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SYNERGISTIC LUNG TOXICITY OF PM2.5 AND ENNIATIN B1 IN A CO-CULTURE MODEL

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In real-world scenarios, the human respiratory system is exposed to multiple pollutants. Fine particulate matter (PM2.5) and emerging mycotoxins like Enniatin B1 (ENN B1), currently not regulated, represent significant environmental and indoor stressors. However, their combined impact on the respiratory barrier remains poorly understood. This study investigates whether priming the lung epithelium with PM2.5 activates the immune-epithelial responses to ENN B1 exposure.

An in vitro human lung co-culture (A549 epithelial cells and differentiated THP-1 macrophages, 10:1 ratio) was established at the Air-Liquid Interface (ALI). The protocol consisted of a 72-hour pre-activation of A549 cells with PM2.5 (2.5 µg/cm²). Following this, dTHP-1 cells were integrated (1:10 ratio), and the system was sequentially exposed to ENN B1 (50 ng/cm²) for 24 hours. Cytotoxicity and pro-inflammatory responses (IL-6, IL-8) were quantified via LDH and ELISA, respectively. Mechanistic insights into inflammation, oxidative stress, xenobiotic metabolism, and cell fate are being elucidated through RT-qPCR.

Our findings reveal a significant synergistic effect. While individual exposures to PM2.5 and ENN B1 induced moderate cytotoxicity, the sequential exposure led to a significant decrease in cell viability and an increase in IL-8 secretion, when compared to single-treatment groups. This suggests that PM2.5-induced pre-activation determines a status that may amplify or may be amplified by secondary stressors. Gene expression analysis will elucidate the specific pathways involved in this synergistic interaction.

The study demonstrates that PM2.5 might act as a priming agent, exacerbating the toxicological profile of ENN B1, strengthening the importance of investigating more deeply the relationship between air pollutants and bioaerosol in environmental health sciences.