47346
Tyndallized bacteria prime bronchial epithelial cells to mount an effective innate immune response against infections

Epithelial cell, Inflammation, Immunology

S. Di Vincenzo[1], C. Di Sano[1], C. D'Anna[1], M. Ferraro[1], V. Malizia[1], A. Bruno[1], M. Cristaldi[2], C. Cipollina[2], V. Lazzara[3], P. Pinto[4], S. La Grutta[1], E. Pace[1]

[1]Institute of Translational Pharmacology (IFT) - National Research Council (CNR) - Palermo (PA) (Italy), [2]Rimed Foundation - Palermo (PA) (Italy), [3]Dipartimento di Scienze Economiche, Aziendali e Statistiche - Università degli Studi di Palermo - Palermo (PA) (Italy), [4]Department of Public Health, Experimental and Forensic Medicine, University of Pavia - Pavia (Italy)

Airway epithelium represents a physical barrier against toxic substances and pathogens but also presents pattern recognition receptors on the epithelial cells that detect pathogens leading to molecule release and sending signals that activate both the innate and adaptive immune responses. Thus, impaired airway epithelial function and poor integrity may increase the recurrence of infections. Probiotic use in respiratory diseases as adjuvant of traditional therapy is increasingly widespread. There is growing interest in the use of non-viable heat-killed bacteria, such as tyndallized bacteria (TB), due to safety concerns and to their immunomodulatory properties.

This study explores in vitro the effects of a TB blend on the immune activation of airway epithelium. A bronchial epithelial cell line, 16HBE, was exposed to different concentrations of TB. Cell viability, TB internalization, TLR2 protein expression, IL-6, IL-8 and TGF-β1 gene expression and release, E-cadherin expression and wound healing were assessed. We found that TB were well tolerated, internalized, increased TLR2 and E-cadherin expression, increased IL-6 release and wound healing but decreased both IL-8 and TGF-β1 release.

In conclusion, TB activate TLR2 pathway without inducing a relevant pro-inflammatory response and improve barrier function. In this way, TB preserve epithelial homeostasis and could be used as strategy to prevent and to manage respiratory infection, exacerbations included.