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Metabolomics (Los Angel) 2018, Volume 8
DOI: 10.4172/2153-0769-C2-044

13th International Conference on

Metabolomics and Systems Biology

October 11-12, 2018 | Zurich, Switzerland

Metabolic reprogramming of macrophages exposed to *Pseudomonas aeruginosa* biofilms

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Currently, the annual economic cost of chronic wounds exceeds $1 billion in the United States, and the incidence of non-healing human wounds is expected to dramatically increase in the next several years due to emerging Type 2 diabetes epidemics. Several common characteristics typical of chronic wounds include tissue colonization by persistent antibiotic-resistant pathogenic microbial biofilms, excessive inflammation, and failure of human cells to resolve the wound. *Pseudomonas aeruginosa* is one of the predominant opportunistic pathogens that colonizes greater than 50% of all chronic wounds in the US and is a serious health threat. To better understand the molecular processes by which *P. aeruginosa* biofilms interfere with human macrophage immune responses, we have undertaken nuclear magnetic resonance (NMR)-based metabolomics studies of activated and resting macrophages. The studies aim to probe the metabolic reprogramming of these immune cells as result of exposure to secreted molecules produced by *P. aeruginosa* biofilms, using an *in vitro* host-pathogen co-culture model. Herein, we present our recent NMR-based metabolomics results demonstrating the presence of a significant metabolic shift between different macrophage phenotypes. This metabolic phenotyping is correlated with fluorescence-activated cell sorting (FACS) analysis which has been employed to characterize the relationship between metabolic profiles and macrophage immunomodulation (i.e. macrophage polarization into pro- or anti-inflammatory M1 or M2 subpopulations, respectively), resulting from macrophage exposure to secreted molecules from co-culture *P. aeruginosa* biofilms.

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