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

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ABSTRACT

Micro- and nanoplastics (MNPs) are increasingly recognized as pervasive environmental contaminants with major implications for human health. Their ubiquity across air, water, food chains and indoor environments, together with the release of plastic-associated chemicals—including bisphenols, phthalates, PFAS and heavy metals—creates a complex and continuous exposure scenario. MNPs can enter the human body through ingestion, inhalation, dermal contact and medical devices; nanoplastics, in particular, may cross biological barriers, reach the systemic circulation and accumulate in target organs such as the kidney.

Experimental evidence identifies multiple mechanisms of nephrotoxicity. MNPs induce oxidative stress, mitochondrial dysfunction, ferroptosis, epithelial barrier disruption, inflammatory activation and tubulo-interstitial fibrosis. Adsorbed contaminants further amplify these effects through endocrine disruption, immunotoxicity and metabolic dysregulation. The detection of MNPs in human kidney tissue and urine supports their systemic bioavailability and renal handling. This review synthesizes current knowledge on environmental and clinical exposure sources, routes of absorption, toxicokinetics and pathogenic pathways of MNP-induced renal injury. Understanding these processes is essential to correctly interpret clinical observations and to recognize MNPs as emerging environmental determinants of kidney health.

Keywords: Environmental nephrotoxins, Exposure pathways, Microplastics, Nanoplastics, Renal injury mechanisms, Toxicokinetics

Introduction

Plastic has become a defining structural element of the Anthropocene. Global production reached approximately 435 million tonnes in 2020 and, in the absence of effective interventions, is projected to increase by >70% by 2040 (1-3). Throughout their entire life cycle—production, use, release and degradation—plastic materials generate a continuous flux of microplastics (MPs, <5 mm) and nanoplastics (NPs, <1 µm), which are now ubiquitous in air, water, soil, food chains and indoor environments (1,4-6). This burden is further compounded by the use of more than 13,000 chemical substances in polymer manufacturing, including phthalates, bisphenols, PFAS, flame retardants and heavy metals, many of which are recognized for their toxicity and endocrine-disrupting properties (1,7).

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This complex mixture is reflected in the growing documentation of MNPs in human compartments, including the placenta, lungs, liver, kidneys, gastrointestinal tract and cardiovascular system (1,2,4,8). Longitudinal studies indicate a progressive increase in plastic burden within human tissues over time (1,8).

Sources of exposure are multiple. Ingestion through drinking water, food and packaged beverages represents a primary route (1,5,9). Numerous industrial beverages have been shown to contain MNPs, as reported in JAMA Insights 2025 (1). Cosmetics and personal care products further contribute to exposure (1,10,11). The inhalation route plays a significant role: synthetic fibres and indoor-derived MNPs can reach the alveoli and cross the alveolar–capillary barrier (6,8,12). The use of medical devices—catheters, infusion lines and dialysis membranes—represents an additional clinical source of exposure (13-17).

The surfaces of MNPs can adsorb persistent contaminants such as PFAS, pesticides, heavy metals and polycyclic aromatic hydrocarbons (PAHs), as well as bisphenols and phthalates, generating a synergistic toxicological effect (5,8,18,19). An analysis by Kelly et al. highlighted how climate change renders plastics “more mobile, more persistent and more

dangerous" (20), with extreme weather events capable of increasing environmental concentrations by 30-40-fold (20).

Experimental studies have documented that MNPs induce oxidative stress, mitochondrial dysfunction, DNA damage, tight junction alterations, ferroptosis and fibrosis (5,7, 21-23). Cellular models and organoids confirm these mechanisms even at low concentrations (1,21-23). NPs can cross biological barriers and may accumulate in proximal tubules or traverse the glomerular filtration barrier and be detected in urine (3-6).

Clinical evidence further demonstrates associations between microplastics and cardiovascular and neurological risk: MPs detected in carotid plaques are associated with a fivefold increased risk of major cardiovascular events (7), and plastic particles have been identified in brain tissue of patients with dementia (8). Overall, MNPs are emerging as relevant environmental determinants of human health in general, and of kidney health in particular (5,7,21).

Sources of exposure to micro- and nanoplastics

Human exposure to MNPs arises from multiple environmental sources. It is an inevitable, daily and cumulative exposure: particles are present in drinking water, food, indoor air, cosmetics and numerous medical devices. Their ubiquity renders accumulation a new form of "background exposure" for modern humans.

Drinking water and beverages

Water represents one of the main entry routes into the human body. Numerous studies have documented the presence of microplastics in bottled mineral water, with concentrations ranging from tens to thousands of particles per litre (24,25). Levels may increase following exposure to heat, compression or prolonged storage.

Municipal water supplies may also contain microplastics: PVC, PEX or polyethylene pipes can release polymer fragments during network ageing or pressure fluctuations (25). JAMA Insights 2025 further reported the presence of microplastics in 85% of industrial beverages examined—including soft drinks, energy drinks, iced teas and packaged beverages (1).

Food and trophic chains

Dietary intake represents a substantial contribution. Marine food chains are a major vector of MNP transfer to humans: molluscs, crustaceans and fish accumulate particles in relation to ecosystem contamination levels (26). Terrestrial foods and packaged products may also be contaminated through environmental exposure or during industrial processing and packaging (27). Sea salt contains measurable quantities of microplastics, illustrating the pervasiveness of contamination.

Cosmetics and personal care products

Cosmetics constitute a direct source of exposure. Microplastics have been detected in approximately 15–20% of products examined, including exfoliants, cleansers and make-up formulations (10,11). Exposure routes include dermal contact, inhalation (particularly aerosolized cosmetics) and inadvertent ingestion, as with lip products.

Indoor environments and inhalation

Indoor environments represent one of the main sources of inhalational exposure. Synthetic fibres released from clothing, household textiles, furnishings and carpets generate micro- and nanoplastic particles that remain suspended in indoor air. Particles ≤2.5 µm can reach the alveoli, cross the alveolar–capillary barrier and enter systemic circulation (28,29). Given that individuals spend over 90% of their time indoors, this route contributes substantially to the overall MNP burden.

Healthcare settings and medical devices

Clinical exposure is particularly relevant in vulnerable patients. Medical devices such as catheters, plasticized PVC infusion lines, nutritional bags, infusion sets and—within nephrology—dialysis membranes composed of polysulfone, polyethersulfone and polycarbonate may release phthalates, bisphenols and polymer fragments (14-16,18).

This exposure pathway is unique in that it adds to an already multivectorial and persistent environmental exposure—mediated by ingestion, inhalation and dermal contact—while bypassing biological barriers and affecting populations with reduced renal excretory capacity. Such patients represent a "sentinel population" for investigating accumulation mechanisms and biological effects of MNPs.

Particles as vectors of contaminants: the "chemical corona" and endocrine disruptors

Micro- and nanoplastics (MNPs) do not act as inert particles. Their surfaces readily adsorb persistent contaminants (PFAS, pesticides, PAHs, metals and microorganisms), forming a so-called chemical corona that modifies biodistribution and toxicity (20,30). Some contaminants, such as bisphenols and phthalates, act as endocrine-disrupting chemicals (EDCs) and may interfere with thyroid, reproductive, metabolic and cardiovascular processes (31). The particle–contaminant combination generates a "cocktail effect" that amplifies the impact on vulnerable organs such as the kidney.

Absorption, translocation, and distribution of micro- and nanoplastics

Entry of MNPs into the human body occurs primarily via enteral and inhalational routes, while dermal exposure and medical device–related exposure become particularly relevant under specific clinical conditions. Once physiological barriers are crossed, particles may access systemic circulation and distribute to multiple organs and tissues, including the kidney.

Gastrointestinal absorption

The gastrointestinal tract represents a major portal of entry. Larger MPs tend to remain within the intestinal

lumen or traverse the epithelium via M cells located in Peyer's patches, through endocytic processes or paracellular passage, which becomes more likely in the presence of tight junction alterations (32). NPs, owing to their nanoscale size and high surface area, exhibit significantly greater absorption capacity: they may be internalized through clathrin- or caveolin-mediated endocytosis or transported by dendritic cells and macrophages via trans-epithelial pathways, particularly active under conditions of inflammation or dysbiosis (33,34).

Once the intestinal epithelium is crossed, particles may access capillaries or the lymphatic system, thereby reaching the liver, spleen and systemic circulation. The documented presence of MNPs in human faeces, blood and urine suggests not only that absorption is possible, but also that a cycle of distribution and partial excretion exists that remains incompletely defined (35).

Inhalational absorption

The inhalation route substantially contributes to daily exposure, particularly in indoor environments, where synthetic fibres and derived plastic fragments—now ubiquitous in homes and clothing—can persist suspended in inhaled air for prolonged periods. Particles ≤2.5 µm, including many NPs, are capable of crossing the alveolar–capillary barrier and entering systemic circulation through mechanisms analogous to those described for atmospheric pollutants and engineered nanoparticles (36).

JAMA Insights 2025 further confirmed the ability of NPs to cross complex biological barriers and reach deep organs, including the cardiovascular system and kidneys (1). Once in circulation, formation of the so-called protein corona—a coating composed of plasma proteins—modifies biodistribution, immunogenicity and interactions with endothelium, tubular cells and tissues (37).

Systemic biodistribution and organ accumulation

Numerous experimental and clinical studies have documented the multi-organ distribution of MNPs. Particles have been identified in the liver, likely through uptake by Kupffer cells; in the spleen via lymphatic sequestration; and in the lungs through both direct deposition and systemic recirculation. They have also been detected in the placenta, with evidence of potential transplacental transfer and more recently in the heart, blood vessels, kidney and brain tissue (24,38-40).

Of particular relevance is the observation—reported in JAMA 2025—of a progressive increase in plastic burden within human tissues over recent decades, suggesting continuous exposure in the context of limited elimination capacity (1). Particle nature, size and surface charge substantially influence biodistribution. NPs, in particular, display greater ability to cross biological barriers and to be internalized by cells of the mononuclear phagocyte system. The presence of adsorbed contaminants on particle surfaces—PFAS, metals, PAHs and microorganisms—further shapes biological behaviour (41,42).

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