Poster presentation
Biofilm

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Biological cost of antibiotic pressure in *Pseudomonas aeruginosa* biofilm phenotype

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Objectives: *P. aeruginosa* (PA) has become recognized as an opportunistic pathogen of clinical relevance mainly when encased in biofilms. Bacteria facing adverse conditions (e.g. in the course of antibiotic chemotherapy) can survive, multiply and form biofilms as a defensive tactic. This work aimed to evaluate the role of antibiotic pressure in biofilm phenotype and in the selection of persisters.

Methods: *P. aeruginosa* from collections (PAO, ATCC, CGCT) and isolated from an endoscope (PAI) were used to form biofilms in the presence of several doses of rifampicin (RIF) and ciprofloxacin (CIP). Biofilm phenotype was evaluated by biomass (CV), respiratory activity (XTT) and number of biofilm-grown cells. MIC and MBC of RIF and CIP against all bacterial strains were assessed.

Results: For all strains, RIF MIC was 32 µg/ml and MBC 64 µg/ml. MIC and MBC of CIP were strain dependent, varying from, respectively, 0.1-0.4 µg/ml and 0.8-3 µg/ml. However, *P. aeruginosa* strains did not loose the ability to form biofilms even when facing extreme doses of CIP and RIF. Biofilm mass was only altered at MIC value, being noticed a big reduction but not total inability of all strains to form biofilms. PAI and PAO were the strains more able to form biofilms even at excessive doses of RIF (64 µg/ml) and CIP (6 µg/ml). Concerning activity, biofilms developed by bacteria that survive in the presence of both antibiotics still able to colonize surfaces, giving rise to metabolically active biofilms also at MBC and 2×MBC. PAI, ATCC and CGCT were the strains that give rise to more active biofilms in CIP and PAI, ATCC and CGCT in RIF. Despite having a good reduction in activity and a reasonable reduction in biofilm mass, results are still worrisome as the number of viable biofilm-cells was very high. There was only a 2-log reduction at MIC and a 3-log reduction at MBC for both antibiotics, being the final cell number around 1E4 CFU/cm^2.

Conclusions: The presence of antibiotics during biofilm formation seems to enforce a selective pressure on bacteria members, eliminating planktonic cells, slowing down biofilm formation and interfering with biofilm phenotype. But cells that survived to antibiotic attack by surface adhesion were the ones among the communities that were more resistant to antimicrobials. These cells may give rise to phenotypically altered biofilms with increasing virulence factors and be the cause of persistent biofilm infections.