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THE ROLE OF CAPSULAR POLYSACCHARIDES AND OUTER MEMBRANE VESICLES IN THE PATHOGENESIS OF OPPORTUNISTIC PATHOGEN ACINETOBACTER **BAUMANNII**

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Acinetobacter baumannii is considered one of the most crucial opportunistic pathogens, causing various medical-related infections worldwide. It commonly presents resistance to multiple antimicrobial agents, and hence, it is considered as a multidrug-resistant [1]. This opportunistic pathogen possesses multiple virulence factors, which contribute to the bacterial survival during the stress. A. baumannii ability to form biofilms on abiotic surfaces, such as catheters and endotracheal tubes, poses a great threat to the immunocompromised patients, therefore a better insight into A. baumannii pathogenesis could enhance the treatment of such individuals. Bacteria in biofilms can be 10-1,000 times less susceptible to various antimicrobial agents compared to planktonic bacteria [2]. The goal of this research is to investigate the impact of capsular polysaccharides and outer membrane vesicles produced by clinical A. baumannii isolates on biofilm formation and resistance under stress conditions, such as exposure to antimicrobial agents. The virulence properties of A. baumannii isolates and their $\Delta galU$ and $\Delta ompA$ mutants were assessed in this research. The deletion of galU gene provides capsule-less phenotype, whereas ompA knockout leads to a hyperproduction of outer membrane vesicles. Biofilm formation was evaluated by crystal violet staining, which revealed significant differences between mutant and wild-type isolates. The ability of various antimicrobial compounds to inhibit A. baumannii biofilms was tested. The results showed that the resistance of mutant isolates' biofilms was compromised in most cases compared to their wild-type isolates. Quantitative analysis demonstrated, that capsule production can alter the survival of A. baumannii in biofilms after exposure to antimicrobial agents.

^[1] Michalopoulos, A. and Falagas, M. E. (2010). Treatment of Acinetobacter infections. Expert opinion on pharmacotherapy, 11(5), 779-788.

^[2] Davies, D. (2003). Understanding biofilm resistance to antibacterial agents. Nature Reviews Drug Discovery 2, 114-122