

Cellular Microbiology and Pathogenesis

P-058 - BIOFILM PHENOTYPE POTENTIATES VIRULENCE TRANSDUCTION IN ACINETOBACTER BAUMANNII

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Background

An alarming increase of bacterial resistance to antibiotics has occurred recently. Many factors contribute to this, mainly antibiotics misuse but also an intrinsic capacity of bacteria to trade genetic material. These exchanges are often prophage-mediated. Prophages are bacteriophages that integrate into the bacterial genome, being able to excise and enter other bacteria. They are found in many bacterial species, being particularly frequent in *Acinetobacter baumannii*, an important multidrug-resistant nosocomial pathogen. This species is known to produce biofilms; these protect bacteria against antibiotics and host defenses and are responsible for bacteria persistence in clinical environments. This work aimed at evaluating the contribution of biofilms for virulence spread among *A. baumannii* strains.

Method

Transduction was evaluated in both planktonic and biofilm cultures. A donor strain (ANC 4097) containing a prophage coding for a beta-lactam resistance gene, and a receptor strain (NIPH 146) susceptible to beta-lactams, were chosen for the assays. To distinguish strains, NIPH 146 was modified with an erythromycin-resistant plasmid, antibiotic to which the donor strain was susceptible. For transduction assays in both planktonic and biofilm cultures, strains were inoculated together or sequentially (receptor strain first), and transducing phenomena was evaluated at different time points of growth or biofilm formation; the effect of antibiotic pressure was evaluated using sub-minimal inhibitory concentrations of meropenem. Transduction was evaluated by plating cells in agar plates (to count all viable cells), and agar supplemented with meropenem (donor and transduced receptor strains), erythromycin (receptor strain), and meropenem+erythromycin (transduced receptor strain).

Results & Conclusions

Transduction occurred only in biofilms, being higher for cells subjected to antibiotic pressure (36% transduced cells compared to 14% in the absence of selective pressure). Furthermore, the transduced receptor cells displayed an increased resistance to meropenem and other beta-lactams, with levels similar to the donor strain, revealing an important effect of prophages in the bacterial phenotype. This work reveals that biofilms contribute to the spread of virulence via transduction among *A. baumannii*, probably by exerting a protective environment to the prophages that are exited upon exposure to antibiotics.

References & Acknowledgments

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