IDENTIFICATION OF THE TOXIN-ANTITOXIN SYSTEMS IN THE OPPORTUNISTIC PATHOGEN *STENOTROPHOMONAS MALTOPHILIA*

Laurita Klimkaitė, Julija Armalytė, Edita Sužiedėlienė

Institute of Biosciences, Life Sciences Center, Vilnius University, Vilnius, Lithuania
lauritaklimkaite@gmail.com

Bacterial toxin-antitoxin (TA) systems are small genetic elements, coding a stable protein toxin and an unstable molecule, neutralizing its toxic effect – antitoxin. In stress conditions (e.g. starvation, antibiotic pressure, host immune system attack) the unstable antitoxin molecule is degraded and the toxin inhibits the main cellular processes – DNA replication or protein, cell wall, ATP synthesis [1]. TA systems are potentially associated with the virulence traits of pathogenic bacteria, such as persistence, biofilm formation, host colonization [2]. Furthermore, TA systems are proposed as new targets for antimicrobial therapy especially important for the multidrug-resistant pathogen treatment [3].

Stenotrophomonas maltophilia is an environmental bacterium found in aqueous habitats, the rhizosphere of plants, on animals, in foods. In clinical settings *S. maltophilia* is known as an opportunistic multidrug-resistant nosocomial pathogen causing respiratory tract, bloodstream, urinary tract infections [4]. At present there is no information about the TA systems of this pathogen, thus our goal is to identify and characterize the TA systems of *S. maltophilia.*

Bioinformatic analysis was performed on 21 *S. maltophilia* genome sequences available to this date and 50 putative TA systems were predicted. 7 genes pairs best matching TA systems criteria were selected for further analysis. All selected TA systems were detected in clinical or environmental *S. maltophilia* isolates from laboratory collection. Interestingly, the frequency and spread of detected TA systems differed from bioinformatic analysis predictions. Detection results showed that several selected TA systems are present only in clinical *S. maltophilia* bacteria and are not found in environmental *S. maltophilia* isolates.

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[3] Williams, J. J. & Hergenrother, P. J. Artificial activation of toxin–antitoxin systems as an antibacterial strategy. *Trends Microbiol.* 20, 291–298 (2012).
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