CATÓLICA
ESCOLA SUPERIOR DE BIOTECNOLOGIA
PORTO

PSEUDOMONADOTA ON HUMAN SKIN: EFFECTS OF ANTIMICROBIAL AGENTS

by

Saravanan Murugesan

November 2024

CATÓLICA
ESCOLA SUPERIOR DE BIOTECNOLOGIA
PORTO

PSEUDOMONADOTA ON HUMAN SKIN: EFFECTS OF ANTIMICROBIAL AGENTS

Thesis presented to *Escola Superior de Biotecnologia* of the *Universidade Católica Portuguesa to fulfil requirements of Master of science in Applied Microbiology*

By

Saravanan Murugesan

November 2024

Supervisor - Doctor Ivone Vaz Moreira

Co-Supervisor – Doctor Inês Maria Ribeiro

ACKNOWLEDGEMENT:

To Universidade Católica Portuguesa, for giving me academic knowledge, teaching me ethics. To all the teachers I had the honour in getting taught everyday, for all the knowledge and life practicality challenges they made me competent, educationally skillfull.

In particular, to Professor Doctor Célia Manaia, for her valuable advice and guidance with her phenomenal experience, has guided the project in right direction, in bringing out the real essence and values with the project.

In particular, to Professor Doctor Ivone Vaz Moreira, for all her kindness, patiently available in valuable teaching throughout these months, and practical lab guidance, invaluable for the completion of this work.

In particular, to Professor Doctor Inês Ribeiro, for her timely helps, concerns which helped me with these works.

To all my laboratory colleagues, for all their humbleness, help, advice, constant concern for making these works completed in time.

To my parents, family, brother and friends, thank you for giving me this opportunity.

"A dúvida é o princípio da sabedoria", Aristóteles

Resumo

Pseudomonadota são reconhecidos como importantes portos de resistência aos antibióticos, e algumas delas também são bactérias ubíquas capazes de sobreviver em múltiplos ambientes. Objetivos- compreender se bactérias do filo *Pseudomonadota*, que são colonizadores comuns de uma pele humana saudável, podem ser portadoras de resistência a antibióticos e se a sua sobrevivência pode ser afetada pelo uso de cosméticos ou agentes antimicrobianos e a resistência a carbapenemos entre espécies de *Acinetobacter* é afetada pela filogenómica. Para responder a esta questão, foi realizada uma análise da diversidade bacteriana da pele humana saudável com base em dados metagenómicos disponíveis em bases de dados públicas, e identificado os géneros e espécies mais comuns de *Pseudomonadota*. Dessa análise, identificou-se o género *Acinetobacter* como um dos mais frequentes na pele humana, a saber, as espécies *Acinetobacter johnsonii, Acinetobacter lwoffii* e *Acinetobacter junii.* Os genomas dessas três espécies foram recolhidos de bases de dados públicas (n=232) e comparados em termos de perfis de resistência a antibióticos e virulência, visando entender se os perfis de resistência/virulência são determinados pela espécie ou por outros fatores, como a filogenia ou a origem dos isolados (clínica, clinicamente associada e ambiental) podem interferir. Com base na comparação de genomas foi possível concluir que são importantes portadores de genes de resistência a antibióticos. Os isolados de origem ambiental apresentaram maior diversidade de genes de resistência, enquanto os isolados de origem clínica apresentam menores perfis de resistência mas maior abundância. Os genomas bla_{NDM} positivos (n=23) foram processados para compreender o ambiente genómico do gene da carbapenemase e se ele varia de acordo com a espécie ou a origem da estirpe. Foi observado que o gene bla_{NDM} estava associado principalmente a elementos genéticos móveis, nomeadamente a família IS66, e associado a outros genes de resistência (p.e. aminoglicosideos, macrólidos). Alguns isolados de *Acinetobacter* da coleção de culturas do laboratório, todos de origem ambiental, foram testados quanto à sua tolerância a antissépticos (peróxido de hidrogénio, iodo, lixivia) e cosméticos, utilizando o método de difusão em disco. Para esses isolados não foi possível inferir qualquer correlação entre o perfil de resistência aos antibióticos e a tolerância aos compostos cosméticos, mas observou-se alguma variância entre as estirpes na tolerância ao peróxido de hidrogénio. No futuro, seria interessante estender estes testes a isolados de diferentes origens (p. e., isolados cutâneos) e testar concentrações mais elevadas dos cosméticos.

Palavras-chave: Origem, Espécie, Resistência aos carbapenemos, Filogenómica, Fenotípica

Abstract

Pseudomonadota are recognized as important harbours of antibiotic resistance, and some of them are also ubiquitous bacteria able to survive in multiple environments. The aim of this study was to understand if bacteria of the phylum *Pseudomonadota* that are common colonizers of a healthy human skin may be carriers of antibiotic resistance, and if their survival may be affected by the use of cosmetics or antimicrobial agents and if carbapenems resistance among *Acinetobacter* species is affected by phylogenomics. To answer this question, it was first done an analysis of the human healthy skin bacterial diversity based on metagenomic data available in public databases, and identified the most common *Pseudomonadota* genera and species. From this analysis, was identified the genus *Acinetobacter* as one of the most frequent in human skin, namely the species *Acinetobacter johnsonii*, *Acinetobacter lwoffii* and *Acinetobacter junii*. The genomes of those three species were collected from public databases (n=232) and compared in terms of antibiotic resistance and virulence profiles, aiming to understand if the resistance/virulence profiles were determined by the species or if other factors, such as the phylogeny or the strains origin (clinical, clinical associated, and environmental) may interfere. Based on this genome's comparison it was possible to conclude that they are important carriers of antibiotic resistance genes. The strains from environmental origin presented a higher diversity of resistance genes, while the strains from clinical origin have poorer profiles of resistance but higher abundance. The bla_{NDM} positive genomes (n=23) were processed to understand the genomic environment of the carbapenemase gene and if it varies according to the species or the strain origin. It was observed that the bla_{NDM} gene was mainly associated to mobile genetic elements, namely the IS66 family, and associated with other genes of resistance (e.g. aminoglycosides, macrolides). Some *Acinetobacter* isolates in the laboratory culture collection, all from environmental origin, were tested for their tolerance to antiseptics (hydrogen peroxide, iodine, bleach, water) and cosmetics, using the disk diffusion method. For these isolates it was not possible to infer any correlation between the antibiotic resistance profile and the tolerance to cosmetic compounds, but it was observed some variance between strains in the tolerance to hydrogen peroxide. In the future, it would be interesting to extend these tests to strains from different origins (e.g. skin isolates) and test higher concentration of the cosmetics.

Keywords: Origin, Species, Carbapenems resistance, Filogenomics, Phenotypic

LIST OF FIGURES

LIST OF TABLES-

APPENDIX

1. Introduction

1.1. The Human skin

The human skin extends to a total of 1.8 m² with various folds with special niches in which a variety of microorganisms, including bacteria, fungi, viruses and mites live (Grice & Segre, 2011). The main function of human skin is to provide a strong physical barrier that resists penetration by microorganisms and potential toxins, while retaining moisture and nutrients in the body. It also represents an interface to the external environment and most colonized symbiotic microorganisms are harmless and protects against invasion by pathogenic or harmful organisms. These microorganisms may also play a role in educating skin immune system for better immunity (Grice & Segre, 2011).

Disturbances affecting the host-microorganism relationship can be endogenous in nature (e.g. genetic variation that selects a particular microbial community) or exogenous in nature (e.g. hand washing). Microbiologists, immunologists, and dermatologists have collaborated with genomic scientists to develop a more comprehensive characterization of the skin microbiota, to understand skin health, diseases, and infections (Grice & Segre, 2011). A balanced skin microbiome is known to play an important role in skin health, as any alterations lead to the overgrowth of pathogenic strains linked to various skin diseases (Kim et al., 2018; Kong et al., 2012).

Skin diseases are closely related to skin microorganisms and these two elements can regulate each other (Cundell, 2018). For example, the skin can release neurohormones such as peptides and catecholamines that alter the activity of skin microorganisms. Research has also shown that skin microorganisms can release neurohormonal compounds such as histamine, glutamic acid and R-aminobutyric acid to improve the physiological condition of the skin (Cundell, 2018). Various factors, including the use of antibiotics, cosmetics, soaps, personal care products, and living conditions such as life styles and alimentations can have influence on the skin microbiome (Perez et al., 2016).

1.2. Bacterial diversity in human skin

Skin microbial communities have been studied using culture-based methods. Because this approach selects microorganisms that thrive under artificial growth conditions, the overall diversity of the community is underestimated. For example, the cutaneous genus *Staphylococcus* is easier to culture than the *Cutibacterium* or *Corynebacterium* species, which have often been underestimated in culture-based studies (Byrd et al., 2018). To circumvent cultural biases and capture the full diversity of the microbiome, researchers began using next generation sequencing (NGS) methods (Byrd et al., 2018).

There are two main methodologies used for NGS: shotgun metagenomics and 16S rDNA sequencing (Laudadio et al., 2018). Shotgun metagenomics involves the sequencing of bacterial DNA taken from whole microbial community. The sequencing reads are analysed with metagenomics databases as reference, assigning them to a particular taxon. 16S rDNA sequencing relies on the polymerase chain reaction (PCR) amplification of specific region of the 16S rRNA gene, utilizing degenerated primers allowing amplification of rDNA from large species list (Laudadio et al., 2018).

Both methods have some limitations. The 16S rRNA gene amplicon sequencing is well-typified, means that yields enough information about microbial communities' arrangement, using a short fragment of the gene (~300 bp). Taxa are designated on the basis of single region of the bacterial genome, and primer bias can result in over- or under-representation of specific taxa. Shotgun metagenomics needs more coverage (10–30 million of reads) and complicated data analysis. Thus, by collecting sequence information about whole genomic regions, shotgun metagenomics allows a more accurate definition at the species level (Laudadio et al.,2018).

The bacterial diversity in human skin varies due to various factors like age, sex, site, climate, geographical site, immune conditions like previous infections, environmental factors (e.g. smoking or food habits), and certain conditions like diseases of the host (Grice & Segre, 2011; Bay et al., 2020). On the skin surface, *Actinomycetota*, *Pseudomonadota*, *Bacillota* and *Bacteroidota* are described as the most common inhabitants of skin (Table 1). Interpersonal variation occurs between individuals. The most consistent sites with bacterial community were

inside the ear, nostril and inguinal crease. The antecubital fossa, back, nare and plantar heel are more similar to the same site on another individual, than other areas (Oh et al., 2017).
The bacterial diversity varies according to the moisture content, sebum level in human skin. Sebum lubricates the skin, and contains no dissolved oxygen, hence it helps in the growth of facultative anaerobes like *Cutibacterium acnes*, a common skin commensal bacterium (Grice & Segre, 2011). While *Corynebacterium* and *Staphylococcus* species are found in moist areas, *Micrococcus*, *Enhydrobacter*, and *Streptococcus* species in dry areas (Callewaert et al., 2020).

The decrease in the skin pH may inhibit common pathogens, like *Staphylococcus aureus* and *Streptococcus pyogenes* and helps the growth of coagulase-negative staphylococci and corynebacteria (Grice and Segre, 2011). The age is also an important factor in microbial colonisation (Howard et al., 2022). Skin microbiota is established in first years of life, as a newborn baby explores its surrounding. During puberty, and the changes observed in sweat, sebum and hormone production, affects the change in composition later in life (Howard et al., 2022). From young to old age, are observed some changes: (i) a decrease in the relative abundance of *Cutibacterium* and *Lactobacillus* at all the buttock, face, forearm body sites; (ii) an increase in the relative abundance of *Corynebacterium* and *Enhydrobacter* at the facial sampling site; (iii) an increase in the relative abundance of *Streptococcus* and *Anaerococcus* and a decrease in the relative abundance of *Staphylococcus* at the buttocks sampling site (Howard et al., 2022).

Environmental factors specific to the individual, such as occupation, clothing choice, antibiotic usage, or cosmetics use affect the bacterial composition (Grice and Segre, 2011). Some studies investigated the effect of some conditions in the skin microbiota, such as swimming in the ocean, playing a derby, or cosmetics application. The predominating phyla on the skin change after ocean swimming with *Actinomycetota* decreasing from 34% to 6.7%, *Bacillota* decreasing from 32.3% to 9.4%, *Pseudomonadota* keeping stable (24.8% to 24%), and *Bacteroidota* increasing from 7% to 41.8% (Nielsen & Jiang, 2021). After playing roller derby, it was detected in the players skin *Arthrobacter and Xanthomonas* organisms, that are usually associated with the plant and soil communities (Meadow et al., 2013). The cosmetics application, increases the skin hydration increasing the bacterial diversity. For example, the abundance of *Cutibacterium* may be modulated by the cosmetics (Moskovicz et al., 2020).

Table 1. Bacterial diversity of a healthy human skin, based on what is described in the literature

Reference	Year	Methodology	Major bacterial groups	Observations
Grice & Segre, 2011 ("The skin microbiome") https://doi.org/10.1038/nrmicro2537	2011	CI: 16S ribosomal RNA sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Cutibacterium* spp. (in sebaceous areas); *Corynebacterium* and *Staphylococcus* spp. (in moist areas)	Healthy skin
Byrd et al., 2018 ("The human skin microbiome") https://doi.org/10.1038/nrmicro.2017.157	2018	CI:16S ribosomal RNA sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Cutibacterium* acnes, *Corynebacterium Staphylococcus epidermidis* and *Staphylococcus aureus*	Healthy skin
Oh et al., 2016 ("Temporal stability of the human skin microbiome") https://doi.org/10.1016/j.cell.2016.04.008	2016	CI: Metagenomic shotgun sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Cutibacterium acnes* and *Cutibacterium* phage (in sebaceous sites, moist popliteal crease), *Staphylococcus epidermidis* (in Plantar heel), *Staphylococcus hominis*, *Staphylococcus epidermidis*	Healthy skin
Howard et al., 2022 ("Aging-associated changes in the adult human skin microbiome") https://doi.org/10.1016/j.jid.2021.11.029	2022	CI:16S ribosomal RNA gene sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Cutibacterium, Lactobacillus, Enhydrobacter, Acinetobacter, Corynebacterium, Staphylococcus, and Streptococcus*	Healthy skin

Bay et al., 2020 ("Universal Dermal Microbiome in Human Skin") https://doi.org/10.1128/mBio.02945-19	2020	CI: 16S ribosomal RNA sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Corynebacterium* spp., *Staphylococcus* spp., *Streptococcus* spp., *Micrococcus* spp.	Healthy skin
Callewaert et al., 2020 ("Skin Microbiome and its Interplay with the Environment") https://doi.org/10.1007/s40257-020-00551-x	2020	CI: Metagenomic sequencing.	*Actinomycetota*, *Pseudomonadota*, *Bacillota* *Cutibacterium* (in sebaceous sites), *Corynebacterium*, *Staphylococcus* spp. (in moist areas), *Micrococcus*, *Enhydrobacter* and *Streptococcus* spp. (in dry areas)	Healthy skin
Nielsen & Jiang,2019 ("Alterations of the human skin microbiome after ocean water exposure") https://doi.org/10.1016/j.marpolbul.2019.06.047	2019	CI: 16S ribosomal RNA sequencing	*Actinomycetota*, *Bacillota*, *Bacteroidota*, followed by *Pseudomonadota*	Post-swim in healthy skin
Meadow et al., 2013 ("Significant changes in the skin microbiome mediated by the sport of roller derby") https://doi.org/10.7717/peerj.53	2013	CI: 16S ribosomal RNA sequencing	*Actinomycetota, Pseudomonadota, Bacillota* *Corynebacterium, Micrococcus, Staphylococcus, and Acinetobacter. Arthrobacter and Xanthomonas* (in Post-bout samples)	Before and after roller derby sport in healthy skin
Moskovicz et al., 2020 ("Extrinsic Factors Shaping the Skin Microbiome") https://doi.org/10.3390/microorganisms8071023	2020	CI:16S ribosomal RNA sequencing	*Actinomycetota, Pseudomonadota, Bacillota, Bacteroidota* *Corynebacterium* (in moist skin) *Cutibacterium* and *Staphylococcus* (in sebaceous skin), *Betaproteobacteria* and *Flavobacteriales* (most on dry skin)	Healthy skin

1.3. *Pseudomonadota* diversity in healthy human skin

Human skin is colonized by a complex microbial community, long thought to be composed of gram-positive bacteria such as staphylococci, micrococci, corynebacteria, with *Cutibacterium* spp. dominating (Cosseau & Bertrand, 2016). Besides known bacteria, culture-independent approaches demonstrate that Gram-negative bacteria, especially *Pseudomonadota*, represent major component of microbial community diversity (Cosseau & Bertrand, 2016). An analysis of the healthy skin microbiota, based on the 16S rRNA gene amplicon, sequenced on Illumina MiSeq platform, revealed the following structure: *Bacillota* (40.7%), *Bacteroidota* (34.7%), *Pseudomonadota* (6.1%), *Synergistota* (3.9%), *Fusobacteriota* (3.5%), *Actinomycetota* (3.4%), *Spirochaetota* (1.2%), and *Cyanobacteria* (0.5%) (Ramadan et al., 2016).

Despite their detection in numerous metagenomic studies and under various pathophysiological conditions, cutaneous *Pseudomonadota* remained poorly described, mainly due to the lack of available isolates and NGS-generated sequences were generally too short to obtain precise species affiliation (Cosseau & Bertrand, 2016). In Table 2 are listed some of the *Pseudomonadota* genera previously reported in human skin.

Pseudomonadota abundance was significantly higher in the older group (Kim et al., 2021). Cheek moisture content was significantly higher among the elderly, being positively associated with the occurrence of *Enhydrobacter*, wich was most abundant in older age group. *Pseudomonadota*, including *Pseudomonas* and *Acinetobacter*, are epidermal microbial communities found in longer existence and they are involved in opportunistic pathogenic skin infections (Peng & Biswas, 2020). Workers in farms containing various animals were associated with higher abundances of epidermal *Pseudomonadota*, mainly *Pseudomonas* and *Acinetobacter*, but lower *Actinomycetota* particularly *Corynebacterium* and *Cutibacterium* due to frequent operations on various livestock. Hence microbial dysbiosis leads to various epidermal illness like acne or eczema.

Table 2. *Pseudomonadota* diversity in healthy human skin, based on the literature

Class	Genus	References
Alpha-Proteobacteria	*Agrobacterium*	Cosseau & Bertrand, 2016; Diptaraj & Chaudhari, 2020
	Aurantimonas	Cosseau & Bertrand, 2016
	Bradyrhizobium	Cosseau & Bertrand, 2016
	Novosphingobium	Diptaraj & Chaudhari, 2020
	Paracoccus	Cosseau & Bertrand, 2016; Kim et al., 2021; Peng & Biswas, 2020; Mukherjee et al., 2016; Diptaraj & Chaudhari, 2020
	Rhizobium	Peng & Biswas, 2020
	Roseomonas	Mukherjee et al., 2016
	Sphingomonas	Cosseau & Bertrand, 2016; Peng & Biswas, 2020; Diptaraj & Chaudhari, 2020
	Tardiphaga	Kim et al., 2021
Beta-Proteobacteria	*Acidovorax*	Cosseau & Bertrand, 2016
	Burkholderia	Peng & Biswas , 2020
	Cupriavidus	Diptaraj & Chaudhari, 2020
	Lautropia	Diptaraj & Chaudhari, 2020; Mukherjee et al., 2016
	Massilia	Peng & Biswas, 2020
	Neisseria	Cosseau & Bertrand, 2016; Diptaraj & Chaudhari, 2020; Mukherjee et al., 2016
	Pelomonas	Cosseau & Bertrand, 2016
	Tepidimonas	Mukherjee et al., 2016
Gamma-Proteobacteria	*Acinetobacter*	Cosseau & Bertrand, 2016; Peng & Biswas, 2020; Ramadan et al., 2016; Luqman et al., 2020
	Aeromonas	Cosseau & Bertrand, 2016
	Alcanivorax	Cosseau & Bertrand, 2016
	Enhydrobacter	Diptaraj & Chaudhari, 2020; Peng & Biswas, 2020; Mukherjee et al., 2016; Kim et al., 2021
	Enterobacter	Cosseau & Bertrand, 2016
	Erwinia	Diptaraj & Chaudhari, 2020
	Escherichia	Ramadan et al., 2016
	Haemophilus	Mukherjee et al., 2016; Diptaraj & Chaudhari, 2020; Cosseau & Bertrand, 2016
	Halomonas	Kim et al., 2021
	Idiomarina	Cosseau & Bertrand, 2016
	Klebsiella	Cosseau & Bertrand, 2016
	Luteimonas	Ramadan et al., 2016
	Marinobacter	Cosseau & Bertrand, 2016
	Moraxella	Cosseau & Bertrand, 2016; Diptaraj & Chaudhari, 2020
	Morganella	Kim et al., 2021
	Pantoea	Kim et al., 2021; Peng & Biswas, 2020
	Pasteurella	Cosseau & Bertrand, 2016
	Pseudomonas	Cosseau & Bertrand, 2016; Luqman et al., 2020; Ramadan et al., 2016; Peng & Biswas, 2020; Mukherjee et al., 2016; Diptaraj & Chaudhari, 2020
	Serratia	Cosseau & Bertrand, 2016; Ramadan et al., 2016; Diptaraj & Chaudhari, 2020; Peng & Biswas, 2020
	Shewanella	Diptaraj & Chaudhari, 2020
	Stenotrophomonas	Cosseau & Bertrand, 2016; Peng & Biswas, 2020

1.4 How cosmetics can affect the skin bacterial diversity profile

Although skin is frequently exposed to various topical care products, it has the capacity to remove contaminants from its surface and prevent bacteria from entering the body and cause infection. Disequilibrium of skin microbiota is known to cause some diseases, including for example acne, eczema, rosacea, and psoriasis (Bouslimani et al., 2019). Daily use of skin care products can modify the diversity of skin microorganisms by adding active ingredients, that have a beneficial or inhibitory effect to certain microorganisms and, with prolonged or regular use, produce healing effects (Paluszak et al., 2011). The application of Sodium Lauryl Sulfate (SLS), a cleansing agent, was observed to increase the abundance of *Actinomycetota*, *Bacillota*, and *Pseudomonadota*, microorganisms with a protective effect against UV (Okombi et al., 2021). The treatment with fermented oils, containing several antioxidants, showed an increase in the biodiversity indicating healthier microbial ecosystem (Ciardiello et al., 2020). In subjects with acne, the application of Rhodomyrtus tomentosa Fruit Extract, a antibacterial, showed reduced the abundance of *Cutibacterium acnes* and *Cutibacterium granulosum*, with a reduction of the inflammatory lesions (Gervason et al., 2020). These and other examples are detailed in Table 3, with a special focus on the effects observed in *Pseudomonadota*.

Table 3. Effect of cosmetics on bacteria diversity, especially bacteria of the phylum *Pseudomonadota*, of a healthy and acne human skin

Reference	Cosmetics effects on Bacteria (Increase, Decrease, Same)	Condition
Okombi et al., 2021 https://doi.org/10.3390/cosmetics8010006	INCREASE: *Pantoea*, *Enhydrobacter* DECREASE: *Acinetobacter*, *Pseudomonas*, *Paracoccus*	Sodium Lauryl Sulphate on Healthy skin
Cui et al., 2023 https://doi.org/10.3389/fcimb.2023.1210724	INCREASE: *Ralstonia* DECREASE: *Neisseria*, *Comamonas*, *Acinetobacter*, *Pseudomonas*, *Alcaligenes*, *Janthinobacterium*, *Diaphorobacter*	Tremella, Dendrobium, Snow lotus polysaccharides on Healthy skin Forehead
Filaire et al., 2019 https://doi.org/10.3390/cosmetics6040069	DECREASE: *Acinetobacter*	Halymenia Durvillei on Sensitive skin
Ciardiello et al., 2020 https://doi.org/10.3390/cosmetics7020034	DECREASE: *Acinetobacter*, *Pseudomonas*	Shiunko and Artemisia Oils on Healthy skin
Gervason et al., 2020 https://doi.org/10.3390/cosmetics7030053	INCREASE: *Paracoccus*	Rhodomyrtus Tomentosa extract on Acne skin
Hong et al., 2020 https://doi.org/10.3390/jpm10030091	INCREASE: *Sphingomonas*, *Burkholderia* DECREASE: *Enhydrobacter*	Probiotics Serum on Healthy skin
Hwang et al., 2021 https://doi.org/10.1002/mbo3.1236	DECREASE: *Acinetobacter*, *Sphingomonas*, *Haemophilus*, INCREASE: *Moraxella*, *Neisseria*, *Pseudomonas*	Secret essence Skincare product on Healthy skin
Chaiyasut et al., 2022 https://doi.org/10.3390/app122312483	INCREASE: *Mesorhizobium*, *Legionella*, *Stenotrophomonas* DECREASE: *Pseudomonas*, *Acinetobacter*, *Haemophilus*, *Roseomonas, Paracoccus, Sphingomonas, Enhydrobacter*	Paraprobiotics Moisturiser On Healthy skin
Lee et al., 2018 https://doi.org/10.1002/mbo3.557	DECREASE: *Paracoccus*, *Pseudomonas*, *Haemophilus*, *Burkholderia* INCREASE: *Neisseria*, *Enhydrobacter*, *Cupriavidus*, *Acinetobacter*, *Ralstonia*	High Hydration cosmetic On Healthy skin

Note: all the studies did the bacteria diversity analysis based on a culture-independent methods (16S rRNA gene sequencing)

1.5 Acinetobacter spp.

Acinetobacter are nearly 3% of all bacteria in intensive care units, and nearly 55% of them show resistance to commonly used antibiotics (Tsay et al., 2020). *Acinetobacter* are Gram-negative bacteria, that have characteristic of being present everywhere and are commensals of human skin. *Acinetobacter* spp. are organisms naturally present in human skin and can be around 35-45% of bacteria present in a healthy skin (Yavankar et al., 2007). Few species of *Acinetobacter* are skin commensals and few species are cause of serious nosocomial diseases (Patil & Chopade, 2001). There are nearly 50 species of *Acinetobacter*, most of them being non disease causing environmental organisms. The pathogenic species are *Acinetobacter baumannii*, *Acinetobacter calcoaceticus* and *Acinetobacter lwoffii*. While *Acinetobacter haemolyticus*, *Acinetobacter johnsonii*, *Acinetobacter junii*, *Acinetobacter nosocomialis*, *Acinetobacter pittii*, *Acinetobacter schindleri*, and *Acinetobacter ursingii* cause diseases in very few scenario (Wong et al., 2017).

Nosocomial diseases caused by *Acinetobacter* spp. are commonly seen all over the world. Diseases caused by *Acinetobacter* in hospitalized patients have been found to infect almost all vital parts of human body (Patil & Chopade, 2001). *Acinetobacter* forms biofilms and are common in intensive care patients and hospital equipments like catheter or ventilation devices (Gedefie et al., 2021). *Acinetobacter* have been studied in detail and have been found to have commonly used anti-microbial- and metal-resistance plasmids (Patil & Chopade, 2001). The reason why *Acinetobacter* are considered a problem in clinical settings is because of this high percentage of antibiotics resistance (Tsay et al., 2020). The antibiotic resistance is so effective because of the low permeability to these drugs and efflux pumps like AdeABC, AdeIJK that can actively efflux these drugs (Wong et al., 2017). Our focus is on *Acinetobacter junii*, *Acinetobacter johnsonii* and *Acinetobacter lwoffii* as they are among the common commensals of skin (Seifert et al., 1997).

The mechanisms how *Acinetobacter* enter or exit hospital environments are not clearly known and the natural reservoir source of multidrug resistant *Acinetobacter* are not proven. It is estimated that the digestive tracts of the hospital patients become the reservoir for these multidrug resistant *Acinetobacter* from where they get discharged into wastewater environment (Hrenovic et al.,2014). Usually, drug resistance in *Acinetobacter* strains from tap water is scarce, but some non-wildtype *Acinetobacter* (drug resistance) for tetracycline, meropenem,

and ceftazidime were reported in tap water, suggesting the potential of some *Acinetobacter* spp. to acquire and disseminate resistance via drinking water (Narciso-da-Rocha et al., 2012).

1.6 Thesis objectives

The objective of this study was to understand if bacteria of the phylum *Pseudomonadota* that are common colonizers of a healthy human skin may be carriers of antibiotic resistance, and if their survival may be affected by the use of cosmetics or antimicrobial agents.

This study postulates that 1) Analysis of evolution and response of *Pseudomonadota* phylum *Acinetobacter* to cosmetics 2) carbapenem resistance among *Acinetobacter* species is affected by phylogenomics, with uniqueness within clinical, clinical associated, environment origin isolates and uniqueness within species.

2. *Materials and Methods*

2.1. Analysis of the Human skin bacterial diversity

The bacterial diversity of healthy human skin was investigated based on 16S rRNA gene sequence data deposited in public databases. For that, it was searched in the public database MGnify (https://www.ebi.ac.uk/metagenomics, accessed on 20th October 2023) for human skin microbiomes. From the search resulted 15 studies. From those, were selected the three studies that analysed the healthy skin microbiota (MGYS00000520; MGYS00001295; MGYS00005037). These studies analysed the bacterial diversity of skin of mostly healthy subjects, under normal conditions without any cosmetic, drug skin, effects (Table 4). From those, three studies on the skin bacterial microbiome were selected, based on study which has more samples and samples involving healthy individuals. For the three studies was downloaded the taxonomic annotation data of each study in the .xls format. In total, were analysed 204 samples (2 samples from MGYS00000520; 96 samples from MGYS00001295; and 106 samples from MGYS00005037), excluding the samples that were not from healthy individuals or that were analysed after the exposition to any stress. Taxonomic annotation was in number of reads per operational taxonomic unit (OTU). To normalise the data, the relative abundance (number of reads of the OTU per total number of reads of the sample) of each OTU was calculated.

Table 4. Publicly available bacterial studies included in this work.

MGnify accession Number	**Study title**	**Subjects involved**
MGYS00000520	Metagenomes study from palm of two individuals	Metagenome study of skin bacteria of palms of two healthy individuals
MGYS00001295	Human Skin bacterial and fungal microbiota analysis	Skin bacterial microbiota from 11 healthy and 13 dandruff subjects, comparing scalp and forehead from both lesional and non-lesional skin sites.
MGYS00005037	Human Skin Microbiome Metagenome	Human skin microbiome of 46 male and 248 female healthy volunteers.

The taxa average values of relative abundance of all three studies was unified. From here were identified the genus from *Pseudomonadota* phylum that are more common in healthy human skin following the criteria: 1) Presence in the three studies; and 2) genus with a relative abundance >1%. The list of significant selected genus is made. The most common genus of *Pseudomonadota* in human healthy skin were represented in a phylogenetic tree. For the phylogenetic construction, the 16S rRNA gene sequence of the type species of each selected genus, was collected from the NCBI public database with the help of the List of Prokaryotic names with Standing in Nomenclature (LPSN, https://www.bacterio.net/) in FASTA format. The MEGA Software (Version 10.2) was used to align the sequences, using the MUSCLE algorithm, and construct the dendrogram.

2.2 Acinetobacter spp. genomes comparison

After the analysis of the human healthy skin bacterial diversity, three species of *Acinetobacter* were identified as always present in human healthy skin (identified in the three studies): *A. junii*, *A. johnsonii* and *A. lwoffii*. For these three species were collected all the genomes available in the public NCBI genomes database (https://www.ncbi.nlm.nih.gov/datasets/genome/) on 2nd January 2024: *Acinetobacter junii* (n= 107), *Acinetobacter johnsonii* (n=113), and *Acinetobacter lwoffii* (n=72). Of these, only the genomes with a known origin (*Acinetobacter junii*, n= 91; *Acinetobacter johnsonii*, n=101; and *Acinetobacter lwoffii*, n=53) were included in the study. The strains were classified according to the origin as clinical, clinical associated (when isolated from clinical environments, e.g. hospital surfaces), or environmental.

For all the genomes was determined the multilocus sequence type (MLST), with MLST finder (https://cge.food.dtu.dk/services/MLST/) and the Average nucleotide identity (ANI) with ANI matrix calculator. Wherever there were possible duplicates, as determined by same origin/institution, high ANI value (>99.5%) and same MLST or same MLST gene variants, duplicates were excluded. After duplicates removal, a total of 232 genomes were selected to proceed: 86 *Acinetobacter junii* genomes (clinical n= 46, clinical associated n=6, environmental n=34), 95 *Acinetobacter johnsonii* genomes (clinical n=20, clinical associated n=6, environmental n=69), and 51 *Acinetobacter lwoffii* genomes (clinical n=19, clinical associated n=3, environmental n=29). Antibiotic resistance genes and virulence factors were annotated for all these genomes with ResFinder (https://genepi.food.dtu.dk/resfinder) and the

Virulence factors database (https://.mgc.ac.cn/cgi-bin/VFs/v5/main.cgi), respectively. The profiles of antibiotic resistance with relative gene abundance and virulence were compared considering the species and origin of the isolates.

2.3 bla_{NDM} positive genomes

From the 232 genomes, 23 were *bla_{NDM}* positive: *Acinetobacter junii*, 9 genomes (clinical n= 2, clinical associated n=2, environmental n=5), *Acinetobacter johnsonii*, 13 genomes (clinical n=1, clinical associated n=1, environmental n=11), and *Acinetobacter lwoffii,* 1 genome (clinical associated n=1). For these 23 genomes, antibiotic resistance genes with gene abundance was compared with species and origin of isolates. For wgMLST dendogram analysis, except for Acinetobacter johnsonii environment genome GCA_013186005.1 which had only *rplB* 128, with rest 22 genomes, was constructed a dendrogram with Hierarchical cluster using IBM SPSS Statistics Software (29 version), based on the gene variants of the MLST scheme (*cpn60, fusA, gltA, pyrG, recA, rplB, rpoB* genes), to understand the phylogenetic relationship of the *bla_{NDM}* positive isolates.

The 23 *bla_{NDM}* positive genomes were processed for functional annotation using RASTtk pipeline (https://rast.nmpdr.org/rast.cgi), to identify the location of the carbapenemase gene. For the region of interest, around 3000 bp were sectioned before and after the *bla_{NDM}* gene. This region of interest was analysed for insertion sequence, transposon associated DNA endonuclease, transposase using IS finder (https://isfinder.biotoul.fr/blast.php), and aligned using the MEGA Software (Version 10.2) with MUSCLE algorithm. Then phylogeny is selected, neighbouring tree method is done with default settings and Phylogeny tree is created.

2.4 Acinetobacter profiles of tolerance to antimicrobials and cosmetics

From the Bacterial Ecology Lab culture collection were selected some isolates of the *A. junii*, *A. johnsonii* and *A. lwoffii* to perform tests of tolerance to antibiotics and cosmetics. These strains were tested using the disk diffusion method with the antiseptics (hydrogen peroxide 3%, iodine 5%, bleach5%) and cosmetics. Water is used as negative control. The antibiograms were performed on Mueller-Hinton agar and incubated for 24h at 30ºC.

3 *Results and Discussion*

3.1 Healthy human skin bacterial diversity

Three studies, comprising 204 samples, were considered for the analysis of the healthy human skin microbiota. From this analysis it was possible to conclude that the most abundant phylum was *Bacillota*, which is in agreement with literature (Pérez-Losada et al., 2023). Skin microbiota included the phyla *Bacillota* (40.7%), *Bacteroidota* (34.7%), *Pseudomonadota* (6.1%), *Synergistota* (3.9%), *Fusobacteriota* (3.5%), *Actinomycetota* (3.4%), *Spirochaetota* (1.2%), *Cyanobacteria* (0.5%) and other minoritary (6%) (Figure 1).

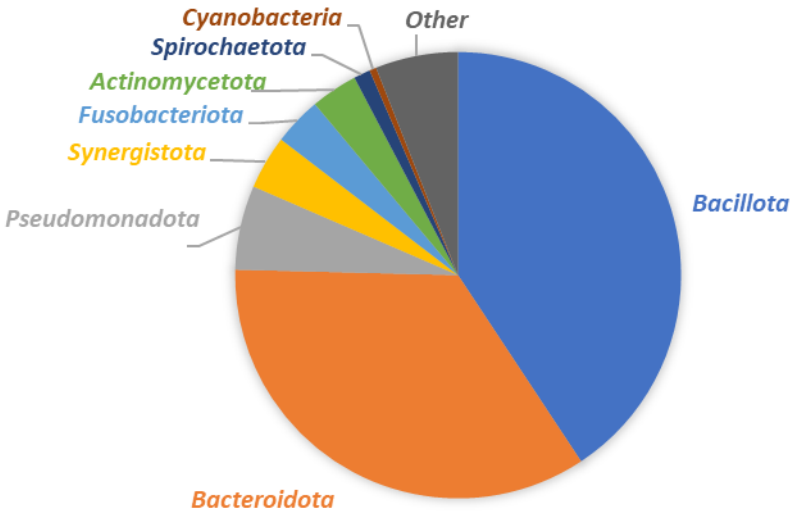

Figure 1. Most abundant phyla in human healthy skin samples.

As described in the literature we observed that the phyla *Bacteroidota* and *Bacillota* are the predominant in healthy human skin (Ramadan et al., 2016). After these two phyla, *Pseudomonadota* is also an abundant phylum, and among them were highlighted the genera *Acinetobacter* for their frequent presence in skin samples.

We focused our study in the three species of *Acinetobacter* more commonly observed in healthy skin samples: *A. johnsonii*, *A. junii* and *A. lwoffii*.

Pseudomonadota are among the most abundant phyla, although it was not among the top bacteria present in human skin. Our interest in this phylum is justified by their relevance in terms of antibiotic resistance. A high diversity of genus belonging to the *Pseudomonadota*

phylum were identified in the human skin samples. Among them some were more frequent being identified in the three studies samples (Figure 3, genera highlighted in yellow). The calculation of the genus relative abundance gave a clearer picture that the percentage of *Acinetobacter* was reduced in comparison to other genera, but some species of *Acinetobacter* are very commonly described as colonizers of the human skin. Also, *Acinetobacter* are commonly associated with resistance to antimicrobials, and are ubiquitous bacteria for example in aquatic habitats (Vaz-Moreira et al., 2014). Three species of *Acinetobacter* were consistently identified in the skin microbiota: *A. junii*, *A. johnsonii* and *A. lwoffi*. For that reason, we decided to proceed the study with focus on those three *Acinetobacter* species. For the selection, it was also relevant to select species for which we had available isolates in the laboratory culture collection to perform the microbiology tests. The phylogenetic tree was analysed for *Acinetobacter (Figure 2)*, the *Acinetobacter* had a genetic change which was significantly correlated with all notable genomes which was in similar evolutionary timeline, suggesting that it had indistinguishable evolutionary relationship with all other important genomes.

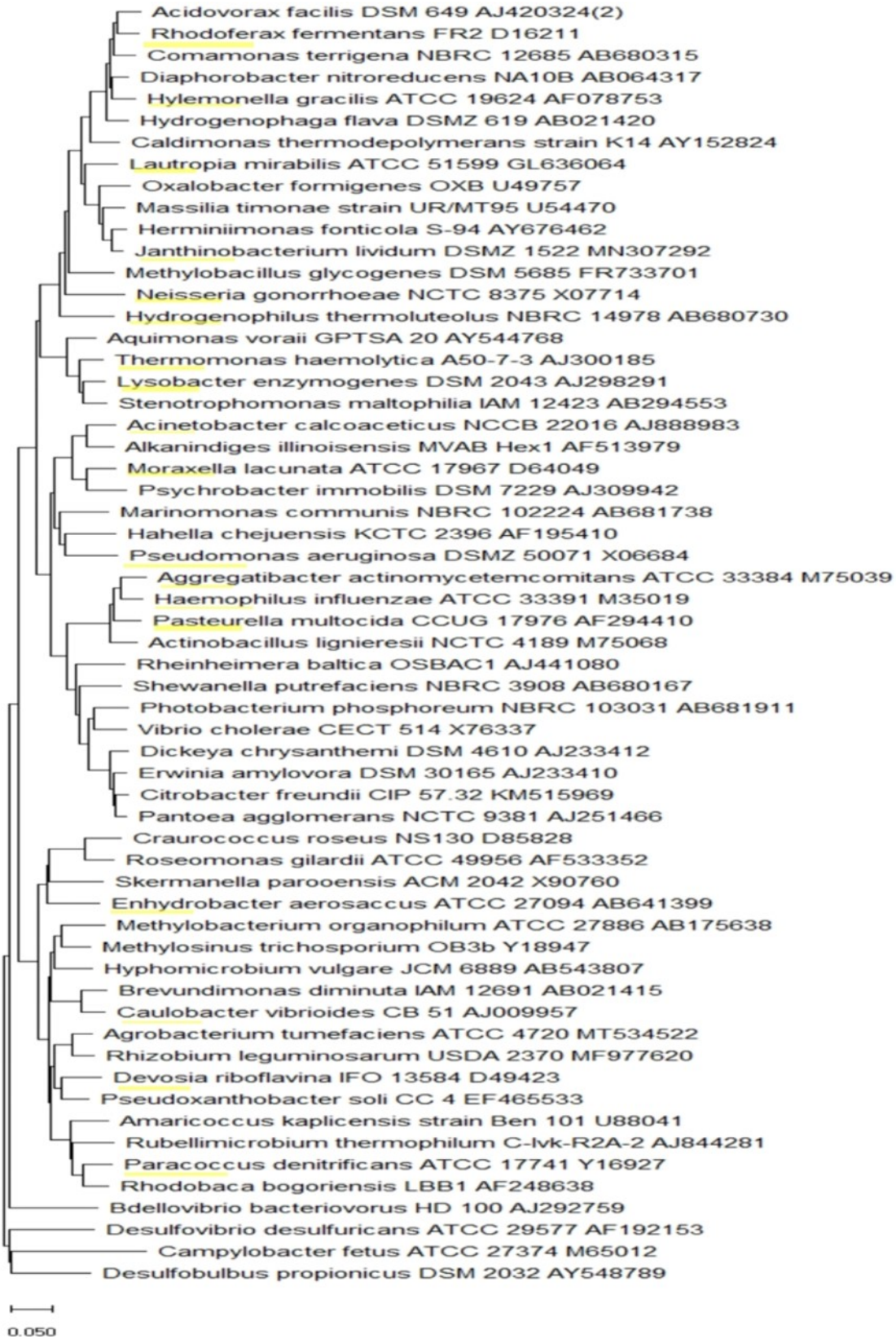

Figure 2. Phylogenetic tree, based on the 16S rRNA gene, of the *Pseudomonadota* genera diversity observed in healthy human skin, represented by the type species of each genus. The genus consistently identified in the three studies are highlighted in yellow.

3.2 Acinetobacter spp. genomes comparison

After exclusion of the possible repetitions of the genomes, a total of 232 genomes was considered for the genomes comparison. The 232 genomes were classified as clinical, clinical associated or environmental according to the origin of the *Acinetobacter junii*, *Acinetobacter johnsonii*, and *Acinetobacter lwoffii* strains (Figure 3). The clinical isolates were taken from human specimens like sputum, blood, urine, faeces while clinical associated cases were taken from hospital sink, hospital basin, hospital floors and surfaces. The environmental isolates were taken from areas like lakes, sewages, river. The most common origin for *A. johnsonii* and *A. lwoffi* was the environmental, while for *A. junii* was the clinical.

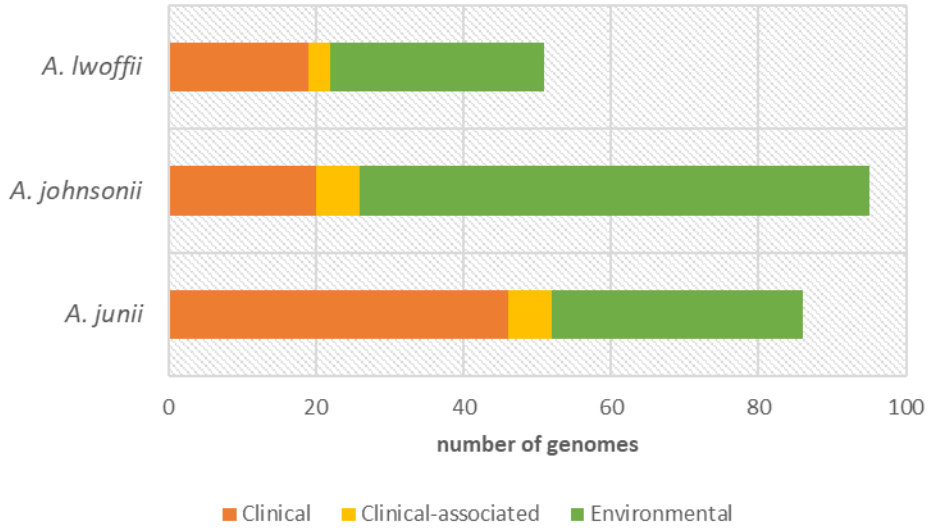

Figure 3. Origin of the Acinetobacter strains used for the genomes comparison.

Acinetobacter johnsonii, *Acinetobacter lwoffii, Acinetobacter junii* are found in human skin as commensal. All these three species cause blood infections via catheter if hospital acquired infection occur. (Dijkshoorn et al., 2007). *Acinetobacter junii*, clinically more relevant, have been recorded to cause sepsis in newborn in hospital via infected intravenous fluids (Beaufort et al.,2007). In a study, It has caused bacteremia in 43 patients in a tertiary care center in northern Taiwan, in which 2 patients went for shock, where 35% of cases were resistant to colistin (Tsai et Al., 2012). These *Acinetobacter* forms biofilms, hence more horizontal mobile genetic elements transfer, thereby capable of transferring the virulence factors, increased survival and multidrug antibiotics resistance genes to other disease causing pathogens. (Mendes et al., 2023). The *Acinetobacter junii* species samples analysed from a Shanghai

hospital in China from August 2011 to August 2016, showed around 35% resistance to Gentamicin with around 4% resistance to Carbapenems (Tang et al.,2017). There was a case of ***bla*$_{NDM-1}$** positive *Acinetobacter junii* in a 37 year old hospital patient with leg ulcer in Argentina (Montaña et al., 2023).

Acinetobacter johnsonii has been identified as a chronic pathogen for chronic rhinosinusitis (Cleland et al.,2016), meningitis (Mónica P Gutiérrez-Gaitán,2022) and is also used for malathion biodegradation (Shan et al., 2008). *Acinetobacter johnsonii* Aj2199 had been identified to have more number of mobile genetic elements, with around 10 resistance genes *bla*$_{PER2}$, *bla*$_{OXA58}$, *bla*$_{TEM-1}$, *strA, strB, ereA, sul1, aacC2, msrE* and *mphE*, thereby showing it could be a source for horizontal gene transfer for all other clinical relevant pathogens (Sabrina Montaña et al.,2022). *Acinetobacter johnsonii* isolates XBB1 and XBC1 were found in hospital sewage carrying ***bla*$_{NDM}$** gene by Tn125 transposon suggesting carbapenem resistance (Zong et al., 2013). *Acinetobacter johnsonii* MA19 isolated from malathion soils, was successfully used to remove malathion from polluted wastewater (Shan et al., 2008). *Acinetobacter johnsonii* are found in environments like soil, wastewater (Hrenovic et al.,2014).

Acinetobacter lwoffii had involved organs like lungs, urinary tract, a case of gastroenteritis (Regalado et al., 2009), a case of peritonitis was reported (Yasar Tas et al., 2017). *Acinetobacter lwoffii* is a known soil bacterium that is found in forest soil, agricultural land soil, lake, sea water (Rakitin et al.,2021). *Acinetobacter lwoffii* was able to survive with toxic metals like mercury, arsenic due to larger and more plasmids with multiple resistance genes with a study done with frozen permafrost isolates (Mindlin et al.,2016) and survive post desiccation 21days (Houang et al.,1998). *Acinetobacter* lwoffii samples gives an antimicrobial resistance with gentamicin (29%) and Carbapenems (around 12-15%) (Tang et al.,2017). 37-year-old Chinese female with urinary tract infection was diagnosed with *bla*$_{NDM}$ positive *Acinetobacter lwoffii*, resistant to imipenem and meropenem, showing attention need (Hu et al.,2012).

3.2.1 Antibiotic resistance genes profiles

A high diversity of antibiotic resistance genes was identified in the genomes of the selected *Acinetobacter* genomes. Most of them were associated with resistance to beta-lactam, aminoglycosides, tetracyclines and macrolides (Figure 4). The beta-lactams resistance formed around 44% of total antibiotics resistance genes, which was significantly higher comparing to other drugs resistance genes. Aminoglycosides resistance genes occupied nearly 27.5% of the total genes whereas the Macrolides, Tetracycline, and quinolone resistance genes represented around 17% of the total antibiotic's resistance genes. Comparison of inter origin and inter species of 232 *Acinetobacter* genomes was brought by seeing the diversity of antibiotic resistance genes in them, relative abundance of antibiotic resistance genes and analyse of the clinically significant antibiotic resistance genes in them.

On comparing all the genomes considering the origin (Figure 4A), the clinical strains presented aminoglycoside resistance genes with 16 resistance genes out of 19 aminoglycoside resistance genes (84%), 75% of the tetracycline resistance genes (9 genes out of 12 genes), and macrolides (3 genes of 4 genes, 75%). For Beta lactam was observed a diversity of 23 genes out of 31 beta lactam genes identified (74%). Genes conferring resistance to other antibiotic classes were also frequent in clinical strains (85%).

The trend of lower relative abundance was observed in clinical genomes with *aac(6')-Ib* (1.29%), *tetM* (0.43%), bla_{NDM} (1.29%), bla_{TEM-1} (0.43%), bla_{OXA-58} (2.58%), *floR* (1.29%), *sul2* (3.87%), *sul1*(3.44%). This pattern disagreed with the environmental genomes which had higher relative abundance and diversity, indicating that the pattern of resistance in the environmental isolates is more conserved. The clinical associated strains followed a different trend with lesser diversity (aminoglycosides 31%, beta lactam 38% with exception of macrolides 75%) and lower relative gene abundance with *aph(3')-Via* (1.72%), *msr(E)* (2.58%), *mph(E)* (2.58%), bla_{PER-1}(0.43%), bla_{NDM-1}(1.72%), *floR*(0.86%), *aac (6')-Ib* (1.72%), *tetM* (0%), bla_{NDM} (1.72%). bla_{TEM-1} (0.43%), bla_{OXA-58} (0.86%), *floR* (0.86%), *sul2* (1.72%), *sul1*(1.72%) for clinical associated genomes.

Similarly on comparing the three species of *Acinetobacter* (Figure 4B), it became clear that *Acinetobacter lwoffii* had higher number in relative gene abundance of antibiotic resistance genes than *Acinetobacter junii* and lower genes than *Acinetobacter junii,* with much the same design for clinically remarkable antibiotic resistance genes. The *Acinetobacter johnsonii* had a soaring diversity and inflated relative resistance gene abundance.

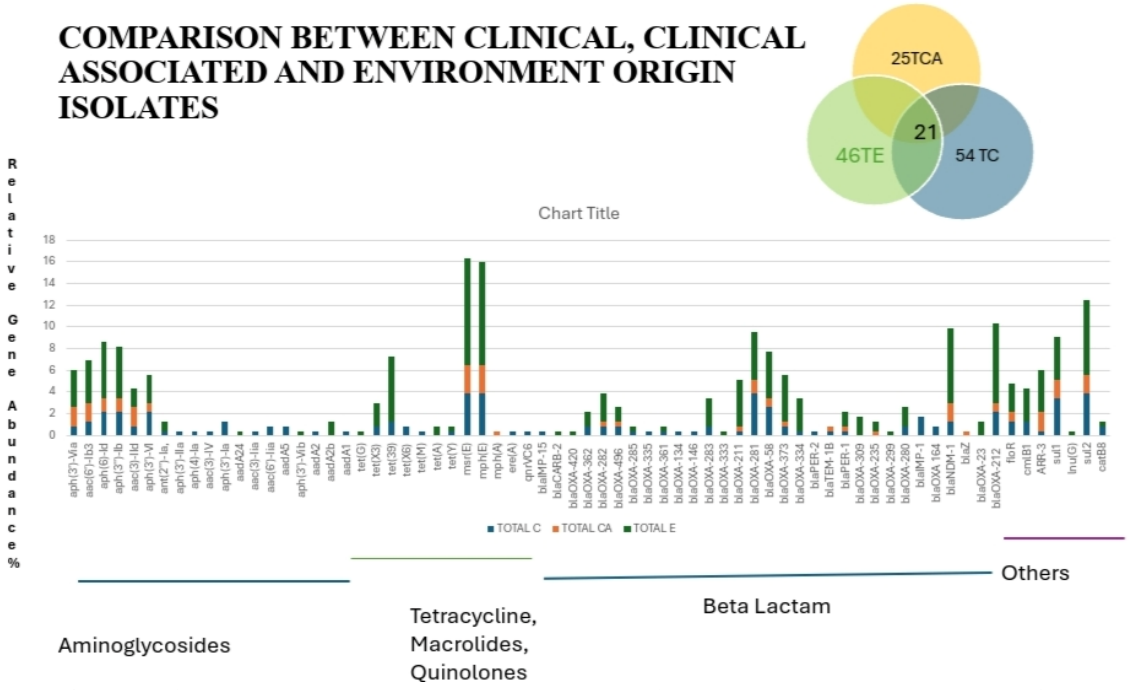

Figure 4 A. Comparison between clinical (TC), clinical associated (TCA) and environmental (TE) origin isolates of all *Acinetobacter* genomes for antibiotic resistance genes

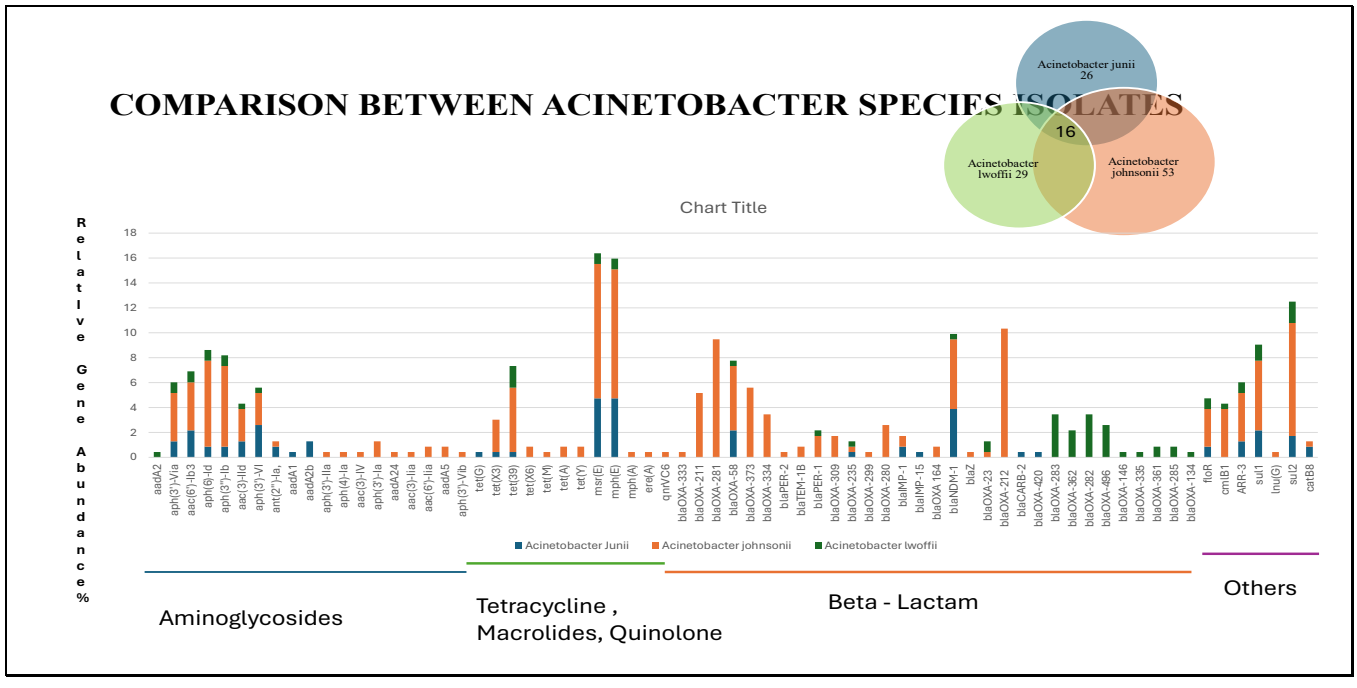

Figure 4B. Comparison of the antibiotic resistance profiles of the *Acinetobacter junii, Acinetobacter johnsonii, and Acinetobacter lwoffi* genomes.

Some genes were common to the different species and environments such as aminoglycosides resistance genes, macrolides and beta-lactams. 21 genes were identified in all the origins (Figure 4A, Venn diagram), and 16 genes in the three species under study (Figure 4B, Venn diagram). Among these ubiquitous genes was the carbapenemase NDM-1, a gene with high clinical relevance.

From 124 isolates, collected from clinical samples in two teaching hospitals in Ahvaz, distribution of *qnrA* (52.6%)*, qnrS* (3.2%)*, tetA* (93.5%)*, tetB* (69.2%) and *sul1* genes (6.42%) were observed. Our results go in hand with study showing environmental group has more diverse antibiotic resistance genes with high relative abundance. This goes in hand positively with a study where wastewater treatment process in Kokšov-Bakša was a potential source from which antibiotic resistance spreads (Kisková et al., 2023). Our study has positive correlation with the study *Acinetobacter* spreads surprisingly high beta lactam resistance in environment posing threat (Hubeny et al., 2022). These results in our study implicated there were similarity within a group based on the origin but are distinct and have differences between corresponding groups, which was also seen in species level. *Acinetobacter johnsonii* had lofty presentation in whole genomes species inspection, whereas *Acinetobacter junii* and *Acinetobacter lwoffii* had a different demonstration. This study has positive association with a study showing *Acinetobacter johnsonii* had many important antibiotic resistance genes and have horizontal transfer capacity (Castillo-Ramirez et al., 2020).

3.2.2 Virulence factors profiles

The virulence factors profiles of the 232 *Acinetobacter* genomes were analysed. The clinical strains showed that they lacked the unique virulence factors and pili of environmental origin, (Table 5A). The clinical associated strains mimicked clinical strains virulence pattern. Environmental strains, on the other hand, showed a diverse range of *pilR*, *pilG*, *pilJ* pili virulence genes, virulence associated proteins, *Lvh Legionella* secretion system virulence gene (Table 5A).

The Inter-species analysis showed that *Acinetobacter junii* had more *LPS O-antigen* virulence, while *Acinetobacter lwoffii* had a unique capsule and a cell surface gene (Table 5B).

Table 5A. Comparison of virulence factors profile between clinical, clinical associated and environmental genomes

Clinical	***Clinical associated***	***Environmental***
Lack of unique pili and virulence factors associated with environment origin strains. Shares *SodCl* gene with environment strains.	Shares common virulence factors of clinical strains	Diverse of *pilR*, *pilG*, *pilJ* unique pili virulence gene. Virulence associated proteins *vapA4*, *Bacillibactin* dhbE *Lvh Legionella* secretion system virulence gene.

Table 5B. Comparison of virulence factors profile between species *Acinetobacter junii*, *Acinetobacter johnsonii*, and *Acinetobacter lwoffii* genomes

Acinetobacter junii	***Acinetobacter johnsonii***	***Acinetobacter lwoffii***
More *LPS O-antigen* virulence gene than *Acinetobacter johnsonii and Acinetobacter lwoffii.* Nitrate reductase *narH* virulence gene Virulence associated proteins, *vapA4*	Biotin metabolism *bioB* virulence gene *Bacillibactin* dhbE, *SodCl* virulence gene *pili R, pili J, pili G, tapW* pili virulence gene are seen	Capsule biosynthesis and transport, *kpsT* virulence gene. Cell surface *GPL* locus *rmt4* virulence gene.

These results indicated that there is uniqueness in clinical origin pili virulence containing strains, proving its ability as a clinically significant pathogen. The virulence gene for environment strains was distinct and showed its ability to mediate cells interactions, DNA uptake with unique secretion system gene, neutral pH phagosome helping *vapA4* gene, iron transferring *Bacillibactin* gene. Our study adds positive light to this study which shows environmental strains have more virulence factors (Tobin et al.,2023). Environmental *Acinetobacter baumannii* isolate DSM30011 may serve as source for potential virulence factors more divergent than clinical strains (Repizo et al., 2017). Similarly, *Acinetobacter johnsonii* showed *pil R*, *pilG*, *pilJ* pili virulence genes, dhbE *bacillibactin* gene, stress adaptation *SodCl* virulence gene. *Acinetobacter junii* is clinically relevant with more *LPS O-antigens,* which helps evade host immune response (Shadan et al., 2023).

3.3 bla_{NDM} positive genomes

For the reasons described before, the clinical relevance and ubiquitous presence in the three *Acinetobacter* species and origins, were selected the genomes of the bla_{NDM} positive strains (n=23) to proceed with a deepest genomic analysis of these isolates, aiming to understand if the gene is present in the same genomic environment independently of the species and origin.

The 23 bla_{NDM} positive genomes of *Acinetobacter junii* (n=9 genomes)*, Acinetobacter johnsonii* (n=13 genomes)*, and Acinetobacter lwoffii* (n=1 genome), have rich profiles of antibiotic resistance with the aminoglycosides contributing around 25% of all antibiotic resistance genes, Macrolides, tetracycline, and quinolone (20%), beta-lactams (37%) and others (17%) (Figure 8A).

Considering the origin, the environmental bla_{NDM} positive strains revealed a lofty diversity with higher gene abundance for *aph(3')-Via* (6 genes), *aac(6')-Ib3* (7 genes), *aph(6)-Id* (9 genes), *aph(3'')-Ib* (8 genes), *aph(3')-VI* (6 genes), *tet(X3)* (5 genes), *tet(39)* (4 genes), *msr(E)* (8 genes), *mph(E)* (13 genes), *aac(6')-Ib* (7 genes), *tetA*(2 genes), *floR* (4 genes), *sul2* (10 genes), *sul1*(6 genes). The clinical bla_{NDM} positive genomes conflicted this result with smaller diversity for antibiotic resistance genes, smaller relative abundance percentage and also for clinically significant antibiotic resistance genes. The clinical associated bla_{NDM} positive genomes followed a distinct path with moderate relative abundance , with lesser gene diversity. In the same way, the species of bla_{NDM} genomes of *Acinetobacter*, the *Acinetobacter lwoffii* had a

squatted relative gene abundance with stubby gene diversity than *Acinetobacter junii*. *Acinetobacter johnsonii* dominated the picture, as seen in figure 8B.

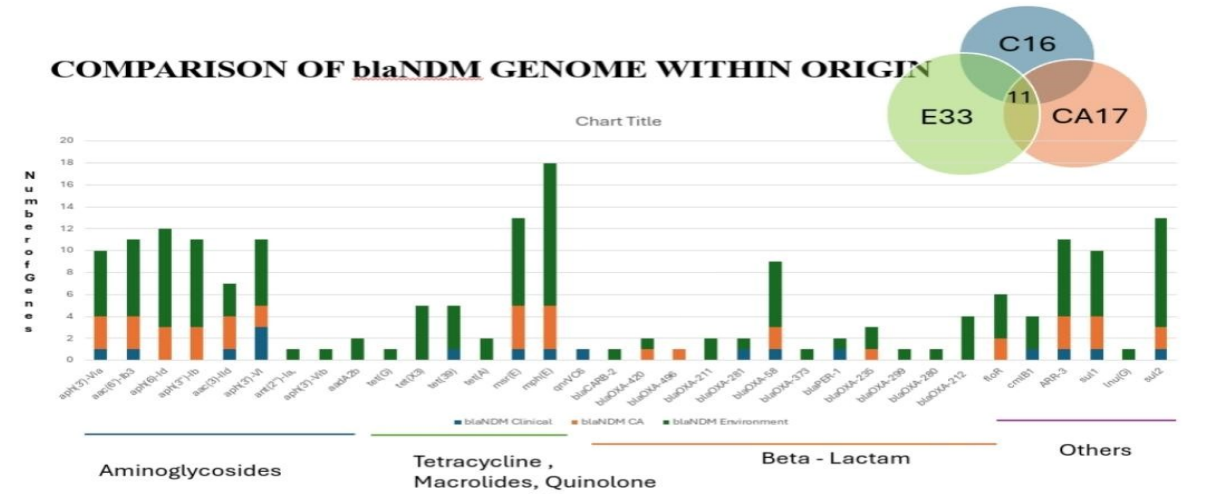

Figure 5A. Comparison of the antibiotic resistance genes profiles of *bla*$_{NDM}$ positive genomes considering the origin of the isolates: clinical, clinical associated or environmental.

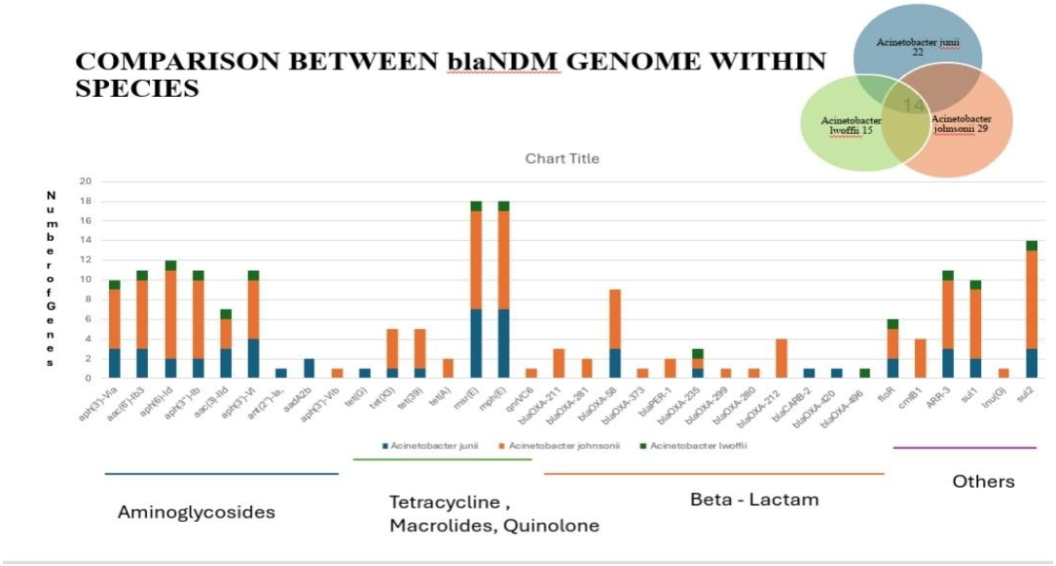

Figure 5B. Comparison of the antibiotic resistance profiles of the *bla*$_{NDM}$ positive *Acinetobacter junii*, *Acinetobacter johnsonii*, and *Acinetobacter lwoffii* genomes considering the species.

The Carbapenem resistant *bla*$_{NDM}$ *Acinetobacter* displayed that environment genomes and *bla*$_{NDM}$ clinical genomes disagreed with same course. *Acinetobacter junii* presented a halfway picture in *bla*$_{NDM}$ species approach. Our study findings goes in hand that environmental Acinetobacter species may serve as reservoir (Tobin et al., 2024). This study gives positive value to carbapenemase Acinetobacter from environment sources like wastewater treatment plants, biogas management system with strong spread to clinical MDR strains (Pulami et al.,2022). Similarly *Acinetobacter johnsonii* with *bla*$_{NDM}$ resistance is important source of dissemination of clinical strains (Zong et al., 2013). This pattern suggested inter origin and inter species based differences in both *Acinetobacter* with and without acquired carbapenemase gene was based on phylogeny. This showed how groups, based on phylogeny, acquire and determined drug resistance (Ranjbar et al., 2020).

The virulence factor analysis in *bla*$_{NDM}$ positive genomes showed that there were some distinctions between clinical, clinical associated and environmental origin strains. Nitrate reductase (*narH*), Bartonella virulence associated proteins *vapA4*, *Bacillibactin dhbE*, SodCl gene was seen in environmental strains (Table 6A). The clinical origin as well as clinical

associated strains on the other hand showed a diverse of *pilT2*, *pilU*, *pilA* and *pilE*. This study goes in hand with environmental Acinetobacter study with diverse virulence genes found capable of causing virulent antibiotic resistant infections(Doughari et al., 2012).

Table 6A. Comparison of virulence factors profile for *bla*$_{NDM}$ positive genomes from clinical, clinical associated and environmental origin

Clinical	***Clinical associated***	***Environmental***
diverse of *pilT2, pilU, pilA* and *pilE*	diverse of *pilT2, pilU, pilA* and *pilE*	*pilR, pilG* virulence gene for Type IV pili. Nitrate reductase(*narH*) Virulence associated proteins *vapA4*, *Bacillibactin dhbE, SodCl* gene

There were some differences regarding inter-species comparison of *bla*$_{NDM}$ positive genomes. The *LPS O-antigen* virulence gene was commonly found in *Acinetobacter junii,* whereas its frequency was less in *Acinetobacter johnsonii*. The *Catalase-peroxidase katG* virulence gene was more commonly found in *Acinetobacter johnsonii* than *Acinetobacter junii*. Bartonella virulence associated proteins *vapA4*, *Bacillibactin dhbE*, *SodCl* gene are present in *Acinetobacter johnsonii* (Table 6B). The *Acinetobacter lwoffii bla*$_{NDM}$ genome cannot be commented as it was a single genome.

Table 6B. Comparison of virulence factors profile for *bla*$_{NDM}$ positive genomes from *Acinetobacter junii, Acinetobacter johnsonii, Acinetobacter lwoffi* isolates

Acinetobacter junii	***Acinetobacter johnsonii***	***Acinetobacter lwoffii***
More *LPS O-antigen* virulence gene	More *Catalase-peroxidase katG* virulence gene than Acinetobacter junii. *Bartonella* virulence associated proteins vapA4, *Bacillibactin dhbE, SodCl* gene	Cannot be commented with a single genome

Acinetobacter junii which had more clinical isolations has more *LPS-O* antigen, which was important cause septic shock in patients, via toll like receptor-4 activation, whose ligand was LPS antigen (Moffatt et al., 2013). This uniqueness seen in carbapenemase gene acquired *Acinetobacter* where the virulence changed according to where it was isolated and which species it was from, suggesting phylogeny relationship. These results indicated there was a connection between virulence and phylogeny and based on the necessity, genetic background helped in formation of virulence factor clone, thereby making them pathogenic (Picard et al., 1999). Particular phylogeny groups had particular virulence which help them to be clinically significant pathogens (Rezatofighi et al., 2021).

Aiming to understand the phylogenetic relationship among the *bla*$_{NDM}$ positive strains, it was attempted to do an analysis based on the comparison of the conserved genes with wgMLST Dendrogram for 22 genomes. The dendrogram results gave close distance relationship between a total of 7 *bla*$_{NDM}$ positive *Acinetobacter* genomes formed a cluster for environmental origin samples, while clinical origin also has same distance cluster (Figure 6).

Considering the *bla*$_{NDM}$ gene *Acinetobacter* inter-species analysis, it showed that *Acinetobacter johnsonii* formed 6 close distance clusters. On the other hand, *Acinetobacter junii* formed two pairs of 2 genome cluster. So with this analysis, we can say though there are some similarities within origin and species, one big conclusion we can get is inter-origin and inter-species differences is not seen in wgMLST dendrogram analysis.

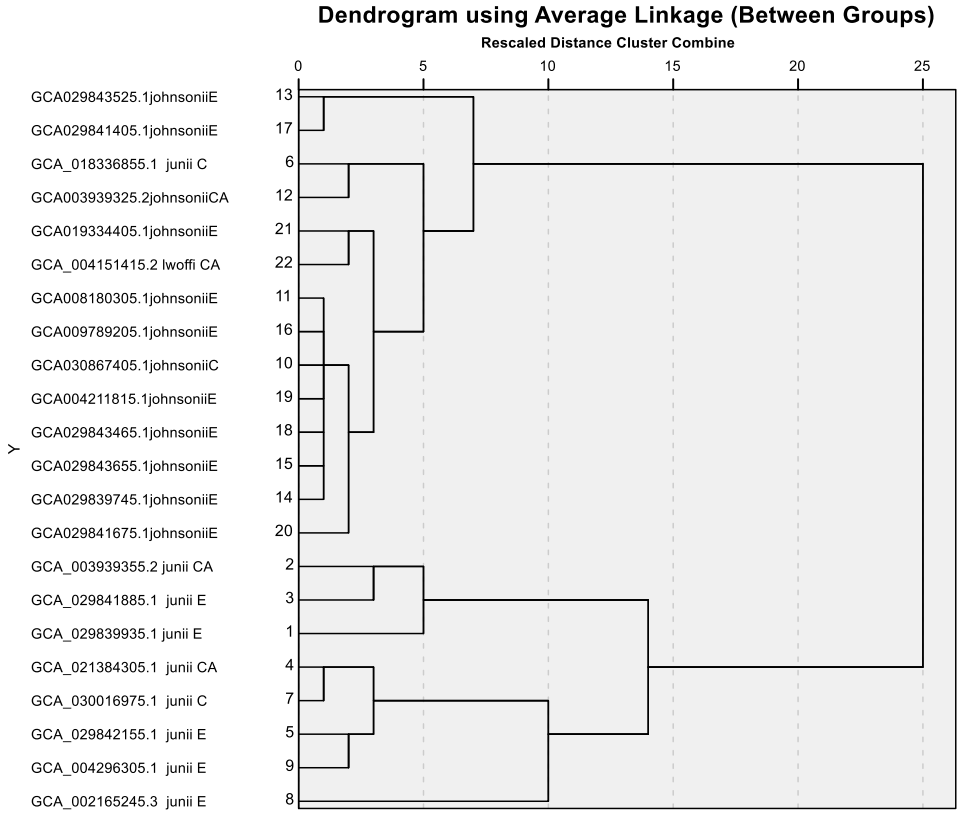

Figure 6. wgMLST Dendrogram analysis for the *bla*$_{NDM}$ positive genomes

Our study implicated that origin and species group is not associated with phylogeny based on this MLST analysis. These MLST like for these variable target genes that helped to differentiate various strains based on evolution (Losada et al., 2013), can also help in providing knowledge into their phylogeny behaviour. MLST analysis done with the DNA sequences of various housekeeping genes helped in bringing out the phenotypic relationship between the studied genomes (Boers et al.,2012). This knowledge into the MLST based genetic structure of the isolates helped us to better understand their pathogenicity and other phylogenetic functions (Martino et al., 2011).

3.4 blaNDM gene environment

The location of the gene *bla*$_{NDM}$ was studied for the 21 out of the 23 genomes. Two bla$_{NDM}$ positive genomes were excluded from the analysis due to not significant scores, values, origin. For the remaining 21 genomes inter-origin comparison showed that the genomes of clinical origin have the insertion sequences of family IS4, IS3, IS66 (Table 7). The clinical associated

genomes show the insertion sequences of family IS66 (for 3 genomes), IS1202. The environmental genomes reveal IS66 (for 5 genomes), IS1595, IS3 (for 3 genomes), IS4, IS21, IS5 (for 2 genomes), IS982.

The inter species analysis conveyed that the *Acinetobacter junii* had three common IS66 out of eight insertion sequences family while *Acinetobacter johnsonii* revealed five IS66 family sequences out of ten.

Table7. Insertion sequence, transposon associated DNA endonuclease, transposase analysis of *bla*$_{NDM}$ positive genomes

Genome	**Insertion sequence**	**IS Family**	***Acinetobacter* high Score bits and Significant E value**
GCA_029839935.1 *A. junii* Environmental	ISAba16- 65% (ORF1), 98% (ORF2) and 96% (ORF3)	IS66	high Score(bits)- 4377 E value- 0.0
GCA_003939355.2 *A. junii* Clinical associated	ISAba17- 61% (ORF1), 78% (ORF2) and 67% (ORF3)	IS66	high Score(bits)-5095 E value-0.0
GCA_029841885.1 *A. junii* Environmental	ISAba16- 65% (ORF1), 98% (ORF2) and 96%(ORF3)	IS66	high Score(bits)-4329 E value-0.0
GCA_021384305.1 *A. junii* Clinical associated	ISApi2 Found in GR13-type plasmid p2012N21-164-1 in pdif site and flanked by a 5 bp target site.	IS1202	High Score(bits)- 153 E value- 3e-36
GCA_029842155.1 *A. junii* Environmental	ISAju1	IS1595	High Score(bits)- 143 E value- 1e-31
GCA_018336855.1 *A. junii* Clinical	ISAba33	IS4	High Score(bits)- 2244 E value-0.0

	Third ORF is a considered ORFAB transposase		
GCA_030016975.1 *A. junii* Clinical	ISAcsp12 Third ORF is a considered ORFAB transposase	IS3	High Score(bits)-40.1 E value- 0.99
GCA_002165245.3 *A. junii* Environmental	ISAba14 Third ORF is a considered ORFAB transposase	IS3	High Score(bits)-2399 E value-0.0
GCA_004296305.1 *A. junii* Environmental	InSignificance scores,values, origin		
GCA_030867405.1 *A. johnsonii* Clinical	ISAjo6	IS66	High Score(bits)-4766 E value-0.0
GCA_008180305.1 *A. johnsonii* Environmental	ISAlw10	IS4	High Score(bits)-2730 E value-0.0
GCA_003939325.2 *A. johnsonii* Clinical associated	ISAjo4 found in the GR13-type plasmid pAJ_082-3, flanked by 8 bp target site.	IS66	High Score(bits)-4900 E value-0.0
GCA_029843525.1 *A. johnsonii* Environmental	ISAba25 99%(ORF1), 100%(ORF2) and 95% (ORF3)	IS66	High Score(bits)-4314 E value-0.0
GCA_029839745.1 *A. johnsonii* Environmental	ISAjo6	IS66	High Score(bits)-3903 E value-0.0
GCA_029843655.1 *A. johnsonii* Environmental	ISAba8	IS21	High Score(bits)-3487 E value-0.0
GCA_009789205.1 *A. johnsonii* Environmental	ISAba66- Found in the GR13-type plasmids pE47_002 and pBspH2, flanked by a 3 bp target site. The third ORF is considered ORFAB transposase.	IS3	High Score(bits)-577 E value-8e-165
GCA_029841405.1 *A. johnsonii* Environmental	ISAba12	IS5	High Score(bits)-615 E value- 2e-173
GCA_029843465.1 *A. johnsonii* Environmental	ISAjo3 ISAjo3 was found in the A. johnsonii GR13-type plasmid pXBB1-8.	IS3	High Score(bits)-38.2 E value- 7.5

	Copies flanked by 4 bp target site		
GCA_004211815.1 *A. johnsonii* Environmental	Insignificant values scores origin		
GCA_029841675.1 *johnsonii* Environment	ISAba68 GR13-type plasmid, flanked by 9 bp target site	IS5	High Score(bits)- 42.1 E value- 0.30
GCA_019334405.1 *A. johnsonii* Environmental	ISAcsp2	IS982	High Score(bits)- 771 E value-0.0
GCA_013186005.1 *A. johnsonii* Environmental	ISAlw34	IS66	High Score(bits)- 3927 E value-0.0
GCA_004151415.2 *A. lwoffi* Clinical associated	ISAba17	IS66	High Score(bits)- 4587 E value-0.0

With two insignificant insertion sequences, the number of genomes analysed may be less, to get significant correlations for inter-origin and inter-species insertion sequences and phylogeny. *ISAba125* of IS30 family has universally known to harbour carbapenemase bla_{NDM} gene (Tolemon et al.,2012). The transposon *Tn125* is one of key vehicles of transmission of bla_{NDM} genes (Poiral et al.,2012) . The IS3 families is responsible for carbapenem resistant strains and successful dissemination of carbapenemase resistance in a study in India (Vijayakumar et al., 2013). The *ISaba14* belonging to IS3 family is responsible for carbapenemase bla_{NDM} *Acinetobacter* strains from USA patient (Hamed et al., 2022). The IS26 is known to cause carbapenemase dissemination in Acinetobacter in a bla_{NDM} study (Jones et al., 2014). Regarding the evolution of bla_{NDM} gene, it was first isolated in a Swedish patient in New Delhi, 2008 in a Klebsiella pneumoniae (Yong et al., 2009). First report of bla_{NDM} in Acinetobacter was reported in 2012 in civilians wounded in civil war in Syria (Rafei et al., 2012). The fast spread of bla_{NDM} with incompatible plasmid groups joined together like IncF, IncA/C and IncX (Wu et al., 2019). In last few years emerging new bla_{NDM} plasmids with new structures have been discovered (Li et al., 2022).

The presence of ble hints the hypothesis that blaNDM–1 arrived from *Acinetobacter*, while the presence of TnpA suggests bla_{NDM-1} may have got from transposition and remained for greater period in Klebsiella strains(Xiang et al.,2020). The insertion sequence was salient because the

gene expression played an important role in transposition activity like translation commence signal, RNA transcription, Tpase stability, host factors (Mahillon & Chandler., 1998), that finally decided the organism pathogenicity and virulence. The Insertion sequence showed homology between bacteria when they have phylogenetic closeness between them and there was also genus specific insertion sequence (Ouahrani et al., 1993). Even a composition of simple lipid A and the similarity in them signal us an evolutionary intimacy between these bacteria (Moreno et al., 1990).

The functional annotations of the *bla*$_{NDM}$ positive genomes showed other Antibiotics resistance genes (ARG) like Macrolide phosphotransferase genes, Aminoglycoside nucleotidyltransferase, Aminoglycoside acetyl transferase, Tetracycline inactivating enzyme that were found similar and cotransporting among bla$_{NDM}$ genomes in same genetic element. The inter-origin analysis of Clinical, Clinical associated and Environment genomes had shown that the *bla*$_{NDM}$ genetic element had similar Aminoglycoside, Macrolide resistance, while Tetracycline and Bleomycin resistance occuring in Environment origin genomes. The inter-species analysis of *Acinetobacter junii*, *Acinetobacter johnsonii*, *Acinetobacter lwoffii* genomes of *bla*$_{NDM}$ genetic element display similar Aminoglycoside, Macrolide resistance, with Tetracycline and Bleomycin resistance occurring in *Acinetobacter johnsonii* samples.

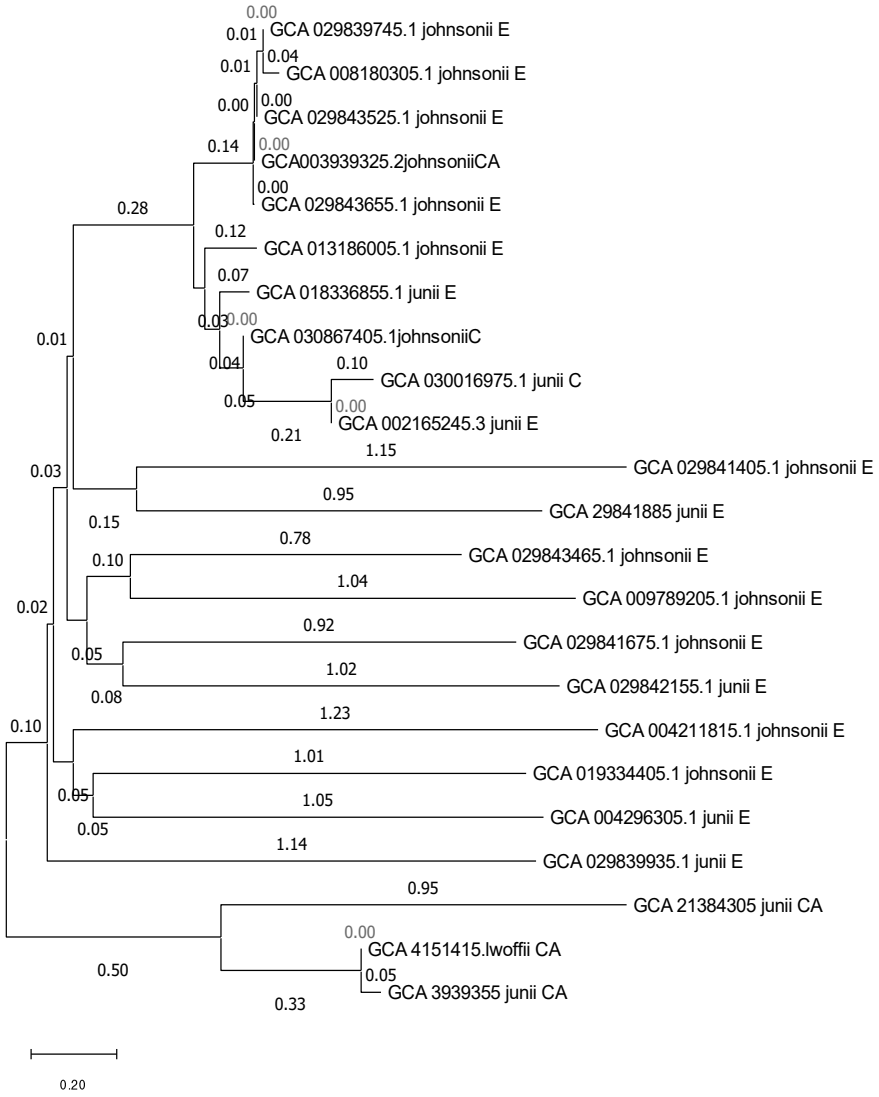

Figure.7 Alignment of the *bla*$_{NDM}$ genes

Table8 - Other Antibiotics resistance

S.No	***Genomes***	***Origin and Species***	***Genetic element***	***Other Antibiotics resistance carried***
1	GCA 29839935	*Acinetobacter junii Enviromental*	NZ_JAOCBN010000002.1	Macrolides specific efflux protein *MacA*,Aminoglycoside3,39 phosphotransferase, *APH*
2	GCA 3939355	*Acinetobacter junii Clinical Associated*	NZ_CP078043.1	Macrolides specific efflux protein *MacA*,Aminoglycoside3,39 phosphotransferase, *APH*, Chloramphenicol
3	GCA 29841885	*Acinetobacter junii Enviromental*	NZ_JAOCFJ010000001.1	Macrolide phosphlotransferase,*Mph(E)*,Aminoglycoside3,39 phosphotransferase, *APH*
4	GCA 21384305	*Acinetobacter junii Clinical associated*	NZ_JAHNFA010000086.1	-
5	GCA 29842155	*Acinetobacter junii Environmental*	NZ_JAOCFW010000046.1	Aminoglycoside3,39 phosphotransferase, *APH*
6	GCA 18336855	*Acinetobacter junii Clinical*	NZ_CP059559.1	Aminoglycoside3,39 phosphotransferase, *APH*
7	GCA 30016975	*Acinetobacter junii Clinical*	NZ_JASDED010000082.1	-
8	GCA 2165245	*Acinetobacter junii Enviromental*	NZ_CP028800.2	Macrolides specific efflux protein *MacA*

9	GCA 4296305	*Acinetobacter junii Enviromental*	NZ_SGST01000061.1	-
10	GCA 30867405	*Acinetobacter johnsonii Clinical*	NZ_CP121779.1	Aminoglycoside3,39 phosphotransferase, *APH*
11	GCA 8180305	*Acinetobacter johnsonii Enviromental*	NZ_CP043307.1 NZ_CP043309.1	Macrolide phosphotransferase *Mph(E)*,Aminoglycoside3,39 phosphotransferase, *APH*
12	GCA 3939325	*Acinetobacter johnsonii Clinical associated*	NZ_CP079793.1	Macrolide phosphotransferase *Mph(E)*, Aminoglycoside3,39 phosphotransferase, *APH*
13	GCA 29843525	*Acinetobacter johnsonii Enviromental*	NZ_JAOCIL010000002.1	Macrolides phosphotransferase *Mph(E),* ,Aminoglycoside3,39 phosphotransferase, *APH*
14	GCA 29839745	*Acinetobacter johnsonii Enviromental*	NZ_JAOCBE010000002	Macrolides specific efflux protein *MacA*,Aminoglycoside3,39 phosphotransferase, *APH*, Teracycline inactivating enyme *Tet(X)*
15	GCA 29843655	*Acinetobacter johnsonii Enviromental*	NZ_JAOCIS010000002.1	Aminoglycoside3,39 phosphotransferase, *APH*,Teracycline inactivating enyme *Tet(X),* Macrolides specific efflux protein *MacA*
16	GCA 9789205	*Acinetobacter johnsonii Enviromental*	NZ_WSRY01000026.1	-

17	GCA 29841405	*Acinetobacter johnsonii Enviromental*	NZ_JAOCEJ010000052.1	Aminoglycoside3,39 phosphotransferase, *APH* bleomycin resistance protein
18	GCA 29843465	*Acinetobacter johnsonii Enviromental*	NZ_JAOCIJ010000063.1	Aminoglycoside3,39 phosphotransferase, *APH*, bleomycin resistance protein
19	GCA 4211815	*Acinetobacter johnsonii Enviromental*	SGVZ01000005.1	Bleomycin resistance protein
20	GCA 29841675	*Acinetobacter johnsonii Enviromental*	JAOCEX010000092.1	Bleomycin resistance protein
21	GCA 19334405	*Acinetobacter johnsonii Enviromental*	CP079750.1	Aminoglycoside3,39 phosphotransferase, *APH* Bleomycin resistance protein
22	GCA 13186005	*Acinetobacter johnsonii Enviromental*	JABAGU010000003.1	Aminoglycoside acetyl transferase *AAC(3)*, Tetracycline Résistance Protein *Tet(X)*
23	GCA 4151415	*Acinetobacter lwoffii Clinical associated*	CP078046.1	Aminoglycoside3,39 phosphotransferase, *APH*

The fasta analysis results hinted that in origin and species of carbapenem defending Acinetobacter genomes, there was resemblance within origin and within species, but contrasts persisted between the different origin and species. The evolutionary adaptation occurred when the genes are stimulated, for the organism to survive in that selective environment (Arnold et al., 2001). These results implicated that there may be changes in phylogenetic lineages when the external environment changed over time. So, the adaptation phenotypic behaviour only stimulated the switch in evolutionary changes in traits (Revell et al., 2009).

3.5 Acinetobacter profiles of tolerance to antimicrobials and cosmetics

A set of 32 strains of *Acinetobacter johnsonii* (10 strains), *lwoffii* (13strains) and *junii* strains (9 strains) from the lab culture collection were tested for their tolerance to some antimicrobials, aiming to understand if the tolerance to different groups of antimicrobials (e.g. antibiotics, antiseptics) could be associated with a higher tolerance to cosmetics. All the strains were already characterized for their profiles of tolerance to a set of 12 antibiotics, and were now tested for antiseptics (hydrogen peroxide, iodine, bleach) and cosmetics (Ecoskin 3% m/v, Naturstar antimicrobial 0.6% - 5% m/v, Carboxymethylhalocellulose (CMH) 2-4% m/v, Ectoin 1-2% m/v , Bovine Lactoferrin). Water was used as negative control.

The *Acinetobacter* strains with antibiogram showed that water had no reactions, while the highest inhibition was caused by Hydrogen peroxide (Table 9) , with lineant hampering by compound Iodine, while Bleach had a modest response .

Table 9 – Antibiogram results

Strains	**Water(cm)**	**Hydrogen peroxide(cm)**	**Iodine (cm)**	**Bleach (cm)**	**Cosmetics**
Acinetobacter junii					
E.Coli Reference	0	2.3	1.2	1.4	No inhibition observed
EF1	0	3	1.3	2	No inhibition observed
EF6	0	3.4	1.7	2.5	No inhibition observed
EF8	Inhibition not measurable due to repeated poor growth				
EF14	0	3.4	1.4	2	No inhibition observed

EF19	0	3.4	1.6	2.2	No inhibition observed
E. coli Reference	0	2.2	1.1	1.1	No inhibition observed
BT34	0	3.4	1.4	1.4	No inhibition observed
A2R10	0	2.5	1.4	0.8	No inhibition observed
BF26	0	3.8	2.5	3	No inhibition observed
BF53	0	3.5	1.2	1.5	No inhibition observed
Acinetobacter johnsonii					
E. coli Reference	0	2.5	1.1	1.6	No inhibition observed
T3CT9	0	3.5	1.6	2.5	No inhibition observed
T3CP1	0	4	2	3	No inhibition observed
T7AP9	0	3	1.2	1.8	No inhibition observed
T7BP10	0	2.2	1.1	1.5	No inhibition observed
E. coli reference	0	2	1.1	1.5	No inhibition observed
T5CP2	0	3.7	1.3	2.2	No inhibition observed
T5CP3	0	3.5	1.6	2	No inhibition observed
T3BR8	0	1.3	1.1	1.6	No inhibition observed

T3BR9	0	2.2	1.2	1.7	No inhibition observed
T7BR14	0	2.6	1.8	2.5	No inhibition observed
T3CR2	0	3.3	1.2	1.2	No inhibition observed
Acinetobacter lwoffii					
E. coli Reference	0	2.5	1.1	1.6	No inhibition observed
T10AT20	0	3.1	1.4	2.5	No inhibition observed
T5CT5	0	2.5	1.1	2.1	No inhibition observed
T1BT10	0	2.3	1.3	2.3	No inhibition observed
E. coli reference	0	2	1.1	1.5	No inhibition observed
T5BT15	0	1.8	1.5	2.7	No inhibition observed
T6BT3	0	2	1.1	2.1	No inhibition observed
T7BT2	0	2	1.1	1.6	No inhibition observed
T5BT12	0	1.8	1.6	2.6	No inhibition observed
T8BT11	0	2.1	1.3	2.6	No inhibition observed
T10BT5	0	2	1.4	1.7	No inhibition observed
T5BP20	0	2.1	1.4	1.7	No inhibition observed

T10BP2	0	2.1	1.6	2.3	No inhibition observed
T8BR19	0	1.9	1.3	2.3	No inhibition observed
T9BR1	Original source Contaminated				

Though *Acinetobacter* occured at a diluted abundance, it was significant in terms of phylogenetic evolutionship which is alike the most other notable genus. The reactions of *Acinetobacter junii, Acinetobacter johnsonii, Acinetobacter lwoffii* to certain anti septics showed the phenotypic behaviour of *Acinetobacter* with cosmetic compounds mimicking human skin microbiome. These results added positivity to other similar studies like the study done which showed *Acinetobacter* species changes the immune response with T helper cells and other immune cells and modulated composition of skin microbiota. (Fyhrquist et al., 2014).

4 Conclusions

With this study we could conclude that *Acinetobacter*, namely *A. johnsonii*, *A. junii* and *A. lwoffii* are among the most common Pseudomonadota present in healthy human skin. Based on a genome's comparison of strains of those three species it was also possible to conclude that they are important carriers of antibiotic resistance genes. *Acinetobacter junii* had more clinical relevance, with more LPS virulence antigens. The strains from environmental origin presented a higher diversity of resistance genes and virulence, while the strains from clinical origin have poorer profiles of resistance but higher abundance. *Acinetobacter johnsonii* is important source for dissemination for resistance for cases. These were observed for bla_{NDM} positive cases also. Some of the genes detected in the genomes under study are of clinical relevance, as for example the $bla_{NDM\text{-}1}$ gene, responsible for resistance to carbapenems that was observed to be common to the three *Acinetobacter* species and also to the different origins (clinical, clinical associated, and environmental). The $bla_{NDM\text{-}1}$ gene was mainly associated to mobile genetic elements, namely the IS66 family, and associated with other genes of resistance (e.g.Aminoglycosides, Macrolides).

With the *Acinetobacter* available in the lab culture collection, all from environmental origin, was not possible to infer any correlation between the antibiotic resistance profile and the tolerance to cosmetic compounds, but it was observed some variance between strains in the tolerance to hydrogen peroxide.

5 Future work

Our study findings gives green signal and promotes additional positivity with identical results of similar studies. This study further adds support to current phylogenetic research studies. With some limitations such as fewer bla_{NDM} strains were able to be collected from NCBI genomes, technical difficulties to test more cosmetics in *Acinetobacter* strains, future works is suggested to be carried on additional cosmetics, and to test on strains of clinical origin (if possible isolated from skin and carbapenem resistant), on larger timescale. One recommendation, which would like to give for future research, is to conduct with various other antibiotic resistant strains, to help us to better understand the bacteria phenotype and phylogeny traits. Hence future research should be done on a mega scale.

6 Appendices and Annexes-

Appendices

Antibiotic Resistance genes of all *Acinetobacter* Genomes

Acinetobacter junii	Isolation	aph(3')-Via	aac(6')-Ib3	blaOXA-235	blaPER-1	, msr(E)	mph(E)	floR	ARR-3	sul1	qacE	blaOXA-58	blaOXA-420	tet(X3)
GCA_003939335.2	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839935.1	Environment	1	1	1	0	1	1	0	1	0	1	0	0	1
GCA_003939355.2	CA	1	1	0	0	1	1	1	1	1	1	1	0	0
GCA_029841885.1	Environment	1	1	0	0	1	1	0	1	1	1	0	0	0
GCA_003939835.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843865.1	Environment	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_029842755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003940305.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021384305.1	CA	0	0	0	0	1	1	0	0	0	0	1	0	0
GCA_029842155.1	Environment	0	0	0	0	1	1	1	0	0	0	0	1	0
GCA_029840725.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016888805.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_018336855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_018281725.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016743155.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_900444875.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368765.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000831735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009001395.1	Clinical	0	1	0	0	1	1	0	0	1	1	1	0	0
GCA_016502415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009014175.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004055.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003957055.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009013435.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008979805.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031461105.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008984045.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008980005.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008999855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_030016975.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009014455.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008980735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027854915.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012375.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009013555.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025513885.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008999055.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030056035.1	Clinical	0	0	0	0	1	1	0	0	1	1	0	0	0
GCA_000775815.1	Clinical	0	1	0	0	1	1	0	0	1	1	1	0	0
GCA_009013895.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008988725.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_902374105.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025562355.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012295.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012455.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008996015.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009011915.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008988745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008981685.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004095.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004115.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008983685.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008981205.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012575.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_958369725.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963517235.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032480795.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001941805.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002165245.3	Environment	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_030665425.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_002761875.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021491935.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368665.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000430225.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_012271855.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004123275.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004296305.1		Environment	0	0	0	0	1	1	0	0	0	0	1	0	0
GCA_004921845.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000813495.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004920505.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293755.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293735.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293765.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019048765.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002328335.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031996545.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937927325.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016790125.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003484365.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027321245.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002387225.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002380985.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003452175.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023255055.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter junii	**Isolation**	**aadA2b**	**sul2**	**aph(6)-Id**	**aph(3'')-Ib**	**aac(3)-IId**	**aph(3')-VI**	**blaNDM-1**	**ant(2'')-Ia,**	**tet(G)**	**blaIMP-1**	**catB8**	**blaIMP-15**	**aadA1**	**blaCARB-2**	**tet(39)**
GCA_003939335.2	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839935.1	Environment	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0
GCA_003939355.2	CA	0	1	1	1	1	1	1	0	0	0	0	0	0	0	0
GCA_029841885.1	Environment	1	0	0	0	0	0	1	0	0	0	0	0	0	0	0
GCA_003939835.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_029843865.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003940305.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021384305.1	CA	0	0	0	0	1	0	1	0	0	0	0	0	0	0	0
GCA_029842155.1	Environment	1	0	0	0	0	1	1	1	1	0	0	0	0	1	0
GCA_029840725.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016888805.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_018336855.1	Clinical	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0
GCA_018281725.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016743155.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_900444875.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368765.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000831735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009001395.1	Clinical	0	0	0	0	0	1	0	0	0	1	1	0	0	0	0
GCA_016502415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009014175.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004055.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003957055.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009013435.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008979805.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031461105.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008984045.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008980005.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008999855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030016975.1	Clinical	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0
GCA_009014455.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008980735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027854915.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012375.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009013555.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025513885.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_008999055.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030056035.1		Clinical	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0
GCA_000775815.1		Clinical	0	0	0	0	0	1	0	1	0	1	1	0	1	0	0
GCA_009013895.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008988725.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_902374105.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025562355.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012295.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012455.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008996015.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009011915.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008988745.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008981685.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004095.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009004115.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008983685.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008981205.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012575.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368745.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_958369725.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963517235.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032480795.1		Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001941805.1		Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002165245.3		Environment	0	0	0	0	1	0	1	0	0	0	0	0	0	0	1
GCA_030665425.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002761875.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021491935.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368665.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000430225.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_012271855.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004123275.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004296305.1		Environment	0	1	1	1	0	0	1	0	0	0	0	0	0	0	0

GCA_004921845.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000813495.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004920505.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293755.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293735.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003293765.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019048765.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002328335.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031996545.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937927325.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016790125.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003484365.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027321245.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002387225.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002380985.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003452175.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023255055.1		Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter johnsonii	**Isolation**	**aph(3')-Via**	**aac(6')-Ib3**	**blaOXA-280**	**blaPER-1**	**msr(E)**	**mph(E)**	**floR**	**cmlB1**	**ARR-3**	**sul1**	**qacE**	**blaOXA-23**	**blaOXA-212**
GCA_000368805.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_004337595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	1
GCA_021496365.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_003952785.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030867405.1	Clinical	1	1	0	1	1	1	0	1	1	1	1	0	0
GCA_020162195.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_015602645.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_017068175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008180305.1	Environment	1	0	0	0	1	1	1	1	0	0	0	0	0
GCA_003335165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021432745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_021496345.1	Environment	0	0	0	0	1	0	0	0	0	0	0	0	1
GCA_021496295.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003939325.2	CA	1	1	0	0	1	1	0	0	1	1	1	0	0
GCA_001484935.1	Environment	1	1	0	1	1	1	0	1	1	1	1	0	0
GCA_900444855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000949655.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368045.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843525.1	environment.	1	1	0	0	1	1	0	0	1	1	1	0	1
GCA_029839745.1	environment.	1	1	0	0	1	1	0	0	1	1	1	0	0
GCA_029843655.1	environment.	1	1	0	0	1	1	0	0	1	1	1	0	0
GCA_900162715.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009828985.1	Clinical	0	0	0	0	1	1	1	0	0	0	0	0	1
GCA_009789205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029839405.1	environment.	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_031157845.1	Environment	0	0	0	0	1	1	0	1	0	0	0	0	0
GCA_029842765.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009823385.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842705.1	environment.	0	0	0	0	1	1	1	0	0	0	0	0	0
GCA_001483265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_033023695.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029842825.1	environment.	0	0	0	0	0	0	0	0	0	1	1	0	0
GCA_029841405.1	environment.	0	1	0	1	1	1	0	0	1	1	1	0	1
GCA_947498685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029845205.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839085.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029842835.1	environment.	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_029843465.1	environment.	0	0	0	0	1	1	0	0	0	0	0	0	1
GCA_029840405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_030325455.1	Clinical	0	0	0	0	1	1	1	0	0	0	0	0	1
GCA_029842585.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029840425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843005.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_029838425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004211815.1	Environment	0	0	0	0	1	1	1	1	0	0	0	0	0
GCA_001632325.1	Clinical	0	0	1		1	1	0	1	0	1	1	0	0
GCA_003940285.1	CA	1	1	0	1	1	1	0	0	1	1	1	0	1
GCA_029841175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027675825.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025717035.1	Environment	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_029841075.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841675.1	environment.	0	1	1	0	1	1	1	1	1	1	1	0	0
GCA_025513415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029836775.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032841755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021735885.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_009012975.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029844685.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000302335.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000301735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023822225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019334405.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029168455.1	Clinical	0	0	0	0	1	1	0	1	0	1	0	0	1
GCA_029168395.1	Clinical	1	0	0	0	1	1	1	0	0	1	1	0	0
GCA_028614115.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_028614535.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_013186005.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032078225.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_963528365.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963523135.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_031981085.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002333325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963517035.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_943787325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_946478025.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_963529475.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937872055.1	Environment	0	0	0	0	1	1	0	1	0	0	0	0	1
GCA_003523415.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_003241525.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_943912695.1	Clinical	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_950067465.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016790205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950068975.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031978345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003446165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_032102905.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002365955.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_937980675.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020741955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031999705.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_003240765.1	CA	0	0	0	0	1	1	0	0	0	0	0	0	0
GCA_950070875.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032084265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023257685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter johnsonii	**Isolation**	**tet(X3)**	**sul2**	**aph(6)-Id**	**aph(3'')-Ib**	**aac(3)-IId**	**aph(3')-VI**	**blaNDM-1**	**ant(2'')-Ia,**	**blaIMP-1**	**catB8**	**tet(39)**	**blaOXA-333**	**blaOXA-211**	**blaOXA-281**	**blaOXA-58**
GCA_000368805.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004337595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496365.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003952785.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0
GCA_021496325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_030867405.1	Clinical	0	1	0	0	1	1	1	0	0	0	1	0	0	1	1
GCA_020162195.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_015602645.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_017068175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008180305.1	Environment	0	1	1	0	0	0	1	0	0	0	0	0	0	0	1

GCA_003335165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021432745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_021496345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496295.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_003939325.2	CA	0	1	1	1	0	0	1	0	0	0	0	0	1	0	0
GCA_001484935.1	Environment	0	1	1	1	1	0	0	0	0	0	0	0	0	1	1
GCA_900444855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_000949655.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_000368045.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029843525.1	environment.	1	1	1	1	0	0	1	0	0	0	0	0	0	0	0
GCA_029839745.1	environment.	1	1	1	1	0	0	1	0	0	0	1	0	1	0	1
GCA_029843655.1	environment.	1	1	1	1	0	0	1	0	0	0	0	0	1	0	0
GCA_900162715.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009828985.1	Clinical	1	1	1	1	0	0	0	0	0	0	1	0	0	0	0
GCA_009789205.1	Environment	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0
GCA_029839405.1	environment.	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
GCA_031157845.1	Environment	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0
GCA_029842765.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_009823385.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029842705.1	environment.	0	1	1	1	0	0	0	0	0	0	1	0	1	0	0
GCA_001483265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_033023695.1	Environment	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0
GCA_029842825.1	environment.	0	0	0	0	0	0	0	1	0	1	0	0	0	0	0
GCA_029841405.1	environment.	0	1	1	1	0	1	1	0	0	0	1	0	0	0	0
GCA_947498685.1	Environment	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029845205.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029839085.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842835.1	environment.	0	0	0	0	0	0	0	0	0	0	1	0	1	0	1
GCA_029843465.1	environment.	0	0	0	0	0	1	1	0	0	0	1	0	0	0	1
GCA_029840405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030325455.1	Clinical	1	1	0	0	0	0	0	0	0	0	1	0	0	0	0
GCA_029842585.1	environment.	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0

GCA_029840425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029843005.1	environment.	0	1	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029838425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004211815.1	Environment	0	1	1	1	1	1	1	0	0	0	0	0	0	1	1
GCA_001632325.1	Clinical	0	0	1	1	0	0	0	0	0	0	0	0	0	1	1
GCA_003940285.1	CA	0	1	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_029841175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027675825.1	Clinical	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
GCA_025717035.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029841075.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029841675.1	environment.	0	1	1	1	0	0	1	0	0	0	0	0	0	0	0
GCA_025513415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_029836775.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032841755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_021735885.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012975.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029844685.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000302335.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_000301735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_023822225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019334405.1	Environment	0	0	1	1	0	1	1	0	0	0	0	0	0	0	0
GCA_029168455.1	Clinical	0	1	0	0	0	0	0	0	1	0	0	0	0	0	1
GCA_029168395.1	Clinical	0	1	1	1	1	0	0	0	1	0	0	0	0	0	1
GCA_028614115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_028614535.1	Environment	0	0	0	0	0	0	0	0	0	0	1	0	0	1	0
GCA_013186005.1	Environment	1	1	0	0	1	0	1	0	0	0	0	0	0	0	1
GCA_032078225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963528365.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_963523135.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031981085.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002333325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_963517035.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_943787325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_946478025.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963529475.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_937872055.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_003523415.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003241525.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_943912695.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950067465.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016790205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_950068975.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031978345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003446165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032102905.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002365955.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937980675.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020741955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031999705.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003240765.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_950070875.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032084265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023257685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter johnsonii	**Isolation**	**blaOXA-373**	**aph(3')-IIa**	**blaOXA-334**	**tet(X6)**	**aph(4)-Ia**	**aac(3)-IV**	**aph(3')-Ia**	**aadA24**	**aac(3)-Iia**	**tet(M)**	**blaPER-2**	**blaTEM-1B**	**ere(A)**	**blaOXA-309**
GCA_000368805.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004337595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496365.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003952785.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030867405.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020162195.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_015602645.1	CA	1	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_017068175.1	environment.	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008180305.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003335165.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021432745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496295.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003939325.2	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001484935.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_900444855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000949655.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368045.1	Clinical	0	1	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843525.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839745.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843655.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_900162715.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_009828985.1	Clinical	0	0	0	1	1	1	1	0	0	0	0	0	0	0
GCA_009789205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839405.1	environment.	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031157845.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842765.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009823385.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842705.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001483265.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_033023695.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842825.1	environment.	1	0	0	0	0	0	0	1	0	0	0	0	0	0
GCA_029841405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_947498685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029845205.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839085.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842835.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843465.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029840405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_030325455.1	Clinical	0	0	0	1	0	0	1	0	0	1	0	0	0	0
GCA_029842585.1	environment.	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029840425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843005.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029838425.1	environment.	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_004211815.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001632325.1	Clinical	0	0	0	0	0	0	0	0	1	0	1	1	1	0
GCA_003940285.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841175.1	environment.	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027675825.1	Clinical	0	0	1	0	0	0	1	0	0	0	0	0	0	0
GCA_025717035.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841075.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841675.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025513415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029836775.1	environment.	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_032841755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021735885.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012975.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029844685.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_000302335.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000301735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023822225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_019334405.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029168455.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029168395.1	Clinical	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_028614115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_028614535.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_013186005.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032078225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963528365.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963523135.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031981085.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0

GCA_002333325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963517035.1	Clinical	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_943787325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_946478025.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_963529475.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937872055.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003523415.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003241525.1	CA	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_943912695.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950067465.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_016790205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950068975.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_031978345.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_003446165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032102905.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002365955.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937980675.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020741955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031999705.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003240765.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950070875.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032084265.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_023257685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter johnsonii	**Isolation**	**blaOXA-235**	**blaOXA-299**	**tet(A)**	**aac(6')-Iia**	**aadA5**	**blaOXA 164**	**lnu(G)**	**mph(A)**	**blaZ**	**qnrVC6**	**tet(Y)**	**aph(3')-Vib**	**blaOXA 146**
GCA_000368805.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004337595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496365.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003952785.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030867405.1	Clinical	0	0	0	0	0	0	0	0	0	1	0	0	0

GCA_020162195.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_015602645.1	CA	0	0	0	0	0	0	0	0	0	0	0	0
GCA_017068175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_008180305.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003335165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021432745.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021496295.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003939325.2	CA	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001484935.1	Environment	0	0	0	0	0	0	0	0	0	1	0	0
GCA_900444855.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000949655.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368045.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843525.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839745.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843655.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_900162715.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009828985.1	Clinical	0	0	0	0	0	0	0	0	0	1	0	0
GCA_009789205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031157845.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842765.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009823385.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842705.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001483265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_033023695.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842825.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841405.1	environment.	0	0	0	0	0	0	0	0	0	0	1	0
GCA_947498685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029845205.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029839085.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842835.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0

GCA_029843465.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029840405.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030325455.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029842585.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029840425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029843005.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029838425.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004211815.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_001632325.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003940285.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	1
GCA_029841175.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_027675825.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025717035.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841075.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029841675.1	environment.	1	0	1	0	0	0	0	0	0	0	0	0	0
GCA_025513415.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029836775.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032841755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_021735885.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009012975.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_029844685.1	environment.	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000302335.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000301735.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023822225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019334405.1	Environment	0	1	1	0	0	0	0	0	0	0	0	0	0
GCA_029168455.1	Clinical	0	0	0	1	1	1	0	0	0	0	0	0	0
GCA_029168395.1	Clinical	0	0	0	1	1	1	0	0	0	0	0	0	0
GCA_028614115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_028614535.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_013186005.1	Environment	0	0	0	0	0	0	1	0	0	0	0	0	0
GCA_032078225.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963528365.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_963523135.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031981085.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002333325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963517035.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_943787325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_946478025.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963529475.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937872055.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003523415.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003241525.1	CA	0	0	0	0	0	0	0	1	1	0	0	0	0
GCA_943912695.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950067465.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_016790205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950068975.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031978345.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003446165.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032102905.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002365955.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_937980675.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020741955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031999705.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_003240765.1	CA	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_950070875.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032084265.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_023257685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter lwoffii	**Isolation**	**aph(3')-Via**	**aac(6')-Ib3**	**blaOXA-283**	**blaPER-1**	**, msr(E)**	**mph(E)**	**floR**	**cmlB1**	**ARR-3**	**sul1**	**qacE**	**blaOXA-362**	**blaOXA-282**	**blaOXA-58**
GCA_019343495.1	Environment	1	1	1	1	1	1	1	1	1	1	1	0	0	0
GCA_019787645.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_019787625.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_015602705.1	CE	0	0	0	0	0	0	0	0	0	0	0	0	1	0

GCA_013349125.1	Clinical	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_011058205.1	Environment	0	0	0	0	0	0	0	0	0	0	1	0	1
GCA 12393445.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129635.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA 24129855	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129435.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129115.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA 22967985	Clinical	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_024129715.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_024129685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_902363005.1	Clinical	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_024129355.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_014769185.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129735.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030678775.1	Environment	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_025514015.1	Clinical	0	0	0	0	0	0	0	0	0	1	0	0	0
GCA_025515075.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_025513955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030678755.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030678665.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_005924195.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_000369145.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000369125.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368165.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_000301755.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_004151415.2	CE	1	1	0	0	1	1	1	0	1	1	1	0	0	0
GCA_002119785.3	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_009829005.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963516025.1	Clinical	0	0	0	0	0	0	0	0	0	0	1	0	0
GCA_007678965.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002321025.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_963518635.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002332985.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_013530545.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_020742465.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_945638835.1	CE	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_943912775.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_031992985.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_963529775.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032087195.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0
GCA_003519025.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032106985.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032107325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032107605.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032098625.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	1	0
GCA_032013825.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Acinetobacter lwoffii	**Isolation**	**blaOXA-496**	**tet(39)**	**blaOXA-23**	**blaOXA-335**	**aadA2**	**blaOXA-285**	**sul2**	**blaOXA-361**	**aph(6)-Id**	**aph(3'')-Ib**	**aac(3)-IId**	**aph(3')-VI**	**blaOXA-235**	**blaNDM-1**	**blaOXA-134**	**blaOXA-146**
GCA_019343495.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019787645.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_019787625.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_015602705.1	CE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_013349125.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_011058205.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA 12393445.1	Clinical	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129635.1	Environment	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA 24129855	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129435.1	Environment	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129115.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA 22967985	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129715.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129685.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_902363005.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129355.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129595.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_014769185.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_024129735.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030678775.1	Environment	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025514015.1	Clinical	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0
GCA_025515075.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_025513955.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_030678755.1	Environment	0	1	1	0	0	1	0	0	0	0	0	0	0	0	0	0
GCA_030678665.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_005924195.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000369145.1	Clinical	0	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0
GCA_000369125.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_000368165.1	Clinical	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0
GCA_000301755.1	Clinical	0	0	0	0	0	1	1	0	1	1	0	0	0	0	0	0
GCA_004151415.2	CE	1	0	0	0	0	0	1	0	1	1	1	1	1	1	0	0
GCA_002119785.3	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_009829005.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963516025.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_007678965.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002321025.1	Environment	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963518635.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_002332985.1	Environment	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0
GCA_013530545.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_020742465.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_945638835.1	CE	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_943912775.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
GCA_031992985.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_963529775.1	Clinical	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032087195.1	Environment	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0

GCA_003519025.1	Clinical	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032106985.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032107325.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032107605.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032098625.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
GCA_032013825.1	Environment	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

Bibliography

AJ de Beaufort, AT Bernards, L Dijkshoorn, CPA van Boven. Acinetobacter junii causes life-threatening sepsis in preterm infants. Acta PaediatricaVolume 88, Issue 7 p. 772-775. 02 January 2007. https://doi.org/10.1111/j.1651-2227.1999.tb00041

Aguilar-Vera. A, Bello-López E, Iván Pantoja-Nuñez G, M. Rodríguez-López G, Morales-Erasto V, Castillo-Ramírez S. Acinetobacter junii: an emerging One Health pathogen.12 April 2024. mSphere 9:e00162-24. https://doi.org/10.1128/msphere.00162-24

Al-Agamy. Molecular resistance mechanisms to older antimicrobial agents in Escherichia coli isolates. J African Microbiol, 6, 106-111. 09 January 2012. https://doi.org/10.5897/AJMR11.1051

Alghamdi S. Isolation and identification of the oral bacteria and their characterization for bacteriocin production in the oral cavity. Saudi Journal of Biological Sciences. 2022. https://doi.org/10.1016/j.sjbs.2021.08.096

Andrey L. Rakitin, Alexandra Y. Ermakova, Alexey V. Beletsky, Mayya Petrova, Andrey V. Mardanov and Nikolai V. Ravin. Genome Analysis of Acinetobacter lwoffii Strains Isolated from Permafrost Soils Aged from 15 Thousand to 1.8 Million Years Revealed Their Close Relationships with Present-Day Environmental and Clinical Isolates.2021 Sep 4. Biology (Basel) . 2021 Sep 4;10(9):871. PMID: 34571748, PMCID: PMC8472584 https://doi: 10.3390/biology10090871

Arnold, S.J., Pfrender, M.E. & Jones, A.G. The adaptive landscape as a conceptual bridge between micro- and macroevolution. Genetica 112, 9–32 (2001). https://doi.org/10.1023/A:1013373907708

Boers SA, van der Reijden WA, Jansen R. High-throughput multilocus sequence typing: bringing molecular typing to the next level. PLoS One. 2012;7(7):e39630. Epub 2012 Jul 18. PMID: 22815712; PMCID: PMC3399827. https://doi.org/10.1371/journal.pone.0039630

Borer A, Saidel-Odes L, Riesenberg K, et al. Attributable Mortality Rate for Carbapenem-Resistant Klebsiella pneumoniae Bacteremia. Infection Control & Hospital Epidemiology. 2009;30(10):972-976. Infection Control & Hospital Epidemiology>Volume 30 Issue 10>Attributable Mortality Rate for Carbapenem-Resistant. https://doi:10.1086/605922

Broniek, G., Langwińska-Wośko, E., Szaflik, J. et al. Acinetobacter junii as an aetiological agent of corneal ulcer. Infection 42, 1051–1053 (2014). https://doi.org/10.1007/s15010-014-0647-8

Castillo-Ramírez S, Mateo-Estrada V, Gonzalez-Rocha G, Opazo-Capurro A. Phylogeographical Analyses and Antibiotic Resistance Genes of *Acinetobacter johnsonii* Highlight Its Clinical Relevance. mSphere. 2020 Jul 1;5(4):e00581-20. PMID: 32611704; PMCID: PMC7333577. https://doi: 10.1128/mSphere.00581-20.

Cleland EJ, Bassiouni A, Vreugde S, Wormald P-J. The Bacterial Microbiome in Chronic Rhinosinusitis: Richness, Diversity, Postoperative Changes, and Patient Outcomes. American Journal of Rhinology & Allergy. Am J Rhinol Allergy . 2016 Jan-Feb;30(1):37-43. PMID: 26867528. 2016;30(1):37-43. https://doi:10.2500/ajra.2016.30.4261

Dijkshoorn, L., Nemec, A. & Seifert, H. An increasing threat in hospitals: multidrug-resistant Acinetobacter baumannii. PMID: 18007677. Nat Rev Microbiol 5, 939–951 (2007). https://doi.org/10.1038/nrmicro1789

Doughari, H. J. (2012, January 28). Virulence, Resistance Genes, and Transformation Amongst Environmental Isolates of Escherichia coli and Acinetobacter spp. Journal of Microbiology and Biotechnology. Korean Society for Microbiology and Biotechnology. https://doi.org/10.4014/jmb.1107.07029

El Far SW, Abukhatwah MW. Prevalence of Aminoglycoside Resistance Genes in Clinical Isolates of Pseudomonas aeruginosa from Taif, Saudi Arabia—An Emergence Indicative Study. Journals Microorganisms Volume 11 Issue 9 10.3390/microorganisms11092293. https://doi.org/10.3390/microorganisms11092293

.

Feuerriegel S, Köser C, Niemann S, Phylogenetic polymorphisms in antibiotic resistance genes of the Mycobacterium tuberculosis complex, Journal of Antimicrobial Chemotherapy, Volume 69, Issue 5, May 2014, Pages 1205–1210 https://doi.org/10.1093/jac/dkt535

Gedefie A, Demsis W, Ashagrie M, Kassa Y, Tesfaye M, Tilahun M, Bisetegn H and Sahle Z. Acinetobacter baumannii Biofilm Formation and Its Role in Disease Pathogenesis: A Review.2021; Infect Drug Resist. 2021; 14: 3711–3719, PMCID: PMC8439624, PMID: 34531666 https://doi: 10.2147/IDR.S332051

Hamed S.M, Amira F.A. Hussein, Mohamed H. Al-Agamy, Hesham H. Radwan, Mai M. Zafer, Tn7382, a novel composite transposon harboring blaNDM-1 and aphA6 in Acinetobacter baumannii, Journal of Global Antimicrobial Resistance, Volume 30, 2022, Pages 414-417, ISSN 2213-7165, https://doi.org/10.1016/j.jgar.2022.08.001.

Houang ET, Sormunen RT, Lai L, Chan CY, Leong AS. Effect of desiccation on the ultrastructural appearances of Acinetobacter baumannii and Acinetobacter lwoffii. Journal of Clinical Pathology 1998;51:786-788.PMID: 10023344 PMCID: PMC500936. 1998 Oct. https://doi.org/10.1136/jcp.51.10.786

- Howard B, Bascom C.C, Hu. P, Wehmeyer K, Kimball AB, Isfort R.J. Aging-Associated Changes in the Adult Human Skin Microbiome and the Host Factors that Affect Skin Microbiome Composition. J Invest Dermatol. 2022 Jul;142(7):1934-1946.e21. PMID: 34890626. December 07, 2021.

  https://doi.org/10.1016/j.jid.2021.11.029

Hubeny J, Korzeniewska E, Buta-Hubeny M, ZieliÅ W, Rolbiecki D, Harnisz M, Characterization of carbapenem resistance in environmental samples and Acinetobacter spp. isolates from wastewater and river water in Poland, Science of The Total Environment, Volume 822, 2022, 153437, ISSN 0048-9697, https://doi.org/10.1016/j.scitotenv.2022.153437.

Hu H, Hu Y, Pan Y, Liang H, Wang H, Wang X, Hao Q, Yang X, Yang X, Xiao X, Luan C, Yang Y, Cui Y, Yang R, Gao GFSong Y, Zhu B.2012.Novel Plasmid and Its Variant Harboring both a blaNDM-1 Gene and Type IV Secretion System in Clinical Isolates of Acinetobacter lwoffii. Antimicrobial Chemotherapy Research Article. 27 October 20. https://doi.org/10.1128/aac.06199-11

Hrenovic J, Durn G, Goic-Barisic I, and Kovacic A. Appl Environ Microbiol. 2014 May; 80(9): 2860–2866. Occurrence of an Environmental Acinetobacter baumannii Strain Similar to a Clinical Isolate in Paleosol from Croatia. PMCID: PMC3993276.PMID: 24584245.
https://doi: 10.1128/AEM.00312-14

Hujer KM, Hujer AM, Hulten EA, Bajaksouzian S, Adams JM, Donskey CJ, Ecker DJ, Massire C, Eshoo MW, Sampath R, Thomson JM, Rather PN, Craft DW, Fishbain JT, Ewell AJ, Jacobs MR, Paterson DL, Bonomo RA2006.Analysis of Antibiotic Resistance Genes in Multidrug-Resistant Acinetobacter sp. Isolates from Military and Civilian Patients Treated at the Walter Reed Army Medical Center. AntimicrobAgents Chemother50.
https://doi.org/10.1128/aac.00778-06

Jiang Shiang S, Fan Q, Zhang Z , Deng Y, Dai Q, Wang J, Lin M, Zhou J, Long Z, He G, Zhou Z. Biodegradation of Oil by a Newly Isolated Strain Acinetobacter junii WCO-9 and Its Comparative Pan-Genome Analysis. PMCID: PMC9967506.PMID: 36838372.2023 Feb 6.
https://doi: 10.3390/microorganisms11020407

J R Patil, B A Chopade. Distribution and in vitro antimicrobial susceptibility of Acinetobacter species on skin of healthy humans.
Natl Med J India. 2001 Jul-Aug;14(4):204-8.
PMID: 11547525
https://pubmed.ncbi.nlm.nih.gov/11547525/

Kansal S1, Gupta G, Chouhan A, Mahajan S. An Extremely Rare Case of Asymptomatic Acinetobacter junii. J Microbil Infect Dis. September 2022;12(03):136-138.
https://doi:10.5799/jmid.1176559

Karshenas AE, Salehi TZ, Adabi M, Asghari B and Yahyaraeyat R. Prevalence of main quinolones and carbapenems resistance genes in clinical and veterinary Escherichia coli strains. Iran J Microbiol. 2022 Dec; 14(6): 841–849. https://doi: 10.18502/ijm.v14i6.11259

Kisková, J.; Juhás, A.; Galušková, S.; Maliničová, L.; Kolesárová, M.; Piknová, M.; Pristaš, P. Antibiotic Resistance and Genetic Variability of *Acinetobacter* spp. from Wastewater Treatment Plant in Kokšov-Bakša (Košice, Slovakia). *Microorganisms* **2023**, *11*, 840. https://doi.org/10.3390/microorganisms11040840

Kollimuttathuillam S, Bethel N, Shaaban H, Alexander Muacevic and John R Adler. A Case of Acinetobacter junii Cavitary Pneumonia with Bacteremia in a Patient With Systemic Lupus Erythematosus. Cureus. 2021 Nov; 13(11): e19711. 2021 Nov 18. PMCID: PMC8681891. PMID: 34976481. https://doi:10.7759/cureus.19711

Kumarasamy K.K, MPhil, Toleman MA , Walsh T.R, Bagaria J, Butt F, Balakrishnan R. Emergence of a new antibiotic resistance mechanism in India, Pakistan, and the UK: a molecular, biological, and epidemiological study. August 11, 2010. Lancet Infect Dis. 2010 Sep; 10(9): 597–602. PMCID: PMC2933358. PMID: 20705517. https://doi.org/10.1016/S1473-3099(10)70143-2

Li Jia , Li Yang , Cao Xiaoli , Zheng Jie , Zhang Yan , Xie Hui , Li Chuchu , Liu Chang , Shen Han. Genome-wide identification and oxacillinase OXA distribution characteristics of Acinetobacter spp. based on a global database, Frontiers in Microbiology, VOLUME=14, YEAR 2023 https://doi.org/10.3389/fmicb.2023.1174200

Linde H.J, Hahn J, Holler E, Reischl U and Lehn N. Septicemia Due to Acinetobacter junii. J Clin Microbiol. 2002 Jul; 40(7): 2696–2697. PMCID: PMC120562, PMID: 12089313. 2002 Jul.
https://doi: 10.1128/JCM.40.7.2696-2697.2002

Li X, He J,,Jiang Y,,Peng M,Yu Y, Fu Y,2021.Genetic Characterization and Passage Instability of a Hybrid Plasmid Co-Harboring blaIMP-4 and blaNDM-1 Reveal the Contribution of Insertion Sequences During Plasmid Formation and Evolution. Microbiol Spectr9:e01577 21.
https://doi.org/10.1128/Spectrum.01577-21

L.S. Jones, M.A. Toleman, J.L. Weeks, R.A. Howe, T.R. Walsh, K.K. Kumarasamy. Plasmid carriage of bla NDM-1 in clinical Acinetobacter baumannii isolates from India. Antimicrob Agents Chemother, 58 (2014), pp. 4211-4213
http://dx.doi.org/10.1128/AAC.02500-14

M.F. Elbadawy, W.M. Tawakol, S.W. Elfar, I.A. Maghrabi, S. Alghamdi, M.S. Mansy, M.S. Ashour, M.M. Shohayeb. Molecular identification of aminoglycoside-modifying enzymes and plasmid-mediated quinolone resistance genes among Klebsiella pneumoniae clinical isolates recovered from Egyptian patients. Int. J. Microbiol., 2017, p. 8050432, PMID: 28638412 PMCID: PMC5468591. 2017 May 30.
https://doi.org/10.1155/2017/8050432

Mahillon J, Chandler M1998.Insertion Sequences. Microbiol Mol Biol Rev62: PMID: 9729608 PMCID: PMC98933
https://doi.org/10.1128/mmbr.62.3.725-774.1998

Martino ME, Fasolato L, Montemurro F, Rosteghin M, Manfrin A, Patarnello T, Novelli E, Cardazzo B.2011.Determination of Microbial Diversity of Aeromonas Strains on the Basis of Multilocus Sequence Typing, Phenotype, and Presence of Putative Virulence Genes. Appl Environ Microbiol. 2011 Jul; 77(14): 4986–5000. PMCID: PMC3147379. PMID: 21642403. 2011
https://doi.org/10.1128/AEM.00708-11

Mendes SG, Combo SI, Allain T, Domingues S, Buret AG, Da Silva GJ. Co-regulation of biofilm formation and antimicrobial resistance in Acinetobacter baumannii: from mechanisms to therapeutic strategies. Eur J Clin Microbiol Infect Dis. 2023 Dec;42(12):1405-1423. Epub 2023 Oct 28. PMID: 37897520; PMCID: PMC10651561.
https://doi: 10.1007/s10096-023-04677-8.

Mindlin S, Petrenko A, Kurakov A, Beletsky A, Mardanov A, Petrova M, "Resistance of Permafrost and Modern Acinetobacter lwoffii Strains to Heavy Metals and Arsenic Revealed by Genome Analysis", BioMed Research International, vol. 2016, Article ID 3970831, 9 pages, 2016.
https://doi.org/10.1155/2016/3970831

Moffatt JH, Harper M, Mansell A, Crane B, Fitzsimons TC, Nation RL, Li J, Adler B, Boyce JD. Lipopolysaccharide-deficient Acinetobacter baumannii shows altered signaling through host Toll-like receptors and increased susceptibility to the host antimicrobial peptide LL-37. Infect Immun. 2013 Mar;81(3):684-9. Epub 2012 Dec 17. PMID: 23250952; PMCID: PMC3584870.
https://doi: 10.1128/IAI.01362-12

Montaña S, Sareda T. J. Schramm, Traglia M, Chiem K, Parmeciano Di Noto G, Almuzara M, Barberis C, Vay C, Quiroga C, Marcelo E. Tolmasky, Iriarte A, and Soledad Ramírez M. The Genetic Analysis of an Acinetobacter johnsonii Clinical Strain Evidenced the Presence of Horizontal Genetic Transfer. PLoS One . 2016 Aug 22;11(8):e0161528. 2016 Aug 22.

https://doi: 10.1371/journal.pone.0161528

Moosavian M, Ahmadi K, Shoja S, Mardaneh J, Shahi F, Afzali M, Antimicrobial resistance patterns and their encoding genes among clinical isolates of Acinetobacter baumannii in Ahvaz, Southwest Iran, MethodsX, Volume 7, 2020, 101031, ISSN 2215-0161, https://doi.org/10.1016/j.mex.2020.101031

Moreno E, Stackebrandt E, Dorsch M, Wolters J, Busch M, Mayer H.1990.Brucella abortus 16S rRNA and lipid A reveal a phylogenetic relationship with members of the alpha-2 subdivision of the class Proteobacteria. J Bacteriol. 1990 Jul;172(7):3569-76. PMID: 2113907, PMCID: PMC213329. 1990 Jul. https://doi.org/10.1128/jb.172.7.3569-3576.1990

Mónica P Gutiérrez-Gaitán, Andrés D Montoya-Moncada, José M Suescún-Vargas, Javier Y Pinzón-Salamanca, Brianna L Aguirre-Borrero. Emerging species in pediatrics: a case of Acinetobacter johnsonii meningitis. Bol Med Hosp Infant Mex 2022;79(1):51-55.PMID: 35086130 https://doi:10.24875/BMHIM.21000041

Narciso-da-Rocha C, Vaz-Moreira I, Svensson-Stadler L, Moore E.R.B and Manaia C.M. Diversity and antibiotic resistance of Acinetobacter spp. in water from the source to the tap. Springer-Verlag 2012.Appl Microbiol Biotechnol. 2013 Jan;97(1):329-40. PMID: 22669636. 6 June 2012. https://doi.org/10.1007/s00253-012-4190-1

Ouahrani S, Michaux S, Widada J, Bourg G, Tournebize R, Ramuz M and Liautard J. Identification and sequence analysis of IS6501, an insertion sequence in Brucella spp.: relationship between genomic structure and the number of IS6501 copies. Microbiology Volume 139, Issue 12. 01 December 1993. https://doi.org/10.1099/00221287-139-12-3265

Pandey, P.K., Verma, P., Kumar, H. et al. Comparative analysis of faecal microflora of healthy full-term Indian infants born with different methods of delivery (vaginal vs cesarean): Acinetobacter sp. prevalence in vaginally born infants. J Biosci 37 (Suppl 1), 989–998 (2012). PMID: 23151789. 2012 Dec. https://doi.org/10.1007/s12038-012-9268-5

Peleg A.Y, Seifert H and Paterson D.L . Acinetobacter baumannii: Emergence of a Successful Pathogen. Clin Microbiol Rev
. 2008 Jul;21(3):538-82, PMID: 18625687, PMCID: PMC2493088
https://doi: 10.1128/CMR.00058-07

Perewari, Otis D, Otokunefor, Kome, Agbagwa, Ejiro O, Tetracycline-Resistant Genes in Escherichia coli from Clinical and Nonclinical Sources in Rivers State, Nigeria, International Journal of Microbiology, 2022, 9192424, 5pages, 2022. PMID: 35855811, PMCID: PMC9288291. 2022 Jul 9.
https://doi.org/10.1155/2022/9192424

Pérez-Losada M, Crandall KA. Spatial diversity of the skin bacteriome. Front Microbiol. 2023 Sep 19;14:1257276. PMID: 37795302; PMCID: PMC10546022.
https://doi: 10.3389/fmicb.2023.1257276.

Pérez-Losada M, Cabezas P, Castro-Nallar E, Crandall K.A. Pathogen typing in the genomics era: MLST and the future of molecular epidemiology. Infection, Genetics and Evolution. Volume 16. 2013. Pages 38-53, ISSN 1567-1348. PMID: 23357583. Infect Genet Evol. 2013 Jun:16:38-53
https://doi.org/10.1016/j.meegid.2013.01.009

Picard B, Garcia JS, Gouriou S, Duriez P, Brahimi N, Bingen E, Elion J, Denamur E1999.The Link between Phylogeny and Virulence in Escherichia coli Extraintestinal Infection. Infect Immun67. PMID: 9916057, PMCID: PMC96353. 1999 Feb

https://doi.org/10.1128/iai.67.2.546-553.1999

Pillonetto M, Arend L, Vespero EC, Pelisson M, Chagas TP, Carvalho-Assef AP, Asensi MD. First report of NDM-1-producing Acinetobacter baumannii sequence type 25 in Brazil. Antimicrob Agents Chemother. 2014 Dec;58(12):7592-4. Epub 2014 Oct 6. PMID: 25288087; PMCID: PMC4249496.
http://doi: 10.1128/AAC.03444-14.
.

P J van den Broek,T J K van der Reijden, E van Strijen, A V Helmig-Schurter, A T Bernards, L Dijkshoorn. Endemic and epidemic Acinetobacter species in a university hospital: an 8-year survey.2009 Nov;47(11):3593-9. Epub 2009 Sep 30.
https://doi:10.1128/JCM.00967-09

Poirel, L. et al. Tn125-related acquisition of blaNDM-like genes in Acinetobacter baumannii. Antimicrobial agents and chemotherapy 56, 1087–1089, 2012.
https://doi:10.1128/aac.05620-11

Pulami D, KÃmpfer P, Stefanie P. Glaeser,
High diversity of the emerging pathogen Acinetobacter baumannii and other Acinetobacter spp. in raw manure, biogas plants digestates, and rural and urban wastewater treatment plants with system specific antimicrobial resistance profiles, Science of The Total Environment, Volume 859, Part 1, 2023, 160182, ISSN 0048-9697,
https://doi.org/10.1016/j.scitotenv.2022.160182

Pustijanac E, Hrenović J, Vranić-Ladavac M, Močenić M, Karčić N, Stefanović L.L, Hrstić I, Lončarić J, Šeruga Musić M, Drčelić M, Majstorović D. Dissemination of Clinical Acinetobacter baumannii Isolate to Hospital Environment during the COVID-19 Pandemic. PMID: 36986332 PMCID: PMC10057452. Pathogens
. 2023 Mar 3;12(3):410. 25 January 2023.

https://doi.org/10.3390/pathogens12030410

Rafei R, Dabboussi F, Hamze M, Eveillard M, Lemarié C, Mallat H, Rolain JM, Joly-Guillou ML, Kempf M. First report of blaNDM-1-producing Acinetobacter baumannii isolated in Lebanon from civilians wounded during the Syrian war. Int J Infect Dis. 2014 Apr;21:21-3. Epub 2014 Feb 19. PMID: 24560830. https://doi: 10.1016/j.ijid.2014.01.004

RANJBAR R, NAZARI S and FARAHANI O. Phylogenetic Analysis and Antimicrobial Resistance Profiles of Escherichia coli Strains Isolated from UTI-Suspected Patients. Iran J Public Health
. 2020 Sep; 49(9): 1743–1749. PMID: 33643950, PMCID: PMC7898090. 2020 Sep.
https://doi: 10.18502/ijph.v49i9.4094

Regalado N.G, Martin G, Antony S.J,
Acinetobacter lwoffii: Bacteremia associated with acute gastroenteritis,
Travel Medicine and Infectious Disease,
Volume 7, Issue 5, 2009, Pages 316-317, ISSN 1477-8939, PMID: 19747669. 2009 Sep.
https://doi.org/10.1016/j.tmaid.2009.06.001

Repizo G.D, Viale A.M, Borges V, Cameranesi M.M, Taib N, Espariz M, Brochier-Armanet C, Gomes J.P, Salcedo S.P, The Environmental *Acinetobacter baumannii* Isolate DSM30011 Reveals Clues into the Preantibiotic Era Genome Diversity, Virulence Potential, and Niche Range of a Predominant Nosocomial Pathogen, *Genome Biology and Evolution*, Volume 9, Issue 9, September 2017, Pages 2292-2307,
https://doi.org/10.1093/gbe/evx162

Revell L.J, Collar D.C, PHYLOGENETIC ANALYSIS OF THE EVOLUTIONARY CORRELATION USING LIKELIHOOD, Evolution, Volume 63, Issue 4, 1 April 2009, Pages 1090–1100
https://doi.org/10.1111/j.1558-5646.2009.00616.x

Rezatofighi, S.E., Mirzarazi, M. & Salehi, M. Virulence genes and phylogenetic groups of uropathogenic Escherichia coli isolates from patients with urinary tract infection and uninfected control subjects: a case-control study. BMC Infect Dis 21, 361 (2021). PMID: 33865334, PMCID: PMC8052790. 2021 Apr 17
https://doi.org/10.1186/s12879-021-06036-4

S.C. Ku, P.R. Hsueh, P.C. Yang, K.T. Luh. Clinical and Microbiological Characteristics of Bacteremia Caused by Acinetobacter lwoffii. Eur J Clin Microbiol Infect Dis (2000) 19 :501–505. Springer-Verlag 2000 .
http://doi: 10.1007/s100960000315

S. Fahy, J. A. O'Connor, B. Lucey and R. D. Sleator. Hospital Reservoirs of Multidrug Resistant Acinetobacter Species—The Elephant in the Room. 2023 Mar 20. PMCID: PMC10069268. PMID: 37020476.
https://doi: 10.3389/bjbs.2023.11098

S. Montaña, R.Cittadini, M.del Castillo, S.Uong, T.Lazzaro, M.Almuzara, C. Barberis, C. Vay, M.S. Ramírez. Presence of New Delhi metallo-β-lactamase gene (NDM-1) in a clinical isolate of Acinetobacter junii in Argentina. New Microbes New Infect. 2016 May; 11: 43–44. PMCID: PMC4877398
PMID: 27257491. 2016 Feb 23.
https://doi: 10.1016/j.nmni.2016.02.008

Schmitz FJ, Sadurski R, Kray A, Boos M, Geisel R, Köhrer K, Verhoef J, Ad C. Fluit, Prevalence of macrolide-resistance genes in Staphylococcus aureus and

Enterococcus faecium isolates from 24 European university hospitals, Journal of Antimicrobial Chemotherapy, Volume 45, Issue 6, June 2000, Pages 891–894, https://doi.org/10.1093/jac/45.6.891

Schwarz S, Kehrenberg C, Doublet B, Cloeckaert A, Molecular basis of bacterial resistance to chloramphenicol and florfenicol, FEMS Microbiology Reviews, Volume 28, Issue 5, November 2004, Pages 519–542, https://doi.org/10.1016/j.femsre.2004.04.001

Seifert H, Dijkshoorn L, Gerner-Smidt P, Pelzer N, Tjernberg I, Vaneechoutte M. Distribution of Acinetobacter species on human skin: comparison of phenotypic and genotypic identification methods. J Clin Microbiol. 1997 Nov;35(11):2819-25. PMID: 9350741; PMCID: PMC230069. http://doi: 10.1128/jcm.35.11.2819-2825.1997

Shadan A, Pathak A, Ma Y, Pathania R, Singh RP. Deciphering the virulence factors, regulation, and immune response to *Acinetobacter baumannii* infection. Front Cell Infect Microbiol. 2023 Feb 23;13:1053968. PMID: 36968113; PMCID: PMC10038080. https://doi: 10.3389/fcimb.2023.1053968.

SP Yavankar, KR Pardesi, BA Chopade. Species distribution and physiological characterization of Acinetobacter genospecies from healthy human skin of tribal population of India. 2007. Indian J Med Microbiol. 2007 Oct;25(4):336-45. PMID: 18087081. https://doi.org/10.1016/S0255-0857(21)02047-8

Tobin LA, Jarocki VM, Kenyon J, Drigo B, Donner E, Djordjevic SP, Hamidian M. Genomic analysis of diverse environmental *Acinetobacter* isolates identifies

plasmids, antibiotic resistance genes, and capsular polysaccharides shared with clinical strains. Appl Environ Microbiol. 2024 Feb 21;90(2):e0165423. Epub 2024 Jan 11. PMID: 38206028; PMCID: PMC10885009. https://doi: 10.1128/aem.01654-23.

Toleman MA, Spencer J, Jones L, Walsh TR. blaNDM-1 is a chimera likely constructed in Acinetobacter baumannii. Antimicrob Agents Chemother. 2012 May;56(5):2773-6. Epub 2012 Feb 6. PMID: 22314529; PMCID: PMC3346620. https://doi: 10.1128/AAC.06297-11

Rong Tang T, Chen W, Yi J, Yu Q, Hong Q, Shu W, Liu Q, Ll L, Cui Z. Antimicrobial resistances and clinical distributions of Acinetobacter junii and Acinetobacter lwoffii. Journal of Shanghai Jiaotong University(Medical Science) ; (12): 386-389, 2017.

Tsai, HY., Cheng, A., Liu, CY. *et al.* Bacteremia caused by *Acinetobacter junii* at a medical center in Taiwan, 2000–2010. *Eur J Clin Microbiol Infect Dis* **31**, 2737–2743 (2012). https://doi.org/10.1007/s10096-012-1622-x

Tsay MD , Tseng CC, Wu NX, Lai C. Size distribution and antibiotic-resistant characteristics of bacterial bioaerosol in intensive care unit before and during visits to patients. November 2020 Environment International. Volume 144(3):106024. https://doi:10.1016/j.envint.2020.106024

Vaz-Moreira.I, Olga C. Nunes, Célia M. Manaia, Bacterial diversity and antibiotic resistance in water habitats: searching the links with the human microbiome, FEMS Microbiology Reviews, Volume 38, Issue 4, July 2014, Pages 761–778, https://doi.org/10.1111/1574-6976.12062

Vázquez-López R, Hernández-Martínez T, Larios-Fernández SI, Piña-Leyva C, Lara-Lozano M, Guerrero-González T, Martínez-Bautista J, Gómez-Conde E, González-Barrios JA. Characterization of Beta-Lactam Resistome of Escherichia coli Causing Nosocomial Infections. Antibiotics (Basel). 2023 Aug 23;12(9):1355.
https://doi: 10.3390/antibiotics12091355

Vijayakumar S, Anandan S, Ms DP, Kanthan K, Vijayabaskar S, Kapil A, Ray P, Sistla S, Bhattacharya S, Wattal C, Thirunarayan, Deotale V, Mathur P, Walia K, Ohri VC, Veeraraghavan B. Insertion sequences and sequence types profile of clinical isolates of carbapenem-resistant A. baumannii collected across India over four year period. J Infect Public Health. 2020 Jul;13(7):1022-1028. Epub 2019 Dec 23. PMID: 31874816.
https://doi: 10.1016/j.jiph.2019.11.018

Wong D, Nielsen T.B, Bonomo R.A, Pantapalangkoor P, Luna B, Spellberg B. Clinical and Pathophysiological Overview of Acinetobacter Infections: a Century of Challenges. Clin Microbiol Rev
. 2017 Jan;30(1):409-447.PMID: 27974412 PMCID: PMC5217799
https://doi: 10.1128/CMR.00058-16

WHO publishes-list-of-bacteria-for-which-new-antibiotics- are-urgently-needed. News release GENEVA. 27 February 2017.

Wisplinghoff H, Bischoff T, Tallent S.M, Seifert H, Wenzel R.P, Edmond M.B. Nosocomial bloodstream infections in US hospitals: analysis of 24,179 cases from a prospective nationwide surveillance study. Clin Infect Dis 2004 Aug 1;39(3):309-17. PMID: 15306996. 2004 Oct.
https://doi: 10.1086/421946

Wu WFeng YTang GQiao F, McNally A, Zong Z2019.NDM Metallo-β-Lactamases and Their Bacterial Producers in Health Care Settings. Clin Microbiol Rev 32:10.1128/cmr.00115-18.
https://doi.org/10.1128/cmr.00115-18

Xiang T, Chen C, Wen J, Liu Y, Zhang Q, Cheng N, Wu X, Zhang W. Resistance of Klebsiella pneumoniae Strains Carrying blaNDM-1 Gene and the Genetic Environment of blaNDM-1. Front Microbiol. 2020 Apr 30;11:700. PMID: 32425903; PMCID: PMC7203411.
https://doi: 10.3389/fmicb.2020.00700.
.

XIE Shan, LIU Junxin, LI Lin, QIAO Chuanling. Biodegradation of malathion by Acinetobacter johnsonii MA19 and optimization of cometabolism substrates, Journal of Environmental Sciences,2008, ISSN 1001-0742.
https://doi.org/10.1016/S1001-0742(09)60014-0

Yasar Tas M , Merve Oguz M, Ceri M, "Acinetobacter lwoffii Peritonitis in a Patient on Automated Peritoneal Dialysis: A Case Report and Review of the Literature", Case Reports in Nephrology, vol. 2017, Article ID 5760254, 2 pages, 2017.
https://doi.org/10.1155/2017/5760254

Yong D, Toleman MA, Giske CG, Cho HS, Sundman K, Lee K, Walsh TR2009.Characterization of a New Metallo-β-Lactamase Gene, blaNDM-1, and a Novel Erythromycin Esterase Gene Carried on a Unique Genetic Structure in Klebsiella pneumoniae Sequence Type 14 from India . Antimicrob Agents Chemother53:.
https://doi.org/10.1128/aac.00774-09

Zong Z, Zhang X, blaNDM-1-carrying Acinetobacter johnsonii detected in hospital sewage, Journal of Antimicrobial Chemotherapy, Volume 68, Issue 5, May 2013, Pages 1007-1010, https://doi.org/10.1093/jac/dks505