x						x	CP	68055113	1,62	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8N1N4	KRT78 K5B KB40	Keratin, type II cytoskeletal 78 (Cytokeratin-78) (CK-78) (Keratin-5b) (Keratin-78) (K78) (Type-II keratin Kb40)	Homo sapiens (Human)						x					x	CP	68055113	3,34	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8N201	INTS1 KIAA1440 UNQ1821/PRO3434	Integrator complex subunit 1 (Int1)	Homo sapiens (Human)						x					x	CP	68055113	46,81	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8N257	HIST3H2BB	Histone H2B type 3-B	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q8N3Z0	PRSS35 C6orf158 UNQ522/PRO1057	Protease, serine, 35	Homo sapiens (Human)							x					CP	68055113	32(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q8N4F0	BPIFB2 BPIL1 C20orf184 LPLUNC2 UNQ248	BPI fold-containing family B member 2 (Bactericidal/permeability-increasing protein-like 1) (BPI-like 1) (Long palate, lung and nasal epithelium carcinoma-associated protein 2) (RYSR)	Homo sapiens (Human)					x						x	CP	68055113	-1,08	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8N4F0	BPIFB2 BPIL1 C20orf184 LPLUNC2 UNQ248	BPI fold-containing family B member 2 (Bactericidal/permeability-increasing protein-like 1) (BPI-like 1) (Long palate, lung and nasal epithelium carcinoma-associated protein 2) (RYSR)	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8N4G4	CA6	CA6 protein (Carbonic anhydrase 6)	Homo sapiens (Human)						x					x	CP	68055113	9,47	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8N4N8	KIF2B	Kinesin-like protein KIF2B	Homo sapiens (Human)						x					x	CP	68055113	2,69	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8N614	TMEM156	Transmembrane protein 156	Homo sapiens (Human)							x					CP	68055113	27(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q8N655	C10orf12	Uncharacterized protein C10orf12	Homo sapiens (Human)						x					x	CP	68055113	2,80	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8N7Z2	GOLGA6L1	Golgin subfamily A member 6-like protein 1	Homo sapiens (Human)					x						x	CP	68055113	1,31	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8NA04	0	cDNA FLJ35984 fis, clone TESTI2014097, highly similar to V_segment translation product	Homo sapiens (Human)						x					x	CP	68055113	6,87	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8NB25	FAM184A C6orf60	Protein FAM184A	Homo sapiens (Human)					x						x	CP	68055113	1,48	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8NB66	UNC13C	Protein unc-13 homolog C (Munc13-3)	Homo sapiens (Human)							x				x	CP	68055113	4,27	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8NBJ4	GOLM1 C9orf155 GOLPH2 PSEC0242 UNQ6	Golgi membrane protein 1 (Golgi membrane protein GP73) (Golgi phosphoprotein 2)	Homo sapiens (Human)					x						x	CP	68055113	-1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8NCL6	0	cDNA FLJ90170 fis, clone MAMMA1000370, highly similar to Ig alpha-1 chain C region	Homo sapiens (Human)							x				x	CP	68055113	3,51	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8NCM8	DYNC2H1 DHC1B DHC2 DNCH2 DYH1B KIA	Cytoplasmic dynein 2 heavy chain 1 (Cytoplasmic dynein 2 heavy chain) (Dynein cytoplasmic heavy chain 2) (Dynein heavy chain 11) (hDHC11) (Dynein heavy chain isotype 1B)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8NFQ5	BPIFB6 BPIL3	BPI fold-containing family B member 6 (Bactericidal/permeability-increasing protein-like 3)	Homo sapiens (Human)						x					x	CP	68055113	4,52	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8NFW1	COL22A1	Collagen alpha-1(XXII) chain	Homo sapiens (Human)						x					x	CP	68055113	1,46	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8NFY9	KBTBD8 KIAA1842 TAKRP	Kelch repeat/BTB domain-containing protein 8	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8NG31	CASC5 KIAA1570 KNL1	Protein CASC5	Homo sapiens (Human)						x					x	CP	68055113	1,06	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q8NG94	OR11H1	Olfactory receptor 11H1	Homo sapiens (Human)						x					x	CP	68055113	1,08	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8NHU2	CFAP61 C20orf26	Uncharacterized protein C20orf26	Homo sapiens (Human)						x					x	CP	68055113	5,28	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8NI98	OK/SW-cl.5	Cytoskeletal tropomyosin TM30	Homo sapiens (Human)						x					x	CP	68055113	1,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8TAQ5	ZNF420	Zinc finger protein 420	Homo sapiens (Human)					x						x	CP	68055113	1,51	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8TAT5	NEIL3	Endonuclease 8-like 3 (EC 3.2.2.-) (EC 4.2.99.18) (DNA glycosylase FPG2) (DNA glycosylase/AP lyase Neil3) (Endonuclease VIII-like 3) (Nei-like protein 3)	Homo sapiens (Human)					x						x	CP	68055113	1,66	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8TAX7	MUC7 MG2	Mucin-7 (MUC-7) (Apo-MG2) (Salivary mucin-7)	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8TAX7	MUC7 MG2	Mucin-7 (MUC-7) (Apo-MG2) (Salivary mucin-7)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8TCG1	KIAA1524 CIP2A	Protein CIP2A (Cancerous inhibitor of PP2A) (p90 autoantigen)	Homo sapiens (Human)							x				x	CP	68055113	3,59	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8TD26	CHD6 CHD5 KIAA1335 RIGB	Chromodomain-helicase-DNA-binding protein 6	Homo sapiens (Human)						x					x	CP	68055113	1,48	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8TD57	DNAH3 DNAHC3B	Dynein heavy chain 3, axonemal (Axonemal beta dynein heavy chain 3) (HsADHC3) (Ciliary dynein heavy chain 3) (Dnahc3-b)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8TE73	DNAH5 DNAHC5 HL1 KIAA1603	Dynein heavy chain 5	Homo sapiens (Human)						x					x	CP	68055113	-1,02	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8TEP8	CEP192 KIAA1569 PP8407	Centrosomal protein of 192 kDa (Cep192)	Homo sapiens (Human)					x						x	CP	68055113	-1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8TEY4	APBB1	Adaptor protein FE65a2 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	27,94	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8WU39	MZB1 MEDA7 PACAP HSPC190	Marginal zone B and B1 cell-specific protein	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q8WUM4	PDCD6IP AIP1 ALIX KIAA1375	Programmed cell death 6-interacting protein (PDCD6-interacting protein) (ALG-2-interacting protein 1) (ALG-2-interacting protein X) (Hp95)	Homo sapiens (Human)						x					x	CP	68055113	2,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8WVQ1	CANT1 SHAPY	Soluble calcium-activated nucleotidase 1 (SCAN-1) (EC 3.6.1.6) (Apyrase homolog) (Putative MAPK-activating protein PM09) (Putative NF-kappa-B-activating protein 107)	Homo sapiens (Human)					x						x	CP	68055113	-1,17	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q8WVZ9	KBTBD7	Kelch repeat and BTB domain-containing protein 7	Homo sapiens (Human)						x					x	CP	68055113	-1,22	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WWJ8	0	Integrin beta	Homo sapiens (Human)						x					x	CP	68055113	3,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8WXH0	SYNE2 KIAA1011 NUA	Nesprin-2	Homo sapiens (Human)						x					x	CP	68055113	2,04	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WXH0	SYNE2 KIAA1011 NUA	Nesprin-2 (Nuclear envelope spectrin repeat protein 2) (Nucleus and actin connecting element protein) (Protein NUANCE) (Synaptic nuclear envelope protein 2) (Syne-2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8WXI7	MUC16 CA125	Mucin-16	Homo sapiens (Human)						x					x	CP	68055113	1,14	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WXX0	DNAH7 KIAA0944	Dynein heavy chain 7, axonemal	Homo sapiens (Human)						x					x	CP	68055113	-1,05	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WXX0	DNAH7 KIAA0944	Dynein heavy chain 7, axonemal (Axonemal beta dynein heavy chain 7) (Ciliary dynein heavy chain 7) (Dynein heavy chain-like protein 2) (hDHC2)	Homo sapiens (Human)						x					x	CP	68055113	6,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q8WY64	MYLIP BZF1 IDOL BM-023 PP5242	E3 ubiquitin-protein ligase MYLIP	Homo sapiens (Human)						x					x	CP	68055113	1,62	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WZ42	TTN	Titin	Homo sapiens (Human)						x					x	CP	68055113	2,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q8WZ42	TTN	Titin (EC 2.7.11.1) (Connectin) (Rhabdomyosarcoma antigen MU-RMS-40.14)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q8WZ64	ARAP2 CENTD1 KIAA0580	Arf-GAP with Rho-GAP domain, ANK repeat and PH domain-containing protein 2 (Centaurin-delta-1) (Cnt-d1) (Protein PARX)	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q92616	GCN1L1 KIAA0219	Translational activator GCN1 (HsGCN1) (GCN1-like protein 1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q92621	NUP205 C7orf14 KIAA0225	Nuclear pore complex protein Nup205	Homo sapiens (Human)						x					x	CP	68055113	1,32	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q92736	RYR2	Ryanodine receptor 3	Homo sapiens (Human)						x					x	CP	68055113	1,09	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q92738	USP6NL KIAA0019	USP6 N-terminal-like protein (Related to the N-terminus of tre) (RN-tre)	Homo sapiens (Human)						x					x	CP	68055113	10,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q92841	DDX17	DEAD (Asp-Glu-Ala-Asp) box helicase 17	Homo sapiens (Human)							x					CP	68055113	,24(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q92896	GLG1 CFR1 ESL1 MG160	Golgi apparatus protein 1 (CFR-1) (Cysteine-rich fibroblast growth factor receptor) (E-selectin ligand 1) (ESL-1) (Golgi sialoglycoprotein MG-160)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q92900	UPF1 KIAA0221 RENT1	Regulator of nonsense transcripts 1 (EC 3.6.4. -) (ATP-dependent helicase RENT1) (Nonsense mRNA reducing factor 1) (NORF1) (Up-frameshift suppressor 1 homolog) (hUpf1)	Homo sapiens (Human)						x					x	CP	68055113	5,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q93073	SECISBP2L KIAA0256	Selenocysteine insertion sequence-binding protein 2-like (SECIS-binding protein 2-like)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q93077	HIST1H2AC H2AFL	Histone H2A type 1-C	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q93079	HIST1H2BH H2BFJ	Histone H2B type 1-H	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q969H8	MYDGF C19orf10 IL25	UPF0556 protein C19orf10 (Interleukin-25) (IL-25) (Stromal cell-derived growth factor SF20)	Homo sapiens (Human)					x						x	CP	68055113	1,10	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96A08	HIST1H2BA TSH2B	Histone H2B type 1-A	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q96A08	HIST1H2BA TSH2B	Histone H2B type 1-A	Homo sapiens (Human)						x					x	CP	68055113	1,34	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96AG3	SLC25A46 TB1	Solute carrier family 25 member 46	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96AM7	0	SOD2 protein	Homo sapiens (Human)						x					x	CP	68055113	2,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96DA0	ZG16B UNQ773/PRO1567	Zymogen granule protein 16 homolog B	Homo sapiens (Human)					x						x	CP	68055113	-1,70	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96DA0	ZG16B UNQ773/PRO1567	Zymogen granule protein 16 homolog B	Homo sapiens (Human)						x						CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics			20215060
Q96DA0	ZG16B UNQ773/PRO1567	Zymogen granule protein 16 homolog B	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96DR5	BPIFA2 C20orf70 SPLUNC2 UNQ510/PRO10	BPI fold-containing family A member 2 (Parotid secretory protein) (PSP) (Short palate, lung and nasal epithelium carcinoma-associated protein 2)	Homo sapiens (Human)					x						x	CP	68055113	-2,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96DR5	BPIFA2 C20orf70 SPLUNC2 UNQ510/PRO10	BPI fold-containing family A member 2 (Parotid secretory protein) (PSP) (Short palate, lung and nasal epithelium carcinoma-associated protein 2)	Homo sapiens (Human)					x						x	CP	68055113	-1,50	35-66	M/F	General good health, non-smoker, non-diabetic and no intake of antibiotics in the last 6 months	Participants were provided with a parafilm bolus to chew and provided 5ml of saliva by expectoration. Colected between 08:00 and 10:00 hours following overnight fasting.	2D SDS-PAGE + MALDI-TOF or (LC)-MS/MS	Proteomics			20149214
Q96DR5	BPIFA2 C20orf70 SPLUNC2 UNQ510/PRO10	BPI fold-containing family A member 2 (Parotid secretory protein) (PSP) (Short palate, lung and nasal epithelium carcinoma-associated protein 2)	Homo sapiens (Human)						x					x	CP	68055113	4,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q96DR5	BPIFA2 C20orf70 SPLUNC2 UNQ510/PRO10	BPI fold-containing family A member 2 (Parotid secretory protein) (PSP) (Short palate, lung and nasal epithelium carcinoma-associated protein 2)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96EB1	ELP4 C11orf19 PAXNEB	Elongator complex protein 4 (hELP4) (PAX6 neighbor gene protein)	Homo sapiens (Human)						x					x	CP	68055113	40,78	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96FV2	SCRN2	Secernin-2	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96G03	PGM2 MSTP006	Phosphoglucomutase-2 (PGM 2) (EC 5.4.2.2) (Glucose phosphomutase 2) (Phosphodeoxyribomutase) (Phosphopentomutase) (EC 5.4.2.7)	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96G03	PGM2 MSTP006	Phosphoglucomutase-2 (PGM 2) (EC 5.4.2.2) (Glucose phosphomutase 2) (Phosphodeoxyribomutase) (Phosphopentomutase) (EC 5.4.2.7)	Homo sapiens (Human)						x					x	CP	68055113	2,82	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96GM1	LPPR2 PRG4	Lipid phosphate phosphatase-related protein type 2 (EC 3.1.3.4) (Plasticity-related gene 4 protein) (PRG-4)	Homo sapiens (Human)						x					x	CP	68055113	3,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96GX5	MASTL GW GWL THC2	Serine/threonine-protein kinase greatwall (GW) (GWL) (hGWL) (EC 2.7.11.1) (Microtubule-associated serine/threonine-protein kinase-like) (MAST-L)	Homo sapiens (Human)					x						x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96H55	MYO19 MYOHD1	Unconventional myosin-XIX (Myosin head domain-containing protein 1)	Homo sapiens (Human)						x					x	CP	68055113	5,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96HA8	WDYHV1 C8orf32 NTAQ1	Protein N-terminal glutamine amidohydrolase (EC 3.5.1.-) (Protein NH2-terminal glutamine deamidase) (N-terminal Gln amidase) (Nt(Q)-amidase) (WDYHV motif-containing protein 1)	Homo sapiens (Human)					x						x	CP	68055113	-1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96HE7	ERO1L UNQ434/PRO865	ERO1-like protein alpha (ERO1-L) (ERO1-L-alpha) (EC 1.8.4.-) (Endoplasmic oxidoreductin-1-like protein) (Oxidoreductin-1-L-alpha)	Homo sapiens (Human)					x						x	CP	68055113	-1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96HE7	ERO1L UNQ434/PRO865	ERO1-like protein alpha (ERO1-L) (ERO1-L-alpha) (EC 1.8.4.-) (Endoplasmic oxidoreductin-1-like protein) (Oxidoreductin-1-L-alpha)	Homo sapiens (Human)						x					x	CP	68055113	4,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q96HE9	PRR11	Proline-rich protein 11	Homo sapiens (Human)						x					x	CP	68055113	2,17	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96JP2	MYO15B KIAA1783 MYO15BP	Putative unconventional myosin-XVB (Myosin XVBP) (Unconventional myosin-15B)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96KK3	KCNS1	Potassium voltage-gated channel subfamily S member	Homo sapiens (Human)						x					x	CP	68055113	-1,37	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96KK5	HIST1H2AH HIST1H2AI	Histone H2A type 1-H	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q96KK5	HIST1H2AH HIST1H2AI	Histone H2A type 1-H (Histone H2A/s)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96KP4	CNDP2 CN2 CPGL PEPA	Cytosolic non-specific dipeptidase (EC 3.4.13.18) (CNDP dipeptidase 2) (Glutamate carboxypeptidase-like protein 1) (Peptidase A)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96M89	CCDC138	Coiled-coil domain-containing protein 138	Homo sapiens (Human)						x					x	CP	68055113	10,35	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96MS0	ROBO3	Roundabout homolog 3	Homo sapiens (Human)						x					x	CP	68055113	-1,39	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96MV8	ZDHHC15 UNQ1969/PRO4501	Palmitoyltransferase ZDHHC15 (EC 2.3.1.225) (Zinc finger DHHC domain-containing protein 15) (DHHC-15)	Homo sapiens (Human)						x					x	CP	68055113	2,66	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96N87	SLC6A18 XTRP2	Sodium-dependent neutral amino acid transporter B(0)AT3 (Sodium- and chloride-dependent transporter XTRP2) (Solute carrier family 6 member 18) (System B(0) neutral amino acid transporter AT3)	Homo sapiens (Human)					x						x	CP	68055113	1,04	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96NG3	TTC25	Tetratricopeptide repeat protein 25 (TPR repeat protein 25)	Homo sapiens (Human)						x					x	CP	68055113	9,07	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R _Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96NL6	SCLT1 SAP1	Sodium channel and clathrin linker 1 (Sodium channel-associated protein 1)	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96NM4	TOX2 C20orf100 GCX1	TOX high mobility group box family member 2 (Granulosa cell HMG box protein 1) (GCX-1)	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96NY7	CLIC6 CLIC1L	Chloride intracellular channel protein 6 (Parchorin)	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96PD5	PGLYRP2 PGLYRPL PGRPL UNQ3103/PRO	N-acetylmuramoyl-L-alanine amidase (EC 3.5.1.28) (Peptidoglycan recognition protein 2) (Peptidoglycan recognition protein long) (PGRP-L)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q96PR1	KCNC2	Potassium voltage-gated channel subfamily C member 2 (Voltage-gated potassium channel Kv3.2)	Homo sapiens (Human)						x					x	CP	68055113	1,46	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q96PZ7	CSMD1 KIAA1890 UNQ5952/PRO19863	CUB and sushi domain-containing protein 1 (CUB and sushi multiple domains protein 1)	Homo sapiens (Human)					x						x	CP	68055113	1,30	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96Q91	SLC4A9 AE4 SBC5	Anion exchange protein 4 (AE 4) (Anion exchanger 4) (Sodium bicarbonate cotransporter 5) (Solute carrier family 4 member 9)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96QU1	PCDH15 USH1F	Photocadherin 15	Homo sapiens (Human)						x					x	CP	68055113	1,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96QV6	HIST1H2AA H2AFR	Histone H2A type 1-A	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q96RL7	VPS13A CHAC KIAA0986	Vacuolar protein sorting-associated protein 13A	Homo sapiens (Human)						x					x	CP	68055113	2,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96RR4	CAMKK2 CAMKKB KIAA0787	Calcium/calmodulin-dependent protein kinase kinase 2 (CaM-KK 2) (CaM-kinase kinase 2) (CaMKK 2) (EC 2.7.11.17) (Calcium/calmodulin-dependent protein kinase kinase beta) (CaM-KK beta) (CaM-kinase kinase beta) (CaMKK beta)	Homo sapiens (Human)					x						x	CP	68055113	1,35	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q96S42	NODAL	Nodal homolog	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q96SI9	STRBP SPNR	Spermatid perinuclear RNA-binding protein	Homo sapiens (Human)						x					x	CP	68055113	5,24	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96ST3	SIN3A	Paired amphipathic helix protein	Homo sapiens (Human)						x					x	CP	68055113	1,28	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q96T23	RSF1 HBXAP XAP8	Remodeling and spacing factor 1 (Rsf-1) (HBV pX-associated protein 8) (Hepatitis B virus X-associated protein) (p325 subunit of RSF chromatin-remodeling complex)	Homo sapiens (Human)						x					x	CP	68055113	2,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q96TA1	FAM129B C9orf88	Niban-like protein 1 (Meg-3) (Melanoma invasion by ERK) (MINERVA) (Protein FAM129B)	Homo sapiens (Human)						x					x	CP	68055113	3,23	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q99459	CDC5L KIAA0432 PCDC5RP	Cell division cycle 5-like protein (Cdc5-like protein) (Pombe cdc5-related protein)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q99497	PARK7	Protein DJ-1 (EC 3.4.-.-) (Oncogene DJ1) (Parkinson disease protein 7)	Homo sapiens (Human)					x						x	CP	68055113	1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q99497	PARK7	Protein DJ-1 (EC 3.4.-.-) (Oncogene DJ1) (Parkinson disease protein 7)	Homo sapiens (Human)						x					x	CP	68055113	1,85	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q99590	SCAF11 CASP11 SFRS2IP SIP1 SRSF2IP	Protein SCAF11 (CTD-associated SR protein 11) (Renal carcinoma antigen NY-REN-40) (SC35-interacting protein 1) (SR-related and CTD-associated factor 11) (SRSF2-interacting protein) (Serine/arginine-rich splicing factor 2-interacting protein) (Splicing factor, arginine/serine-rich 2-interacting protein) (Splicing regulatory protein 129) (SRrp129)	Homo sapiens (Human)						x					x	CP	68055113	5,54	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q99616	CCL13 MCP4 NCC1 SCYA13	C-C motif chemokine 13	Homo sapiens (Human)						x					x	CP	68055113	4,97						Non-proteomics		x	23375121
Q99732	LITAF PIG7 SIMPLE	Lipopolysaccharide-induced tumor necrosis factor-alpha factor (LPS-induced TNF-alpha factor) (Small integral membrane protein of lysosome/late endosome) (p53-induced gene 7 protein)	Homo sapiens (Human)						x					x	CP	68055113	2,81	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q99759	MAP3K3 MAPKKK3 MEKK3	Mitogen-activated protein kinase kinase kinase 3 (EC 2.7.11.25) (MAPK/ERK kinase kinase 3) (MEK kinase 3) (MEKK 3)	Homo sapiens (Human)					x						x	CP	68055113	1,44	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q99856	ARID3A DRIL1 DRIL3 DRX E2FBP1	AT-rich interactive domain-containing protein 3A (ARID domain-containing protein 3A) (B-cell regulator of IgH transcription) (Bright) (Dead ringer-like protein 1) (E2F-binding protein 1)	Homo sapiens (Human)					x						x	CP	68055113	1,45	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q99877	HIST1H2BN H2BFD	Histone H2B type 1-N	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q99878	HIST1H2AJ H2AFE	Histone H2A type 1-J	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q99879	HIST1H2BM H2BFE	Histone H2B type 1-M	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q99879	HIST1H2BM H2BFE	Histone H2B type 1-M (Histone H2B.e) (H2B/e)	Homo sapiens (Human)						x					x	CP	68055113	2,31	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q99880	HIST1H2BL H2BFC	Histone H2B type 1-L	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q99880	HIST1H2BL H2BFC	Histone H2B type 1-L (Histone H2B.c) (H2B/c)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q99969	RARRES2 TIG2	Chemerin	Homo sapiens (Human)					x						x	CP	68055113	2		MF	Exclusion criteria were systemic disease, use of medication in the last 6 months, pregnancy, smoking, and periodontal therapy within the last 6 months.	The saliva samples were collected according to the unstimulated saliva collection procedure.	ELISA	on-proteomics		x	25164155
Q99996	AKAP9 AKAP350 AKAP450 KIAA0803	A-kinase anchor protein 9 (AKAP-9) (A-kinase anchor protein 350 kDa) (AKAP 350) (hgAKAP 350) (A-kinase anchor protein 450 kDa) (AKAP 450) (AKAP 120-like protein) (Centrosome- and Golgi-localized PKN-associated protein) (CG-NAP) (Protein hyperion) (Protein kinase A-anchoring protein 9) (PRKA9) (Protein yotiao)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9BQ75	CMSS1 C3orf26	Protein CMSS1 (Cms1 ribosomal small subunit homolog)	Homo sapiens (Human)						x					x	CP	68055113	1,86	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9BQR3	PRSS27 MPN UNQ1884/PRO4327	Serine protease 27 (EC 3.4.21.-) (Marapsin) (Pancreasin)	Homo sapiens (Human)					x						x	CP	68055113	-1,16	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BQS8	FYCO1 ZFYVE7	FYVE and coiled-coil domain-containing protein 1 (Zinc finger FYVE domain-containing protein 7)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9BRA2	TXNDC17 TXNL5	Thioredoxin domain-containing protein 17 (14 kDa thioredoxin-related protein) (TRP14) (Protein 42-9-9) (Thioredoxin-like protein 5)	Homo sapiens (Human)						x					x	CP	68055113	-1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BRF8	CPPED1 CSTP1	Calcineurin-like phosphoesterase domain-containing protein 1 (EC 3.1.-.-) (Complete S-transactivated protein 1)	Homo sapiens (Human)						x					x	CP	68055113	1,43	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BRX8	FAM213A C10orf58 PAMM PRO2290 PSEC0	Family with sequence similarity 213, member A	Homo sapiens (Human)							x					CP	68055113	.25(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9BT49	THAP7	THAP domain-containing protein 7	Homo sapiens (Human)						x					x	CP	68055113	1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9BTC0	DIDO1 C20orf158 DATF1 KIAA0333	Death-inducer obliterator 1	Homo sapiens (Human)						x					x	CP	68055113	3,75	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BTE3	MCMBP C10orf119	Mini-chromosome maintenance complex-binding protein (MCM-BP) (MCM-binding protein)	Homo sapiens (Human)						x					x	CP	68055113	26,00	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9BTM1	H2AFJ	Histone H2A.J	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q9BUH6	C9orf142 PAXX	Uncharacterized protein C9orf142	Homo sapiens (Human)					x						x	CP	68055113	-1,21	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BUH6	C9orf142 PAXX	Uncharacterized protein C9orf142	Homo sapiens (Human)						x					x	CP	68055113	1,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BUJ2	HNRNPUL1 E1BAP5 HNRPUL1	Heterogeneous nuclear ribonucleoprotein U-like protein 1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BVA1	TUBB2B	Tubulin beta-2B chain	Homo sapiens (Human)						x					x	CP	68055113	-					Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q9BWK5	MRI C7orf49	Modulator of retrovirus infection homolog	Homo sapiens (Human)					x						x	CP	68055113	1,43	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BXL5	HEMGN EDAG NDR PRO1037 PRO1620	Hemogen	Homo sapiens (Human)						x					x	CP	68055113	2,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BXT5	TEX15	Testis-expressed sequence 15 protein (Cancer/testis antigen 42) (CT42)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9BXW9	FANCD2 FACD	Fanconi anemia group D2 protein subunit BPTF	Homo sapiens (Human)						x					x	CP	68055113	2,16	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BY64	UGT2B28	UDP-glucuronosyltransferase 2B28 (UDPGT 2B28) (EC 2.4.1.17)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9BYE2	TMPRSS13 MSP TMPRSS11	Transmembrane protease, serine 13	Homo sapiens (Human)						x					x	CP	68055113	1,53	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9BYJ1	ALOXE3	Hydroperoxide isomerase ALOXE3 (EC 5.4.4.-) (Epidermis-type lipoxygenase 3) (Epidermal LOX-3) (e-LOX-3) (eLOX-3) (EC 1.13.11.-)	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BYX7	POTEKP ACTBL3 FKSG30	Putative beta-actin-like protein 3	Homo sapiens (Human)						x						CP	68055113	10,00						Proteomics			24098404
Q9BYZ2	LDHAL6B LDHAL6 LDHL	L-lactate dehydrogenase A-like 6B (EC 1.1.1.27)	Homo sapiens (Human)						x					x	CP	68055113		30-67	M/F	All subjects were systematically healthy, non-smokers and not taking medication known to affect periodontal tissues. Subjects reporting antibiotic intake during the previous six months and pregnant or lactating women were excluded from this study.	1Each participant contributed with one pooled GCF sample from four pre-selected sites. For periodontitis cases, the sample was taken from sites which displayed probing depth >6 mm and <8 mm. For periodontally healthy individuals, the samples were taken from the mesiobuccal sites of first molars. GCF samples were obtained as previously described (Sakellari et al. 2008).	high-performance liquid chromatography, tandem mass spectrometry and the PILOT_PROTEIN algorithm. A mixed-integer linear optimization (MILP) model was then developed to identify the optimal combination of biomarkers which could clearly distinguish a blind subject sample as healthy or diseased.	Proteomics			23190455
Q9BZF2	OSBPL7 ORP7	Oxysterol-binding protein-related protein 7 (ORP-7) (OSBP-related protein 7)	Homo sapiens (Human)						x					x	CP	68055113	2,13	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9BZJ6	GPR63 PSP24B	Probable G-protein coupled receptor 63 (PSP24-2) (PSP24-beta)	Homo sapiens (Human)					x						x	CP	68055113	1,49	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9BZZ5	API5 MIG8	Apoptosis inhibitor 5 (API-5) (Antiapoptosis clone 11 protein) (AAC-11) (Cell migration-inducing gene 8 protein) (Fibroblast growth factor 2-interacting factor) (FIF) (Protein XAGL)	Homo sapiens (Human)					x						x	CP	68055113	1,98	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9C010	PKIB PRKACN2	cAMP-dependent protein kinase inhibitor beta (PKI-beta)	Homo sapiens (Human)						x					x	CP	68055113	2,96	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9C0B0	UNK KIAA1753 ZC3H5 ZC3HDC5	RING finger protein unkempt	Homo sapiens (Human)						x					x	CP	68055113	3,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9C0D5	TANC1 KIAA1728	Protein TANC1	Homo sapiens (Human)						x					x	CP	68055113	1,45	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9GZU2	PEG3 KIAA0287 ZSCAN24	Paternally-expressed gene 3 protein (Zinc finger and SCAN domain-containing protein 24)	Homo sapiens (Human)						x					x	CP	68055113	-1,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9GZV4	EIF5A2	Eukaryotic translation initiation factor 5A-2 (eIF-5A-2) (eIF-5A2) (Eukaryotic initiation factor 5A isoform 2)	Homo sapiens (Human)						x					x	CP	68055113	1,50	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9GZX6	IL22 ILTIF ZCYTO18 UNQ3099/PRO10096	Interleukin-22	Homo sapiens (Human)					x						x	CP	68055113	1,07		MF		Prior initial periodontal treatment period, about 5 mL of unstimulated whole saliva was collected from each subject into a 50 mL sterile plastic centrifuge tube (Greiner Bio-one1, Frickenhausen, Germany) before breakfast intake and any dental hygiene procedure. No antiseptic mouth rinse was used before collection. Immediately after collection, whole saliva was clarified by centrifugation for 5-min at 800 g (IEC1 Centra CL2 Centrifuge, Thermo Electron Corporation, Milford, MA, USA). The supernatants were collected and aliquoted into 500 mL volumes and frozen at .75 8C for until processed.	ELISA	on-proteomics			25285903
Q9H0U4	RAB1B	Ras-related protein Rab-1B	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals	using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9H160	ING2 ING1L	Inhibitor of growth protein 2 (Inhibitor of growth 1-like protein) (ING1Lp) (p32) (p33ING2)	Homo sapiens (Human)					x						x	CP	68055113	1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9H1A4	ANAPC1 TSG24	Anaphase-promoting complex subunit 1 (APC1) (Cyclosome subunit 1) (Mitotic checkpoint regulator) (Testis-specific gene 24 protein)	Homo sapiens (Human)					x						x	CP	68055113	-1,03	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9H1E3	NUCKS1 NUCKS JC7	Nuclear ubiquitous casein and cyclin-dependent kinase substrate 1 (P1)	Homo sapiens (Human)						x					x	CP	68055113	3,64	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H1X3	DNAJC25	DnaJ homolog subfamily C member 25	Homo sapiens (Human)						x					x	CP	68055113	1,28	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9H239	MMP28 MMP25 UNQ1893/PRO4339	Matrix metallopeptidase 28	Homo sapiens (Human)							x					CP	68055113	,34(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9H254	SPTBN4 KIAA1642 SPTBN3	Spectrin beta chain, non-erythrocytic 4 (Beta-IV spectrin) (Spectrin, non-erythroid beta chain 3)	Homo sapiens (Human)						x					x	CP	68055113	8,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H299	SH3BGRL3 P17125	SH3 domain-binding glutamic acid-rich-like protein 3 (SH3 domain-binding protein 1) (SH3BP-1)	Homo sapiens (Human)					x						x	CP	68055113	1,07	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9H2C0	GAN GAN1 KLHL16	Kelch-like family member 6	Homo sapiens (Human)							x					CP	68055113	46 (Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9H2G2	SLK KIAA0204 STK2	STE20-like serine/threonine-protein kinase	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9H3K6	BOLA2 BOLA2A My016; BOLA2B	BolA-like protein 2	Homo sapiens (Human)						x					x	CP	68055113	2,12	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H4M9	EHD1 PAST PAST1 CDABP0131	EH domain-containing protein 1 (PAST homolog 1) (hPAST1) (Testilin)	Homo sapiens (Human)						x					x	CP	68055113	2,60	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H6T3	RPAP3	RNA polymerase II-associated protein 3	Homo sapiens (Human)						x					x	CP	68055113	3,03	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H799	C5orf42	Uncharacterized protein C5orf42	Homo sapiens (Human)					x						x	CP	68055113	-1,86	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9H7F0	ATP13A3 AFURS1	Cation-transporting ATPase 13A3	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9H7Q6	FLJ00011	FLJ00011 protein (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	8,48	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R ,Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9H7Z5	0	cDNA FLJ14048 fis, clone HEMBA1006650, weakly similar to ARP2/3 COMPLEX 20 KD SUBUNIT	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9H7Z7	PTGES2 C9orf15 PGES2	Prostaglandin E synthase 2 (EC 5.3.99.3) (Microsomal prostaglandin E synthase 2) (mPGES-2) [Cleaved into: Prostaglandin E synthase 2 truncated form]	Homo sapiens (Human)							x					CP	68055113		35-70		The biopsies were taken during surgery as part of the normal course of periodontal therapy.	For the immunostaining of proteins expression in the biopsies, the sections were deparaffinized using xylene and then were rehydrated through an ethanol series. Immunohistochemical staining was performed using a cell and tissue staining kit (R&D Systems, Minneapolis, MN) according to the manufacturer's instructions.	on-proteomics			21435451

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9H825	METTL8	Methyltransferase-like protein 8	Homo sapiens (Human)						x					x	CP	68055113	1,24	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9HB83	0	MLL protein (Fragment)	Homo sapiens (Human)							x				x	CP	68055113	2,57	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9HBA0	TRPV4 VRL2 VROAC	Transient receptor potential cation channel subfamily V member 4 (TrpV4) (Osm-9-like TRP channel 4) (OTRPC4) (Transient receptor potential protein 12) (TRP12) (Vanilloid receptor-like channel 2) (Vanilloid receptor-like protein 2) (VRL-2) (Vanilloid receptor-related osmotically-activated channel) (VR-OAC)	Homo sapiens (Human)					x						x	CP	68055113	1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9HBG4	ATP6V0A4 ATP6N1B ATP6N2	V-type proton ATPase 116 kDa subunit a isoform 4 (V-ATPase 116 kDa isoform a4) (Vacuolar proton translocating ATPase 116 kDa subunit a isoform 4) (Vacuolar proton translocating ATPase 116 kDa subunit a kidney isoform)	Homo sapiens (Human)					x						x	CP	68055113	1,33	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9HC84	MUC5B MUC5	Mucin 5b	Homo sapiens (Human)							x				x	CP	68055113	1,26	25-60	MF	Exclusion criteria included subjects with history of any systemic diseases or conditions, subjects with history of any salivary gland diseases, subjects with any apparent oral infections (i.e. herpes or candida) or injuries or bleeding in the oral cavity unrelated to	Subjects were instructed not to brush their teeth, or eat or drink one hour before the time of saliva collection. Unstimulated whole saliva was collected between 11am and 12 Noon to avoid diurnal variation with patients seated with instructions to allow saliva to accumulate in the floor of the mouth and to spit without stimulation into the sterile container. The collected samples were immediately taken for biochemical analysis.	Mucin estimation was performed using Alcian Blue method [5,8,9].	on-proteomics			25478449
Q9HC84	MUC5B MUC5	Mucin-5B	Homo sapiens (Human)						x					x	CP	68055113	1,63	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9HC84	MUC5B MUC5	Mucin-5B (MUC-5B) (Cervical mucin) (High molecular weight salivary mucin MG1) (Mucin-5 subtype B, tracheobronchial) (Sublingual gland mucin)	Homo sapiens (Human)					x						x	CP	68055113	1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9HC84	MUC5B MUC5	Mucin-5B (MUC-5B) (Cervical mucin) (High molecular weight salivary mucin MG1) (Mucin-5 subtype B, tracheobronchial) (Sublingual gland mucin)	Homo sapiens (Human)					x						x	CP	68055113		25-50	M/F	All study subjects were systemically healthy. Subjects were excluded from the study if they were nursing or pregnant, smoked, drank alcohol, had received periodontal treatment in the last 2 years or taken antibiotics in the previous year.	Unstimulated. Rest for 15 min before saliva collection (at morning, 2 h after tooth brushing), sitting in an upright position and were asked not to speak	2-DE + MALDI-TOF/TOF + LC-ESI-MS + nLC-Q-TOF	Proteomics		x	20215060
Q9HCB6	SPON1 KIAA0762 VSGP	Spondin-1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9HCJ0	TNRC6C KIAA1582	Trinucleotide repeat-containing gene 6C protein	Homo sapiens (Human)							x				x	CP	68055113	1,46	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9HCJ0	TNRC6C KIAA1582	Trinucleotide repeat-containing gene 6C protein	Homo sapiens (Human)						x					x	CP	68055113	-1,35	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9HCY8	S100A14 S100A15	Protein S100-A14 (S100 calcium-binding protein A14) (S114)	Homo sapiens (Human)							x				x	CP	68055113	5,91	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9HD89	RETN FIZZ3 HXCP1 RSTN UNQ407/PRO1157	Resistin (Adipose tissue-specific secretory factor) (ADSF) (C/EBP-epsilon-regulated myeloid-specific secreted cysteine-rich protein) (Cysteine-rich secreted protein A12-alpha-like 2) (Cysteine-rich secreted protein FIZZ3)	Homo sapiens (Human)							x				x	CP	68055113	2,43	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NP62	GCM1 GCMA	Chorion-specific transcription factor GCMa (hGCMa) (GCM motif protein 1) (Glial cells missing homolog 1)	Homo sapiens (Human)							x				x	CP	68055113	1,77	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from occlusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NPE6	SPAG4 SUN4	Sperm associated antigen 4	Homo sapiens (Human)								x				CP	68055113	29(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9NQ25	SLAMF7 CS1 UNQ576/PRO1138	SLAM family member 7	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9NQ38	SPINK5	Serine protease inhibitor Kazal-type 5 (Lympho-epithelial Kazal-type-related inhibitor) (LEKTI) [Cleaved into: Hemofiltrate peptide HF6478; Hemofiltrate peptide HF7665]	Homo sapiens (Human)					x						x	CP	68055113	-1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NQC7	CYLD CYLD1 KIAA0849 HSPC057	Ubiquitin carboxyl-terminal hydrolase CYLD (EC 3.4.19.12) (Deubiquitinating enzyme CYLD) (Ubiquitin thioesterase CYLD) (Ubiquitin-specific-processing protease CYLD)	Homo sapiens (Human)							x				x	CP	68055113	4,62	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NQR7	CCDC177 C14orf162 PLPL	Coiled-coil domain-containing protein 177 (Myelin proteolipid protein-like protein)	Homo sapiens (Human)							x				x	CP	68055113	3,34	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NR99	MXRA5	Matrix-remodeling-associated protein 5 (Adhesion protein with leucine-rich repeats and immunoglobulin domains related to perlecan) (Adlican)	Homo sapiens (Human)							x				x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9NRL2	BAZ1A ACF1 WCRF180 HSPC317	Bromodomain adjacent to zinc finger domainprotein 1A	Homo sapiens (Human)							x				x	CP	68055113	1,03	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9NRS6	SNX15	Sorting nexin-15	Homo sapiens (Human)							x				x	CP	68055113	4,69	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NS15	LTBP3	Latent-transforming growth factor β-binding protein 3	Homo sapiens (Human)							x					CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9NS62	THSD1 TMTSP UNQ3010/PRO9769	Thrombospondin type-1 domain-containing protein 1 (Transmembrane molecule with thrombospondin module)	Homo sapiens (Human)							x				x	CP	68055113	6,83	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NSB2	KRT84 KRTHB4	Keratin, type II cuticular Hb4	Homo sapiens (Human)						x					x	CP	68055113							Proteomics			24098404
Q9NSC2	SALL1 SAL1 ZNF794	Sal-like protein 1	Homo sapiens (Human)						x					x	CP	68055113	-1,18	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9NT68	TENM2 KIAA1127 ODZ2 TNM2	Teneurin-2 (Ten-2) (Protein Odd Oz/ten-m homolog 2) (Tenascin-M2) (Ten-m2) (Teneurin transmembrane protein 2) [Cleaved into: Ten-2, soluble form; Ten-2 intracellular domain (Ten-2 ICD)]	Homo sapiens (Human)					x						x	CP	68055113	-1,20	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NU22	MDN1 KIAA0301	Midasin	Homo sapiens (Human)							x				x	CP	68055113	-1,49	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9NU22	MDN1 KIAA0301	Midasin (MIDAS-containing protein)	Homo sapiens (Human)						x					x	CP	68055113	22,79	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NUP1	BLOC1S4 CNO	Biogenesis of lysosome-related organelles complex 1 subunit 4 (BLOC-1 subunit 4) (Protein cappuccino homolog)	Homo sapiens (Human)					x						x	CP	68055113	1,34	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NVM4	PRMT7 KIAA1933	Protein arginine N-methyltransferase 7 (EC 2.1.1.-) (Histone-arginine N-methyltransferase PRMT7) (EC 2.1.1.125) ([Myelin basic protein]-arginine N-methyltransferase PRMT7) (EC 2.1.1.126)	Homo sapiens (Human)					x						x	CP	68055113	-1,14	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NVP1	DDX18	ATP-dependent RNA helicase DDX18	Homo sapiens (Human)						x					x	CP	68055113	2,1	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9NVR0	KLHL11	Kelch-like protein 11	Homo sapiens (Human)					x						x	CP	68055113	-1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NVR2	INTS10 C8orf35	Integrator complex subunit 10 (Int10)	Homo sapiens (Human)					x						x	CP	68055113	1,15	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9NWM3	CUEDC1	CUE domain-containing protein 1	Homo sapiens (Human)						x					x	CP	68055113	1,46	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NXL9	MCM9 C6orf61 MCMDC1	DNA helicase MCM9 (hMCM9) (EC 3.6.4.12) (Mini-chromosome maintenance deficient domain-containing protein 1) (Minichromosome maintenance 9)	Homo sapiens (Human)						x					x	CP	68055113	2,04	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NY33	DPP3	Dipeptidyl peptidase 3 (EC 3.4.14.4) (Dipeptidyl aminopeptidase III) (Dipeptidyl arylamidase III) (Dipeptidyl peptidase III) (DPP III)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9NY41	CHI3L1	Cartilage glycoprotein-39 (Fragment)	Homo sapiens (Human)						x					x	CP	68055113	2,25	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NY74	ETAA1 ETAA16	Ewing's tumor-associated antigen 1	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9NYF0	DACT1 DPR1 HNG3	Dapper homolog 1 (hDPR1) (Dapper antagonist of catenin 1) (Hepatocellular carcinoma novel gene 3 protein)	Homo sapiens (Human)					x						x	CP	68055113	-1,13	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9NZD4	AHSP EDRF ERAF	Alpha-hemoglobin-stabilizing protein (Erythroid differentiation-related factor) (Erythroid-associated factor)	Homo sapiens (Human)						x					x	CP	68055113	-2,70	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9NZN5	ARHGEF12 KIAA0382 LARG	Rho guanine nucleotide exchange factor 12 (Leukemia-associated RhoGEF)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9NZY2	KIAA0125 C14orf110 FAM30A HSPC053	KIAA0125	Homo sapiens (Human)							x					CP	68055113	35 (Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9P041	0	HSPC-109	Homo sapiens (Human)						x					x	CP	68055113	1,12	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9P135	0	PRO3077 (Uncharacterized protein)	Homo sapiens (Human)						x					x	CP	68055113	10,05	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9P1Y6	PHRF1 KIAA1542	PHD and RING finger domain-containing protein 1	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9P212	PLCE1 KIAA1516 PLCE PPLC	1-Phosphatidylinositol-phosphodiesterase ε-1	Homo sapiens (Human)						x					x	CP	68055113	1,65	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9P270	SLAIN2 KIAA1458	SLAIN motif-containing protein 2	Homo sapiens (Human)						x					x	CP	68055113	1,73	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9P2D1	CHD7 KIAA1416	Chromodomain-helicase-DNA-binding protein 7	Homo sapiens (Human)						x					x	CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9P2E3	ZNFX1 KIAA1404	NFX1-type zinc finger-containing protein 1	Homo sapiens (Human)						x					x	CP	68055113	-1,85	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UBC9	SPRR3 SPRC	Small proline-rich protein 3 (22 kDa pancornulin) (Cornifin beta) (Esophagin)	Homo sapiens (Human)					x						x	CP	68055113	-1,37	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UBC9	SPRR3 SPRC	Small proline-rich protein 3 (22 kDa pancornulin) (Cornifin beta) (Esophagin)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q9UBG3	CRNN C1orf10 DRC1 PDRC1 SEP53	Cornulin (53 kDa putative calcium-binding protein) (53 kDa squamous epithelial-induced stress protein) (58 kDa heat shock protein) (Squamous epithelial heat shock protein 53) (Tumor-related protein)	Homo sapiens (Human)					x						x	CP	68055113	-1,26	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UBG3	CRNN C1orf10 DRC1 PDRC1 SEP53	Cornulin (53 kDa putative calcium-binding protein) (53 kDa squamous epithelial-induced stress protein) (58 kDa heat shock protein) (Squamous epithelial heat shock protein 53) (Tumor-related protein)	Homo sapiens (Human)						x					x	CP	68055113						Samples were trypically digested with trypsin, eluted using high-performance liquid chromatography, and fragmented using tandem mass spectrometry (MS/MS). MS/MS spectra were analysed using PILOT_PROTEIN to identify all unmodified proteins within the samples	Proteomics			22092770
Q9UBR2	CTSZ	Cathepsin Z (EC 3.4.18.1) (Cathepsin P) (Cathepsin X)	Homo sapiens (Human)						x					x	CP	68055113	1,28	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UBV2	SEL1L TSA305 UNQ128/PRO1063	sel-1 suppressor of lin-12-like (C. elegans)	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9UBW7	ZMYM2 FIM RAMP ZNF198	Zinc finger MYM-type protein 2	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UBX7	KLK11 PRSS20 TLSP UNQ649/PRO1279	Kallikrein-11 (hK11) (EC 3.4.21.-) (Hippostasin) (Serine protease 20) (Trypsin-like protease) (Cleaved into: Kallikrein-11 inactive chain 1; Kallikrein-11 inactive chain 2]	Homo sapiens (Human)					x						x	CP	68055113	-1,56	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UBX7	KLK11 PRSS20 TLSP UNQ649/PRO1279	Kallikrein-11 (hK11) (EC 3.4.21.-) (Hippostasin) (Serine protease 20) (Trypsin-like protease) (Cleaved into: Kallikrein-11 inactive chain 1; Kallikrein-11 inactive chain 2]	Homo sapiens (Human)						x					x	CP	68055113	1,10	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9UDR5	AASS	Alpha-aminoadipic semialdehyde synthase,	Homo sapiens (Human)						x					x	CP	68055113	1,62	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UFH2	DNAH17 DNAHL1 DNEL2	Dynein heavy chain 17, axonemal	Homo sapiens (Human)						x					x	CP	68055113	-1,25	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UGM3	DMBT1 GP340	Deleted in malignant brain tumors 1 protein (Glycoprotein 340) (Gp-340) (Hensin) (Salivary agglutinin) (SAG) (Surfactant pulmonary-associated D-binding protein)	Homo sapiens (Human)					x						x	CP	68055113	-1,25	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UGM3	DMBT1 GP340	Deleted in malignant brain tumors 1 protein (Glycoprotein 340) (Gp-340) (Hensin) (Salivary agglutinin) (SAG) (Surfactant pulmonary-associated D-binding protein)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UHC9	NPC1L1	Niemann-Pick C1-like protein 1	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UII2	ATPIF1 ATPI	ATPase inhibitor, mitochondrial (Inhibitor of F(1)F(o)-ATPase) (IF(1)) (IF1)	Homo sapiens (Human)						x					x	CP	68055113	4,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9UIQ6	LNPEP OTASE	Leucyl-cystinyl aminopeptidase	Homo sapiens (Human)						x					x	CP	68055113	-1,06	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UIV8	SERPINB13 PI13	Serpin B13 (HaCaT UV-repressible serpin) (Hurpin) (Headpin) (Peptidase inhibitor 13) (PI-13) (Proteinase inhibitor 13)	Homo sapiens (Human)					x						x	CP	68055113	-1,01	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UJ70	NAGK	N-acetyl-D-glucosamine kinase (N-acetylglucosamine kinase) (EC 2.7.1.59) (GlcNAc kinase)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UKK3	PARP4 ADPRTL1 KIAA0177 PARPL	Poly [ADP-ribose] polymerase 4 (PARP-4) (EC 2.4.2.30) (193 kDa vault protein) (ADP-ribosyltransferase diphtheria toxin-like 4) (ARTD4) (PARP-related/talphal-related H5/proline-rich) (PH5P) (Vault poly(ADP-ribose) polymerase) (VPARP)	Homo sapiens (Human)						x					x	CP	68055113	3,78	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R .Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9UKM7	MAN1B1 UNQ747/PRO1477	Endoplasmic reticulum mannosyl-oligosaccharide 1,2-alpha-mannosidase (EC 3.2.1.113) (ER alpha-1,2-mannosidase) (ER mannosidase 1) (ERMan1) (Man9GlcNAc2-specific-processing alpha-mannosidase) (Mannosidase alpha class 1B member 1)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UKP5	ADAMTS6	A disintegrin and metalloproteinase with thrombospondin motifs 6 (ADAM-TS 6) (ADAM-TS6) (ADAMTS-6) (EC 3.4.24.-)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UKR3	KLK13 KLKL4	Kallikrein-13 (EC 3.4.21.-) (Kallikrein-like protein 4) (KLK-L4)	Homo sapiens (Human)					x						x	CP	68055113	-1,06	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UKT4	FBXO5 EMI1 FBX5	F-box only protein 5 OS=Homo sapiens	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9UKX3	MYH13	Myosin-13	Homo sapiens (Human)						x					x	CP	68055113	1,67	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UKZ4	TENM1 ODZ1 TNM1	Teneurin-1 (Ten-1) (Protein Odd Oz/ten-m homolog 1) (Tenascin-M1) (Ten-m1) (Teneurin transmembrane protein 1) [Cleaved into: Ten-1 intracellular domain (ID)ten-1) (Ten-1 ICD); Teneurin C-terminal-associated peptide (TCPA-1) (Ten-1 extracellular domain) (Ten-1 ECD)]	Homo sapiens (Human)						x					x	CP	68055113				isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9UL36	ZNF236	Zinc finger protein 236	Homo sapiens (Human)					x						x	CP	68055113	-1,41	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UL58	ZNF215 BAZ2 ZKSCAN11	Zinc finger protein 215	Homo sapiens (Human)							x					CP	68055113	28(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9UL89	0	Myosin-reactive immunoglobulin heavy chain variable region (Fragment)	Homo sapiens (Human)							x				x	CP	68055113	2,20	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9UL96	0	Myosin-reactive immunoglobulin heavy chain variable region (Fragment)	Homo sapiens (Human)							x				x	CP	68055113	4,29	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9ULL4	PLXNB3 KIAA1206 PLXN6	Plexin-B3	Homo sapiens (Human)					x						x	CP	68055113	-1,27	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at .80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9ULU4	ZMYND8 KIAA1125 PRKCBP1 RACK7	Protein kinase C-binding protein 1	Homo sapiens (Human)						x					x	CP	68055113	1,18	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9ULX6	AKAP8L NAKAP NAKAP95 HRIHFB2018	A-kinase anchor protein 8-like	Homo sapiens (Human)						x					x	CP	68055113	1,57	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9ULZ2	STAP1 BRDG1	Signal transducing adaptor family member 1	Homo sapiens (Human)							x					CP	68055113	27(Vs Ag Per)		MF	non-smoking, systemically healthy individuals	We used 310 Affymetrix HG-U133Plus2.0 microarray samples (GEO accession number GSE16134) of 'healthy' [n = 69, no bleeding on probing (BoP), probing depth (PD) ≤ 4 mm, and clinical attachment loss (CAL) ≤ 4 mm)] or 'diseased' gingival tissue samples (n = 241; with BoP, PD ≥ 4 mm, and CAL ≥ 3 mm), obtained from 120 non-smoking, systemically healthy individuals with moderate/severe periodontitis (65 with CP and 55 with AgP), as previously described (Demmer et al., 2008; Kebschull and Papapanou, 2010).	We used molecular profiling to explore biological differences between CP and AgP and subsequently carried out supervised classification using machine-learning algorithms including an internal validation.	on-proteomics			24122488
Q9UM07	PADI4 PAD4 PADI5 PDI5	Protein-arginine deiminase type-4 (EC 3.5.3.15) (HL-60 PAD) (Peptidylarginine deiminase IV) (Protein-arginine deiminase type IV)	Homo sapiens (Human)							x				x	CP	68055113	2,87	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9UNX4	WDR3	WD repeat-containing protein 3	Homo sapiens (Human)						x					x	CP	68055113	-1,28	22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UPA5	BSN KIAA0434 ZNF231	Protein bassoon	Homo sapiens (Human)						x						CP	68055113		22-61	MF		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9UPY3	DICER1 DICER HERNA KIAA0928	Endoribonuclease Dicer	Homo sapiens (Human)						x					x	CP	68055113	2,42	22-61	M/F		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9UQ16	DNM3 KIAA0820	Dynamin-3 (EC 3.6.5.5) (Dynamin, testicular) (T-dynamin)	Homo sapiens (Human)					x						x	CP	68055113	1,09	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9UQ35	SRRM2 KIAA0324 SRL300 SRM300 HSPC07	Serine/arginine repetitive matrix protein 2 (300 kDa nuclear matrix antigen) (Serine/arginine-rich splicing factor-related nuclear matrix protein of 300 kDa) (SR-related nuclear matrix protein of 300 kDa) (Ser/Arg-related nuclear matrix protein of 300 kDa) (Splicing coactivator subunit SRm300) (Tax-responsive enhancer element-binding protein 803) (TaxREB803)	Homo sapiens (Human)						x					x	CP	68055113	-1,50	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y2Q0	ATP8A1 ATPIA	Probable phospholipid-transporting ATPase IA (EC 3.6.3.1) (ATPase class I type 8A member 1) (Chromaffin granule ATPase II)	Homo sapiens (Human)					x						x	CP	68055113	-1,02	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9Y2V2	CARHSP1	Calcium-regulated heat stable protein 1 (Calcium-regulated heat-stable protein of 24 kDa) (CRHSP-24)	Homo sapiens (Human)						x					x	CP	68055113	1,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y2X3	NOP58 NOL5 NOP5 HSPC120	Nucleolar protein 58 (Nucleolar protein 5)	Homo sapiens (Human)						x					x	CP	68055113	3,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y463	DYRK1B MIRK	Dual specificity tyrosine-phosphorylation-regulated kinase 1B (EC 2.7.12.1) (Minibrain-related kinase) (Mirk protein kinase)	Homo sapiens (Human)						x					x	CP	68055113	3,58	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y483	MTF2 PCL2	Metal-response element-binding transcription factor 2 (Metal regulatory transcription factor 2) (Metal-response element DNA-binding protein M96) (Polycomb-like protein 2) (hPCl2)	Homo sapiens (Human)					x						x	CP	68055113	-1,39	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9Y487	ATP6V0A2	V-type proton ATPase 116 kDa subunit a isoform 2	Homo sapiens (Human)						x						CP	68055113		22-61	M/F		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9Y490	TLN1 KIAA1027 TLN	Talin-1	Homo sapiens (Human)						x					x	CP	68055113	2,75	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from cclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y490	TLN1 KIAA1027 TLN	Talin-1	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y493	ZAN	Zonadhesin	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q9Y4E6	WDR7 KIAA0541 TRAG	WD repeat-containing protein 7 (Rabconnectin-3 beta) (TGF-beta resistance-associated protein TRAG)	Homo sapiens (Human)						x					x	CP	68055113	3,33	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y4G2	PLEKHM1 KIAA0356	Pleckstrin homology domain-containing family M member 1 (PH domain-containing family M member 1) (162 kDa adapter protein) (AP162)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y5H6	PCDHA8	Protocadherin alpha-8 (PCDH-alpha-8)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y5K2	KLK4 EMSP1 PRSS17 PSTS	Kallikrein-4 (enamel matrix serine proteinase 1)	Homo sapiens (Human)						x					x	CP	68055113	2,16	22-61	M/F		Gingival crevicular fluid was collected from 9:00 am to 12:00 pm to minimize the effect of circadian rhythmic variation on the composition of the fluids. All subjects were asked not to eat, drink, brush their teeth or use any type of mouthwash 2 h prior to fluid collection. The sites selected for collection were subjected to washing by the dental unit's air–water syringe. The areas were isolated from salivary contamination with cotton rolls, air-dried and GCF was collected with Periopaper strips (Oraflow, Plainview, NY, USA).	Quantitative proteome of GCF was established using stable isotope-labelling reagents, ICAT and mTRAQ, with MS technology and validated by enzyme-linked immunosorbent methods.	Proteomics			24738839
Q9Y5Z4	HEBP2 C6orf34 SOUL	Heme-binding protein 2 (Placental protein 23) (PP23) (Protein SOUL)	Homo sapiens (Human)					x						x	CP	68055113	1,05	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9Y5Z4	HEBP2 C6orf34 SOUL	Heme-binding protein 2 (Placental protein 23) (PP23) (Protein SOUL)	Homo sapiens (Human)						x					x	CP	68055113	1,57	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
Q9Y5Z7	HCFC2	Host cell factor 2 (HCF-2) (C2 factor)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y618	NCOR2 CTG26	Nuclear receptor corepressor 2 (N-CoR2) (CTG repeat protein 26) (SMAP270) (Silencing mediator of retinoic acid and thyroid hormone receptor) (SMRT) (T3 receptor-associating factor) (TRAC) (Thyroid-, retinoic-acid-receptor-associated corepressor)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y623	MYH4	Myosin-4 (Myosin heavy chain 2b) (MyHC-2b) (Myosin heavy chain 4) (Myosin heavy chain IIb) (MyHC-IIb) (Myosin heavy chain, skeletal muscle, fetal)	Homo sapiens (Human)						x					x	CP	68055113					isolated GCF from periodontitis patients and healthy individuals using a gel loading tip	LC-ESI-MS/MS analysis following in-gel digestion	Proteomics			21794177
Q9Y6R7	FCGBP	IgGFc-binding protein (Fcgamma-binding protein antigen) (FcgammaBP)	Homo sapiens (Human)					x						x	CP	68055113	-1,23	35-64	M/F	non-smoking and non-diabetic sub-jects with at least 10 natural teeth	Stimulated saliva was collected with a commercially available collection sys-tem (Salivette). The subjects chewed a plain cotton role exactly for 1 min. to stimulate salivation. The roles with the absorbed saliva were placed into the Salivette and immediately centrifuged at 1000 g for 20 min. at 4°C to remove food remnants, insol-uble material and cell debris. The resulting supernatant was stored at 80°C.	Whole saliva proteins were analysed after trichloroacetic acid (TCA) precipitation and proteolytic digestion with trypsin by LC-MS/MS. MS-data were analysed and quantified using the Rosetta Elucidator software package.	Proteomics			23790309
Q9Y6W5	WASF2 WAVE2	Wiskott-Aldrich syndrome protein family member 2 (WASP family protein member 2) (Protein WAVE-2) (Verprolin homology domain-containing protein 2)	Homo sapiens (Human)						x					x	CP	68055113	13,84	46,3	M/F	Subjects examined were those without systemic diseases, treatment for periodontal disease, juvenile periodontal diseases, acute gingival inflammation, or trauma from oclusion. All were in good general health with no history of antimicrobial or antiinflammatory therapy or periodontal treatment for 6 months before the start of the study. As smoking is a risk factor for periodontal disease, the current study did not include smokers.	All GCF samples were collected and processed as described previously [10]. Briefly, supra-gingival plaque was carefully removed from the tooth with a curette, teeth were rinsed with saline, and sampling sites were isolated with cotton rolls and dried. Each sample site was carefully isolated using cotton rolls to avoid saliva contamination. Absorbent Paper points (Zipperer R . Munich, Germany) were gently inserted into the gingival crevice and left in place for 30 s. GCF was collected from the labial side of axillary incisors without crown and restoration. For severe periodontal cases, the sample was taken from four preselected sites, which displayed probing depths >6 mm and <8 mm. Mechanical irritation avoided and absorbent paper points contaminated with blood were discarded. Paper points were stored at –80 C for further processing. GCF obtained from the 31 patients was randomly divided into two groups; a test set for proteomic analysis using TMT, and a validation set (Table 1). The test set included GCF from four patients and GCF from two healthy volunteers. The validation set included GCF from 27 patients and GCF from 14 healthy volunteers.	LC-MS/MS analyses using tandem mass tag (TMT) labeling	Proteomics			23696425
P05120	SERPINB2	PAI-2	Homo sapiens (Human)						x					x	CP	68055113	+		M/F	None of the patients had received any antibiotics in the past 3 months or primary periodontal therapy in the past 6 months before the sampling and recording. The following additional exclusion criteria were as follows: presence of systemic disease that could affect the periodontal tissues(diabetes mellitus,cancer,cardiovascular and respiratory diseases) and need for antibiotic prophylaxis for dental treatment, use of antibiotics, phenytoin, calcium antagonists, cyclosporine or anti-inflammatory drugs, current pregnancy or lactation and postmenopausal women and smokers.	Gingival crevicular fluid samples were collected from the deepest six pockets the day after clinical examination of patients.	Enzyme-linked immunosorbent	Proteomics			24690077
P00750	PLAT	t-PA	Homo sapiens (Human)						x					x	CP	68055113	+		M/F	None of the patients had received any antibiotics in the past 3 months or primary periodontal therapy in the past 6 months before the sampling and recording. The following additional exclusion criteria were as follows: presence of systemic disease that could affect the periodontal tissues(diabetes mellitus,cancer,cardiovascular and respiratory diseases) and need for antibiotic prophylaxis for dental treatment, use of antibiotics, phenytoin, calcium antagonists, cyclosporine or anti-inflammatory drugs, current pregnancy or lactation and postmenopausal women and smokers.	Gingival crevicular fluid samples were collected from the deepest six pockets the day after clinical examination of patients.	Enzyme-linked immunosorbent	Proteomics			24690077
P01584	IL1B	IL-1b	Homo sapiens (Human)						x					x	CP	68055113	+		M/F	None of the patients had received any antibiotics in the past 3 months or primary periodontal therapy in the past 6 months before the sampling and recording. The following additional exclusion criteria were as follows: presence of systemic disease that could affect the periodontal tissues(diabetes mellitus,cancer,cardiovascular and respiratory diseases) and need for antibiotic prophylaxis for dental treatment, use of antibiotics, phenytoin, calcium antagonists, cyclosporine or anti-inflammatory drugs, current pregnancy or lactation and postmenopausal women and smokers.	Gingival crevicular fluid samples were collected from the deepest six pockets the day after clinical examination of patients.	Enzyme-linked immunosorbent	Proteomics			24690077

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P08254	MMP3	MMP-3	Homo sapiens (Human)						x					x	CP	68055113	+		M/F	None of the patients had received any antibiotics in the past 3 months or primary periodontal therapy in the past 6 months before the sampling and recording. The following additional exclusion criteria were as follows: presence of systemic disease that could affect the periodontal tissues(diabetes mellitus,cancer,cardiovascular and respiratory diseases) and need for antibiotic prophylaxis for dental treatment, use of antibiotics, phenytoin, calcium antagonists, cyclosporine or anti-inflammatory drugs, current pregnancy or lactation and postmenopausal women and smokers.	Gingival crevicular fluid samples were collected from the deepest six pockets the day after clinical examination of patients.	Enzyme-linked immunosorbent	Proteomics			24690077
Q5QEX9	IL17	Interleukin-17	Homo sapiens (Human)					X						x	CP	68055113	2,3	35-65	M/F	healthy untreated nonsmokers with chronic periodontitis	In the morning following an overnight fast, during which subjects were requested not to drink (except water) or to chew gum, whole saliva samples were obtained by expectorating into polypropylene tubes; clinical periodontal measurements and any necessary periodontal interventions were then carried out.	The ELISA development kits (Bender MedSystems, Vienna, Austria) were used to analyze the concentrations of both IL-17 and IL-18 in the saliva samples.	Proteomics			21635252
Q14116	IL18	Interleukin-18	Homo sapiens (Human)					X						x	CP	68055113	1,59	35-65	M/F	healthy untreated nonsmokers with chronic periodontitis	In the morning following an overnight fast, during which subjects were requested not to drink (except water) or to chew gum, whole saliva samples were obtained by expectorating into polypropylene tubes; clinical periodontal measurements and any necessary periodontal interventions were then carried out.	The ELISA development kits (Bender MedSystems, Vienna, Austria) were used to analyze the concentrations of both IL-17 and IL-18 in the saliva samples.	Proteomics			21635252
P49913	CAMP	ll-37	Homo sapiens (Human)					X						x	CP	68055113	10,22	19-83	M/F	All subjects were in good general health with no history of systemic diseases or medication known to affect periodontal tissues	From each participant, a sample of unstimulated whole saliva was obtained by spitting. Sampling was performed in the morning, around 10 am, to avoid a possible variation in peptide concentration due to circadian rhythm in salivary flow rate and composition.	by an enzyme-linked immunosorbent assay (ELISA)	Proteomics			23778112
Q9HC84	MUC5B	Mucin-5B	Homo sapiens (Human)					X						x	CP	68055113	1,55	32-40	M/F	subjects who had not had a periodontal checkup in the previous 6 months and all of the subjects gave their informed consent. Exclusion criteria included: smokers, those with cardiovascular or respiratory diseases, systemic inflammatory conditions or non-plaque induced oral inflammatory conditions, immunodeficiency, pregnant or breast feeding individuals and those using medicine.	Saliva collection Unstimulated saliva was collected at 10 am the day after the periodontal diagnosis. Subjects were asked to refrain from eating or drinking two hours prior to collection.	Colorimetric methods were used for all determinations in unstimulated saliva.	Proteomics			26221923
P04745	AMY1A	Alpha-amylase 1	Homo sapiens (Human)					X						x	CP	68055113	1,55	32-40	M/F	subjects who had not had a periodontal checkup in the previous 6 months and all of the subjects gave their informed consent. Exclusion criteria included: smokers, those with cardiovascular or respiratory diseases, systemic inflammatory conditions or non-plaque induced oral inflammatory conditions, immunodeficiency, pregnant or breast feeding individuals and those using medicine.	Saliva collection Unstimulated saliva was collected at 10 am the day after the periodontal diagnosis. Subjects were asked to refrain from eating or drinking two hours prior to collection.	Colorimetric methods were used for all determinations in unstimulated saliva.	Proteomics			26221923
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X						x	CP	68055113	-1,44	35-55		Exclusion criteria included subjects with history of habits like usage of tobacco and/or consumption of alcohol, chronic inflammatory disorders of the skin and oral mucosa, known systemic illnesses; subjects on antibiotics and/or anti-inflammatory drugs within the last 6 months, subjects who underwent professional cleaning/periodontal treatment within the last 6 months, and pregnant women and lactating mothers.	Saliva Sampling - Whole unstimulated saliva of 2mL was collected from the subjects of both the groups in eppendorf tubes and stored at −80◦C, which was subjected to further analysis in order to evaluate the presence of the biomarker TIMP-1. A detailed clinical examination of the selected diseased subjects with periodontitis	ELISA was the method used to evaluate salivary TIMP-1 levels in this study, with the intention to provide an economical alternative for periodontal	Proteomics			26464855
P01583	IL1A	Interleukin-1 alpha	Homo sapiens (Human)						X						CP	68055113	6,24		M/F	all the subjects were non smokers, with the exception of two periodontal disease subjects who smoked ≤10 cigarettes/day. Exclusion criteria included pregnancy or lactation, systemic diseases or intake of medication, such as antibiotics, anti inflammatory agents or immunosuppressors, for six months prior to the study due to their possible effects on the immune or inflammatory response.	samples were collected from disease sites of chronic periodontal disease subjects and ten samples were collected from non disease sites of subjects without periodontitis. Subjects received instruction to not eat, drink or brush the teeth for 1 h prior to GCF sampling. Prior to GCF sampling, the individual tooth was isolated with cotton rolls, supragingival plaque was carefully removed and the site was gently air dried with an air syringe.	concentrations were determined using a commercial multiplex fluorescent bead based immunoassay kit (	Proteomics			24944641
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)						X						CP	68055113	57		M/F	all the subjects were non smokers, with the exception of two periodontal disease subjects who smoked ≤10 cigarettes/day. Exclusion criteria included pregnancy or lactation, systemic diseases or intake of medication, such as antibiotics, anti inflammatory agents or immunosuppressors, for six months prior to the study due to their possible effects on the immune or inflammatory response.	samples were collected from disease sites of chronic periodontal disease subjects and ten samples were collected from non disease sites of subjects without periodontitis. Subjects received instruction to not eat, drink or brush the teeth for 1 h prior to GCF sampling. Prior to GCF sampling, the individual tooth was isolated with cotton rolls, supragingival plaque was carefully removed and the site was gently air dried with an air syringe.	concentrations were determined using a commercial multiplex fluorescent bead based immunoassay kit (	Proteomics			24944641
P05231	IL6	Interleukin-6	Homo sapiens (Human)						X						CP	68055113	0		M/F	all the subjects were non smokers, with the exception of two periodontal disease subjects who smoked ≤10 cigarettes/day. Exclusion criteria included pregnancy or lactation, systemic diseases or intake of medication, such as antibiotics, anti inflammatory agents or immunosuppressors, for six months prior to the study due to their possible effects on the immune or inflammatory response.	samples were collected from disease sites of chronic periodontal disease subjects and ten samples were collected from non disease sites of subjects without periodontitis. Subjects received instruction to not eat, drink or brush the teeth for 1 h prior to GCF sampling. Prior to GCF sampling, the individual tooth was isolated with cotton rolls, supragingival plaque was carefully removed and the site was gently air dried with an air syringe.	concentrations were determined using a commercial multiplex fluorescent bead based immunoassay kit (	Proteomics			24944641
P22301	IL10	Interleukin-10	Homo sapiens (Human)						X						CP	68055113	13		M/F	all the subjects were non smokers, with the exception of two periodontal disease subjects who smoked ≤10 cigarettes/day. Exclusion criteria included pregnancy or lactation, systemic diseases or intake of medication, such as antibiotics, anti inflammatory agents or immunosuppressors, for six months prior to the study due to their possible effects on the immune or inflammatory response.	samples were collected from disease sites of chronic periodontal disease subjects and ten samples were collected from non disease sites of subjects without periodontitis. Subjects received instruction to not eat, drink or brush the teeth for 1 h prior to GCF sampling. Prior to GCF sampling, the individual tooth was isolated with cotton rolls, supragingival plaque was carefully removed and the site was gently air dried with an air syringe.	concentrations were determined using a commercial multiplex fluorescent bead based immunoassay kit (	Proteomics			24944641
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)						X						CP	68055113	6		M/F	all the subjects were non smokers, with the exception of two periodontal disease subjects who smoked ≤10 cigarettes/day. Exclusion criteria included pregnancy or lactation, systemic diseases or intake of medication, such as antibiotics, anti inflammatory agents or immunosuppressors, for six months prior to the study due to their possible effects on the immune or inflammatory response.	samples were collected from disease sites of chronic periodontal disease subjects and ten samples were collected from non disease sites of subjects without periodontitis. Subjects received instruction to not eat, drink or brush the teeth for 1 h prior to GCF sampling. Prior to GCF sampling, the individual tooth was isolated with cotton rolls, supragingival plaque was carefully removed and the site was gently air dried with an air syringe.	concentrations were determined using a commercial multiplex fluorescent bead based immunoassay kit (	Proteomics			24944641
P26022	PTX3	Pentraxin-related protein PTX3	Homo sapiens (Human)						X					X	CP	68055113	10,66	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)						X					X	CP	68055113	24,65	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007
P05231	IL6	Interleukin-6	Homo sapiens (Human)						X					X	CP	68055113	4,64	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007
P10145	CXCL8	Interleukin-8	Homo sapiens (Human)						X					X	CP	68055113	2	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007
P22301	IL10	Interleukin-10	Homo sapiens (Human)						X					X	CP	68055113	2,48	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)						X					X	CP	68055113	1,94	40-72	M/F	All subjects were systemically healthy and were not receiving any medication at the time of study. Exclusion criteria included: (1) subjects with systemic diseases such as diabetes mellitus, immunologic disorders, hepatitis, and cardiac disease; (2) pregnant and lactating women and women taking oral contraceptive drugs; (3) subjects who had received antibiotics or treatment for periodontal disease within the previous 3 months; and (4) individuals who self-reported the use of any tobacco products.	In each subject, GCF samples were collected using paper strips) from one diseased site and one healthy site. After isolation of the site with cotton rolls to prevent contamination with saliva, supragingival plaque was removed with cotton pellets and the tooth was air-dried. The paper strips were carefully inserted into the crevice until mild resistance was felt and were left in place for 30 seconds.	assay (ELISA) for PTX3 and Luminex assay for cytokine levels	Proteomics			21932007
P01137	TGFB1	Transforming growth factor beta-1	Homo sapiens (Human)						X					X	CP	68055113	-2,14			All subjects were systemically healthy and wasn't medicatio for all patients at various stages of treatment during the course of the study.	GCF collection the selected site was air dried and isolated with cotton rolls. A standardised volume of 1 µl GCF was collected from the selected sites in Eppendorf tubes using black colour-coded 1-5µl calibrated volumetric microcapillary pipettes (Sigma-Aldrich Chemical Company, USA.) by placing the tip of the pipette extracrevicularly (unstimulated) for 5-20 min.	enzyme immunometric assay (EIA) kit. (DRG Instruments GmbH, Germany).	Proteomics			25859517
P10145	CXCL8	Interleukin-8	Homo sapiens (Human)						X					X	CP	68055113		30-50	M/F	All subjects were systemically healthy andIndividuals who have systemic disease or pregnancy, tobacco use, history of periodontal treatment in previous 6 months, history of systemic antibiotic administration within the 3 months prior to the study or any other regular medication were excluded.	GCF samples were collected at baseline from the proximal defect site of the target tooth treated with bone grafting and before the collection, supragingival plaque was gently removed. Then, the tooth was isolated with cotton rolls for keeping away from contamination, and the cervical region was gently dried with air-spray.	The strips (Periopaper, Amityville, NY, USA) were placed inside the sulcus during 30 s in situ and then transported to a chair-side located Periotron 8000 (Orafl ow Inc., Plainview, NY, USA) calibrated with known volumes of phosphate-buffered saline (PBS), for identifying GCF volume. Four strips from region of interest were immediately placed in a labeled tube containing 500 µl PBS and following 10 s vortexing and 20 min. Shaking, the tubes were centrifuged during 5 min at 5800 rpm for removing plaque and other cellular elements. After the process, the samples were placed into ~ 80°C freezer and stored for subsequent assays.	Proteomics			25713486
Q16552	IL17A	Interleukin-17A	Homo sapiens (Human)						X					X	CP	68055113		30-50	M/F	All subjects were systemically healthy andIndividuals who have systemic disease or pregnancy, tobacco use, history of periodontal treatment in previous 6 months, history of systemic antibiotic administration within the 3 months prior to the study or any other regular medication were excluded.	GCF samples were collected at baseline from the proximal defect site of the target tooth treated with bone grafting and before the collection, supragingival plaque was gently removed. Then, the tooth was isolated with cotton rolls for keeping away from contamination, and the cervical region was gently dried with air-spray.	The strips (Periopaper, Amityville, NY, USA) were placed inside the sulcus during 30 s in situ and then transported to a chair-side located Periotron 8000 (Orafl ow Inc., Plainview, NY, USA) calibrated with known volumes of phosphate-buffered saline (PBS), for identifying GCF volume. Four strips from region of interest were immediately placed in a labeled tube containing 500 µl PBS and following 10 s vortexing and 20 min. Shaking, the tubes were centrifuged during 5 min at 5800 rpm for removing plaque and other cellular elements. After the process, the samples were placed into ~ 80°C freezer and stored for subsequent assays.	Proteomics			25713486

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P05362	ICAM1	Intercellular adhesion molecule 1	Homo sapiens (Human)						X					X	CP	68055113		30-50	M/F	All subjects were systemically healthy andIndividuals who have systemic disease or pregnancy, tobacco use, history of periodontal treatment in previous 6 months, history of systemic antibiotic administration within the 3 months prior to the study or any other regular medication were excluded.	GCF samples were collected at baseline from the proximal defect site of the target tooth treated with bone grafting and before the collection, supragingival plaque was gently removed. Then, the tooth was isolated with cotton rolls for keeping away from contamination, and the cervical region was gently dried with air-spray.	The strips (Periopaper, Amityville, NY, USA) were placed inside the sulcus during 30 s in situ and then transported to a chair-side located Periotron 8000 (Oraflow Inc., Plainview, NY, USA) calibrated with known volumes of phosphate-buffered saline (PBS), for identifying GCF volume. Four strips from region of interest were immediately placed in a labeled tube containing 500 µl PBS and following 10 s vortexing and 20 min. Shaking, the tubes were centrifuged during 5 min at 5800 rpm for removing plaque and other cellular elements. After the process, the samples were placed into − 80°C freezer and stored for subsequent assays.	Proteomics			25713486
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	1,7	42-61	M/F	in the study were regarded as the former smokers and Non-smokers, did not have history of smoking Individuals with known medical disorders, such as diabetes mellitus or immunological disorders and those who had received antibiotic or periodontal treatment in the last 6 months were excluded fromthe study.	Whole saliva samples were obtained simply by expectorating into polypropylene tubes before clinical periodontal measurements or any periodontal intervention and in the morning following an overnight fast during which subjects were requested not to drink (except water) or chew gum.	Analysis by Immunofluorometric Assay and concentrations were determined using commercially available ELISA kits.	Proteomics			24799321
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)					X							CP	68055113	3,3	42-61	M/F	in the study were regarded as the former smokers and Non-smokers, did not have history of smoking Individuals with known medical disorders, such as diabetes mellitus or immunological disorders and those who had received antibiotic or periodontal treatment in the last 6 months were excluded fromthe study.	Whole saliva samples were obtained simply by expectorating into polypropylene tubes before clinical periodontal measurements or any periodontal intervention and in the morning following an overnight fast during which subjects were requested not to drink (except water) or chew gum.	Analysis by Immunofluorometric Assay and concentrations were determined using commercially available ELISA kits.	Proteomics			24799321
P08246	ELANE	Neutrophil elastase	Homo sapiens (Human)					X							CP	68055113	7,25	42-61	M/F	in the study were regarded as the former smokers and Non-smokers, did not have history of smoking Individuals with known medical disorders, such as diabetes mellitus or immunological disorders and those who had received antibiotic or periodontal treatment in the last 6 months were excluded fromthe study.	Whole saliva samples were obtained simply by expectorating into polypropylene tubes before clinical periodontal measurements or any periodontal intervention and in the morning following an overnight fast during which subjects were requested not to drink (except water) or chew gum.	Analysis by Immunofluorometric Assay and concentrations were determined using commercially available ELISA kits.	Proteomics			24799321
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X							CP	68055113	-3,33	42-61	M/F	in the study were regarded as the former smokers and Non-smokers, did not have history of smoking Individuals with known medical disorders, such as diabetes mellitus or immunological disorders and those who had received antibiotic or periodontal treatment in the last 6 months were excluded fromthe study.	Whole saliva samples were obtained simply by expectorating into polypropylene tubes before clinical periodontal measurements or any periodontal intervention and in the morning following an overnight fast during which subjects were requested not to drink (except water) or chew gum.	Analysis by Immunofluorometric Assay and concentrations were determined using commercially available ELISA kits.	Proteomics			24799321
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)						X					X	CP	68055113	+	31-59	M/F	All subjects were systemically healthy and exclusion criteria involved (1) the presence of systemic diseases where administration of NSAIDs would be contraindicated such as stomach or duodenal ulcer, (2) presence of systemic illness or conditions which affect oral tissues such as insulin dependent diabetes mellitus, (3) known hypersensitivity to NSAIDs, (4) pregnancy, (5) smoking, and (6) any antibiotic, systemic corticosteroid, or immunosuppressive drug use within the past six months.	The GCF samples were collected using periopaper (Oraflow, Plainview, NY, USA) strips. The sample sites (the upper 4 incisors) were gently airdried and all supragingival plaque removed. The area was isolated carefully with cotton rolls and a saliva ejector was used to prevent saliva contamination. The paper strip was inserted into the pocket until slight resistance was felt and left in place for 30 s [26]. Care was taken to avoid mechanical injury of the gingival tissues. Strips contaminated by bleeding or exudate were discarded. Four samples per patient were placed into Eppendorf tubes and stored at –80°C until analysis.	samples were assayed using commercially available sandwich enzyme linked immunosorbent assay (ELISA) kits (RayBio, Norcross, GA, USA and Invitrogen, Carlsbad, CA, USA).	Proteomics			25166440
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)						X					X	CP	68055113	+	31-59	M/F	All subjects were systemically healthy and exclusion criteria involved (1) the presence of systemic diseases where administration of NSAIDs would be contraindicated such as stomach or duodenal ulcer, (2) presence of systemic illness or conditions which affect oral tissues such as insulin dependent diabetes mellitus, (3) known hypersensitivity to NSAIDs, (4) pregnancy, (5) smoking, and (6) any antibiotic, systemic corticosteroid, or immunosuppressive drug use within the past six months.	The GCF samples were collected using periopaper (Oraflow, Plainview, NY, USA) strips. The sample sites (the upper 4 incisors) were gently airdried and all supragingival plaque removed. The area was isolated carefully with cotton rolls and a saliva ejector was used to prevent saliva contamination. The paper strip was inserted into the pocket until slight resistance was felt and left in place for 30 s [26]. Care was taken to avoid mechanical injury of the gingival tissues. Strips contaminated by bleeding or exudate were discarded. Four samples per patient were placed into Eppendorf tubes and stored at –80°C until analysis.	samples were assayed using commercially available sandwich enzyme linked immunosorbent assay (ELISA) kits (RayBio, Norcross, GA, USA and Invitrogen, Carlsbad, CA, USA).	Proteomics			25166440
B3VMW0	0	Lactoferrin	Homo sapiens (Human)					X							CP	68055113	2,42		M/F	The CP+ group contained eight smokers and five individuals with diabetes mellitus.	Two millilitres of stimulated whole saliva was collected while chewing on paraffin blocks in standardized conditions. Before sampling, subjects were required to chew for 1 min, and saliva was discarded. Samples were frozen and stored at |80 C until final analysis.	Salivary lactoferrin was analysed using indirect ELISA.	Proteomics			22471324
Q9NRJ3	CCL28	C-C motif chemokine 28	Homo sapiens (Human)						X					X	CP	68055113	+		M/F	All subjects were systemically healthy and Exclusion criteria were the presence of any systemic disease, the use of any anticonvulsant medication or immunosuppressive drugs, periodontal treatment in the last 6 months, any drug addiction, smoking and pregnancy.	Gingival crevicular fluid samples were made in Each tooth zone was dried by air-spraying to prevent irritation and isolated carefully with cotton tampons. To avoid contamination of samples with saliva, saliva absorbents were used.	The levels of CCL28, IL-8, IL-1b and TNF-a were analyzed in gingival crevicular fluid using commercial ELISA kits	Proteomics			22812409
P10145	CXCL8	Interleukin-8	Homo sapiens (Human)						X					X	CP	68055113	+		M/F	All subjects were systemically healthy and Exclusion criteria were the presence of any systemic disease, the use of any anticonvulsant medication or immunosuppressive drugs, periodontal treatment in the last 6 months, any drug addiction, smoking and pregnancy.	Gingival crevicular fluid samples were made in Each tooth zone was dried by air-spraying to prevent irritation and isolated carefully with cotton tampons. To avoid contamination of samples with saliva, saliva absorbents were used.	The levels of CCL28, IL-8, IL-1b and TNF-a were analyzed in gingival crevicular fluid using commercial ELISA kits	Proteomics			22812409
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)						X					X	CP	68055113	+		M/F	All subjects were systemically healthy and Exclusion criteria were the presence of any systemic disease, the use of any anticonvulsant medication or immunosuppressive drugs, periodontal treatment in the last 6 months, any drug addiction, smoking and pregnancy.	Gingival crevicular fluid samples were made in Each tooth zone was dried by air-spraying to prevent irritation and isolated carefully with cotton tampons. To avoid contamination of samples with saliva, saliva absorbents were used.	The levels of CCL28, IL-8, IL-1b and TNF-a were analyzed in gingival crevicular fluid using commercial ELISA kits	Proteomics			22812409
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)						X					X	CP	68055113	+		M/F	All subjects were systemically healthy and Exclusion criteria were the presence of any systemic disease, the use of any anticonvulsant medication or immunosuppressive drugs, periodontal treatment in the last 6 months, any drug addiction, smoking and pregnancy.	Gingival crevicular fluid samples were made in Each tooth zone was dried by air-spraying to prevent irritation and isolated carefully with cotton tampons. To avoid contamination of samples with saliva, saliva absorbents were used.	The levels of CCL28, IL-8, IL-1b and TNF-a were analyzed in gingival crevicular fluid using commercial ELISA kits	Proteomics			22812409
A4D0Y8	LEP	Leptin	Homo sapiens (Human)						X					X	CP	68055113	-		M/F	All subjects were systemically healthy and Exclusion criteria were: aggressive periodontitis; smoking; alcoholism; and/or pregnancy. Lactating women were also excluded from the study. The nature and procedures of the	Samples of gingival crevicular fluid were obtained before probing by placing white color-coded 1–5 IL calibrated volumetric microcapillary pipettes on the the site.From each test site, a extracrevicularly (unstimulated). Test sites from which no gingival crevicular fluid could be obtained, and the micropipette which was contaminated with blood and saliva, were excluded from the study.standardized volume of 1 IL was collected, using the calibration on the micropipette, by placing the tip of the pipette	The assay was performed using the leptin Elisa kit	Proteomics			17559625

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	+	21-64	M/F	smokers or former-smokers were not excluded from the study. Patients with medical disorders, such as immunological disorders, diabetes mellitus, those who received antibiotic treatment within the last 3 months, and periodontal treatment within the last 6 months, or had less than 20 teeth and those wearing removable dentures were also excluded.	All patients were asked to simply expectorate into polypropylene tubes in order to collect unstimulated whole saliva was performed before clinical periodontal measurements and/or any periodontal intervention, in the morning following an overnight fast during which patients were asked not to drink anything except water or chew gum. 500 mL aliquots of saliva samples were placed in sterile polypropylene tubes following clarification by centrifugation (800 g) for 10 min at room temperature.	analyses by immunofluorometric assay, by enzyme-linked immunosorbent assay (ELISA) and Western immunoblotting	Proteomics			25455127
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)					X							CP	68055113	-	21-64	M/F	smokers or former-smokers were not excluded from the study. Patients with medical disorders, such as immunological disorders, diabetes mellitus, those who received antibiotic treatment within the last 3 months, and periodontal treatment within the last 6 months, or had less than 20 teeth and those wearing removable dentures were also excluded.	All patients were asked to simply expectorate into polypropylene tubes in order to collect unstimulated whole saliva was performed before clinical periodontal measurements and/or any periodontal intervention, in the morning following an overnight fast during which patients were asked not to drink anything except water or chew gum. 500 mL aliquots of saliva samples were placed in sterile polypropylene tubes following clarification by centrifugation (800 g) for 10 min at room temperature.	analyses by immunofluorometric assay, by enzyme-linked immunosorbent assay (ELISA) and Western immunoblotting	Proteomics			25455127
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X							CP	68055113	-	21-64	M/F	smokers or former-smokers were not excluded from the study. Patients with medical disorders, such as immunological disorders, diabetes mellitus, those who received antibiotic treatment within the last 3 months, and periodontal treatment within the last 6 months, or had less than 20 teeth and those wearing removable dentures were also excluded.	All patients were asked to simply expectorate into polypropylene tubes in order to collect unstimulated whole saliva was performed before clinical periodontal measurements and/or any periodontal intervention, in the morning following an overnight fast during which patients were asked not to drink anything except water or chew gum. 500 mL aliquots of saliva samples were placed in sterile polypropylene tubes following clarification by centrifugation (800 g) for 10 min at room temperature.	analyses by immunofluorometric assay, by enzyme-linked immunosorbent assay (ELISA) and Western immunoblotting	Proteomics			25455127
P08246	ELANE	Neutrophil elastase	Homo sapiens (Human)					X							CP	68055113	-	21-64	M/F	smokers or former-smokers were not excluded from the study. Patients with medical disorders, such as immunological disorders, diabetes mellitus, those who received antibiotic treatment within the last 3 months, and periodontal treatment within the last 6 months, or had less than 20 teeth and those wearing removable dentures were also excluded.	All patients were asked to simply expectorate into polypropylene tubes in order to collect unstimulated whole saliva was performed before clinical periodontal measurements and/or any periodontal intervention, in the morning following an overnight fast during which patients were asked not to drink anything except water or chew gum. 500 mL aliquots of saliva samples were placed in sterile polypropylene tubes following clarification by centrifugation (800 g) for 10 min at room temperature.	analyses by immunofluorometric assay, by enzyme-linked immunosorbent assay (ELISA) and Western immunoblotting	Proteomics			25455127
P80188	LCN2	Neutrophil gelatinase-associated lipocalin	Homo sapiens (Human)					X							CP	68055113	-	21-64	M/F	smokers or former-smokers were not excluded from the study. Patients with medical disorders, such as immunological disorders, diabetes mellitus, those who received antibiotic treatment within the last 3 months, and periodontal treatment within the last 6 months, or had less than 20 teeth and those wearing removable dentures were also excluded.	All patients were asked to simply expectorate into polypropylene tubes in order to collect unstimulated whole saliva was performed before clinical periodontal measurements and/or any periodontal intervention, in the morning following an overnight fast during which patients were asked not to drink anything except water or chew gum. 500 mL aliquots of saliva samples were placed in sterile polypropylene tubes following clarification by centrifugation (800 g) for 10 min at room temperature.	analyses by immunofluorometric assay, by enzyme-linked immunosorbent assay (ELISA) and Western immunoblotting	Proteomics			25455127
Q15722	LTB4R	Leukotriene B4 receptor 1	Homo sapiens (Human)					X						X	CP	68055113	1,61	30-61	M/F	All subjects were systemically healthy and exclusion criteria included smokers, diabetes, immune suppression and medicine use.	Whole saliva was collected by spiting into an ice-cooled graduated vessel. Subjects spat out every 30 s for 5 min. The volume of saliva was recorded and expressed as mL/min. The resulting saliva was stored in aliquots at 20°C until determinations were performed. Saliva was collected at 10.00 h by one calibrated examiner the day after periodontal diagnosis.	The concentration of LTB4 in saliva was measured by the Enzyme Immuno Assay kit from Cayman Chemicals Co. (Ann Arbor, MI, USA).	Proteomics			23488687
O60603	TLR2	Toll-like receptor 2	Homo sapiens (Human)					X						X	CP	68055113	+		M/F	Twenty systemically healthy nonsmokers	UWS was collected by the drooling method, and epithelial cells were	Epithelial cells isolated from each UWS sample were assessed for TLR-2, TLR-4, peptidoglycan recognition protein (PGRP)-3 and PGRP-4 by quantitative real-time PCR.The culture supernatant was assessed for cytokines by ELISA.	Proteomics			23679005
O00206	TLR4	Toll-like receptor 4	Homo sapiens (Human)					X						X	CP	68055113	+		M/F	Twenty systemically healthy nonsmokers	UWS was collected by the drooling method, and epithelial cells were	Epithelial cells isolated from each UWS sample were assessed for TLR-2, TLR-4, peptidoglycan recognition protein (PGRP)-3 and PGRP-4 by quantitative real-time PCR.The culture supernatant was assessed for cytokines by ELISA.	Proteomics			23679005
Q59E93	0	Membrane alanine aminopeptidase variant	Homo sapiens (Human)					X							CP	68055113	+	25-72	M/F	All subjects were systemically healthy and exclusion criteria included: smoking; use of dental prostheses/orthodontic appliances; pregnancy; nursing; periodontal therapy in the previous 6 months; use of antibiotics or mouthrinse in the previous 3 months; and any systemic condition that might affect the progression of periodontitis	In the periodontitis groups (LCP and GCP), subgingival plaque samples were collected from eight diseased sites (two deepest PDs in each quadrant). In periodontally healthy subjects, samples were collected from the two deepest gingival crevices without BOP in each quadrant. Teeth were gently dried with sterile cotton swabs.	DPPIV enzyme activity was determined by spectroscopic quantification of glycyl-prolyl-p-nitroanilide hydrolysis and at -80 C until DNA extraction. Determination of ALAP and DPPIV Activities in Saliva An ALAP activity assay was by of a colorimetric assay measuring the extent of hydrolysis of L-alanyl-b-naphthylamide	Proteomics			19905950
P27487	DPP4	Dipeptidyl peptidase 4	Homo sapiens (Human)					X							CP	68055113	+	25-72	M/F	All subjects were systemically healthy and exclusion criteria included: smoking; use of dental prostheses/orthodontic appliances; pregnancy; nursing; periodontal therapy in the previous 6 months; use of antibiotics or mouthrinse in the previous 3 months; and any systemic condition that might affect the progression of periodontitis	In the periodontitis groups (LCP and GCP), subgingival plaque samples were collected from eight diseased sites (two deepest PDs in each quadrant). In periodontally healthy subjects, samples were collected from the two deepest gingival crevices without BOP in each quadrant. Teeth were gently dried with sterile cotton swabs.	DPPIV enzyme activity was determined by spectroscopic quantification of glycyl-prolyl-p-nitroanilide hydrolysis and at -80 C until DNA extraction. Determination of ALAP and DPPIV Activities in Saliva An ALAP activity assay was by of a colorimetric assay measuring the extent of hydrolysis of L-alanyl-b-naphthylamide	Proteomics			19905950
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	+	25-75	M/F	A total of 92 patients (47 AMI and 28 non-AMI patients with gingivitis or periodontitis; and 17 systemically and periodontally healthy patients as a control group) were recruited.Edentulous patients were excluded from this study. All participantswere interviewed about their medical status, dental history, and common habits. Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection.	Saliva samples were examined by Western immunoblotting using polyclonal antibodies for MMP-8 and MMP-13 and by gelatinase assay for MMP-942 for identification and quantitation of different molecular forms by scanning image analysis.Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346
P09237	MMP7	Matrilysin	Homo sapiens (Human)					X							CP	68055113	2,1	25-75	M/F	A total of 92 patients (47 AMI and 28 non-AMI patients with gingivitis or periodontitis; and 17 systemically and periodontally healthy patients as a control group) were recruited.Edentulous patients were excluded from this study. All participantswere interviewed about their medical status, dental history, and common habits. Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection.	Saliva samples were examined by Western immunoblotting using polyclonal antibodies for MMP-8 and MMP-13 and by gelatinase assay for MMP-942 for identification and quantitation of different molecular forms by scanning image analysis.Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X							CP	68055113	1,4	25-75	M/F	A total of 92 patients (47 AMI and 28 non-AMI patients with gingivitis or periodontitis; and 17 systemically and periodontally healthy patients as a control group) were recruited.Edentulous patients were excluded from this study. All participantswere interviewed about their medical status, dental history, and common habits. Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection.	Saliva samples were examined by Western immunoblotting using polyclonal antibodies for MMP-8 and MMP-13 and by gelatinase assay for MMP-942 for identification and quantitation of different molecular forms by scanning image analysis.Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P16152	CBR1	Carbonyl reductase [NADPH] 1	Homo sapiens (Human)					X							CP	68055113	1,57	65,4	M/F	atherosclerosis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.		Proteomics			25238219
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)					X							CP	68055113	1,88	65,4	M/F	Chronic periodontitis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.	Salivary inflammatory cytokines were determined by enzymelinked immunosorbent assay (ELISA).	Proteomics			25238219
P05231	IL6	Interleukin-6	Homo sapiens (Human)					X							CP	68055113	1,95	65,4	M/F	Chronic periodontitis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.	Salivary inflammatory cytokines were determined by enzymelinked immunosorbent assay (ELISA).	Proteomics			25238219
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)					X							CP	68055113	1,70	65,4	M/F	Chronic periodontitis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.	Salivary inflammatory cytokines were determined by enzymelinked immunosorbent assay (ELISA).	Proteomics			25238219
P43116	PTGER2	Prostaglandin E2 receptor EP2 subtype	Homo sapiens (Human)					X							CP	68055113	15	65,4	M/F	Chronic periodontitis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.	Salivary inflammatory cytokines were determined by enzymelinked immunosorbent assay (ELISA).	Proteomics			25238219
P16152	CBR1	Carbonyl reductase [NADPH] 1	Homo sapiens (Human)					X							CP	68055113	1,70	65,4	M/F	Chronic periodontitis	Participants were conducted to chew a piece of paraffin gum for 2 min to stimulate salivary flow. Subsequently, subjects were asked to spit the saliva into a test tube. Samples, which were stored at 80 C until use, were delivered to the laboratory for analyses.	Salivary inflammatory cytokines were determined by enzymelinked immunosorbent assay (ELISA).	Proteomics			25238219
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P05231	IL6	Interleukin-6	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P22301	IL10	Interleukin-10	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P43116	PTGER2	Prostaglandin E2 receptor EP2 subtype	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P05112	IL4	Interleukin-4	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)						X						CP	68055113	+	20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257
P45452	MMP13	Collagenase 3	Homo sapiens						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs (prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA, .tobacco smokers were excluded.			Proteomics			22414257

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
Q14116	IL18	Interleukin-18	Homo sapiens (Human)						X						CP	68055113		20-76	M/F	Patients with rheumatoid arthritis.patients were prescribed antimetabolites (methotrexate), nonsteroidal anti-inflammatory drugs (indomethacin), immunosuppressant drugs ((prednosine) and pyrimidine synthesis inhibitors (leflunomide) for the treatment of RA.the drugs used for RA treatment were not reported.anti-TNF-a therapy was recommended to the patients for the treatment of RA. ,tobacco smokers were excluded.			Proteomics			22414257
O95760	IL33	Interleukin-33	Homo sapiens (Human)						X						CP	68055113	-1,09	35-65	M/F	All subjects were systemically healthy and all individuals with CP had >20 teeth present. Individuals with medical disorders, such as diabetes mellitus, immunologic disorders, hepatitis, and those who had antibiotic or periodontal treatment in the previous 6 months were excluded from the study.	GCF samples were collected using filter paper strips.‡ Before GCF sampling, supragingival plaque was removed from the interproximal surfaces with a sterile curet; these surfaceswere dried gently by an air syringe and were isolated by cotton rolls. Paper strips were carefully inserted 1mmnto the crevice and left there for 30 seconds.Carewas taken to avoidmechanical injury. Strips contaminated with blood were discarded. The absorbed GCF volume was estimated by a calibrated instrument.§ Then, the two strips fromtwo sites of each individual were placed into one polypropylene tube and pooled before freezing at -40 C.	IL-33 levels in the biofluid samples were determined by enzyme-linked immunosorbent assay.	Proteomics			21859321
P10645	CHGA	Chromogranin-A	Homo sapiens (Human)					X							CP	68055113	+	31,5	M/F	Exclusion criteria for all groups included the following: <20 teeth;any apparent oral infection (i.e., herpes or candida); injuries or bleeding in the oral cavity unrelated to periodontitis; periodontal treatment and antibiotic medication within the past 3 months; salivary gland dysfunction; acute illness (fever, sore throat);systemic disease, mental diseases, immune-suppressive medication, or immunodeficiency;pregnancy or lactation; allergies to benzoic acid; and in particular, intake of sedatives, tranquilizers, or antidepressants.	Stimulated whole saliva from all groups was collected from 8:00 am to 11:00 am using a rinsing solution of a saliva collection system,according to the instructions of the manufacturer. Participants were not allowed to eat, drink, smoke, brush their teeth, or put anything else in their mouth from midnight on the day before sampling so as to avoid contamination of the saliva sample. Sampling in the periodontitis groups was exclusively made before a planned conservative periodontal therapy.	CgA levels were analyzed with a CgA enzyme-linked immunosorbent assay (ELISA) kit for saliva and AA activity was measured according to the kinetic test for quantitative determination of AA.‡‡ following a dilution protocol for saliva	Proteomics			22264209
P04745	AMY1A	Alpha-amylase 1	Homo sapiens (Human)					X							CP	68055113	-	31,5	M/F	Exclusion criteria for all groups included the following: <20 teeth;any apparent oral infection (i.e., herpes or candida); injuries or bleeding in the oral cavity unrelated to periodontitis; periodontal treatment and antibiotic medication within the past 3 months; salivary gland dysfunction; acute illness (fever, sore throat);systemic disease, mental diseases, immune-suppressive medication, or immunodeficiency;pregnancy or lactation; allergies to benzoic acid; and in particular, intake of sedatives, tranquilizers, or antidepressants.	Stimulated whole saliva from all groups was collected from 8:00 am to 11:00 am using a rinsing solution of a saliva collection system,according to the instructions of the manufacturer. Participants were not allowed to eat, drink, smoke, brush their teeth, or put anything else in their mouth from midnight on the day before sampling so as to avoid contamination of the saliva sample. Sampling in the periodontitis groups was exclusively made before a planned conservative periodontal therapy.	CgA levels were analyzed with a CgA enzyme-linked immunosorbent assay (ELISA) kit for saliva and AA activity was measured according to the kinetic test for quantitative determination of AA.‡‡ following a dilution protocol for saliva	Proteomics			22264209
P08246	ELANE	Neutrophil elastase	Homo sapiens (Human)					X							CP	68055113	+	25-68	M/F	Exclusion criteria were: patients with severe medical disorders, including diabetes mellitus or immunological disorders; patients with any history of any other systemic disease; patients who had received antibiotics or other medicines or periodontal treatment within the past 6 mo; and pregnant women.	Stimulated whole-saliva samples were obtained from all individuals. In order to stimulate saliva production, the participants chewed a piece of paraffin wax for 7 min. Saliva produced during the first 2 min was discarded and the saliva produced during the following 5 min was collected.	Saliva samples were analyzed using the chromogenic substrates 1 mM succinyl-alanyl-alanyl-valine-p-nitroanilide (SAAVNA) for PMN elastase activity (22) and 1 mM N-succinyl-Ala-Ala-Pro-Phe p-nitroanilide (SAAPPNA) for PMN cathepsin G activity, the increase of absorbance was detected by spectrophotometry at 405 nm.	Proteomics			22181012
P08311	CTSG	Cathepsin G	Homo sapiens (Human)					X							CP	68055113	+	25-68	M/F	Exclusion criteria were: patients with severe medical disorders, including diabetes mellitus or immunological disorders; patients with any history of any other systemic disease; patients who had received antibiotics or other medicines or periodontal treatment within the past 6 mo; and pregnant women.	Stimulated whole-saliva samples were obtained from all individuals. In order to stimulate saliva production, the participants chewed a piece of paraffin wax for 7 min. Saliva produced during the first 2 min was discarded and the saliva produced during the following 5 min was collected.	Saliva samples were analyzed using the chromogenic substrates 1 mM succinyl-alanyl-alanyl-valine-p-nitroanilide (SAAVNA) for PMN elastase activity (22) and 1 mM N-succinyl-Ala-Ala-Pro-Phe p-nitroanilide (SAAPPNA) for PMN cathepsin G activity, the increase of absorbance was detected by spectrophotometry at 405 nm.	Proteomics			22181012
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	-	61	M/F	smokers and non-smokers.study participants were excluded if they had undergone cardiac valvular surgery or had language barriers preventing them to complete study procedures.	Sample collection: Stimulated saliva samples were obtained by chewing paraffin wax up to 10 minutes. The produced saliva was collected into a graded test-tube. The saliva collection continued until 2 mL of saliva was obtained or until 10 minutes had passed. The collected amount was determined, excluding the foam. Collected samples were immediately frozen at -20°C or lower until processing.	The biomarkers MMP -8, -9, MPO and TIMP-1 were analyzed by time-resolved immunofluorescence assay (IFMA), Western blot and Enzyme-Linked ImmunoSorbent Assay (ELISA).	Proteomics			26132583
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)					X							CP	68055113	-	61	M/F	smokers and non-smokers.study participants were excluded if they had undergone cardiac valvular surgery or had language barriers preventing them to complete study procedures.	Sample collection: Stimulated saliva samples were obtained by chewing paraffin wax up to 10 minutes. The produced saliva was collected into a graded test-tube. The saliva collection continued until 2 mL of saliva was obtained or until 10 minutes had passed. The collected amount was determined, excluding the foam. Collected samples were immediately frozen at -20°C or lower until processing.	The biomarkers MMP -8, -9, MPO and TIMP-1 were analyzed by time-resolved immunofluorescence assay (IFMA), Western blot and Enzyme-Linked ImmunoSorbent Assay (ELISA).	Proteomics			26132583
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X							CP	68055113	-	61	M/F	smokers and non-smokers.study participants were excluded if they had undergone cardiac valvular surgery or had language barriers preventing them to complete study procedures.	Sample collection: Stimulated saliva samples were obtained by chewing paraffin wax up to 10 minutes. The produced saliva was collected into a graded test-tube. The saliva collection continued until 2 mL of saliva was obtained or until 10 minutes had passed. The collected amount was determined, excluding the foam. Collected samples were immediately frozen at -20°C or lower until processing.	The biomarkers MMP -8, -9, MPO and TIMP-1 were analyzed by time-resolved immunofluorescence assay (IFMA), Western blot and Enzyme-Linked ImmunoSorbent Assay (ELISA).	Proteomics			26132583
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)					X							CP	68055113	+	61	M/F	smokers and non-smokers.study participants were excluded if they had undergone cardiac valvular surgery or had language barriers preventing them to complete study procedures.	Sample collection: Stimulated saliva samples were obtained by chewing paraffin wax up to 10 minutes. The produced saliva was collected into a graded test-tube. The saliva collection continued until 2 mL of saliva was obtained or until 10 minutes had passed. The collected amount was determined, excluding the foam. Collected samples were immediately frozen at -20°C or lower until processing.	The biomarkers MMP -8, -9, MPO and TIMP-1 were analyzed by time-resolved immunofluorescence assay (IFMA), Western blot and Enzyme-Linked ImmunoSorbent Assay (ELISA).	Proteomics			26132583
P14780	MMP9	Matrix metalloproteinase-9	Homo sapiens (Human)					X							CP	68055113	+	61	M/F	smokers and non-smokers.study participants were excluded if they had undergone cardiac valvular surgery or had language barriers preventing them to complete study procedures.	Sample collection: Stimulated saliva samples were obtained by chewing paraffin wax up to 10 minutes. The produced saliva was collected into a graded test-tube. The saliva collection continued until 2 mL of saliva was obtained or until 10 minutes had passed. The collected amount was determined, excluding the foam. Collected samples were immediately frozen at -20°C or lower until processing.	The biomarkers MMP -8, -9, MPO and TIMP-1 were analyzed by time-resolved immunofluorescence assay (IFMA), Western blot and Enzyme-Linked ImmunoSorbent Assay (ELISA).	Proteomics			26132583
P09237	MMP7	Matrilysin	Homo sapiens (Human)					X							CP	68055113	+	25-68	M/F	Smoking habits.All participants were interviewed about their medical status, dental history, and common habits.Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection. The saliva samples were weighed and immediately frozen at -40 C.	Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)					X							CP	68055113	+	25-68	M/F	Smoking habits.All participants were interviewed about their medical status, dental history, and common habits.Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection. The saliva samples were weighed and immediately frozen at -40 C.	Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	+	25-68	M/F	Smoking habits.All participants were interviewed about their medical status, dental history, and common habits.Data on hypertension, diabetes, and smoking habits were recorded.	Stimulated whole saliva samples were obtained from all individuals. To stimulate saliva production, the participants chewed a piece of paraffin wax for 7 minutes. Saliva produced during the first 2 minutes was discarded. Then, saliva was collected during the following 5 minutes. The participants chewed the paraffin during the time of saliva collection. The saliva samples were weighed and immediately frozen at -40 C.	Molecular forms and degree of activation of salivary MMP-8, MMP-9, and MMP-13 were analyzed by computerscanned immunoblots.	Proteomics			21091346

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01009	SERPINA1	Alpha-1-antitrypsin	Homo sapiens (Human)						X						CP	68055113	-1,52	20-75	M/F	the patients has arteriosclerosis and chronic periodontitis. Hypertensio Dyslipidemia, Diabetes mellitus,Chronic renal failure. Eight patients were excluded because of malignant tumor comorbidity, consumption of corticosteroids or immunosuppressants, immunodeficiency diseases, undergoing dialysis, or liver cirrhosis	The test involved collecting GCF with a brush (Perio-catcher, Ikagaku Company, Limited, Kyoto, Japan). The subjects were instructed to refrain from eating, smoking, brushing their teeth, and gargling for at least 30 minutes before the sample collection. GCF samples were collected from the gingival crevices of 10 teeth between the left and right maxillary second premolars, taking advantage of the capillary phenomenon using a small brush of 2-mm diameter and 6-mm length. The brush was lightly and smoothly traced back and forth across the border between the teeth and gums for 30 seconds.	The GCF-Lf and GCF-AT measurements were made with Perio-catcher equipment from Ikagaku Company, Limited.	Proteomics			26549390
B3VMW0	0	Lactoferrin	Homo sapiens (Human)						X						CP	68055113	-2,78	20-75	M/F	the patients has arteriosclerosis and chronic periodontitis. Hypertensio Dyslipidemia, Diabetes mellitus,Chronic renal failure. Eight patients were excluded because of malignant tumor comorbidity, consumption of corticosteroids or immunosuppressants, immunodeficiency diseases, undergoing dialysis, or liver cirrhosis	The test involved collecting GCF with a brush (Perio-catcher, Ikagaku Company, Limited, Kyoto, Japan). The subjects were instructed to refrain from eating, smoking, brushing their teeth, and gargling for at least 30 minutes before the sample collection. GCF samples were collected from the gingival crevices of 10 teeth between the left and right maxillary second premolars, taking advantage of the capillary phenomenon using a small brush of 2-mm diameter and 6-mm length. The brush was lightly and smoothly traced back and forth across the border between the teeth and gums for 30 seconds.	The GCF-Lf and GCF-AT measurements were made with Perio-catcher equipment from Ikagaku Company, Limited.	Proteomics			26549390
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)						X						CP	68055113	+		M/F	periodontitis, peri-implantitis and cardiovascular diseases		Western immunoblot analysis	Proteomics			20937384
P45452	MMP13	Collagenase 3	Homo sapiens (Human)						X						CP	68055113			M/F	periodontitis, peri-implantitis and cardiovascular diseases		Western immunoblot analysis	Proteomics			20937384
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)						X						CP	68055113			M/F	periodontitis, peri-implantitis and cardiovascular diseases		Western immunoblot analysis	Proteomics			20937384
P05231	IL6	Interleukin-6	Homo sapiens (Human)					X							CP	68055113	+	35-65	M/F	Patients With Periodontitis and Diabetes.The exclusion criteria were: 1) smoking within the past 5 years; 2) pregnancy or lactation; 3) use of antibiotics or periodontal treatment in the previous 6 months; 4) concomitant medical therapy, except for a diabetic condition; 5) other inflammatory conditions; and 6) major diabetic complications (retinopathy, nephropathy, neuropathy, and atherosclerosis).Patients with diabetes had type 2 DM diagnosed for ±5 years and current metabolic control above the normal range (glycated hemoglobin [HbA1c] test >6%3).	Non-stimulated whole expectorated saliva was collected (;3 ml) from each subject into sterile tubes according to the method described by Navazesh.23 Subjects refrained from eating, drinking, and oral hygiene for 2 hours prior to saliva collection. Saliva samples were placed on ice immediately and aliquoted prior to freezing at -80 C.	Salivary concentrations of IL-6, MMP-8, and OPG were determined in duplicate for each subject using an enzyme-linked immunosorbent assay (ELISA).	Proteomics			20192865
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)					X							CP	68055113	+	35-65	M/F	Patients With Periodontitis and Diabetes.The exclusion criteria were: 1) smoking within the past 5 years; 2) pregnancy or lactation; 3) use of antibiotics or periodontal treatment in the previous 6 months; 4) concomitant medical therapy, except for a diabetic condition; 5) other inflammatory conditions; and 6) major diabetic complications (retinopathy, nephropathy, neuropathy, and atherosclerosis).Patients with diabetes had type 2 DM diagnosed for ±5 years and current metabolic control above the normal range (glycated hemoglobin [HbA1c] test >6%3).	Non-stimulated whole expectorated saliva was collected (;3 ml) from each subject into sterile tubes according to the method described by Navazesh.23 Subjects refrained from eating, drinking, and oral hygiene for 2 hours prior to saliva collection. Saliva samples were placed on ice immediately and aliquoted prior to freezing at -80 C.	Salivary concentrations of IL-6, MMP-8, and OPG were determined in duplicate for each subject using an enzyme-linked immunosorbent assay (ELISA).	Proteomics			20192865
P25090	FPR2	N-formyl peptide receptor 2	Homo sapiens (Human)						X						CP	68055113	-		M/F	smokers and nonsmokers with chronic periodontitis.Inclusion criteria were as follows: (i) ≥18 years of age and having ≥ 16 teeth; (ii) no periodontal therapy in the 6 mo before data collection; and (iii) no systemic problems or chemotherapy within the 6 wk before data collection. Individuals were included as smokers if they had smoked ≥15 cigarettes/d for ≥5 years, whereas individuals were included as nonsmokers if they had no previous history of smoking.	Gingival crevicular fluid samples were collected using periopaper strips (Oraflow Inc., Plainview, NY, USA). Before sample collection, each site was gently air-dried, all supragingival plaque was removed and the area was carefully isolated to prevent samples from being contaminated with saliva. The paper strip was inserted into the gingival crevice up to 1 mm, or until mild resistance was felt, and was left in place for 30 s. Care was taken to avoid mechanical injury of gingival tissue, and any strip contaminated by bleeding or exudate was discarded.	Gingival crevicular fluid IL-8 and LxA4 levels were analyzed by ELISA	Proteomics			26446985
P10145	CXCL8	Interleukin-8	Homo sapiens (Human)						X						CP	68055113	+		M/F	smokers and nonsmokers with chronic periodontitis.Inclusion criteria were as follows: (i) ≥18 years of age and having ≥ 16 teeth; (ii) no periodontal therapy in the 6 mo before data collection; and (iii) no systemic problems or chemotherapy within the 6 wk before data collection. Individuals were included as smokers if they had smoked ≥15 cigarettes/d for ≥5 years, whereas individuals were included as nonsmokers if they had no previous history of smoking.	Gingival crevicular fluid samples were collected using periopaper strips (Oraflow Inc., Plainview, NY, USA). Before sample collection, each site was gently air-dried, all supragingival plaque was removed and the area was carefully isolated to prevent samples from being contaminated with saliva. The paper strip was inserted into the gingival crevice up to 1 mm, or until mild resistance was felt, and was left in place for 30 s. Care was taken to avoid mechanical injury of gingival tissue, and any strip contaminated by bleeding or exudate was discarded.	Gingival crevicular fluid IL-8 and LxA4 levels were analyzed by ELISA	Proteomics			26446985
P20160	AZU1	Azurocidin	Homo sapiens (Human)						X						CP	68055113	-1,37	46	M/F	All subjects were systemically healthy andl the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P42830	CXCL5	C-X-C motif chemokine 5	Homo sapiens (Human)						X						CP	68055113		46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)						X						CP	68055113	-1,04	46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P22894	MMP8	Neutrophil collagenase	Homo sapiens (Human)						X						CP	68055113	-1,14	46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P45452	MMP13	Collagenase 3	Homo sapiens (Human)						X						CP	68055113	-1,28	46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P50281	MMP14	Matrix metalloproteinase-14	Homo sapiens (Human)						X						CP	68055113	-1,22	46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P01033	TIMP1	Metalloproteinase inhibitor 1	Homo sapiens (Human)						X						CP	68055113		46	M/F	All subjects were systemically healthy and the individuals had no history of systemic disorders, such as diabetes mellitus, osteoporosis, pregnancy or lactating females and medications known to influence periodontal tissues, within the past 6 months period prior to the study	GCF samples were consecutively collected with paper strips (ProFlow, Amityville, NY, USA), placed into the pocket until mild resistance was sensed, and left in place for 30 s as previously reported (Hernandez Rios et al. 2009). GCF was extracted from the strips by centrifugation at 18,000 g for 5 min. at 4°C in 80 ll of elution buffer containing 50 mM Tris-HCl pH 7.5, 0.2 M NaCl, 5 mM CaCl2 and 0.01% Trit on X-100.	The levels of azurocidin, CXCL5, MPO, MMP-8 and TIMP-1 were determined by the following commercial ELISA kits,	Proteomics			24382144
P02741	CRP	C-reactive protein	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P05231	IL6	Interleukin-6	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P05164	MPO	Myeloperoxidase	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P01375	TNF	Tumor necrosis factor	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P14780	MMP9	Matrix metalloproteinase-9	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
Q15848	ADIPOQ	Adiponectin	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
Q99930	SICAM-1	sICAM-1	Homo sapiens (Human)					X							CP	68055113		54,2	M/F	Acute myocardial infarction and chronic periodontitis associated.Exclusion criteria were fever, accompanying stroke, immune system disorders, use of steroidal medications, major organ complications/failure, inability to provide saliva, and age < 18 yr	unstimulated whole saliva (UWS)|were made by a single examiner (i.e., study coordinator) at times when the patient was considered stable, per our previously described method (Foley et al., 2012b).	The Luminex IS-100 instrument (Luminex Corp., Austin, TX, USA) was used for detection of 9 biomarkers in serum and saliva	Proteomics			24879575
P15941	MUC1	Mucin-1	Homo sapiens (Human)					X							CP	68055113	+	32-62	M/F	All subjects were systemically healthy and were made xclusion criteria included: smokers, diabetes, immunosuppression and medicine use	Whole saliva was collected by spitting into an ice-cooled graduated vessel (Falcon type). Subjects spat out every 30 s for 5 min. The total volume of saliva was recorded using a glass pipette and divided by the collection time, then expressed as ml per minute. The resulting saliva was stored in aliquots at 20°C until determinations were performed.	Analysis of protein, amylase and mucin concentration Colorimetric methods were used for all analysis in unstimulated saliva collected at the initial visit (new	Proteomics			24879575
P04745	AMY1A	Alpha-amylase 1	Homo sapiens (Human)					X							CP	68055113	+	32-62	M/F	All subjects were systemically healthy and were made xclusion criteria included: smokers, diabetes, immunosuppression and medicine use	Whole saliva was collected by spitting into an ice-cooled graduated vessel (Falcon type). Subjects spat out every 30 s for 5 min. The total volume of saliva was recorded using a glass pipette and divided by the collection time, then expressed as ml per minute. The resulting saliva was stored in aliquots at 20°C until determinations were performed.	Analysis of protein, amylase and mucin concentration Colorimetric methods were used for all analysis in unstimulated saliva collected at the initial visit (new	Proteomics			24879575
P05112	IL4	Interleukin-4	Homo sapiens (Human)						X					X	CP	68055113	1,28	25-61	M/F	All subjects were systemically healthy and the patients with diagnosis of aggressive periodontitis, any known systemic illness, history of routine use of antibiotics/anti-inflammatory therapy within the past 6 months, subjects with oral mucosal lesions and present or past history of smoking were excluded from the study.	For the chronic periodontitis group, UWS was collected before SRP (P0) and at 1 week after SRP (P1) and at 6 weeks after SRP (P6). UWS was collected at approximately the same time of day by the drooling method (Rhodus et al, 2005). Briefly, subjects were asked to refrain from eating or drinking for 2 h prior to saliva collection. At least 2 ml of UWS was collected by passively drooling into a chilled centrifuge tube for 5–10 min.	ELISA for salivary sTLR-2, sCD14, and cytokines	Proteomics			23496245
P22301	IL10	Interleukin-10	Homo sapiens (Human)						X					X	CP	68055113	1,84	25-61	M/F	All subjects were systemically healthy and the patients with diagnosis of aggressive periodontitis, any known systemic illness, history of routine use of antibiotics/anti-inflammatory therapy within the past 6 months, subjects with oral mucosal lesions and present or past history of smoking were excluded from the study.	For the chronic periodontitis group, UWS was collected before SRP (P0) and at 1 week after SRP (P1) and at 6 weeks after SRP (P6). UWS was collected at approximately the same time of day by the drooling method (Rhodus et al, 2005). Briefly, subjects were asked to refrain from eating or drinking for 2 h prior to saliva collection. At least 2 ml of UWS was collected by passively drooling into a chilled centrifuge tube for 5–10 min.	ELISA for salivary sTLR-2, sCD14, and cytokines	Proteomics			23496245
P05231	IL6	Interleukin-6	Homo sapiens (Human)						X					X	CP	68055113	1,89	25-61	M/F	All subjects were systemically healthy and the patients with diagnosis of aggressive periodontitis, any known systemic illness, history of routine use of antibiotics/anti-inflammatory therapy within the past 6 months, subjects with oral mucosal lesions and present or past history of smoking were excluded from the study.	For the chronic periodontitis group, UWS was collected before SRP (P0) and at 1 week after SRP (P1) and at 6 weeks after SRP (P6). UWS was collected at approximately the same time of day by the drooling method (Rhodus et al, 2005). Briefly, subjects were asked to refrain from eating or drinking for 2 h prior to saliva collection. At least 2 ml of UWS was collected by passively drooling into a chilled centrifuge tube for 5–10 min.	ELISA for salivary sTLR-2, sCD14, and cytokines	Proteomics			23496245
Q16552	IL17A	Interleukin-17A	Homo sapiens (Human)						X					X	CP	68055113	-	25-61	M/F	All subjects were systemically healthy and the patients with diagnosis of aggressive periodontitis, any known systemic illness, history of routine use of antibiotics/anti-inflammatory therapy within the past 6 months, subjects with oral mucosal lesions and present or past history of smoking were excluded from the study.	For the chronic periodontitis group, UWS was collected before SRP (P0) and at 1 week after SRP (P1) and at 6 weeks after SRP (P6). UWS was collected at approximately the same time of day by the drooling method (Rhodus et al, 2005). Briefly, subjects were asked to refrain from eating or drinking for 2 h prior to saliva collection. At least 2 ml of UWS was collected by passively drooling into a chilled centrifuge tube for 5–10 min.	ELISA for salivary sTLR-2, sCD14, and cytokines	Proteomics			23496245

UniprotKB AC	Gene name	Name	Organism	Parotid	Parotid Exosome	SM/SL	Minor	Whole Saliva	Crevicular Fluid	Mucosa	Tongue	Biofilm	In Vitro	Health	Disease Name	Disease (MeSH ID)	Regulation	Age group	Gender*	Social Habits*	Methods of Sampling**	Methods of Analysis***	Type of Study	PTM	x	Citation (NCBI ID)
P01584	IL1B	Interleukin-1 beta	Homo sapiens (Human)					X							CP	68055113	1,72			type 2 diabetes mellitus. Dentate patients who had received a dental cleaning within 6 months or those who ever had periodontal surgery, those who took non-steroidal anti-inflammatory drugs or antibiotics during the past 6 months, women who were pregnant, those who had less than 20 teeth, and those with oral mucosal inflammatory lesions, such as lichen planus, candidiasis, etc. were excluded from the study.	A 5 ml of whole, unstimulated saliva sample was collected from all participants by accumulating saliva in the mouth and expectorating into collection tubes. The sample was placed on ice, and after re-suspension, was aliquoted in 1 ml portions and frozen at -80°C.	IL-1b was analysed using an enzyme-linked imm	Proteomics			22420648