



Microplastic and nanoplastic risks for hemodialysis patients and the environment

Faissal Tarrass, Meryem Benjelloun

Center of Hemodialysis 2 Mars, Casablanca, Morocco

The pervasive contamination of dialysate with microplastics (MPs, <5 mm) and nanoplastics (NPs, <1 µm) presents a critical iatrogenic risk in hemodialysis (HD). This life-sustaining treatment represents a significant exposure route due to its dependence on high-volume dialysate preparation (300–600 L/patient/week) and extensive polymer-based circuitry [1]. The patient's anephric state enables bioaccumulation with negligible renal clearance of these particulates (Fig. 1).

Primary contamination originates from the reverse osmosis (RO) water used for dialysate. While effective for larger particles, RO removal diminishes for NPs (<255 nm). Polyamide membranes themselves shed NPs via abrasion and chemical degradation [2]. Chlorine breakthrough can oxidize the polymer matrix, accelerating colloidal nanoplastic release into product water, with endogenous contributions quantified at approximately 2.08 µg/L [1]. Feed water is also a source, as treated drinking water contains 6.40 to 8.80 MP particles/L [3]. A secondary critical source is dialysate concentrates. Recent analyses confirm they are a direct vector, with detected MPs including polyethylene, polyvinyl chloride (PVC), and ethylene-vinyl acetate polymers, consistent with packaging materials [4].

A secondary, endogenous source is the extracorporeal blood and dialysate circuits, comprising di(2-ethylhexyl) phthalate (DEHP)-plasticized PVC tubing, polycarbonate connectors, and polysulfone dialyzer membranes. These materials are degraded by roller pump heads and pressure

fluctuations, releasing particles directly into the blood path [1]. Concurrently, plasticizers such as DEHP leach into the blood circuit, with intradialytic concentrations reaching 0.2 mg/kg of body mass [5]. Notably, while MP concentrations in HD and peritoneal dialysis fluids are similar, the particles in HD solutions are significantly larger [4].

The toxicological implications for HD patients are direct and multifaceted. The nanoscale size of NPs facilitates translocation across biological barriers. This risk is supported by evidence of significant MPs and NPs bioaccumulation in human organs, with the brain exhibiting particularly high concentrations [6]. At a cellular level, NPs can induce mitochondrial dysfunction, generating reactive oxygen species that activate the NOD-like receptor family pyrin domain containing 3 inflammasome and directly worsen chronic inflammation in HD patients [7]. This cellular damage is driven by well-characterized pathways of oxidative stress and programmed cell death. Furthermore, NPs in dialysate may act as carriers for adsorbed contaminants [8]. The leached DEHP metabolites function as endocrine-disrupting chemicals. Critically, the temporal correlation between peak serum plasticizer concentrations (approximately 6 hours post-dialysis) and the window of highest risk for sudden cardiac death suggests a plausible pathophysiological link [9].

Critical knowledge gaps hinder full risk assessment. Standardized methods are needed to quantify NPs in dialysate and blood. Furthermore, studies are urgently required on

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Correspondence: Meryem Benjelloun

Center of Hemodialysis 2 Mars, 466 Boulevard 2 Mars, Haddaouia 20460 Casablanca, Morocco. E-mail: hemodialysis2mars@gmail.com
ORCID: <https://orcid.org/0000-0001-7773-5732>

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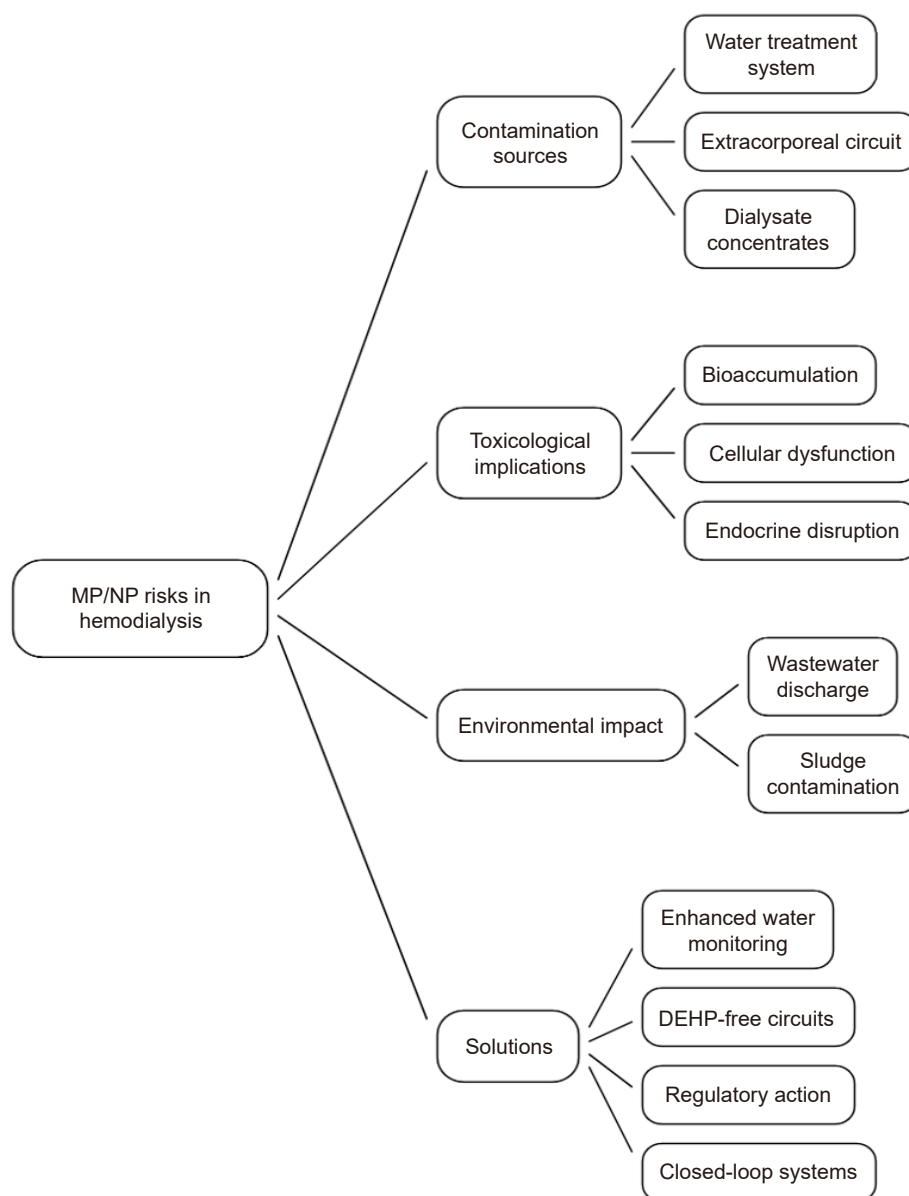


Figure 1. The lifecycle of microplastic (MP) and nanoplastic (NP) contamination in hemodialysis.

DEHP, di(2-ethylhexyl) phthalate.

MP removal in water treatment and whether online substitution fluids in hemodiafiltration act as direct intravenous particle infusions [1,10].

While *in vivo* toxicological evidence is strong, a critical gap between research and clinical practice remains. Prospective longitudinal clinical studies are required to directly link internal MP/NP body burden in HD patients with hard endpoints like cardiovascular mortality and the accelerated loss of residual renal function [8,9], a concern di-

rectly supported by *in vivo* evidence that MP/NP exposure induces glomerular damage, tubular injury, and interstitial fibrosis [8]. From a clinical perspective, research must prioritize the development of alternative, non-reactive materials for dialyzers and circuitry. This is supported by data showing microfiltration membranes achieve higher MP removal than adsorption-only systems [3]. Finally, regulatory frameworks for medical devices must account for chronic exposure to particulates from extracorporeal circuits.

The environmental impact of HD is substantial, creating a cycle where medical practice contributes to the pollution that feeds it. A standard HD center acts as a point-source emitter, discharging an estimated 8.4×10^{11} MP particles annually into wastewater via spent dialysate [1], a mass equivalent to roughly 50 kg of plastic waste. This effluent contains persistent polymer fragments and additive residues like DEHP. Furthermore, the RO water treatment systems themselves concentrate removed MPs into sludge (8,425–11,033 particles/kg), creating a secondary pollution hotspot [3]. This cycle highlights a critical externality, as the sector's emissions contaminate the broader water matrix that supplies the dialysis treatment, thereby inadvertently increasing the toxicological burden on its own vulnerable patient population.

In conclusion, HD is a significant convergence point for human and environmental MP/NP exposure. Mitigation requires a focused approach: enhanced dialysate quality monitoring and DEHP-free circuits in the short term; regulatory action on particulate limits in the mid-term; and the development of closed-loop systems and next-generation biocompatible materials in the long term. Addressing this issue is imperative to reducing an unquantified iatrogenic risk to a vulnerable patient population.

Conflicts of interest

All authors have no conflicts of interest to declare.

Data sharing statement

No datasets were generated or analyzed during the current study.

Authors' contributions

Conceptualization: MB, FT

Supervision: MB

Formal analysis, Resources: FT

Writing–original draft: MB, FT

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All authors read and approved the final manuscript.

ORCID

Faissal Tarrass, <https://orcid.org/0000-0002-9867-2096>

Meryem Benjelloun, <https://orcid.org/0000-0001-7773-5732>

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