28th ECCMID EUROPEAN CONGRESS OF CLINICAL MICROBIOLOGY AND INFECTIOUS DISEASES
Madrid, Spain
21 – 24 April 2018

P1461 Antibiotic efficacy against Mycobacterium abscessus and Pseudomonas aeruginosa dual species biofilm formation on abiotic and biotic surfaces

Graciela Rodríguez Sevilla[1], Charlotte Rigauts[2], Eva Vandeplassche[2], Lisa Ostyn[2], Ignacio Mahillo Fernández[3], Jaime Esteban-Moreno*[1], Concepción Pérez-Jorge[1], Tom Coenye[2], Aurélie Crabbé[2]

[1]IIS- Fundación Jiménez Díaz, Clinical Microbiology, Madrid, Spain, *[2]Ghent University, Laboratory of Pharmaceutical Microbiology, Gent, Belgium*, *[3]IIS- Fundación Jiménez Díaz, Epidemiology, Madrid, Spain*

Background: *Mycobacterium abscessus* lung infection has become a major health problem for cystic fibrosis patients. The antibiotic therapy is chosen according to susceptibility testing results; however, the treatment is long, poorly tolerated and unpredictable. To understand the *in vivo* factors that may influence the outcome of a therapy, mimicking both the *in vivo* bacterial phenotype and the host microenvironment is needed. Our objective is to evaluate the effect of antibiotics in *Mycobacterium abscessus* biofilm formation in the presence of i) another major CF pathogen (i.e. *Pseudomonas aeruginosa)* and ii) *in vivo*-like 3-D lung epithelial cells.

Materials/methods: Dual species biofilms of *M. abscessus* and *P. aeruginosa* (1:1) clinical isolates were cultured on a plastic surface or on 3-D lung epithelial cells. Inhibition of single and dual species biofilms by antibiotics was assessed based on the number of bacteria (CFU/mL) associated with the 3-D cells or with the plastic surface after static incubation for 6 hours, determined after plating serial dilutions on selective media. Antibiotics used were amikacin (17 µg/mL), clarithromycin (1,1 µg/mL), colistin (2 µg/mL), and ceftazidime (256 µg/mL).

Results: None of the antibiotics inhibited *M. abscessus* biofilm formation, while *P. aeruginosa* biofilm formation was effectively inhibited in both models by most antibiotics. This resulted in a competitive advantage for *M. abscessus* in dual species biofilms for all tested antibiotics on plastic and 3-D cells, except for clarithromycin in the latter. Indeed, 3-D lung cells completely abolished the efficacy of clarithromycin against *P. aeruginosa* single and dual species biofilms. In addition, induction of *M. abscessus* single species biofilm formation was observed for all antibiotics when grown on plastic but not on 3-D cells. Interactions with *P. aeruginosa* affected the observed *M. abscessus* biofilm induction in the presence of ceftazidime.

Conclusions: Our results suggest a role of interspecies interactions and/or host epithelial cells in bacterial response to antibiotics, and highlight the importance of developing models that predict *in vivo* outcomes.