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Microplastics as emerging environmental cofactors in autoimmune rheumatic diseases

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Introduction: Microplastics (MPs) are ubiquitous environmental particles with emerging immunotoxic and pro-inflammatory potential. Their persistence in water, food chains and air leads to continuous low-dose human exposure. Increasing evidence suggests MPs may act as environmental cofactors capable of triggering immune dysregulation, oxidative stress and chronic low-grade inflammation. These mechanisms raise concern that MPs could contribute to the initiation or exacerbation of autoimmune inflammation.

The study aims to synthesise peer-reviewed evidence on MPs exposure and its potential role in the development and worsening of autoimmune pathology, with a focus on rheumatic diseases and their underlying immunopathogenic mechanisms.

Material and methods: Narrative review based on structured searches of PubMed/MEDLINE, Scopus and Web of Science, enhanced by manual reference screening. Experimental, translational and clinical studies evaluating MPs exposure in relation to rheumatic outcomes were qualitatively integrated. Exclusion criteria included non-peer-reviewed publications, conference abstracts, and studies lacking immune or clinical outcome assessment.

Results: In murine models, oral MPs exposure induced lupus-like manifestations in C57BL/6 mice and aggravated spontaneous lupus in MRL/lpr mice. These effects were associated with expansion of splenic double-negative T cells and plasma cells, increased anti-dsDNA and anti-nuclear antibodies titres, elevated interleukin-6 and tumour necrosis factor levels, and renal injury with proteomic signatures consistent with lupus-associated pathways and complement-mediated damage. In rheumatoid arthritis models, MPs were internalised by fibroblast-like synoviocytes, enhancing proliferation, migration and invasion, increasing inflammatory mediator release, and promoting cartilage damage.

Discussion: Across experimental systems, MPs appear to function as pro-inflammatory adjuvant-like particles, activating stromal effector cells, amplifying cytokine and protease cascades, and potentially enhancing complement-driven tissue injury. However, human causality remains uncertain due to heterogeneity in exposure assessment, particle composition and size variability, and predominance of high-dose experimental models.

Conclusions: Current evidence, largely preclinical, supports a plausible role of MPs in promoting initiation and progression of autoimmune rheumatic phenotypes. Standardised exposure assessment, biomonitoring strategies and prospective human studies are required to define attributable risk and identify preventive public health interventions.