Micro Biotec'13

PORTUGUESE CONGRESS OF MICROBIOLOGY AND BIOTECHNOLOGY

6th – 8th December | Aveiro Portugal

Abstracts Book

universidade de aveiro
um campus que pensa
1973_2013

SPM
Sociedade Portuguesa de Microbiologia

spbt
sociedade portuguesa de biotecnologia

Microbial Pathogenesis

P338/F09

DETECTION OF PREMATURE STOP CODONS LEADING TO TRUNCATED INTERNALIN A AMONG FOOD AND CLINICAL STRAINS OF *LISTERIA MONOCYTOGENES*

M. Ferreira da Silva[1]; V.Ferreira[2]; A. Correia[1]; P.Teixeira[2]

[1]*Department of Biology, University of Aveiro, 3810-193 Aveiro, Portugal*
[2]*CBQF – Centro de Biotecnologia e Química Fina, Laboratório Associado, Escola Superior de Biotecnologia, Universidade Católica Portuguesa/Porto, Porto, Portugal*

Listeria monocytogenes is a food-borne pathogen capable of causing listeriosis, a severe invasive disease and responsible for sporadic outbreaks. Internalin A protein (InlA) encoded by inlA has a key role in the infection mechanism, being necessary for L. monocytogenes to cross the intestinal barrier and to establish a systemic infection. Studies on inlA in L. monocytogenes have shown that mutations leading to premature stop codons (PMSC) occur naturally and are associated with attenuated virulence of this pathogen. The aim of this study was to evaluate the prevalence of PMSCs in inlA of eleven food and eleven clinical strains of L. monocytogenes from Portugal. The characterization was accomplished genetically by inlA sequencing to find mutations carrying PMSC leading to production of a truncated and secreted InlA. The PMSC were detected in three strains recovered from different food products (namely, from raw chicken, non-fermented sausage, and cheese), one strain recovered from a food-processing environment sample, and in one strain from a human clinical case. These strains belonged to different geno-serogroups, including: 1/2a-3a, 1/2b-3b-7 and 1/2c-3c. The mutations found lead to a predicted truncated InlA with 492, 576 and 685 amino acids, have been previously reported by other authors. The remaining seventeen strains tested did not show PMSC in inlA. Invasion efficiency of strains with PMSC in the human colorectal adenocarcinoma epithelial cell line (Caco-2 cells) was further evaluated. The results show a lower invasiveness efficiency of strains with PMSC in inlA compared to the positive control strain. InlA protein is necessary to cross the intestinal barrier, thus being a critical virulence factor promoting L. monocytogenes infection. When inlA shows PMSC leading to a truncated form of InlA, the isolate has reduced invasion efficiency. Understanding the importance of attenuated-virulence strains with PMSC in inlA, occurring mainly in foods, could be explored to prevent risk of human disease. Despite the important results obtained by Caco-2 invasion assays, it is necessary to study other in vitro and animal models to understand the pathophysiology of listeriosis in vivo.