

Microplastics and Human Health: A Narrative Review of Organ-Specific Effects

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Abstract

Background: Microplastics (MPs; < 5 mm) and nanoplastics (NPs; < 1 μm) are emerging pollutants increasingly causing environmental and health concerns due to their widespread presence and poor biodegradability. Humans are exposed to these particles through ingestion, inhalation, and dermal contact.

Methods: This narrative review explores the impact of microplastics on human health by assessing exposure pathways and organ-specific effects. Studies (2010–2024) from major databases were synthesized by polymer type, exposure route, and observed outcomes across key body systems.

Results: Studies have reported that nylon microfibers impair respiratory health by releasing toxic compounds that inhibit epithelial cell differentiation, disrupt tissue repair, and suppress Hoxa5 transcription factor expression. Evidence also indicates that MPs alter nasal and gut microbiota composition, potentially contributing to respiratory, digestive, and immune disorders. Some studies suggest that exposure to sources such as clothing dryers can modify airway protection gene expression. Additionally, MPs disrupt cellular signaling pathways and have been observed to accumulate in male reproductive tissues, including the prostate and semen, raising concerns about infertility and prostate cancer risk.

Conclusion: Further research is urgently needed to clarify the long-term health effects of MPs, particularly regarding gastrointestinal function and chronic disease. This review highlights that MPs enter the human body through multiple pathways and exert detrimental effects on various organ systems. Effective mitigation strategies and improved public awareness are essential to reduce exposure and protect human and environmental health.

Keywords: Emerging pollutants, Infertility, Microplastics, Respiratory, Microbiota

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Introduction

Every day, humans ingest, inhale, and encounter microplastics (MPs), polymer particles smaller than 5 mm, and nanoplastics (< 1 μm), which have become ubiquitous environmental pollutants (1,2). These particles, composed of synthetic organic polymers such as polyethylene (high and low density), polystyrene, polypropylene, polyvinyl chloride, polyurethane, and polyethylene terephthalate, exhibit variability in size, shape (e.g., spherical, fibrous, or fragmentary), and chemical composition (3). Their small size and persistent nature allow them to permeate various environmental compartments, including air, water, and soil, making their study critical for understanding their implications for ecosystems and human health (2). The absence of a formal lower size limit for MPs, typically classified below 1 μm , underscores the complexity of

assessing their behavior and impact (2).

MPs originate from both primary and secondary sources, contributing to their widespread environmental distribution (4). Primary MPs are intentionally manufactured for use in consumer products, such as microbeads in cosmetics, facial scrubs, toothpastes, and hygiene products (4). Secondary MPs form through the degradation of larger plastics, such as plastic bags, bottles, fishing nets, and car tires, via physical, chemical, and biological processes, including weathering, UV radiation, and microbial activity (4). Synthetic textiles, such as polyester and nylon, release microfibers during washing, significantly contributing to aquatic and atmospheric pollution (5). Additionally, plastics often contain additives like bisphenol A, phthalates, polybrominated diphenyl ethers, and metals, which may act as carcinogens



or endocrine disruptors, further complicating their environmental and health impacts (6,7).

The ubiquitous presence of MPs raises profound concerns for both human health and environmental sustainability. Humans are exposed to MPs through multiple pathways, including ingestion of contaminated food (e.g., fish, shellfish, and vegetables), drinking water (both bottled and tap), inhalation of airborne particles in urban and industrial areas, and dermal contact via cosmetics and textiles (8,9). These exposure routes may induce adverse health effects, such as oxidative stress, chronic inflammation, and disruption of gut microbiota composition and metabolism, potentially leading to respiratory, gastrointestinal, reproductive, and immune system disorders (10,11). Environmentally, MPs contaminate aquatic, terrestrial, and atmospheric ecosystems, disrupting food chains and ecological balance (8). For instance, MPs in marine environments affect aquatic species, which in turn enter the human food chain, amplifying exposure risks (9). The variability in MP characteristics and their ability to transport chemical pollutants, such as heavy metals and polycyclic aromatic hydrocarbons, highlights the urgent need for comprehensive research to elucidate their long-term effects and develop effective mitigation strategies (12,13).

This narrative review comprehensively synthesizes current knowledge on the effects of microplastics on human health, with a particular focus on organ-specific impacts. We also seek to identify existing knowledge gaps and highlight future research priorities to inform risk assessment and mitigation strategies. This review is among the few to comprehensively synthesize evidence on the organ-specific impact of microplastics on human health, integrating findings from both experimental and epidemiological studies. By focusing on affected organs, this work provides a unique perspective that bridges environmental exposure data with clinical implications, offering guidance for future mechanistic research and public health interventions.

Materials and Methods

This review investigates the effects of MPs on human health, focusing on entry pathways and impacts on various organs. The extracted data from selected studies were analyzed using a narrative synthesis approach and thematic grouping, and the findings were categorized based on affected organ systems and exposure characteristics. This narrative review was conducted following the PRISMA guidelines, adapted for narrative synthesis to ensure transparency and methodological rigor. A PRISMA flow diagram is provided in [Figure 1](#) to illustrate the study selection process, including the number of studies identified, screened, and included.

A literature search was conducted in reputable scientific databases, including PubMed, Scopus, Web of Science,

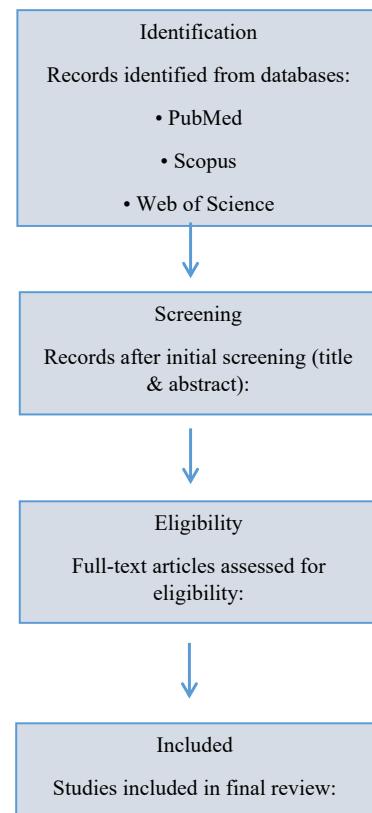


Figure 1. PRISMA flow diagram illustrating the literature search and selection process for studies investigating the effects of microplastics on human health

and Google Scholar, by applying the following keywords: “microplastics,” “human health,” “exposure pathways,” “ingestion,” “inhalation,” and “dermal contact.” Boolean operators (AND, OR, NOT) and temporal filters (2010–2025) were applied for comprehensiveness. A total of 19 studies were included based on their direct investigation of MP exposure and associated health outcomes in human or mammalian models. Studies lacking clearly defined outcome measures or exposure details were excluded. The reference lists of key articles were manually reviewed to identify additional studies. Inclusion criteria encompassed original articles, reviews, and meta-analyses in peer-reviewed journals addressing effects on respiratory, gastrointestinal, reproductive, immune, circulatory, dermal, and skeletal systems. Studies solely on environmental effects or non-human studies without human relevance were excluded. Titles and abstracts were initially screened to eliminate irrelevant studies, followed by full-text evaluation by two independent researchers. Discrepancies were resolved by discussion with a third researcher. Extracted data included author, publication year, study type, MP type, exposure route, reported effects, and proposed mechanisms, which were qualitatively analyzed.

Results

To systematically evaluate the impact of microplastics

(MPs) on human health, findings from 19 studies published between 2016 and 2024 were synthesized. These studies, conducted in diverse regions including China, the Netherlands, Germany, Italy, and the United States, reflect the global scope of this issue. Following PRISMA guidelines, 19 studies were included from an initial pool of 279 identified records (see Figure 1).

Analytical Methods for MP Detection

Advanced analytical techniques were employed to detect MPs in human samples (e.g., blood, feces, semen, skeletal tissues, and laboratory models). Yang et al utilized pyrolysis-Gas Chromatography-Mass Spectrometry (pyrolysis-GC-MS) to characterize MP compositions. The detection limits (LOD) ranged from 0.10 µg/mL to 0.68 µg/mL. The limits of quantification (LOQ) for the same method ranged from 0.33 µg/mL to 2.3 µg/mL (14,15).

Biological Effects of MPs

This evidence shows MPs enter the human body via ingestion, inhalation, and dermal contact, contributing to adverse health outcomes, inducing oxidative stress (reported in 16 studies), inflammation (16 studies), and metabolic disorders (5 studies) (16). The diversity of MP characteristics, including polymer type (e.g., polystyrene, polyethylene, polypropylene, and polytetrafluoroethylene), morphology (spherical, fibrous, fragmentary, or mixed), and size (nanometers to millimeters), influences biological interactions. For instance, small spherical particles (e.g., polystyrene, 0.5–3 µm) are readily internalized by macrophages, inducing cellular toxicity (17), while larger fibrous MPs (e.g., polyester, 200–800 µm) impair airway epithelial cell differentiation, potentially exacerbating respiratory conditions (5). MPs also act as vectors for chemical pollutants, such as phthalates and polycyclic aromatic hydrocarbons (PAHs), increasing risks of carcinogenicity and endocrine disruption. These pollutants increase the risks of carcinogenicity and hormonal disruption (18,19). The global distribution of MP exposure, spanning urban and rural settings in Asia, Europe, and North America (20), underscores global exposure, highlighting the need for coordinated international research.

Quality Control Measures

To ensure data reliability, most studies implemented rigorous quality control measurements, including vacuum filtration with polytetrafluoroethylene (PTFE) membranes, procedural blanks to monitor background contamination, and ethanol rinsing of glassware (21,22). Advanced imaging and validated simulation models further enhanced data accuracy (23). However, a subset of studies, particularly those using laboratory or simulation-based models, lacked detailed reporting of quality control protocols, representing a limitation in methodological

transparency.

Research Gaps and Limitations

Data from 19 studies provide compelling evidence of MPs' ubiquitous and detrimental effects, emphasizing the need for further research into long-term exposure risks and mitigation strategies. Key gaps include limited data on vulnerable populations (e.g., pregnant women, the elderly, and children) and the need to quantify chronic, low-dose exposure risks. Standardized protocols for MP detection are also required to enhance comparability across studies. Table 1 summarizes key findings, including target organs, MP types and morphologies, analytical methodologies, and associated health effects.

Discussion

Table 1 in the results section summarizes the effects of MPs on various organ systems, discussed below.

Respiratory System

MPs enter the respiratory system through inhalation of polluted air, particularly in urban and industrial areas, where concentrations are elevated. Particles smaller than 5 µm penetrate deep into lung alveoli, inducing oxidative stress and inflammation, which may contribute to respiratory disorders, as evidenced by clinical reports of MPs in lung tissue (36). For instance, Winkler et al demonstrated that MP fibers from clothing dryers suppress SCGB1A1 gene expression, compromising the lung's protective barrier and impairing airway repair (35). Similarly, Nylon MP fibers release chemical compounds that upregulate Hoxa5 transcription factor expression, inhibiting airway epithelial cell differentiation and reducing lung organoid formation in human and murine models (24). These effects are particularly concerning for vulnerable populations, such as children and individuals with chronic lung diseases, who rely on robust epithelial repair (24). Particle size, shape, and breathing significantly influence deposition patterns; larger non-spherical particles accumulate in the upper airways and bifurcations at higher breathing rates (23). Furthermore, MPs alter nasal microbiota composition, potentially exacerbating respiratory and immune disorders (30).

Gastrointestinal System

Ingestion of MPs through contaminated food, particularly seafood, and drinking water represents a primary exposure route. During digestion, MPs release heavy metals (e.g., chromium and lead), contributing to toxic exposure (27). They cause structural damage to the gastrointestinal tract, including villi cracking and reduced mucus secretion, which weakens the intestinal barrier. This damage induces systemic inflammation by increasing inflammatory cytokine production and recruiting immune cells, such as lymphocytes and mast

Table 1. Overview of selected studies on microplastic exposure and associated health effects

Author and year	Country	Target organ	Polymer type	Particle shape	Particle size	Analytical methods	Main Effects	DOI	References
Yang et al, 2025	China	Skeletal tissues (bone, cartilage, and intervertebral disc)	Polypropylene, ethylene vinyl acetate, polystyrene, PTFE ^a	Fragments, fibers, microspheres	44.25–39.407 µm	Raman micro spectroscopy	Inflammation in skeletal tissues, disruption of bone metabolism	10.1016/j.envint.2025.109316	(15)
Song et al, 2022	Netherlands, Germany, UK	Lung (airway epithelium)	Nylon, polyester	Not specified	31 µm, 52 µm	Mass spectrometry	Hazardous for children and lung disease patients	10.1164/rccm.202211-2099OC	(24)
Amato-Lourenço et al, 2024	Germany, Brazil	Brain olfactory bulb	Polypropylene, polyamide, nylon	Fragments (75%), fibers (25%)	5.5–4.26 µm	µFTIR	Presence in the olfactory bulb, penetration into the nervous system	10.1001/jamanetworkopen.2024.40018	(21)
Wang et al, 2023	China	Skin (cancerous and normal cells)	Polyethylene	Not specified	1 µm	Fluorescence microscopy, appropriate controls, siRNA, inhibitors	Increased proliferation of skin cancer cells, damage to normal cells	10.1016/j.ecoenv.2023.115636	(25)
Zhang et al, 2024	China	Male reproductive system (semen and urine)	Polystyrene, polypropylene, PTFE	Fragments, spherical, fibers	50–500 µm	Raman micro-spectroscopy analysis	Reduced sperm quality (concentration, count, motility)	10.1016/j.ebiom.2024.105369	(26)
Godoy et al, 2020	Spain	Gastrointestinal system (stomach and intestine)	Polyethylene, polypropylene	Crushed fragments	<5 mm	ICP-mass	Presence in the gastrointestinal system	10.3390/su12114792	(27)
Visalli et al, 2021	Italy	Intestinal epithelial cells	Polystyrene	Spherical	3 and 10 µm	Fluorescence microscope	Increased ROS production, DNA damage, and disruption of the intestinal barrier with prolonged exposure	10.3390/ijerph18115833	(28)
Brynzak-Schreiber et al, 2024	Austria	Gastrointestinal system (colorectal cancer cells)	Polystyrene	Spherical	0.25–10 µm	MTT ^b assay	Presence in cancer cells and increased metastasis risk	10.1016/j.chemosphere.2024.141463	(29)
Zhang et al, 2022	China	Nasal and gut microbiota	Polyethylene, PVC, PTFE	Not specified	<5 mm	Infrared imaging	Alteration of nasal and gut microbiota	10.1097/MD.00000000000030215	(30)
Adler et al, 2024	Germany	Immune system (macrophages)	Polystyrene	Spherical (micro- and nanobeads)	0.5–3 µm	Electron microscopy and live imaging for validation	Uptake by macrophages, increased toxicity, necrosis, and NO production	10.1016/j.jhazmat.2024.134253	(17)
Leslie et al, 2022	Netherlands	Bloodstream	PET, polyethylene, polystyrene	Not specified	≥700 nm	FTIR	Presence in blood	10.1016/j.envint.2022.107199	(14)
Lee et al, 2023	South Korea	Breast cancer cells	Polypropylene	Fragments	4.16 µm	SEM	Effect on cancer cells	10.1038/s41598-023-33393-8	(31)
Deng et al, 2024	China	Prostate (tumorous and para-tumorous tissue)	Polystyrene, polyethylene, PVC	Not specified	Not specified	LDIR and SEM	Presence in the prostate and associated with breast cancer	10.1016/j.ebiom.2024.105360	(22)
Hu et al, 2022	China	Gastrointestinal system	Polyethylene, polypropylene, and polystyrene	Not specified	0.125–0.15 mm	SEM, HPLC, and PBET	PAHs transfer in the intestine	10.1016/j.envint.2022.107459	(32)
Sheikh et al, 2016	Saudi Arabia	Endocrine system	Phthalates	Not specified	Not specified	Molecular modeling	Disruption of steroid homeostasis and hormonal signaling	10.1371/journal.pone.0151444	(19)
Riaz et al, 2024	Pakistan, Australia, Taiwan	Lung (airways)	Not specified	Spherical, cubic, cylindrical, tetrahedral	1.6–56.5 µm	Tracheobronchial airways	Accumulation in lung bifurcations	10.1063/5.0205303	(13)
Wu et al, 2023	China	Testis	Polystyrene	Spherical	1 µm	SEM	Premature testicular aging	10.1186/s12989-023-00546-6	(33)
Wang et al, 2024	China	Heart (cardiomyocytes)	Not specified	Not specified	1–100 nm	Fluorescence microscope	Effect on cardiac cells	10.1186/s12951-024-02375-x	(34)
Winkler et al, 2022	Italy	Human airways	Polyester	Not specified	200–800 µm	SEM and fluorescence microscope	Gene expression changes and airway organoid polarization	10.1016/j.envint.2022.107200	(35)

If any of the fields (e.g., polymer type, particle shape, size, or quality control) were not reported by the original studies, "Not specified" was entered in the table. a: Polytetrafluoroethylene; b: 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl-2H-tetrazolium bromide assay

cells, potentially affecting organs like the brain, though this requires further mechanistic validation (37). Fournier et al found that repeated exposure to polyethylene MPs promotes harmful bacteria and inflammatory metabolites, such as skatole, which may impair gastrointestinal health. However, their study reported no direct effects on intestinal permeability or IL-8 cytokine production, contrasting with other findings of increased permeability, possibly due to differences in MP size or exposure duration (37,38). This discrepancy underscores the need for standardized experimental protocols. MPs also reduce gut microbiota diversity, suppressing anti-inflammatory bacteria, increasing intestinal permeability, and facilitating bacterial toxin transfer to the bloodstream, potentially contributing to conditions like inflammatory bowel disease (37). The presence of plastic-degrading genes in the gut microbiome suggests microbial adaptation to MP pollution, a phenomenon warranting further investigation (39). Additionally, MPs serve as vectors for polycyclic aromatic hydrocarbons (PAHs), releasing up to 98% of these compounds in the intestine, resulting in a high carcinogenic risk (incremental lifetime cancer risk, ILCR $> 10^{-4}$) (32). Brynzak-Schreiber et al demonstrated that polystyrene micro- and nanoplastics exhibit persistent bioaccumulation in colorectal cancer cells, enhancing cell migration and increasing the risk of metastasis, highlighting the role of MPs in cancer progression (29). While these studies provide robust evidence, many rely on *in vitro* or animal models, which may not fully replicate human exposure conditions, necessitating longitudinal human studies.

Collectively, these findings position MPs as a multifaceted threat to gastrointestinal health, contributing to inflammation, microbiota dysbiosis, and elevated cancer risk. These insights emphasize the need for policies to reduce MP contamination in food and water, alongside standardized methods to assess their long-term health impacts (28).

Reproductive System

MPs have been shown to adversely affect reproductive health, with emerging evidence highlighting their presence in reproductive tissues and associated toxicities (40). Codrington et al detected MPs, predominantly polyethylene terephthalate (47.8%) and polypropylene (34.7%), in human penile tissue and semen, suggesting potential entry via the epididymis and seminal vesicles (40,41). However, the cross-sectional design of this study limits causal inferences, underscoring the need for longitudinal research to confirm these findings. Higher concentrations of MPs, including polystyrene, polyethylene, and polyvinyl chloride, were observed in tumorous prostate tissues compared to para-tumorous tissues, with observational studies suggesting a correlation with fast food consumption, potentially increasing

prostate cancer risk (22). Further mechanistic studies are needed to validate this association.

Mechanisms of Reproductive Toxicity

Long-term exposure to polystyrene MPs activates the Ca^{2+} /ROS/NF- κ B signaling pathway, causing premature testicular aging by impairing mitophagy and promoting the accumulation of damaged mitochondria, which may compromise male reproductive health (33). Additionally, Polytetrafluoroethylene (PTFE) from non-stick cookware, likely ingested through contaminated food, reduces sperm count, motility, and quality, with greater MPs diversity exacerbating these effects (26).

Immune System

MPs modify the physical and functional properties of microorganisms, acting as vectors for pathogens and impairing immune recognition and clearance (42). They modulate innate immune responses by activating macrophages, neutrophils, and the complement system (42). For instance, Adler et al demonstrated that polystyrene micro- and nanoplastics, when internalized by human macrophages, induce necrosis and elevate nitric oxide (NO) production, thereby impairing immune homeostasis (17). MP exposure triggers inflammation, damages the intestinal barrier, and alters the gut microbiome, contributing to inflammatory bowel diseases (43). These changes may increase the likelihood of infection and inflammation (44). These disruptions may exacerbate systemic inflammatory response, potentially interacting with effects on other organ systems, such as the gastrointestinal or circulatory system, as discussed elsewhere in this review.

Circulatory System

The detection of MPs in the human blood has serious implications (44). Sun et al showed polyamide and polyurethane (20–100 μm) transfer from maternal blood to umbilical cord blood and fetal appendages, raising concerns for maternal and fetal health (44). Lee et al reported that MPs disrupt blood coagulation and increase inflammatory markers, acting as a risk factor for cardiovascular disorders (31). Polypropylene MPs induce inflammation, histamine release, and ROS production, exerting harmful effects on blood cells, especially with prolonged or high-concentration exposure (45). Polystyrene nanoplastics induce oxidative stress, DNA damage, and cell division disruption, causing cytotoxic and genotoxic effects on peripheral blood lymphocytes (46). A significant correlation exists between plastic food container use and elevated MP levels in blood (31).

Dermal and Skeletal Systems

The skin is a significant exposure route for MPs through contact with textiles and airborne particles (47). Abbasi et

al reported that men in the urban area of Iran experience approximately twice the MP exposure compared to women, likely due to occupational and environmental factors (47). MPs containing flame-retardant additives, such as polybrominated diphenyl ethers, are absorbed through the skin, with up to 8% accumulation by mass under sweating conditions, as measured by dermal absorption assays (48). Furthermore, Wang et al demonstrated that MPs induce oxidative stress and activate the NLRP3 inflammatory pathway, leading to an increased production of pro-inflammatory cytokines (e.g., IL-1 β). This process promotes skin cancer cell proliferation and damages healthy epidermal cells, potentially acting as a tumorigenic trigger (25).

Beyond dermal exposure, MPs infiltrate the skeletal system via systemic circulation, accumulating in intervertebral discs (25.44–407.39 μm) and potentially disrupting skeletal health. These particles elevate inflammatory markers and impair bone metabolism, as evidenced by increased cytokine levels in affected tissues (15).

Conclusion

Microplastics (MPs) pose a significant challenge to human health and environmental sustainability due to their pervasive presence and diverse biological effects. Reviewed studies demonstrate that MPs induce oxidative stress, chronic inflammation, and metabolic disorders across multiple organ systems, including the respiratory, gastrointestinal, reproductive, immune, circulatory, dermal, and skeletal systems. Specifically, fine MPs (<5 μm) trigger inflammation in lung alveoli, with implications for vulnerable populations such as children and those with chronic lung diseases. In the gastrointestinal system, MPs reduce gut microbiota diversity and weaken the intestinal barrier, increasing systemic inflammation and elevating cancer risk through mechanisms such as enhanced cell migration. In the reproductive system, MPs, particularly polystyrene, contribute to premature testicular aging via the $\text{Ca}^{2+}/\text{ROS}/\text{NF-}\kappa\text{B}$ signaling pathway, impairing mitophagy and reducing sperm quality. Immune responses are disrupted by macrophage necrosis and altered pathogen interactions, increasing susceptibility to infections. Additionally, MPs in the blood are associated with cardiovascular risks through inflammation and disrupted coagulation, while dermal and skeletal systems face inflammation and tumorigenesis risks via pathways like NLRP3 activation.

While most studies report the adverse effects of MPs on human health, several inconsistencies exist. For example, some *in vitro* studies demonstrate significant inflammatory responses at low concentrations, while others observe no notable effects under similar exposure levels. These discrepancies may stem from differences in particle size and shape, polymer composition, or

experimental protocols. A major research gap remains in understanding the effects of long-term, low-dose exposure under realistic conditions. Moreover, emerging evidence suggests that MPs may activate signaling pathways such as $\text{Ca}^{2+}/\text{ROS}/\text{NF-}\kappa\text{B}$ and induce mitochondrial dysfunction; however, these mechanisms require further validation in human models. Bridging these gaps through well-designed *in vivo* and longitudinal studies is critical to clarifying the dose-response relationships and biological consequences of MP exposure.

Despite advances in detection methods, limitations such as a lack of standardized protocols, limited data on nanoplastics, and unknown toxicity mechanisms complicate interpretation. Future research should develop precise detection technologies, such as advanced imaging, and conduct long-term cohort studies to investigate chronic effects on cancer, diabetes, and cardiovascular disorders. Studying combined effects with other pollutants and impacts on mental health and sleep is critical. Policy should prioritize banning primary MPs in cosmetics, improving plastic waste management, and promoting biodegradable materials. Raising public awareness through education and reducing single-use plastic consumption are key steps in minimizing exposure. This study underscores the need for collaboration among scientists, policymakers, and industry to develop innovative solutions. MPs affect both human health and ecosystem balance, making coordinated efforts to reduce their production and release a scientific and collective commitment to a sustainable future.

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Competing interests

The authors declare no competing interests, financial or otherwise, that could influence the objectivity or outcomes of this review. No funding was received, and the authors have no affiliations with organizations or entities that could benefit from this work.

Ethical issues

This review was conducted in accordance with ethical guidelines for scientific research. As no human or animal subjects were involved, ethical approval was not required. All data were sourced from publicly available peer-reviewed publications, and proper citation practices ensured academic integrity. The authors adhered to principles of transparency and objectivity in study selection and analysis.

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