



Health Implications of Microplastics in Drinking Water: A Systematic Review of Current Evidence [†]

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Abstract

This systematic review addresses the critical public health concern regarding the pervasive contamination of drinking water by microplastics (MPs). With global plastic production escalating, the degradation of waste into MPs has led to their ubiquity in freshwater sources. This review synthesizes recent findings on the occurrence of MPs in tap and bottled water and evaluates the associated toxicological risks to human health. Results indicate that humans ingest tens of thousands of particles annually, with drinking water being a significant vector. The review highlights that particles, particularly those in the nanoplastic range, can breach biological barriers, leading to bioaccumulation in secondary organs such as the liver and placenta. Toxicological impacts include oxidative stress, inflammation, and metabolic disruption.

Keywords: microplastics; drinking water safety; human health impacts; bioaccumulation; nanoplastics

1. Introduction

Global plastic production reached approximately 390.7 million tons in 2021, leading to widespread contamination of ecosystems and freshwater sources by microplastics (MPs), which are fragments smaller than 5 mm [1,2]. Consequently, human exposure is now inevitable through ingestion, inhalation, and dermal contact, with estimates of annual intake reaching up to 121,000 particles [3]. Drinking water is a major source of exposure, particularly bottled water, where mechanical stress on caps can release additional particles [1,4]. Once inside the body, the fate of MPs depends on their size. While larger particles generally do not cross the intestinal mucosa, nanoplastics (<100 nm) can penetrate the gastrointestinal epithelium, enter the circulation, and accumulate in organs such as the liver, kidneys, and spleen, as well as the placenta and lungs [5–8]. Beyond their physical presence, MPs are



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linked to oxidative stress and inflammation and can act as carriers for toxic chemicals and pollutants, potentially amplifying health risks [3,9,10].

This paper aims to review recent findings regarding the occurrence of MPs in drinking water and the associated toxicological risks to human health, with a specific focus on organ-specific impacts and the limitations of current risk assessment models.

2. Methods

This systematic review was conducted in compliance with PRISMA guidelines, searching PubMed/MEDLINE and Scopus for English-language peer-reviewed articles on microplastic toxicity in drinking water. The search strategy combined keywords regarding plastic particles, water sources, and health outcomes. Selection criteria followed a specific PICO framework (Table 1), including studies on human populations, cell lines, or mammalian models, while excluding non-toxicological or marine-focused research.

Table 1. PICO framework outlining Population (P), Intervention (I), Comparator (C), and Outcome (O).

Population (P)	Human populations, human cell lines (e.g., HEK293, HepG2), or mammalian animal models exposed to microplastics.
Intervention (I)	Exposure to microplastics or nanoplastics via ingestion (drinking water) or direct cellular contact.
Comparator (C)	Non-exposed control groups, healthy age-matched controls, or vehicle controls.
Outcome (O)	Biological impacts including oxidative stress, inflammation, cytotoxicity, metabolic disruption, or organ accumulation.

Screening was managed using Rayyan (version 1.4.3), with two independent, blinded reviewers assessing eligibility and a third resolving conflicts; the process is illustrated in a PRISMA flow diagram (Figure 1). Data extraction was also performed in duplicate to capture study design, polymer characteristics, and toxicological findings. Due to the heterogeneity of the included studies, results were synthesized narratively.

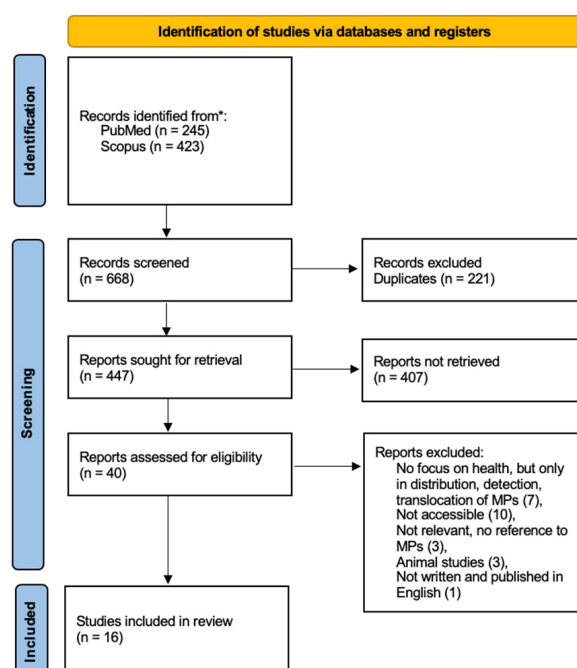


Figure 1. PRISMA flow diagram [11].

3. Results

PRISMA Flow Diagram

The initial search identified 668 articles (245 PubMed, 423 Scopus). After removing 221 duplicates and excluding 407 ineligible records, 40 full-text articles were assessed. A further 34 were excluded due to reasons such as lack of focus on human health, unavailable text, or animal-only models. Ultimately, 16 articles were included for qualitative synthesis. Key results (Table 2) indicate that particles <150 µm penetrate the GI tract [5], nanoplastics accumulate in atherosclerosis sites [12], and MPs act as vectors for pollutants [3], causing oxidative stress [9], hepatic lipid disruption [7], and reproductive toxicity [13,14].

Table 2. Summarizing results of the included papers.

#	Study	Main Results
1	Aarushi A, et al., 2025. [5]	High concentrations in Asian waters; particles <150 µm penetrate GI tract.
2	Li T, et al., 2025. [7]	Chronic exposure disrupts hepatic lipid metabolism via Kupffer cells.
3	Jahan Rakib M, et al., 2025. [9]	Linked to oxidative stress and endocrine disruption.
4	Duggal B, et al., 2025. [12]	Nanoplastics accumulate in atherosclerosis sites; cardiovascular risk.
5	Hoang H, et al., 2025. [3]	MPs act as vectors for pollutants; cause oxidative stress.
6	Shokunbi OS, et al., 2025. [4]	Higher estimated daily intake for children; packaging contamination.
7	Pinillos I, et al., 2025. [6]	Translocation to liver/spleen via Peyer’s patches.
8	Mokgalaka-Fleischmann N, et al., 2024. [13]	Confirmed presence in lung tissue and blood matrix.
9	Bhat M, et al., 2024. [2]	Heavy metal contamination; synergistic carcinogenic risk.
10	Li Y, et al., 2024. [14]	Neurotoxicity and reproductive toxicity (sperm quality reduction).
11	Yang Z, et al., 2023. [8]	Detection in placenta/breast milk; maternal–fetal transfer risks.
12	Yang X, et al., 2022. [15]	Subcellular effects: DNA damage and ROS production.
13	Kumar R, et al., 2022. [16]	Mechanisms of carcinogenesis; cytotoxicity and cytokine secretion.
14	Wu P, et al., 2022. [17]	ADMET processes; smaller particles cross barriers more easily.
15	Râpă M, et al., 2023. [1]	BPA leaching from plastics poses endocrine risks.
16	Goodman KE, et al. [10]	Metabolic decline, cell proliferation inhibition, and ROS generation.

Drinking water has been identified as a significant vector for human exposure to microplastics (MPs), with bottled water presenting heightened risks due to packaging-derived contamination [1,2,4]. Children are particularly vulnerable, showing significantly higher estimated daily intake rates than adults [4,15–19]. Upon ingestion, particle size dictates biological fate [17]: while larger particles (>150 µm) generally remain in the gut, those smaller than 10 µm—especially nanoplastics—can penetrate the gastrointestinal epithelium via endocytosis or paracellular transport [6,14,20–24]. This breach allows MPs to enter the circulatory system and translocate to secondary organs, as evidenced by their detection in human blood, lung tissue, and the placenta [8,25–32]. At the cellular level, toxicity is primarily driven by oxidative stress [3,20,33]. Studies on human liver and kidney cells exposed to polystyrene microspheres reveal dose-dependent increases in Reactive Oxygen Species (ROS) and the downregulation of critical antioxidant enzymes like SOD2 and CAT, resulting in DNA damage, metabolic decline, and severe structural deformation such as cell blebbing [10,16,23,34]. These cellular mechanisms translate into distinct organ pathologies. In the liver, chronic exposure disrupts lipid metabolism by polarizing Kupffer cells toward a pro-inflammatory phenotype, driving hepatic steatosis [7,34–39]. Cardiovascular risks

are also elevated, as nanoplastics can accumulate in atherosclerotic plaques, exacerbating inflammation [12,15,40]. Furthermore, the ability of MPs to cross the placental barrier raises concerns regarding fetal development and metabolic health [9,28,41], while in the gut, they induce dysbiosis and compromise the intestinal barrier, potentially facilitating further systemic contamination [6,29,30].

4. Discussion

The synthesis of data reinforces drinking water as a primary exposure route for microplastics (MPs), characterizing them not merely as physical contaminants but as biologically active stressors capable of inducing oxidative imbalance and systemic toxicity. A critical determinant of this risk is particle size; nanoplastics (<100 nm) pose a heightened threat due to their ability to traverse physiological barriers, including the blood–brain barrier, where they may disrupt neuronal homeostasis and drive neurodegenerative pathologies [42]. This systemic bioavailability has profound implications for cardiovascular health, with a landmark 2024 study linking MP accumulation in carotid artery plaques to a significantly increased risk of myocardial infarction, stroke, and mortality [43]. Concurrently, MPs disrupt metabolic function, acting as potential obesogens that promote insulin resistance and lipid accumulation [22]. Beyond intrinsic toxicity, MPs serve as vectors for chemical pollutants and host “plastisphere” biofilms, which selectively enrich antibiotic-resistant bacteria and facilitate gene transfer to the human microbiome [44,45]. These risks are magnified during early development, as infants face higher intake rates and possess immature physiological barriers, with confirmed placental transfer suggesting exposure begins in utero [46]. However, the assessment of these hazards is currently limited by methodological heterogeneity, a reliance on acute high-dose models [47], and a lack of standardized regulatory protocols for quantification [48,49]. Consequently, future research must prioritize the validation of high-throughput analytical methods and the establishment of longitudinal cohorts to map chronic health outcomes [50].

5. Conclusions

The ubiquity of microplastics in drinking water represents a multifaceted public health challenge. The current body of evidence indicates that these particles are bioavailable, capable of systemic translocation, and act as vectors for chemical and biological contaminants. The emerging clinical associations with cardiovascular pathology and neurotoxicity underscore the urgency of integrating microplastic monitoring into water safety protocols. Developing standardized detection methodologies and advanced filtration technologies is imperative to mitigate the health risks associated with this pervasive pollutant.

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