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Micro- and nanoplastics and the kidney: human evidence, clinical vulnerabilities, dialysis-related exposure, and CKD

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ABSTRACT

Micro- and nanoplastics (MNPs) have recently been identified in human kidney tissue, urine and bloodstream, raising concerns about their potential contribution to renal dysfunction. Epidemiological and clinical studies, although still limited, increasingly link exposure to MNPs and their associated contaminants, such as PFAS, bisphenols and phthalates, are associated with reduced estimated glomerular filtration rate (eGFR), albuminuria, hypertension, metabolic alterations and an increased risk of developing chronic kidney disease (CKD). MNPs may exert both direct effects, through theoretical mechanical clogging, tubular injury, oxidative stress and inflammation, and indirect effects by acting as vectors for toxic co-contaminants.

Patients with CKD represent a high-risk group because of reduced renal clearance, increased susceptibility to oxidative and inflammatory stress, and frequent exposure to medical-grade plastics. Dialysis patients constitute the most informative human model of chronic plastic exposure. Each haemodialysis session involves direct blood contact with PVC tubing spallating in rollerpumps, polysulfone or polyethersulfone membranes and polycarbonate components that may release bisphenols and phthalates. In addition, water treatment systems and peritoneal dialysis bags may contribute to MNP exposure through leaching and material degradation, creating a unique and sustained "plastic burden" with potential systemic and renal consequences.

This review summarizes available evidence of human pathology, discusses the specific vulnerability of CKD, dialysis, transplanted and paediatric populations, and outlines research priorities and public health strategies. Recognizing MNPs and plastic-associated chemicals as emerging, potentially modifiable environmental determinants of kidney health is crucial for risk stratification, device design, regulatory policies and Planetary Health-oriented nephrology practice.

Keywords: Chronic kidney disease, Endocrine-disrupting chemicals, Hemodialysis, Microplastics, Nanoplastics, Peritoneal dialysis, Planetary health

Implications for the kidney

Ingestion, inhalation and dermal contact contribute to a multivectorial and persistent exposure to MNPs. Renal physiology-characterized by high blood flow, elevated mitochondrial density and intense endocytic activity- renders the kidney particularly susceptible to the combined effects of MNPs and the additives they transport. Clinical and experimental studies indicate renal accumulation and significant cellular toxicity (1-5), supporting the concept that micro- and nanoplastics (MNPs) may represent a novel environmental determinant of kidney health.

Translocation and accumulation in the kidney

Following environmental exposure, a fraction of MNPs may undergo translocation, defined as the passage from primary exposure sites—predominantly gastrointestinal or respiratory—to the systemic compartment and subsequently to target tissues. This process entails crossing epithelial and endothelial barriers with access to blood or lymphatic circulation.

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Evidence supporting MNP translocation derives mainly from cellular and animal models, in which micro- and particularly nanometric particles have been shown to cross intestinal and alveolar barriers and distribute to distant organs. In humans, direct demonstration of this process is currently lacking. Available evidence is limited to indirect indicators of systemic bioavailability, including detection of MNPs in blood and human renal parenchyma (5-7). Experimental models further support systemic distribution and organ accumulation of microplastics (8,9).

Although the presence of MNPs in urine and kidney tissue supports the hypothesis that circulating particles may reach the renal compartment, the precise biological pathways involved remain to be elucidated.

Passage across the glomerular filtration barrier has been hypothesized only for ultrasmall nanometric fractions, based on experimental studies using engineered nanoparticles under conditions not fully comparable to real-world environmental exposure (9).

A more robustly supported mechanism is tubular uptake, particularly within proximal tubular epithelial cells, which represent the main endocytic compartment of the nephron. In vitro studies—including human-derived tubular cell lines—and in vivo models have demonstrated internalization of nanoparticles and microplastics through clathrin- and caveolin-mediated endocytic processes (8,10). By contrast, active tubular secretion mechanisms for MNPs have not been demonstrated (see Supplementary Box 1).

The potential vulnerability of the kidney to interaction with MNPs can be plausibly inferred from well-known physiological features, including high renal blood flow and the intense metabolic and mitochondrial activity of proximal tubular cells, which render this compartment particularly susceptible to toxic insults (11,12). Nevertheless, direct

TABLE 1 - The table summarizes the main lines of experimental, environmental and clinical evidence linking exposure to MNPs and plastic-associated chemicals to renal injury. For each exposure setting or population, the table highlights the type of evidence, the predominant renal pathogenic mechanisms and their potential clinical relevance, with particular emphasis on vulnerable groups such as patients with CKD and those undergoing dialysis. The table is intended as an integrative framework bridging pathophysiological insights and clinical nephrology, rather than a comprehensive systematic appraisal of the literature. Representative key references are provided for each exposure setting to facilitate further reading.

Exposure setting/ population	Type of evidence	Main mechanisms involved	Key findings	Clinical relevance	Key references
Environmental exposure/general population	Experimental (cellular, animal, organoids)	Oxidative stress; mitochondrial dysfunction; inflammation; ferroptosis	MNPs induce ROS generation, mitochondrial damage, lipid peroxidation and pro-fibrotic signaling even at low concentrations	Suggests chronic low-grade renal injury and cumulative lifelong risk	(2, 5)
Food and drinking exposure	Environmental & toxicological studies	Systemic bioavailability; possible renal distribution; tubular uptake	MNPs detected in food, beverages and drinking water; indirect evidence of systemic exposure	Represents unavoidable, lifelong background exposure	(6, 7)
Inhalation	Experimental & exposure studies	Barrier crossing; vascular and renal distribution	Nanoparticles cross alveolar–capillary barrier and enter systemic circulation	Relevant for urban populations and indoor lifestyles	(7, 9)
Plastic-associated chemicals (BPA, phthalates)	Epidemiological & clinical studies	Endocrine disruption; oxidative stress; metabolic dysregulation	Associations with altered renal function, inflammation and metabolic disturbances	Synergistic interaction with MNPs (“cocktail effect”)	(18-23)
CKD patients	Pathophysiological inference + limited human data	Reduced clearance; amplified oxidative and inflammatory stress	CKD may favor the accumulation of MNPs and associated toxins	CKD is identified as a vulnerable population	(2, 12)
Hemodialysis patients	Clinical and analytical studies	Direct blood–plastic contact; tubing spallation; chemical leaching	Increased exposure to plastic-derived compounds; microplastics detected in dialysis water and fluids	Represents the most extreme model of chronic medical exposure	(15-19)
Peritoneal dialysis patients	Experimental & clinical observations	Migration from bags; peritoneal absorption; possible spallation	Plastic additives detected in peritoneal dialysate; potential systemic absorption	Long-term cumulative exposure in fragile patients	(16, 21)

Abbreviations: MNPs, micro- and nanoplastics; CKD, chronic kidney disease; BPA, bisphenol A; PFAS, per- and polyfluoroalkyl substances; ROS, reactive oxygen species; eGFR, estimated glomerular filtration rate.

demonstrations of MNP accumulation and MNP-mediated damage in the human kidney remain limited.

Mechanisms of renal toxicity induced by micro- and nanoplastics

mNP-induced renal toxicity results from the simultaneous and convergent activation of multiple pathogenic pathways involving energy metabolism, epithelial integrity, inflammatory responses and fibrotic remodeling. Evidence from cellular studies and animal models, together with emerging observations in human renal tissue, delineates a coherent toxicological framework in which MNPs exert both direct effects on renal cells and indirect toxicity through surface-adsorbed chemical contaminants (2,5,10,13,14). In addition, recent work has demonstrated that combined exposure to microplastics and co-contaminants exacerbates tubular cell injury, highlighting the toxicological relevance of particle–chemical interactions in renal models (14).

One of the earliest and most consistently observed events following MNP exposure is the induction of oxidative stress. Increased reactive oxygen species generation and lipid peroxidation occur even at relatively low concentrations and precede overt structural alterations (2,3,13). Nanoplastics, owing to their higher surface-to-volume ratio and increased chemical reactivity, appear particularly effective in triggering oxidative and inflammatory pathways. These pro-oxidant effects have also been demonstrated in advanced three-dimensional cellular models and human organoid-like systems, reinforcing the biological relevance of these mechanisms and narrowing the conceptual gap between experimental evidence and potential clinical translation (2,3,10,14).

Mitochondria represent a central node of vulnerability, especially in proximal tubular cells, where high energy demand makes cellular function critically dependent on intact oxidative phosphorylation (11,12). In the presence of MNPs, loss of mitochondrial membrane potential, reduced oxygen consumption, cristae fragmentation, decreased ATP synthesis and impairment of fatty acid β-oxidation have been documented. Even subclinical mitochondrial damage may translate into significant functional impairment, given the pivotal role of the proximal tubule in reabsorption and detoxification processes.

Another relevant mechanism involves the integrity of the tubular epithelium. Exposure to MNPs has been associated with reduced expression of tight junction proteins, resulting in increased epithelial permeability. Although much of the available evidence derives from intestinal or non-renal epithelial models, relevance to the kidney is biologically plausible, as tubular epithelium shares similar mechanisms of cellular polarity, barrier function and paracellular trafficking control (2,3,13).

Inflammatory activation represents an additional cornerstone of MNP-associated renal toxicity. Exposure to these particles induces activation of pro-inflammatory pathways, with increased production of cytokines such as IL-6, TNF-α and IL-1β and recruitment of immune cells into the tubulo-interstitial compartment (2,3,13). In prolonged exposure models, this response tends to become chronic, creating a microenvironment conducive to progressive structural damage. Beyond cytokine induction, experimental evidence suggests that sustained MNP exposure may lead to innate immune dysfunction, with persistent macrophage activation and impaired resolution of inflammation, thereby favouring chronic tubulo-interstitial injury (2,3,13,14).

In recent years, ferroptosis has been proposed as an additional relevant mechanism of injury. This iron-dependent, regulated form of cell death, characterized by uncontrolled lipid peroxidation, has been observed in experimental models exposed to nanoplastics (2,3,13). Expansion of the intracellular labile iron pool and impairment of antioxidant systems may promote a shift toward tubular necrosis and fibrosis, particularly under conditions of chronic exposure or reduced elimination capacity.

Supplementary Box 3 summarizes the role of ferroptosis in MNP-associated renal toxicity.

The ultimate outcome of these processes is activation of pro-fibrotic pathways, particularly those mediated by TGF-β and epithelial-to-mesenchymal transition. In experimental models of chronic exposure, increased interstitial collagen deposition, fibroblast activation and distortion of tubular architecture are observed, resulting in a maladaptive remodeling pattern resembling that observed in chronic toxic nephropathies (1,12).

Collectively, these data suggest that MNPs do not act through a single dominant mechanism, but rather function as amplifiers of pre-existing biological vulnerabilities, with particularly relevant effects under conditions of metabolic stress, inflammation or reduced compensatory capacity.

Potential immune pathways implicated in MNP-associated renal toxicity are summarized in Supplementary Table S1.

These convergent mechanisms are schematically illustrated in Figure 1.

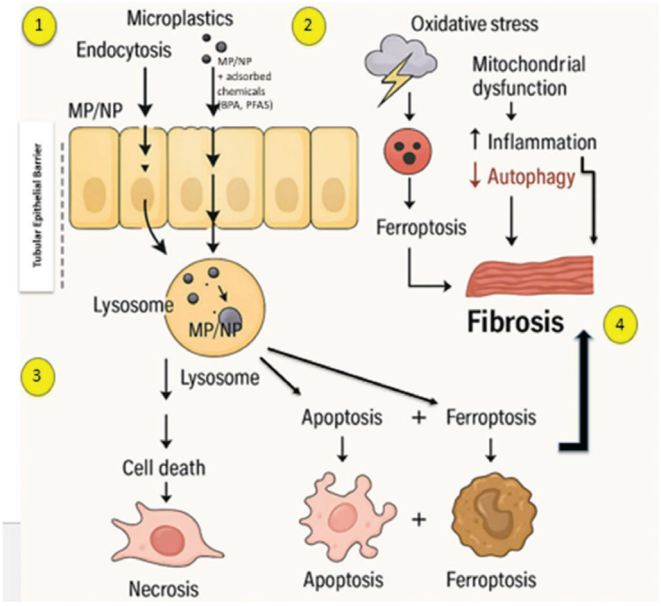

FIGURE 1 - Proposed molecular mechanisms of renal toxicity induced by MNPs.

MNPs, alone or in combination with adsorbed plastic-associated chemicals (e.g., bisphenol A and PFAS), may interact with the tubular epithelial barrier and may be internalized by renal tubular cells through endocytic pathways. Following cellular uptake, MNPs accumulate within lysosomes, contributing to lysosomal dysfunction and impaired intracellular degradation. MNPs exposure induces oxidative stress and mitochondrial dysfunction, which in turn promote pro-inflammatory signaling and dysregulation of autophagy. These processes converge on multiple regulated and non-regulated forms of cell death, including apoptosis, ferroptosis and necrosis. The combined effects of persistent inflammation, ferroptotic signaling and tubular cell loss favor maladaptive repair responses and tubulo-interstitial fibrotic remodeling, ultimately contributing to CKD progression.

Dialysis: the extreme model of plastic overexposure

Why this matters

Dialysis patients experience greater exposure to plastic than other patients. In haemodialysis (and automated peritoneal dialysis), blood passes through synthetic polymers, and tubing spallation in peristaltic roller pumps—caused by repeated mechanical compression and friction of the tubing segment—leads to the release of nano-sized particles and smaller microplastic fragments into the extracorporeal circuit (15). Even in gravity-driven peritoneal dialysis, glucose-based solutions from plastic bags are infused into the peritoneal cavity (16). The use of large volumes of dialysis water and dialysate further contributes to potential exposure, as recent studies have demonstrated the presence of microplastics in dialysis fluids (17). In addition, exposure to plastic-derived chemicals such as bisphenols from dialysis membranes and devices has been documented (18), along with phthalates released from plastic components (19). More recent analyses and reviews have highlighted the broader issue of endocrine-disrupting chemicals in dialysis therapies (20-22), including long-term clinical associations (23). Combined with impaired renal clearance, this may contribute to chronic internal accumulation of plastic-derived chemicals, microplastics, and nanoplastics (Fig. 2).

Repeated contact with plastic-based devices in haemodialysis and peritoneal dialysis may result in sustained internal exposure to MNPs and plastic-associated additives. Mechanical tubing spallation in peristaltic pump segments can generate nano- and micro-sized plastic particles that enter the extracorporeal circuit. Reduced renal clearance and long-term accumulation may enhance vulnerability, with experimental evidence suggesting links to inflammation, oxidative stress, endocrine disruption and immune dysfunction.

Potential biological consequences

Plastic-associated compounds such as bisphenols and phthalates can disrupt endocrine function and promote oxidative stress and inflammation (18-23), while MNPs have been shown in experimental models to cross biological barriers and interact with immune cells (6,8). In dialysis patients, these risks may heighten chronic inflammation, impair immune responses, elevate cardiovascular risk, and worsen metabolic disturbances, potentially contributing to morbidity beyond that attributable to uremia alone.

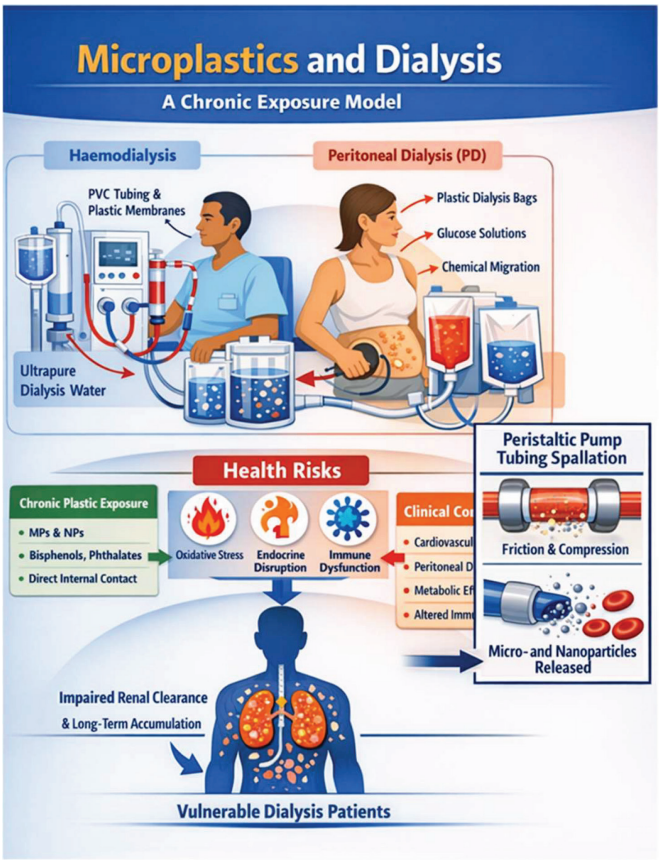

FIGURE 2 - Dialysis as a potential model of chronic exposure to MNPs.

Implications for haemodialysis

Haemodialysis exposes patients to plastics via the extracorporeal circuit, particularly spallation particles generated from peristaltic roller pump segments (15), and large volumes of dialysis water used to generate dialysate. The detection of microplastics in ultrapure water and final dialysate signals a potential risk to patient safety, highlighting that current water-treatment standards are not specifically designed to monitor or eliminate particulate plastic contamination (16,17). This underlines the need for new technical and regulatory approaches to mitigate this risk.

Implications for peritoneal dialysis

Peritoneal dialysis is also a major exposure pathway. Automated peritoneal dialysis devices using roller pumps may similarly generate spallation particles (15). In addition, even purely gravity-driven systems involve dialysis fluids stored in polymer bags and subjected to heat, acidity, and high osmolarity, which may promote migration of plastic additives and

possibly MNPs (16,21). Direct peritoneal infusion exposes these contaminants to immediate contact with the mesothelium and microcirculation, potentially leading to inflammation, membrane damage, and systemic absorption.

Implications for clinical practice and technology

The choice of tubing, membranes, solution bags, and sterilization methods directly affects the long-term risk of plastic exposure. Prioritizing bisphenol-free materials, low-leaching polymers, and enhanced filtration of dialysis water and fluids should be considered alongside traditional biocompatibility and microbiological safety in protecting patient health (21-23) (see Supplementary Box 4).

Future directions

Dialysis provides a uniquely informative human model for studying chronic exposure to MNPs. Integrating environmental toxicology into dialysis research and device development could lead to safer renal replacement therapies and align with broader sustainability and Planetary Health strategies in nephrology.

Interactions between MNPs and chronic kidney disease

chronic kidney disease (CKD) represents a clinical scenario in which exposure to MNPs acquires particular pathophysiological relevance. Reduced renal clearance favours systemic accumulation of particles and associated contaminants, prolonging exposure time in a biological context already characterized by oxidative stress, mitochondrial dysfunction and chronic inflammation (2,12,13).

Integrity of the glomerular barrier constitutes an additional critical point. In CKD, increased glomerular permeability may facilitate the passage of nanometric particles toward the tubular compartment, promoting local accumulation, whereas clogging of glomerular pores by microparticles may theoretically reduce filtration capacity. Demonstration of MNPs in human renal parenchyma and urine strengthens the hypothesis that at least a fraction of circulating particles reaches the kidney (5,7).

Taken together, these observations suggest that MNPs and plastic-associated contaminants do not represent merely an "external" pollutant, but an environmental determinant capable of interacting with CKD pathophysiology and amplifying pre-existing renal vulnerability, as schematically summarized in Figure 3, where CKD-related vulnerability and dialysis-related plastic exposure—including spallation phenomena—are depicted as synergistic amplifiers.

Conclusions

Micro- and nanoplastics (MNPs) are emerging as novel environmental determinants of human health, progressively overcoming the traditional distinction between the external environment and internal biology. Their documented presence in multiple human tissues and organs—including the cardiovascular system, placenta and kidney—indicates that plastic exposure has become an increasingly recognized component of contemporary human pathophysiology (5-7).

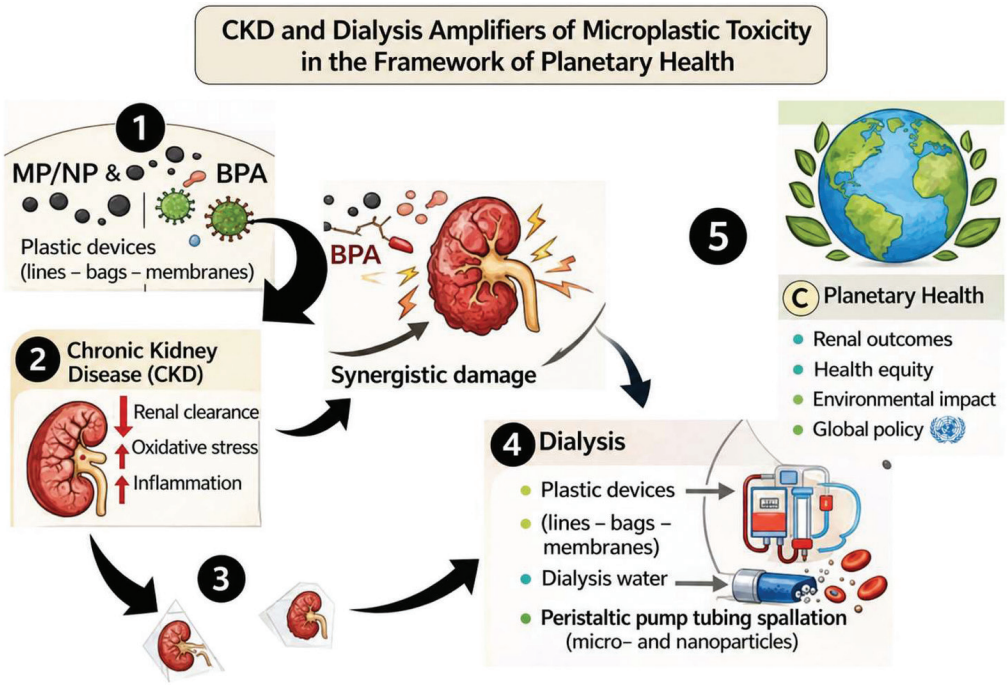

FIGURE 3 - CKD and dialysis as synergistic amplifiers of MNP toxicity within a Planetary Health framework.
The figure illustrates how environmental exposure to micro- and nanoplastics (MNPs) and plastic-associated chemicals (e.g., bisphenol A) interacts with chronic kidney disease (CKD)-related vulnerability—characterized by reduced renal clearance, oxidative stress and inflammation—and dialysis-related exposure (plastic devices, dialysis water and tubing spallation). These convergent pathways promote systemic accumulation, tubular injury and fibrotic remodeling, ultimately contributing to CKD progression and adverse clinical outcomes. The broader implications for Planetary Health, including environmental impact and health equity, are also highlighted.

In nephrology, these observations are particularly relevant. Owing to its filtering function, high blood flow, and intense metabolic and mitochondrial activity, the kidney represents a biologically plausible target for both the direct and indirect effects of MNPs and their associated chemical contaminants. Experimental evidence delineates a coherent framework of renal toxicity mediated by oxidative stress, mitochondrial dysfunction, inflammation, ferroptosis and fibrotic remodeling (2,3,11-14), while early clinical observations—including the detection of microplastics in human renal parenchyma and urine—support the hypothesis of genuine renal exposure in humans, albeit in the absence of definitive causal proof (5,7).

Dialysis represents the most extreme human model of chronic medical exposure to plastic materials. The direct, repeated and prolonged contact of blood or internal biological surfaces with extracorporeal circuits, membranes, tubing and large volumes of treated water calls for consideration beyond the depurative efficacy of dialysis techniques, encompassing material safety, device design and sustainability of healthcare supply chains. In this context, dialysis patients and, more broadly, individuals with CKD emerge as particularly vulnerable populations and as sentinel models for investigating the biological effects of plastic exposure (15-23).

Taken together, MNPs should no longer be regarded solely as environmental pollutants, but rather as emerging determinants capable of interacting with renal pathophysiology and amplifying pre-existing biological vulnerabilities. Addressing this challenge will require an evolution in nephrological thinking, integrating environmental determinants into clinical assessment, translational research and medical device design, in line with the Planetary Health paradigm. Protecting kidney health today also means critically examining the biological and environmental impact of the materials routinely used in clinical practice, with a responsibility that extends to present and future generations.

These considerations are consistent with recent European initiatives calling for environmentally sustainable nephrology practice (24).

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