

Comments on: Micro- and nanoplastics and the kidney: exposure pathways, toxicokinetics, and pathophysiological insights

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We read with great interest the recent article by Lombardi and Bergesio regarding the nephrotoxic effects of micro- and nanoplastics (MNPs) and their potential role as emerging environmental determinants of kidney disease. The authors comprehensively describe the mechanisms of renal injury induced by MNP exposure, including oxidative stress, mitochondrial dysfunction, inflammatory activation, ferroptosis, and tubulo-interstitial fibrosis. These observations are particularly relevant in the context of the growing burden of environmentally mediated chronic diseases (1).

Beyond renal toxicity, we believe that the clinical implications of MNP exposure should also be interpreted within the broader framework of the cardio-reno-metabolic syndrome (CRMS), recently recognized as a pathophysiological continuum linking cardiovascular disease, chronic kidney disease, metabolic dysfunction, and systemic inflammation (2). Indeed, the biological mechanisms activated by MNPs appear to overlap significantly with those involved in CRMS progression.

Experimental and clinical evidence increasingly suggests that MNPs may contribute not only to renal injury but also to endothelial dysfunction, vascular inflammation, metabolic dysregulation, and accelerated atherosclerosis. Of particular concern is the observation that microplastics detected in carotid plaques are associated with a markedly increased risk of major adverse cardiovascular events (3). These findings support the hypothesis that environmental pollutants such as MNPs may represent previously underestimated cardiovascular risk factors.

The kidney and the cardiovascular system are deeply interconnected through shared inflammatory, metabolic, and neurohormonal pathways. Persistent exposure to MNPs may therefore amplify oxidative stress, chronic low-grade inflammation, insulin resistance, endothelial dysfunction,

and activation of the renin-angiotensin-aldosterone system, all hallmark mechanisms of cardio-reno-metabolic disease (3,4). In addition, adsorbed contaminants, including bisphenols, phthalates, PFAS, and heavy metals, may further exacerbate endocrine and metabolic disruption (2).

Importantly, climate change may intensify this phenomenon. As recently highlighted by Marketou et al., climate change is emerging as a major challenge for cardiovascular prevention and cardiovascular risk factor management, influencing exposure to environmental pollutants and contributing to the worsening of cardiometabolic health worldwide (5). The interaction between climate change, environmental plastic pollution, kidney injury, and cardiovascular disease may therefore represent a converging pathogenic axis requiring urgent multidisciplinary attention.

In this perspective, MNPs should no longer be considered exclusively environmental contaminants, but rather novel contributors to the cardio-reno-metabolic continuum. Vulnerable populations, including patients with chronic kidney disease, diabetes, obesity, hypertension, and heart failure, may be particularly susceptible to the cumulative biological effects of chronic plastic exposure.

Future studies should evaluate the long-term cardiovascular and metabolic consequences of MNP accumulation and explore whether reduction of environmental exposure could become part of preventive cardio-renal medicine strategies. Integrating environmental health into cardiovascular and nephrological prevention may become increasingly necessary in the era of climate change.

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