Micro- and nanoplastic interference in THP-1 immune response in an inflammatory context

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Micro- and nanoplastics (MNPLs) are increasingly recognized as threatening environmental pollutants. Their low density and small mass facilitate airborne transport, creating a potential risk to human health as these particles can be inhaled and subsequently accumulate in lung tissue. There, MNPLs harmful effects disclose special threat in inflammatory processes and long-term exposure scenarios.

In vitro human-relevant models, such as the bronchial Calu-3 air–liquid interface (ALI) system, are widely used to evaluate the harmful potential of MNPLs at primary biological barriers. Under ALI conditions, epithelial cells undergo differentiation, produce mucus and form tight junctions, creating a structure that closely mimics the human airway epithelium while also supporting long-term exposure studies. In this context, macrophages play a major role in directing the immune-response and particle clearance.

Understanding MNPLs impact on macrophages is crucial to advance on the development of new strategies to evaluate toxicological risks using the co-culture system. Thereby, in this work we assessed inflammatory-response interference of poly lactic acid (PLA), polyethylene terephthalate (PET) and polytetrafluoroethylene (PTFE, Teflon) MNPLs in THP-1 that have already undergone inflammatory differentiation, as happens in bronchial macrophage recruitment. Ongoing work is focused on evaluation of inflammatory endpoints including proinflammatory cytokine secretion, mitochondrial membrane potential disruption and inflammatory gene expression at short- and long-term timepoints.

Preliminary and ongoing data suggest an increased activation of inflammatory pathways due to the exposure. The in-depth characterization of such impact induced by the diverse real-life polymers tested will contribute to the knowledge base of MNPLs health risk.

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