ANALYSIS OF VIRULENCE FACTORS IN ISOLATES OF OPPORTUNISTIC PATHOGEN *STENOTROPHOMONAS MALTOPHILIA*

Radvilė Drevinskaitė[1], Laurita Klimkaitė[1], Jūratė Skerniškytė[1], Julija Armalytė[1]

[1]Institute of Biosciences, Life Sciences Center, Vilnius University, Vilnius, Lithuania
radvile.drevinskaite@gmc.stud.vu.lt

Stenotrophomonas maltophilia is an aerobic gram-negative bacterium that is widespread in the natural environment including soil, plants, and water sources. This bacterium of environmental origin is becoming an important opportunistic, nosocomial, multidrug-resistant pathogen associated with respiratory, bloodstream and urinary tract infections. Due to *S. maltophilia* innate resistance to various classes of antibiotics infections caused by this bacterium are difficult to treat and have high mortality rates of up to 69% [1]. Despite the wide range of clinical diseases associated with *S. maltophilia*, little information is available on the virulence factors of this bacterium. In addition to antibiotic resistance, the biofilm formation is considered a key virulence factor for *S. maltophilia* [2]. Secreted enzymes such as proteases, lipases and nucleases are thought to play an important role in *S. maltophilia* virulence, contributing to its ability to invade host tissues and degrade host components [3].

The aim of this study was to investigate the presence of genes encoding selected virulence determinants in 34 clinical and 43 natural isolates of *Stenotrophomonas maltophilia* from Lithuania. All clinical isolates were isolated from patients. Environmental isolates were isolated from various sources such as soil, water bodies and fish gut.

Genes encoding extracellular enzymes, toxins, components of secretion systems and iron uptake systems were selected for the detection of virulence factors. The analysis showed that the prevalence of most of the genes studied is similar in *S. maltophilia* isolates of natural and clinical origin. However, genes *stmPr1*, *afaD* and *zot*, encoding putative protease, adhesin and toxin respectively, were found to be exclusively present in isolates of clinical origin. The higher prevalence of virulence genes detected in clinical *S. maltophilia* isolates may be related to their importance in contributing to the ability of the bacterium to infect the host, to degrade various host components and to survive in clinical settings.

Acknowledgements: This research has received funding from the Research Council of Lithuania (LMTLT), project registration no. P-SV-23-156.

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