MOLECULAR CROSS-TALK BETWEEN *Burkholderia cenocepacia* AND HOST CELLS

Gibaldi, D.; Neves, B. C.; Mendonça-Previato, L.; Previato, J. O. *
Laboratório de Glicobiologia, Instituto de Biofísica Carlos Chagas Filho, UFRJ.
* previato@biof.ufrj.br

Chronic bacterial colonization of airways is the major cause of morbidity and mortality within cystic fibrosis patients. Infections with *Burkholderia cenocepacia*, a phytopathogen that shows multi-resistance to antibiotics, exhibit a variable clinical course ranging from asymptomatic carriage to lethal acute necrotizing pneumonia and septicaemia. *B. cenocepacia* is an intracellular pathogen producing several virulence factors, including a type III secretion system that is the most prominent. This system promotes the translocation of effector proteins into host cells, in which they subvert signalling pathways, modulate cell functions (e.g immune cells). Production of oxygen and nitrogen reactive intermediates is the major innate response against bacterial infections. Our data demonstrate a negative modulation of macrophage response by live *B. cenocepacia*. Infected murine macrophages with either wild-type or type III mutant does not release NO. On the other hand, murine macrophages treated with heat-inactivated *B. cenocepacia* displayed a similar response to that of LPS-treated cultures. Furthermore, the release of cytokines was the same in cultures with either type III mutant or wild-type strains. Thus, our results demonstrate that the macrophage-modulation effects by *B. cenocepacia* are not mediated by a functional type III system. Our study is currently focused on the signalling pathways involved and a CF murine model will be used to further assess *B. cenocepacia*-host cell molecular cross-talk.