Microplastic exposure and kidney disease: an emerging environmental nephrotoxin in the Anthropocene

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Background
Microplastics (MPs) and nanoplastics (NPs) are ubiquitous environmental pollutants resulting in universal human exposure. While cardiovascular effects are emerging, a systematic evaluation of their role as a nephrotoxic agent is needed, particularly concerning chronic kidney disease progression.

Objectives
This review critically evaluates the current knowledge on MP/NP exposure and kidney disease, focusing on exposure pathways, renal bioaccumulation, cellular and molecular mechanisms of toxicity, and the emerging clinical and epidemiological evidence.

Methods
A comprehensive literature search of PubMed (National Library of Medicine, National Institutes of Health, Bethesda, MD, USA), Scopus (Elsevier B.V., Amsterdam, The Netherlands), and Web of Science (Clarivate Analytics, Philadelphia, PA, USA) was conducted, with an emphasis on evidence from 2020 to 2025. Human studies, animal models, and *in vitro* experiments were included to synthesize the current understanding of MP-induced nephrotoxicity.

Results
MPs are detected in human kidneys, urine, and blood, with exposure occurring via ingestion (39 000–52 000 particles/year) and inhalation (up to 68 000 particles/day). Mechanistically, MPs induce renal injury through oxidative stress, endoplasmic reticulum stress, inflammation, autophagy dysregulation, and fibrosis. Animal models confirm histological and functional kidney alterations. Dialysis patients represent a high-risk population, with significant weekly exposure from dialysis fluids (9.57–14.28 mp/l). The presence of MPs in atheromas is associated with increased major adverse cardiovascular events, highlighting the cardiorenal risk.

Conclusions
MPs represent a novel and significant nephrotoxic threat. Urgent research is needed to establish the epidemiological link between MP exposure and chronic kidney disease. The development of clinical monitoring strategies and the implementation of public health interventions are essential to mitigate the health impacts of this escalating environmental concern.

Keywords:
chronic kidney disease, environmental exposure, environmental pollutants, kidney diseases, microplastics, nanoplastics, nephrotoxicity, renal dialysis

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Introduction

With over 400 million tons of plastic produced annually, the global environmental burden of plastic waste has reached a critical point [1]. The fragmentation of this waste into microplastics (MPs) and nanoplastics (NPs) has led to their ubiquitous presence in even the most remote ecosystems, from Arctic Sea ice to the deep ocean trenches [2]. This widespread contamination has resulted in continuous human exposure, with recent studies confirming the presence of these particles in various human tissues and fluids, including the kidneys [3]. The escalating crisis of plastic pollution represents a novel and complex challenge for environmental health and clinical medicine, particularly in the context of chronic diseases.

The global prevalence of chronic kidney disease (CKD) affects an estimated 843.6 million individuals worldwide, with a significant portion progressing to end-stage kidney disease (ESKD) requiring life-sustaining dialysis or transplantation [4]. While traditional risk factors such as diabetes, hypertension, and genetic predisposition are well-established drivers of CKD, a substantial portion of the disease burden remains unexplained. This has shifted focus towards the role of environmental exposures, including heavy metals, air pollutants, and now, plastic particles, as potential contributors to the initiation and progression of kidney disease. The intersection of this global environmental toxicant with a highly vulnerable patient population necessitates a comprehensive evaluation of the nephrotoxic potential of MPs and NPs.

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The clinical and economic burden of CKD is immense, consuming a disproportionate share of healthcare resources, particularly for the management of ESKD. Patients on dialysis represent a uniquely vulnerable group, not only due to their compromised ability to clear systemic toxins but also because of their direct and repeated exposure to large volumes of medical-grade plastics used in dialysis circuits and solutions [5]. The recent discovery of MPs in atheromatous plaques and their association with increased major adverse cardiovascular events further amplifies these concerns, given the profound link between cardiovascular disease and kidney disease—the cardiorenal syndrome [6].

Despite the growing body of evidence on the systemic effects of MPs, significant knowledge gaps persist regarding their specific impact on renal pathophysiology. There is a pressing need to understand the mechanisms of renal bioaccumulation, the cellular and molecular pathways of toxicity, and the clinical implications for CKD patients. The lack of robust human epidemiological data and validated clinical biomarkers for MP exposure hinders our ability to accurately assess the risk and formulate effective public health strategies. This review aims to address these critical knowledge gaps by providing a comprehensive synthesis of the current evidence on MP exposure and kidney disease. We will evaluate the environmental sources and human exposure pathways, detail the methods for detecting MPs in biological samples, and dissect the molecular mechanisms of nephrotoxicity. Furthermore, we will examine the emerging epidemiological evidence, with a particular focus on vulnerable populations such as CKD and dialysis patients, and discuss the public health implications and potential mitigation strategies. By integrating findings from environmental science, toxicology, and clinical nephrology, this review seeks to define the scope of this emerging threat and outline a research and policy agenda to protect kidney health in the Anthropocene.

Environmental sources and human exposure pathways

The pervasive nature of plastic pollution has resulted in the contamination of virtually all environmental compartments, leading to complex and continuous human exposure. MPs and NPs are not confined to the oceans but are also found in freshwater systems, soil, and the atmosphere, facilitating their entry into the human food chain and body [7]. The primary routes of human exposure are ingestion, inhalation, and to a lesser extent, dermal contact, with each pathway contributing to the total body burden of these persistent particles [8].

Ingestion is considered the most significant route of exposure, driven by the consumption of contaminated food and water. A foundational 2019 study estimated that annual MP consumption ranges from 39 000 to 52 000 particles, with these estimates increasing substantially when inhalation is considered [9]. Aquatic food webs are a major conduit, with one analysis of 109 countries finding that 57% of plastic particles in foods originate from aquatic sources [10]. Beyond seafood, MPs have been detected in a wide array of consumer products, including salt, sugar, honey, beer, and both bottled and tap water, indicating multiple sources of dietary exposure [11]. The concentration of MPs in bottled water can be particularly high, with some studies reporting levels up to 6,292 MPs/l, highlighting the role of packaging and processing in contamination [12].

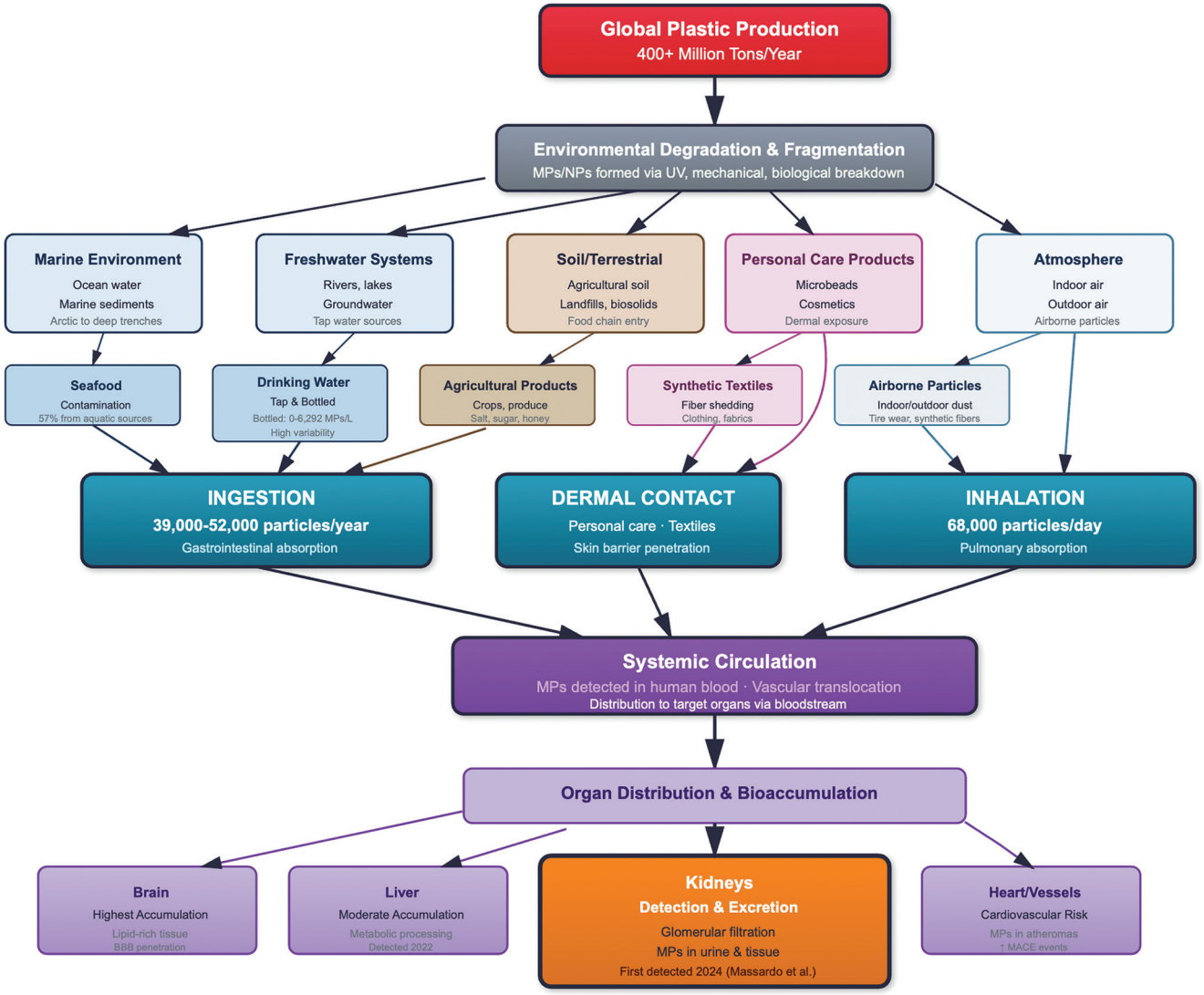
Inhalation represents another critical, and perhaps underappreciated, exposure pathway. Airborne MPs are present in both indoor and outdoor environments, with indoor dust often showing higher concentrations. A recent estimate suggests that humans may inhale as many as 68 000 plastic particles daily, a figure that underscores the importance of air quality in determining an individual's total MP burden [13]. Occupational settings, such as textile factories and waste management facilities, can present even higher risks of airborne exposure. Dermal exposure, while less studied, is also a potential route of entry, particularly from personal care products containing microbeads and from the shedding of synthetic fibers from clothing onto the skin [14]. The cumulative and multi-pathway nature of this exposure complicates risk assessment and underscores the challenge of mitigation (Fig. 1).

Bioaccumulation in kidney tissue: detection methods and evidence

The systemic distribution of MPs following exposure is a critical area of research, with the timeline of their detection in human tissues marking a series of landmark discoveries. While MPs were first identified in human blood in 2021 and liver tissue in 2022, it was not until 2024 that their presence was confirmed in human kidney tissue, a finding that has profound implications for nephrology [3,15,16]. This discovery was made possible by the application of advanced analytical techniques capable of identifying and characterizing these minute particles within complex biological matrices.

Several sophisticated methods are currently employed for the detection of MPs. Micro-Raman Spectroscopy and Fourier-Transform Infrared Spectroscopy are powerful, nondestructive techniques that provide a chemical fingerprint of polymers, allowing for their precise identification and characterization [17]. Pyrolysis-Gas Chromatography-Mass Spectrometry (Py-GC/MS) offers a quantitative approach, capable of detecting both micro- and NPs by thermally decomposing the polymer

Figure 1



Microplastic Environmental Sources, Exposure Pathways, and Renal Bioaccumulation. This figure illustrates the principal sources of global plastic production (>400 million tons/year), the environmental fragmentation pathways generating microplastics (MPs) and nanoplastics (NPs), the primary routes of human exposure (ingestion: 39,000–52,000 particles/year; inhalation: up to 68,000 particles/day; dermal contact), and the subsequent systemic distribution and organ-specific bioaccumulation, with particular emphasis on the kidney as a site of detection, filtration, and excretion. MPs: microplastics; NPs: nanoplastics; UV: ultraviolet radiation

into identifiable fragments [18]. These methods, often used in combination with Electron Microscopy for morphological analysis, have been instrumental in confirming the presence of various polymer types, including polyethylene, polyvinyl chloride (PVC), and polystyrene (PS), in human tissues.

The first direct evidence of MPs in human kidneys, reported by Massardo *et al.* in 2024, identified particles predominantly smaller than 50–100 µm in both kidney tissue and urine, suggesting a potential pathway of renal clearance [3]. Subsequent postmortem studies have provided further quantitative data, with one study detecting up to 40.4 MP particles/g of kidney tissue, indicating significant bioaccumulation [19]. Interestingly, recent research suggests organ-specific accumulation patterns, with the brain potentially harboring higher concentrations of MPs than the liver or kidneys, a finding that may be related to the high vascularization and lipid content of neural tissue [20].

The presence of MPs in the bloodstream is the critical link between exposure and systemic bioaccumulation. Once absorbed, these particles can circulate throughout the body, cross biological barriers, and lodge in various tissues. The detection of MPs in blood has been associated with alterations in coagulation markers, suggesting a potential to induce a prothrombotic state [21]. Furthermore, the identification of MPs in urine not only confirms that the kidneys filter these particles from the blood but also presents an opportunity for the development of noninvasive biomarkers of exposure. However, the translocation of MPs across the glomerular filtration barrier and their

potential for reabsorption and retention within the renal parenchyma remain key areas for future investigation. The challenges of standardizing detection methods, preventing sample contamination, and accurately quantifying NPs continue to be significant hurdles in this rapidly evolving field (Table 1).

Cellular and Molecular Toxicity Mechanisms

The nephrotoxic potential of MPs and NPs is underpinned by a complex interplay of cellular and molecular events, primarily driven by oxidative stress, inflammation, and the disruption of key cellular processes. Experimental studies using animal models and human kidney cell lines have begun to elucidate these intricate pathways. A central and recurring theme in MP-induced toxicity is the generation of reactive oxygen species, which triggers a cascade of downstream pathological events. Exposure to PS MPs has been shown to induce significant oxidative stress in the kidneys of juvenile rats, characterized by the depletion of endogenous antioxidants such as glutathione and superoxide dismutase [22]. This oxidative imbalance can lead to lipid peroxidation, DNA damage, and the activation of stress-related signaling pathways.

One such pathway is Endoplasmic Reticulum (ER) stress, which is activated in response to an accumulation of unfolded or misfolded proteins. Studies have demonstrated that PS NPs can aggravate lipopolysaccharide-induced apoptosis in mouse kidney cells by activating the IRE1/XBP1 ER stress pathway, a response mediated by oxidative stress [23]. This disruption of protein homeostasis can ultimately lead to programmed cell death, or apoptosis, contributing to the loss of renal cells.

Inflammation is another critical mechanism of MP-induced renal injury. MPs can activate the NF-κB signaling pathway, a master regulator of inflammation, leading to the production of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α [24]. This inflammatory milieu can cause direct tissue damage and recruit immune cells, further perpetuating the inflammatory response. Some studies have also pointed to the activation of the NLRP3 inflammasome, a key component of the innate immune system, as a critical event in MP-induced inflammation [25].

Autophagy, the cellular process of self-degradation and recycling of damaged organelles, is also dysregulated by MP exposure. Combined exposure to phthalates and PS MPs has been shown to induce renal autophagy through the reactive oxygen specie/AMPK/ULK1 pathway [26]. While autophagy can initially be a protective response, its chronic activation or impairment can lead to cell death and tissue injury. The interplay between autophagy, apoptosis, and necroptosis (a form of programmed necrosis) is a complex and critical determinant of cell fate in response to MP exposure.

The gut-kidney axis has emerged as another important consideration in MP-induced nephrotoxicity. Ingestion of MPs can disrupt the intestinal barrier, leading to increased gut permeability and the translocation of bacterial products, such as lipopolysaccharide, into the bloodstream. This systemic inflammation can, in turn, exacerbate kidney injury. One study demonstrated that PS MPs induce kidney injury via gut barrier dysfunction and the activation of the C5a/C5aR pathway, a component of the complement system involved in inflammation [27].

Furthermore, MPs can act as vectors for other environmental toxins, such as heavy metals and endocrine-disrupting chemicals, creating synergistic toxic effects. Co-exposure to MPs and cadmium has been shown to exacerbate kidney injury by enhancing

Table 1 Detection methods for microplastics in human biological samples

Method	Principle	Size range	Advantages	Limitations	Clinical applicability
Micro-Raman Spectroscopy	Chemical fingerprinting via molecular vibrations	1–100 µm	Nondestructive; polymer identification; particle-by-particle analysis	Time-consuming; limited throughput; requires expertise	Moderate; suitable for urine and tissue samples
FT-IR Spectroscopy	Infrared absorption by functional groups	10–500 µm	Nondestructive; rapid screening; polymer typing	Lower resolution than Raman; limited to larger particles	High; potential for automated analysis
Py-GC/MS	Thermal decomposition followed by chromatographic separation	<1 nm to >100 µm	Quantitative; detects nanoplastics; high sensitivity	Destructive; requires specialized equipment; complex sample preparation	Moderate; suitable for blood and tissue analysis
SEM-EDS	Electron microscopy with elemental analysis	100 nm to >100 µm	Morphological characterization; elemental composition	Cannot identify polymer type; requires conductive coating	Low; primarily research tool
Fluorescence Microscopy	Fluorescent dye staining of plastics	1–100 µm	Rapid; visual identification; cost-effective	Nonspecific; false positives; limited polymer identification	Low; screening tool only
Nile Red Staining	Lipophilic dye binds to hydrophobic plastics	1–100 µm	Simple; inexpensive; rapid screening	Nonspecific; requires confirmation; variable staining	Low to moderate; preliminary screening

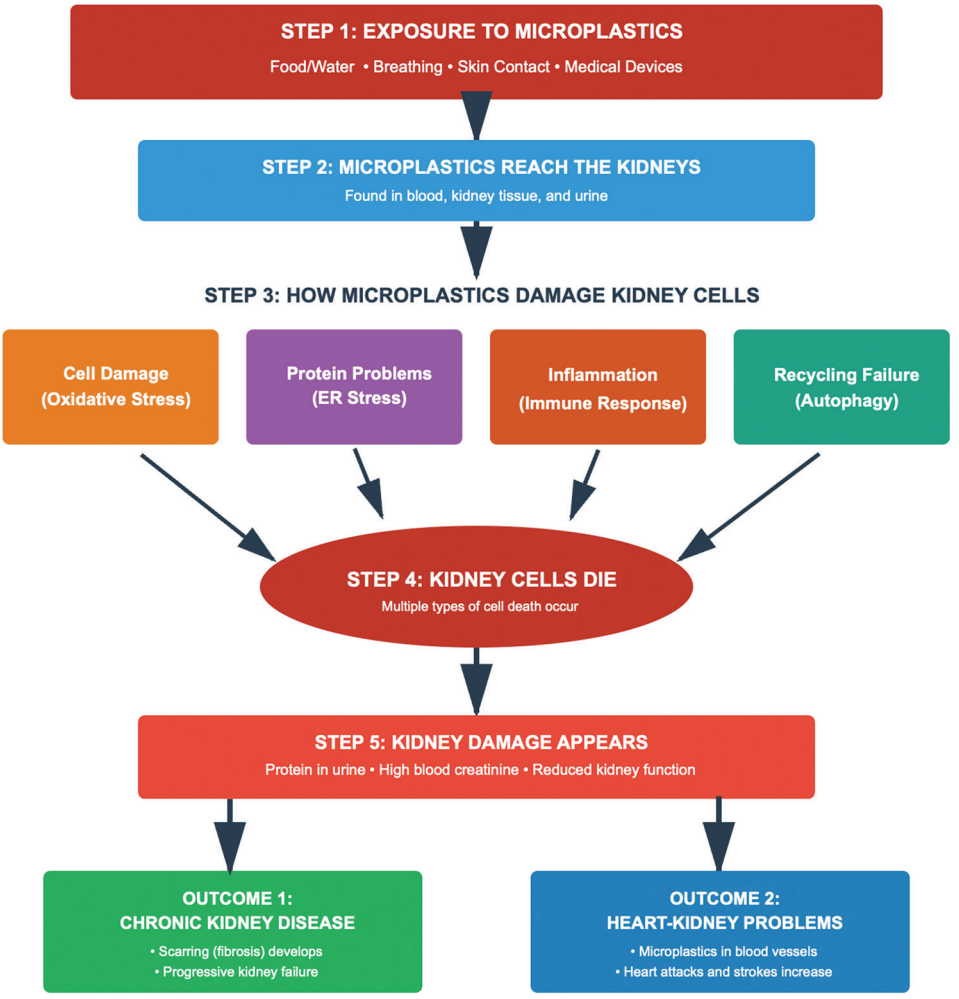
oxidative stress, autophagy, apoptosis, and fibrosis [28]. Similarly, the presence of adsorbed endocrine disruptors like Bisphenol A on the surface of MPs can amplify their toxic effects on renal cells [29]. These findings highlight the complex and multi-faceted nature of MP-induced nephrotoxicity, which involves a convergence of multiple pathological pathways leading to cell death, inflammation, and fibrosis, ultimately compromising kidney function (Fig. 2) (Table 2).

Epidemiologic evidence and clinical associations

Translating the wealth of experimental data on MP toxicity into a clear understanding of its impact on human health requires robust epidemiological evidence, an area that is currently in its infancy. The majority of human studies to date have been cross-sectional in design, making it difficult to establish causality between MP exposure and the development or progression of chronic diseases like CKD. However, the emerging associations between MPs and cardiovascular disease provide a compelling, albeit indirect, line of evidence for their potential role in nephropathy, given the intimate connection between the cardiovascular and renal systems.

The landmark 2024 study by Marfella *et al*. published in the New England Journal of Medicine, which identified MPs in human atheromatous plaques and linked their presence to a significantly higher risk of major adverse cardiovascular events, represents a pivotal moment in this field [6]. This finding is particularly relevant to nephrology, as cardiorenal syndrome is a well-established clinical entity where dysfunction in one organ potentiates dysfunction in the other. The shared pathophysiological mechanisms, including endothelial dysfunction, inflammation, and oxidative stress, are

Figure 2



Microplastic-Induced Nephrotoxicity — Pathophysiological Cascade. This schematic depicts the stepwise sequence of events from microplastic exposure through renal bioaccumulation to cellular injury and clinical outcomes. Key pathophysiological mechanisms include oxidative stress (reactive oxygen species generation), endoplasmic reticulum (ER) stress, innate immune activation and inflammation (NF-κB, NLRP3 inflammasome), and autophagy dysregulation. Downstream consequences encompass renal cell death (apoptosis, necroptosis), fibrosis, and progression to chronic kidney disease (CKD) or cardiorenal disease. ER: endoplasmic reticulum; NF-κB: nuclear factor kappa B; NLRP3: NOD-like receptor family pyrin domain-containing 3; CKD: chronic kidney disease; MACE: major adverse cardiovascular events.

Table 2 Summary of key studies on microplastic-induced renal toxicity

Study	Model	MP type/Size	Exposure duration	Key findings	Mechanisms	Limitations
Wang *et al.* [22]	Juvenile rats	PS-MPs, 5 μm	28 days, oral gavage	Nephrotoxicity, histological damage, oxidative stress	ROS generation, GSH/SOD depletion, ER stress	High dose; short duration; single polymer type
Li *et al.* [23]	Mouse kidney cells (*in vitro*)	PS-NPs, 50 nm	24 h	Enhanced LPS-induced apoptosis	IRE1/XBP1 ER stress pathway, oxidative stress	*In vitro* model; co-exposure with LPS
Meng *et al.* [24]	Chicken kidney cells	PS-MPs, 5 μm	24–48 h	Oxidative stress, inflammation, necroptosis	NF-κB, RIP1/RIP3/MLKL pathways	Non-mammalian model; *In vitro*
Sun *et al.* [26]	Mice	PS-MPs + DEHP	28 days, oral gavage	Renal autophagy, combined toxicity	ROS/AMPK/ULK1 pathway	Co-exposure complicates interpretation
Liang *et al.* [27]	Mice	PS-MPs, 5 μm	28 days, oral gavage	Kidney injury via gut-kidney axis	Gut barrier dysfunction, C5a/C5aR activation	Indirect mechanism; requires validation
Zou *et al.* [28]	Mice	PS-MPs + Cadmium	28 days, oral gavage	Exacerbated kidney injury	Enhanced oxidative stress, autophagy, apoptosis, fibrosis	Co-exposure model; high doses
Verzola *et al.* [29]	Human kidney proximal tubular cells	PS-MPs + BPA	24–72 h	Enhanced cellular damage	Oxidative stress, mitochondrial dysfunction	*In vitro*; co-exposure with BPA
Massardo *et al.* [3]	Human urine and kidney tissue	Various polymers, <100 μm	Cross-sectional	First detection of MPs in human kidneys	Not assessed	Descriptive study; no mechanistic data

central to both atherosclerosis and progressive kidney disease. The presence of MPs in the vasculature, and their potential to promote these pathological processes, suggest a plausible mechanism by which they could contribute to both cardiovascular and renal disease.

While direct epidemiological studies linking MP exposure to CKD are still lacking, research on the health effects of plastic-associated chemicals, such as phthalates and bisphenols, offers valuable insights. A 2024 study reported that exposure to environmental phthalates was associated with alterations in kidney function biomarkers and an increased risk of reduced kidney function [30]. Similarly, longitudinal cohort studies in children with CKD have suggested an association between exposure to these chemicals and a decline in renal function [31]. These studies, while not focused on the plastic particles themselves, highlight the potential for plastic-related exposures to impact kidney health and underscore the need for further research into the combined effects of the particles and their chemical additives.

The primary challenge in conducting epidemiological research in this area is the difficulty in accurately assessing individual exposure to MPs. The development and validation of reliable biomarkers of exposure, such as quantification of MPs in blood or urine, are critical for future prospective cohort studies. Such studies are urgently needed to determine the dose-response relationship between MP exposure and the risk of CKD, as well as its progression. In the interim, the strong mechanistic data from experimental models, combined with the emerging evidence from cardiovascular and chemical-exposure epidemiology, provides a strong rationale for considering MPs as a novel and potentially significant environmental nephrotoxin.

Vulnerable populations: chronic kidney disease patients and dialysis populations

Patients with CKD, particularly those requiring dialysis, represent a population with heightened vulnerability to the toxic effects of MPs. This vulnerability stems from both impaired renal clearance of systemic toxins and direct, repeated exposure to plastic-containing medical equipment. The dialysis procedure itself, a life-sustaining therapy for patients with ESKD, has been identified as a significant and previously underrecognized source of MP exposure.

A groundbreaking 2025 study by Kara *et al.* systematically quantified MP contamination in both hemodialysis (HD) and peritoneal dialysis (PD) fluids. Their findings revealed the presence of MPs in all tested solutions, with concentrations of 0.29 ± 0.16 mp/l in HD solutions and 0.34 ± 0.02 mp/l in PD solutions [5]. While the concentrations were similar, the study estimated that PD patients have a weekly exposure that is ~50% higher than that of HD patients, due to the continuous nature of PD. The primary polymers identified were polyethylene, PVC, and ethylene-vinyl acetate, all common components of medical-grade plastics.

The sources of this contamination are numerous within the dialysis environment. The dialyzer membrane, the extensive tubing of the blood circuit, and the containers for dialysate and infusion fluids are all composed of polymers that can leach particles into the

blood and dialysate. A 2024 study on PVC infusion tubes confirmed the release of both MPs and NPs, highlighting the potential for iatrogenic exposure from a variety of medical devices [32]. For a patient on HD, who is exposed to over 120 l of water and dialysate three times a week, the cumulative burden of this exposure over years of treatment could be substantial.

The clinical implications of this chronic exposure are deeply concerning. With their renal function already compromised, CKD and dialysis patients have a diminished capacity to excrete absorbed MPs, leading to greater systemic bioaccumulation and a higher risk of long-term toxicity. This is particularly alarming given the association between MPs and cardiovascular disease, the leading cause of mortality in the dialysis population. The potential for MPs to exacerbate the already high baseline of inflammation, oxidative stress, and endothelial dysfunction in these patients could contribute to an accelerated progression of atherosclerosis and an increased risk of cardiovascular events, including sudden cardiac death [33].

Beyond the dialysis population, all patients with CKD may have an increased susceptibility to the effects of environmental MP exposure due to their impaired renal clearance and underlying systemic inflammation. Pediatric CKD patients and kidney transplant recipients on immunosuppressive medications also represent uniquely vulnerable subgroups that warrant further investigation. Addressing this challenge will require a multi-pronged approach, including the development of alternative, polymer-free materials for medical devices, the implementation of stricter quality control and filtration standards for dialysate, and enhanced clinical monitoring for the potential long-term consequences of MP exposure in this high-risk population (Table 3).

Key Points:
(a) All dialysis solutions tested contained MPs
(b) PD patients have higher weekly exposure than HD patients despite similar concentrations.
(c) Particle sizes differ significantly between HD and PD ($P = 0.030$).
(d) Primary sources: dialyzer membranes, tubing, dialysate containers.
(e) Vulnerable population with impaired clearance and chronic exposure.

Detection and quantification in clinical settings

The ability to accurately detect and quantify MPs in human biological samples is fundamental to advancing our understanding of their clinical impact and to developing strategies for risk assessment and mitigation. While still an emerging field, several analytical techniques have been adapted for this purpose, each with its own strengths and limitations. The development of reliable, standardized, and clinically feasible methods for MP detection is a critical research priority.

Urinary biomarkers offer a promising avenue for noninvasive exposure assessment. The detection of MPs in urine, first reported by Massardo *et al*. using Micro-Raman Spectroscopy, confirms that the kidneys are involved in the excretion of these particles [3]. This technique can identify the chemical composition and size of individual particles, providing detailed information about the nature of the exposure. However, the development of standardized protocols for urine collection, processing, and analysis is essential to ensure the comparability of results across studies. The quantification of MPs in urine could potentially serve as a proxy for systemic body burden, although the relationship between urinary excretion and tissue bioaccumulation is not yet fully understood.

Blood-based detection methods provide a direct measure of circulating MPs and are therefore a valuable tool for assessing systemic exposure and translocation. Pyrolysis-Gas Chromatography-Mass Spectrometry (Py-GC/MS) has been successfully used to quantify the mass concentration of various polymers in human plasma [15]. This method offers high

Table 3 Microplastic exposure in dialysis populations

Study	Dialysis type	Sample size	MP concentration	Particle characteristics	Clinical implications
Kara *et al*. [5]	Hemodialysis (HD)	15 samples	0.29±0.16 mp/l	Size: 1.31±0.98 mm; Polymers: PE, PVC, EVA; Predominantly fibers	Weekly exposure: 9.57±5.28 mp/L; Chronic cumulative burden
Kara *et al*. [5]	Peritoneal Dialysis (PD)	15 samples	0.34±0.02 mp/l	Size: 0.64±0.43 mm; Polymers: PE, PVC, EVA; Predominantly fibers	Weekly exposure: 14.28±0.84 mp/l (~50% higher than HD, $P<0.001$)
Zheng *et al*. [32]	IV infusion (general)	Laboratory analysis	MPs and NPs released from PVC tubes	Quantitative release from disposable medical devices	Iatrogenic exposure from medical equipment
Tereshchenko *et al*. [33]	Hemodialysis	Hypothesis paper	Not quantified	Plastic chemical-exposure from dialysis equipment	Potential contribution to sudden cardiac death
La Porta *et al*. (review)	General CKD/ dialysis	Literature review	Variable across studies	MPs detected in blood, urine; evidence lacking for kidney tissue at time	Urgent need for human studies on kidney disease-MP correlations

sensitivity and is capable of detecting a wide range of polymer types, including those in the nanometer size range. The main challenge for the clinical implementation of blood-based detection is the need for highly specialized equipment and expertise, as well as the potential for contamination during sample collection and processing.

Advanced imaging techniques may also have a role to play in the future of MP detection. Functional imaging with radiolabeled tracers, such as the use of 99mTc-DMSA (Technetium-99m dimercaptosuccinic acid) and 99mTc-DTPA (Technetium-99m diethylenetriaminepentaacetic acid) in murine models, has shown promise in identifying renal dysfunction associated with MP exposure [34]. While the direct imaging of MPs *in vivo* is not yet feasible, these functional imaging techniques could provide valuable information on the physiological consequences of MP bioaccumulation in the kidneys. The analysis of kidney biopsy tissue, while invasive, provides the most direct evidence of MP deposition and can be used to characterize the types and quantities of polymers present in the renal parenchyma. However, this is primarily a research tool and is not suitable for routine clinical screening.

The ultimate goal is the development of point-of-care technologies that can provide rapid and cost-effective screening for MP exposure. This will require significant innovation in sensor technology and sample processing. The standardization of all aspects of MP analysis, from sample collection to data reporting, is the most pressing challenge facing the field. The establishment of reference materials, inter-laboratory validation studies, and quality assurance programs will be essential for ensuring the accuracy and reliability of MP detection in clinical settings. The successful integration of these methods into clinical practice will be a critical step towards understanding the full spectrum of MP-related diseases and developing effective strategies for their prevention and treatment.

Public health implications and mitigation strategies

The growing evidence linking MP exposure to adverse health outcomes, including potential nephrotoxicity, necessitates a robust public health response. This response must be multi-faceted, encompassing policy interventions, technological solutions, and individual and collective behavioral changes. The goal is to reduce human exposure to MPs and mitigate the long-term health risks associated with these ubiquitous environmental contaminants.

Policy interventions at the national and international levels are crucial for addressing the root causes of plastic pollution. Organizations like the organisation for economic co-operation and development (OECD) and the US United States environmental protection agency (EPA) have outlined strategies that include reducing the production of unnecessary plastics, improving waste management infrastructure, and promoting a circular economy for plastics [35,36]. Specific measures, such as banning single-use plastics, implementing extended producer responsibility schemes, and setting standards for recycled content, can significantly reduce the amount of plastic waste entering the environment. The California Statewide MPs Strategy provides a comprehensive framework for state-level action, including monitoring, source reduction, and public awareness campaigns [37].

Technological solutions, particularly in the area of water treatment, are essential for reducing exposure through drinking water. Advanced filtration technologies, such as reverse osmosis, nanofiltration, and ultrafiltration, are highly effective at removing MPs from water, with some systems achieving removal rates of over 99% [38]. The implementation of these technologies in municipal water treatment plants and as point-of-use devices in homes can significantly reduce dietary exposure to MPs. In the healthcare setting, particularly in dialysis units, the adoption of advanced filtration for water and dialysate, along with the development of alternative, non-leaching materials for medical devices, is a critical priority for protecting vulnerable patients.

Individual actions, while not a substitute for systemic change, can also play a role in reducing personal exposure. These include choosing natural fabrics over synthetic ones to reduce the inhalation of fibers, avoiding plastic tea bags and single-use plastic containers, and using home water filtration systems. Public education and awareness campaigns are essential for empowering individuals to make informed choices and for building broad support for policy changes. Studies have shown that educational interventions, including those delivered via mobile applications, can be effective in improving knowledge, attitudes, and practices related to MP reduction [39].

Ultimately, addressing the public health threat of MPs will require a coordinated and sustained effort from all sectors of society. This includes a commitment from the plastics industry to innovate and design safer, more sustainable materials; a willingness from governments to implement and enforce effective regulations; and a collective effort from the public to reduce plastic consumption and waste. For the medical community, the challenge is to advance our understanding of the health effects of MPs, develop clinical strategies for

diagnosis and management, and advocate for policies that protect the health of our patients and the public.

Challenges, controversies, and future directions

Despite the rapid pace of research into the health effects of MPs, the field is still fraught with challenges, methodological controversies, and unresolved questions that must be addressed to move forward. A clear understanding of the long-term health risks of MP exposure will require a concerted and interdisciplinary research effort focused on overcoming these hurdles.

One of the most significant challenges is the difficulty in establishing a causal link between MP exposure and human disease in the absence of prospective, longitudinal cohort studies. The ethical and practical constraints of conducting human exposure studies necessitate a continued reliance on animal models and *in vitro* systems. However, the translatability of findings from these models to human health is a subject of ongoing debate. The high doses of MPs often used in experimental studies may not accurately reflect the chronic, low-dose exposure experienced by humans, and the physiological differences between species can influence the absorption, distribution, metabolism, and excretion of these particles.

Methodological controversies also abound, particularly in the area of MP detection and quantification. There is currently no universally accepted standard for the analysis of MPs in biological samples, leading to significant inter-study variability and making it difficult to compare findings across different research groups. The prevention of sample contamination during collection and processing is a major technical challenge, and there is ongoing debate about the relative merits of different analytical techniques, such as spectroscopy versus mass spectrometry, and particle counting versus mass measurement. The development of standardized methods and certified reference materials is a critical step for ensuring the reliability and comparability of data in this field.

Looking to the future, several key research priorities emerge. There is an urgent need for well-designed, prospective cohort studies that can track MP exposure over time and correlate it with clinical outcomes, particularly in high-risk populations such as individuals with CKD. The development and validation of sensitive and specific biomarkers of exposure and effect are essential for these studies. Mechanistic research should focus on elucidating the precise molecular pathways of MP-induced toxicity, with a particular emphasis on the effects of NPs, which may have greater toxic potential due to their ability to penetrate deeper into tissues and cells. The use of advanced *in vitro* models, such as human kidney organoids, can provide valuable insights into the cell-specific responses to MP exposure.

Furthermore, the field must move towards a more holistic understanding of the health risks of plastic pollution, considering not only the physical particles but also the complex mixture of chemical additives, adsorbed environmental pollutants, and co-exposures to other environmental stressors. The integration of advanced technologies, such as artificial intelligence for exposure modeling and wearable sensors for personal monitoring, may provide new tools for assessing risk and understanding the complex interplay between environmental exposures and human health. Ultimately, a collaborative, interdisciplinary approach that brings together nephrologists, toxicologists, environmental scientists, and public health professionals will be essential for addressing the multi-faceted challenge of MP pollution and protecting human health in the 21st century.

Conclusion

MP pollution represents an emerging nephrotoxic threat with clear pathways of human exposure, documented bioaccumulation in kidney tissue, and well-characterized mechanisms of cellular toxicity. The identification of MPs in human kidneys and the recognition of dialysis patients as a highly vulnerable population elevate this from an environmental concern to a clinical imperative.

For clinicians, these findings necessitate heightened awareness of environmental factors in CKD management. Practical steps include considering MP exposure in unexplained CKD progression, counseling patients on exposure reduction, and advocating for safer practices in healthcare settings. For policymakers, urgent action is needed: establishing contamination standards for drinking water and medical products, improving waste management infrastructure, and funding critical research.

Critical knowledge gaps remain. Large-scale epidemiological studies are urgently needed to definitively link MP exposure to CKD risk. The development of validated biomarkers and intervention trials assessing mitigation strategies, such as advanced water filtration, are essential next steps.

Addressing MP pollution requires coordinated action: interdisciplinary research collaboration, public health surveillance systems, and collective behavior change to reduce plastic consumption. The ubiquity of MPs reminds us of the intricate connections

between environmental and human health, offering an opportunity to act decisively to protect current and future generations.

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