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REVIEW PAPER

Microplastics – why are they everywhere within the body and where do they come from? A review

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Abstract

Microplastics (MPs) have been detected in nearly all environmental compartments. The crucial routes of human exposure include the indoor environment, where the concentration of airborne fibers is higher than outdoors, the consumption of contaminated food, and contact with cookware, medical equipment, and personal care products made from or containing plastic. Depending on how MPs enter the body, through ingestion, inhalation, or dermal contact, they can reach a variety of organs. In this study, we focus on the detection of MPs and their consequences in several organs, which have not been sufficiently described in literature before, including the kidneys, placenta, testes, heart, brain, bone marrow, and in bodily secretions, namely semen and breast milk. To ensure the most accurate and up-to-date data, the presented results are based on papers published within the past three years. Experimental and clinical studies link exposure to MPs to oxidative stress, inflammation, dysbiosis, mitochondrial damage, fibrosis, and potential carcinogenic and neurotoxic effects. These mechanisms, occurring in the aforementioned tissues, raise concerns regarding fertility, fetal development, metabolic health, and neurodegeneration. However, further improvements in detection of MPs and contamination prevention methods are needed to minimize the potential health risks associated with exposure.

Keywords: microplastics, nanoplastics, environmental exposure, bioaccumulation, toxicity

Introduction

Microplastics (MPs) are an emerging environmental pollutant that has garnered significant attention due to potential adverse effects on human health and ecosystems. They are defined as plastic particles smaller than five millimeters in diameter [1]. Global plastic output is expected to increase from 390.7 million tons in 2021 to up to 590 million tons by 2050. In comparison to the early 1950s, when yearly plastic manufacturing was around 1.7 million tons, this underscores the magnitude of this increase [2]. Due to their minuscule size, they are almost ubiquitous, inevitably resulting in a buildup in ecosystems such as seas, soils, or the atmosphere,

moving up the food chain and eventually making their way into the human body [3]. Microplastics and nanoplastics (MNPs) may gather in different tissues and organs following consumption, inhalation, or absorption through the dermal layer, leading to further negative health impacts [4]. From zooplankton to higher trophic levels, including humans, a variety of species have been shown to bioaccumulate these particles [3]. MPs represent a rapidly expanding issue for human health and well-being in the Anthropocene, an epoch marked by humanity's significant effect on earth. Due in large part to the difficulties in measuring and identifying the degree to which MNPs accumulate in human tissues, the toxicological effects of consumption and inhalation of MPs are still largely unknown [5]. All human tissues and bodily fluids have been shown to contain MPs, indicating that they are actively absorbed, distributed throughout the body, and then either accumulated or removed. The highest concentrations have been reported in lung parenchyma (averaging 14.19±14.57 particles per gram), followed by the tonsils, small intestine, and large intestine. The most common polymer has been identified as polyvinyl chloride (PVC) [6]. Such contamination may affect a substantial proportion of the population based on the results of a study of 36 students, where MPs were detected in 83% of the blood samples [7]. Plastic is regarded as one of the four pillars of modern civilization [8] and plays an indispensable role in health care, where face masks, syringes, catheters, blood bags, sterile packaging, and bedding are used routinely. At present, plastic cannot be produced without endangering both people and the environment [9]. This issue is particularly relevant given the widespread use of plastic in everyday life, ranging from bottled water [10], through cosmetics and personal care products [11], to the kitchen, where plastic items are not only present but also subjected to mechanical forces, heat, and other damaging factors [12]. Preparation, cooking, serving, and storage of food using kitchen equipment made from or with the addition of polypropylene, polyethylene, or other polymers creates a risk of exposure to high amounts of MPs, especially taking into consideration that these items serve for a prolonged time in a repetitive manner [13].

Aim of the work

The aim of this work is to emphasize the significance and extent of accumulation of MPs in organs, highlighting various pathways via which MPs enter the body. Rather than concentrating on its impact on systems with direct exposure contact, such as the digestive or respiratory tracts, which have been extensively described within literature, this study focuses more on the presence of MPs and their impact on the cardiovascular, reproductive, and urinary

systems, also mentioning other specific sites of deposition, namely the brain, bone marrow, and breast milk.

Methods

A literature search strategy for this review was conducted using PubMed, Google Scholar, Elsevier, and Mendeley electronic databases based on a variety of combinations of specific key words: “microplastics”, “nanoplastics”, “bioaccumulation”, “microplastics exposure”, “pulmonary diseases”, “renal diseases”, “dermal barrier”, and “reproductive health”. To enhance the accuracy and relevance of the search, keywords were aligned with Medical Subject Headings (MeSH) terminology. The data gathering period spanned December 2025 to March 2026. To guarantee the review’s applicability and current coverage, primary focus was placed on 35 English-language original and review papers published during the previous three years. Publications unrelated to the article’s scope, or those concentrated exclusively on environmental impacts, were eliminated following an initial examination of the abstracts. All searches were conducted manually using the search engines mentioned above, and no specialized software tools were employed.

Literature review results

Routes of exposure

Entry of MPs into the human body is possible through various exposure routes. While acknowledging well-described reservoirs, including soil, water bodies, and the atmosphere, with the latter demonstrating greater concentrations of suspended MPs in coastal urban regions compared to the ocean [14], this study focuses on more specific indoor sources. The rationale for this approach is based on findings that indoor environments exhibit higher atmospheric concentrations of MPs than outdoor environments, in addition to a nearly threefold greater daily fallout rate observed in the São Paulo megacity [15]. It is worth mentioning that fibers, especially polyester, were more common in both settings compared to particles such as polyethylene (PE) and polypropylene (PP).

Similar conclusions were reached in a study which evaluated environmental and internal exposure to MPs in 26 college students in Changsha, considering dietary intake by assessing a 3-day food record and duplicate meal sampling, water exposure via a 7-day intake questionnaire

and sample collection, and air exposure through active indoor and outdoor sampling [16]. The results showed that MPs, predominantly PE and PVC, were present in 100% of samples and that exposure came mainly from diet, followed by air and water, totaling an average of 460.20 µg/kg bw/day. Here as well, concentration of MPs indoors was reported to be greater than outdoors. Additionally, the quantities of MPs were higher in meat than in rice, and bottled water and beverages contained more MPs than purified or even tap water. Overall, dietary intake was the main contributor to exposure to MPs, with internal accumulation reflecting ingestion patterns.

To enhance understanding of exposure to MPs from dietary intake, attention must also be directed toward cookware. Regarding potential food contamination by MPs, new culinary items made from PP, PE, or polyamide (PA), or those incorporating "non-stick" coatings such as silicone or polytetrafluoroethylene (PTFE), have been observed to release substantial quantities of MPs [17]. Although this number is slightly more than half the amount emitted by older plastic counterparts, it is over three times greater than the emissions from cookware manufactured from stainless steel and glass, which did not release significant amounts of particles, and the observed contamination resulted from their presence in the food. The study presenting these results was conducted using jelly powder as a proxy for food, allowing observations of the release of MPs into this "food simulant" during preparation with different cooking appliances. When extrapolated to daily home cooking, new and old plastic cookware could contribute 2,400-5,000 MPs annually.

A closer examination was conducted specifically on cutting boards [18]. The study model was based on mouse diets prepared by repeated cutting on PP, PE, and wooden boards over 12 cycles, generating increasing the concentrations of MPs, with older boards yielding significantly more particles. Despite minimal or undetectable bioaccumulation of heavy metals, plastic additives, and MPs in tissues, different biological effects were noted. PP MPs primarily stimulated the inflammatory reaction and damage to epithelial integrity, whereas PE MPs largely shifted the microbial and metabolic pathways. The results indicate that MPs from plastic cutting boards are released in increasing amounts with continued use and that negative biological effects can occur with exposure to PP and PE MPs through different mechanisms, thereby presenting potential health hazards due to long-term dietary exposure to MPs from meal preparation surfaces.

Not only can food handling be a source of generation of MPs, but storing food in plastic containers also contributes to this process. A study evaluating the release of nanoplastics (NPs)

from PP food storage containers provided clear evidence that even typical use leads to the emission of NPs and MPs, especially after exposure to high temperatures [19].

Ingestion as a way of exposure has a crucial meaning as it does not only affect the gastrointestinal tract. In an experimental study on murine models, diminutive polystyrene nanoparticles were able to traverse the blood-brain barrier within two hours post-ingestion, a mechanism contingent upon their biomolecular corona, with cholesterol-enriched coronas enhancing membrane penetration [20].

As the initial site where ingestion begins, the oral cavity should also be studied regarding processes occurring there. Particularly, novel findings indicate that orthodontic aligners release MPs compounds [21]. While clear orthodontic aligners offer aesthetic and functional advantages over fixed appliances, their plastic composition poses environmental and health risks. Unlike biocompatible metals or ceramics, aligners may release bisphenol A, leachable chemicals, and MNPs, particularly under extreme oral conditions characterized by fluctuations in temperature and pH, as well as the presence of saliva, enzymes, and masticatory pressures. Intraoral aging results in water absorption, biofilm formation, surface roughness, and material wear, which diminish mechanical performance and elevate particle release [22]. The extensive usage of non-biodegradable aligners contributes to plastic waste and to prolonged direct human exposure, as treatment may vary from months to years, potentially leading to ingestion. Aligners, predominantly made of polyethylene terephthalate glycol (PETG) and polyurethane (PU), have been shown in an *in vitro* study to detach MPs from their surfaces, mainly due to mechanical friction [23]. Furthermore, these devices are classified as medical waste, limiting safe recycling options.

Orthodontic aligners are not the only medical devices posing a risk of exposure to MPs. In fact, they are present in a wide range of medical supplies, such as surgical consumables (like gloves, dressings, and sutures), instruments (including syringes, needles, tissue biopsy kits, and automatic injectors), and patient care items such as bed safeguards and absorbent padding, all of which come into contact with skin, blood, or airways [24]. Face masks have been widely utilized as an accessible, inexpensive, and simple physical protective method to prevent infection since the COVID-19 pandemic. PP is the main component of meltblown fabric, which is the primary material utilized in the manufacture of surgical masks. One surgical mask may emit more than 1 billion MPs, which can be inhaled. It is anticipated that mask use will result in a significant amount of MPs being deposited in the nasal cavity and the lungs due to increased breathing frequency and prolonged wear duration. In a study conducted in China among healthy college students, an abundance of MPs in nasal lavage fluid has been found, which was

comparatively increased by masks usage [25]. Inhalation has also been identified as a route of deposition of MPs in the olfactory bulb via the olfactory pathway. The particles accumulated there were PP and nylon, which are among the most commonly manufactured and processed polymers used for clothing and packaging [26].

Dermatology is also intricately connected to the application of plastics. Many dermatological disorders still require topical treatments, and the vast majority of formulations comprise primary MPs. A significant source of MP deposition on the skin is synthetic textiles, washing of which produces at least 500,000 microfibers for every kilogram of clothes [24]. Other contributors include makeup, shampoos, toothpastes, sunscreens, and soaps. In personal care products, MPs may be present as primary particles intentionally added to formulations or as secondary particles derived from plastic packaging degradation. To reduce skin abrasion and interference with physiological skin processes, these particles are usually designed in spherical forms. Beyond exfoliation, MPs serve a variety of purposes, including binding, emulsifying, regulating viscosity, forming films, and polishing. Although other polymers like PP, polymethyl methacrylate (PMMA), PU, and nylon are also utilized, PE-based microbeads are the most often used [27]. It has been hypothesized that NPs may cross the dermal barrier and that contact of epithelial cells with MNPs can result in oxidative stress, even though the stratum corneum, the outermost layer of skin, offers a protective barrier against potentially damaging compounds in healthy conditions. Particle size, skin state (normal, inflammatory, or injured), and particle load on the skin all affect the extent to which MNPs and their additives are absorbed. Particles under 4 nm may easily permeate skin, whereas NPs between 21-45 nm can only penetrate damaged or impaired skin, such as in conditions of atopic dermatitis, ichthyosis, or bullous dermatoses [24,28].

Presence of MPs within the human body and their effect on selected organs

As the routes of entry of MPs into the body have already been established, this section of the article focuses on their reach and impact within the organs. As previously described, MPs penetrate the dermal layer. When they come into contact with Langerhans cells, dendritic cells, macrophages, and T cells, they interact with their toll-like receptors, thereby causing inflammation and oxidative stress. Additionally, they can be absorbed by phagocytosis or endocytosis, where they aggregate inside lysosomes and induce cellular death. They can exacerbate adolescent acne, allergic contact dermatitis, and contribute to the onset or aggravation of disorders including atopic dermatitis. Furthermore, they have been proven to

cause direct toxicity, oxidative damage, and microbial dysbiosis by accumulating around the cell nuclei by the way of entry through corneal and conjunctival epithelial cells following the use of contact lenses, mascara, or eye shadow [24]. Moreover, by regulating the inflammatory process, MPs promote the proliferation of skin cancer cells while inhibiting the growth of normal skin. The phenomenon has been investigated using two human cutaneous squamous cell carcinoma lines (SCL-1 and A431) and the HaCaT keratinocyte model. MPs were internalized by cancer cells in a time- and dose-dependent manner and significantly promoted tumor cell proliferation, as demonstrated by MTT assays, low cytometry, confocal microscopy, and Western blotting. Mechanistically, MP exposure increased mitochondrial ROS, disrupted mitochondrial membrane potential, induced mitochondrial permeability, pore opening, and triggered mitochondrial DNA release, leading to NLRP3 inflammasome activation and enhanced cancer cell proliferation. In contrast, MPs inhibited proliferation and induced cell cycle arrest and cell death in HaCaT cells through NLRP3-mediated inflammatory pathways, proving their dual biologic effects [29]. Beyond their role in inducing inflammation, airborne particles of MPs mediate the dispersal of antibiotic resistance genes through long-range atmospheric transport, thereby facilitating the global propagation of antibiotic resistance, exacerbating the difficulty of treating infections, while also posing an immediate inhalation risk [30].

MPs detected in higher levels in bronchoalveolar lavage fluid, predominantly in the form of microfibers, have been linked to female gender, smoking, living in upper-floor residences, microbial growth, specific parenchymal lung conditions, and reduced FEV1/FVC ratios [31]. They have also been proven to be a potential risk factor for the development and progression of asthma, chronic obstructive pulmonary disease, pulmonary fibrosis, and lung cancer. Moreover, exposure to polystyrene MPs has been shown to reduce the sensitivity of A549 and PC-9 lung cancer cell lines to radiation in *in vivo* mouse models, suggesting that MPs could limit the effectiveness of radiotherapy by disrupting radiation-induced ferroptosis via induction of oxidative stress [32]. Experimental studies demonstrate that airborne MNPs disrupt mitochondrial function, promote excessive reactive oxygen species generation, and activate other cell death pathways, including apoptosis and autophagy. Key signaling pathways, such as PI3K/Akt/mTOR and MAPK, become dysregulated, leading to mitochondrial dysfunction and epithelial injury. Particle deposition, which is governed by aerodynamic equivalent diameter (AED), occurs through Brownian diffusion, gravitational settling, and inertial impaction. Larger particles (5-10 μm) predominantly deposit in the upper respiratory tract, while intermediate-sized particles (2-5 μm) reach the tracheobronchial region. In contrast, smaller

particles (<1 μm) penetrate deeply into the alveoli, where they may cause irreversible lung injury. Ultrafine particles (<0.5 μm) can translocate into the bloodstream and lymphatic system, enabling systemic distribution [33].

Correspondingly, MPs may be selectively filtered by the glomerular filtration membrane (GFM) based on their size or molecular weight. In contrast to endogenous proteins, MPs are unlikely to be identified or reabsorbed by renal tubular epithelial cells, which facilitates their excretion in urine. Additionally, preliminary data indicates that larger MPs may translocate from peritubular capillaries to tubular epithelial cells in order to reach the urine. Simultaneously, *in vitro* digestion models show that MPs experience significant physicochemical changes in the gastrointestinal system, such as aggregation and creation of a protein corona, which increases their volume and surface charge, making it challenging for them to pass through GFM [34]. Long-term chronic exposure to MPs can result in renal fibrosis mediated by ferroptosis. In particular, renal fibrosis was seen in mice exposed to MPs for six months at human-accessible quantities (10 mg/L), evidenced by GPX4 downregulation, PTGS2 upregulation, altered ferroptosis-related gene expression, mitochondrial damage, and increased renal iron accumulation [35]. Furthermore, endoplasmic reticulum damage is a part of the pathomechanism leading to diseases such as diabetic nephropathy, acute kidney injury, and primary glomerular kidney damage [36]. What is concerning is that even fetuses are already being introduced to MPs induced nephrotoxicity. In China, 1,350 pregnant women were recruited for a large prospective study, in which high-resolution laser direct infrared imaging was used to analyze placental tissues obtained during delivery. Renal biomarkers such as creatinine, cystatin C, blood urea nitrogen test, neutrophil gelatinase-associated lipocalin test, and estimated GFR were measured in umbilical cord serum. All placental samples included MPs, with a median load of seven particles per 10 g of tissue. The mixture of MPs was significantly positively correlated with elevated infant creatinine and cystatin C levels, indicating early renal damage, according to weighted quantile sum regression. The main contributor was found to be PVC, which was followed by PP and polybutylene succinate [37].

Numerous studies have documented the presence of MPs in the placenta; however most relied on techniques with limited resolution and were largely incapable of identifying particles <1 μm, making them unable to estimate the mass concentration of MPs. A recent study isolated MPs for Py-GC-MS analysis by thoroughly and consistently digesting human placental tissue using a method similar to saponification. MPs were identified in every sample examined, indicating a worrisome prevalence that suggests the placental barrier is not immune to these environmental toxins. This analysis revealed an unexpectedly wide range and occasionally high

placental particle concentrations despite rigorous controls. This variability may reflect true environmental and maternal influences [5]. In another study, placental MPs have been linked to unfavorable pregnancy outcomes, such as decreased fetal growth in intrauterine growth restriction (IUGR) pregnancies, suggesting a possible detrimental effect on fetal development. MPs were detected in all 13 IUGR pregnancies, with an average abundance ranging from 2 to 38 particles per placenta. In normal pregnancies, however, MPs were below the limit of detection, with the exception of 3 out of 30 participants. Among individuals with IUGR, inverse relationships between exposure to MPs and delivery outcomes were seen for newborn weight ($r=-0.82$, $p<0.001$), length ($r=-0.56$, $p<0.001$), head circumference ($r=-0.50$, $p=0.001$), and 1-min Apgar score ($r=-0.75$, $p<0.001$) [38]. It is concerning that these MPs were found in such substantial amounts in the placenta (50.09%), meconium (60.22%), and newborn feces (49.67%), as this indicates that growing fetuses and neonates are exposed to these pollutants throughout crucial stages of growth and development [39]. The finding is especially troubling since there is a significant association between newborns' consumption of breast milk and the amount of MPs in their feces. This means that breastfeeding, which is thought to be the gold standard of nourishing infants, may also be a way of exposure to these pollutants. Using Raman microspectroscopy, a pilot observational study assessed human breastmilk samples from 34 nursing mothers and found contamination of MPs in 26 of them. Most of the MPs were colored spherical particles and irregular pieces. The most commonly found polymers were PE, PVC, and PP; fibrous particles were not seen. Maternal variables such as age, use of plastic-containing personal care items, and recent intake of meals and drinks packed in plastic did not show any statistically significant correlations with the presence or concentration of MPs [40].

The disruptive effects of MPs are not limited to the female reproductive system, as they also impact the male reproductive system. In an exploratory research, 10 men residing in a very polluted region of the Campania Region in Southern Italy had their semen tested for contamination with MPs. 6 of the 10 samples had 16 particles of MPs, detected by Raman microspectroscopy. MPs may enter semen through the epididymis and seminal vesicles, which are particularly susceptible to inflammatory processes, or they may translocate across the blood-testis barrier. Smaller particles may enter the seminal fluid more easily due to endothelial hyperpermeability brought on by inflammation and disruption of junctional proteins. Accumulation of MPs may also be influenced by active and passive membrane transport processes that control the composition of seminal plasma. Given the critical role of seminal fluid in supporting sperm function, these findings raise concerns regarding potential adverse effects of exposure to MPs on sperm quality and male fertility [41].

The most extensively described system affected by MPs so far has been the GI tract, as human MP intake is estimated at 39,000-52,000 particles per year, mainly via ingestion and mucociliary clearance. Particles larger than 150 μm are generally not absorbed but may still impair gut function by increasing intestinal permeability and eliciting local inflammation through direct enterocyte contact. Smaller particles can be absorbed depending on surface properties, potentially causing systemic toxicity as nanoparticles distribute throughout the organs. In inflammatory bowel disease patients, fecal MP concentrations were significantly higher than in healthy controls and correlated with disease severity [42]. Barrier impairment may further enhance MP absorption and cumulative toxicity, contributing to villous atrophy, nutrient malabsorption, and metabolic disturbances. In another study, after 4 and 12 weeks of exposure, mice fed PP-derived MP diets exhibited increased levels of systemic inflammation, oxidative stress, decreased expression of tight junction proteins, increased intestinal permeability, and increased serum endotoxin and cancer-related biomarkers indicative of intestinal inflammation and barrier dysfunction. Unlike PE-derived exposure to MPs, it did not provoke significant inflammation; however, marked changes to gut microbiota composition, bile acid profiles, and liver metabolism were observed, indicating alteration and dysfunction of the gut-liver axis [18]. Additionally, a study of plastic factory workers revealed altered gut and nasal microbiota, with increased disease-associated bacteria and reduced beneficial taxa, alongside higher fecal MP burden, particularly PU, underscoring occupational exposure as a risk factor [43].

Although the detection of MPs in organs with direct exposure to the external environment through various body cavities, such as the oral/anal cavity and uterine/vaginal cavity, has already been recorded, data on MPs presence in fully enclosed organs remain sparse. Scanning electron microscopy and laser direct infrared imaging were used to examine samples from 15 patients undergoing heart surgery. This showed that blood and heart tissues contained a variety of MP types. Furthermore, the presence of PMMA in the epicardial adipose tissue, pericardial adipose tissue, and left atrial appendage cannot be explained by unintentional exposure during surgery, establishing solid evidence of MPs in the human circulatory system [44]. This presence poses a potential risk of cardiovascular events, specifically in individuals with MNPs-laden carotid plaques, who had a higher risk of myocardial infarction, stroke, or death from any other cause at 34 months of follow-up compared to those without detectable MNPs [45].

The brain is another organ that does not have direct exposure to MPs. Nevertheless, pyrolysis-GC/MS analysis of postmortem tissues indicated that concentrations of brain MNPs

were even 7-30 times greater than those in the liver or kidney, predominantly consisting of PE and primarily as NPs fragments, with elevated levels observed in dementia cases, suggesting a potential correlation [46].

The conclusive evidence of the pervasive presence of MPs in the human body comes from their detection in bone marrow, observed not merely in isolated instances but across all analyzed samples in the study, underscoring potential ramifications for the hematopoietic system and establishing a foundation for future toxicological research [47]. These findings indicate that MPs have been found in almost all body organs and secretions (Figure 1).

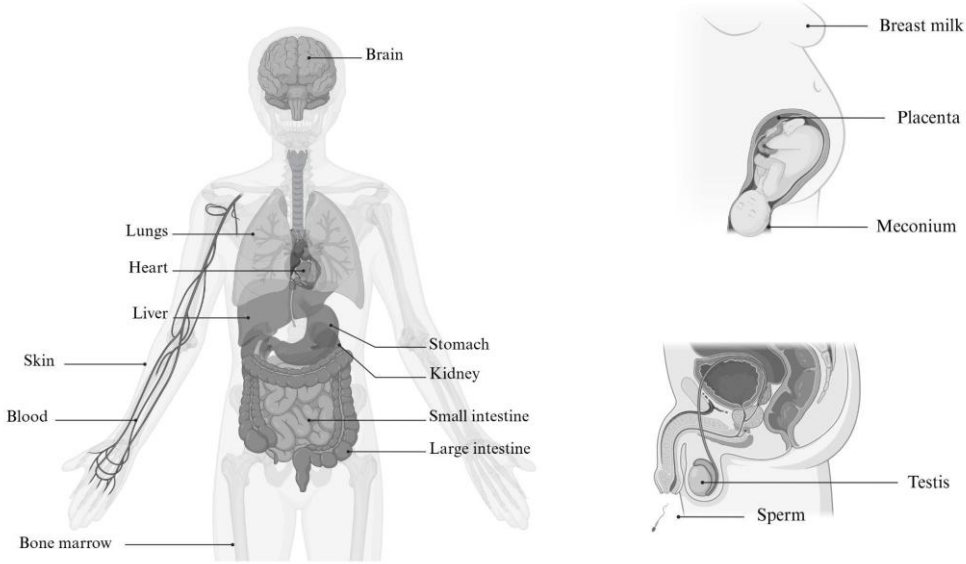

Figure 1. Visual representation of organs and fluids where deposition of MPs has been detected
Notes: Created in BioRender (2026). https://BioRender.com/ab3lgk6

Conclusions

MPs have now been detected across a wide range of human organs. This study focused on their presence and potential effects in less studied systems, including the cardiovascular, reproductive, and urinary systems, as well as the brain, bone marrow, placenta, and breast milk, indicating systemic distribution beyond organs with direct environmental exposure, such as the gastrointestinal and respiratory tracts. This widespread presence raises concerns about potential health consequences, particularly for vulnerable populations such as fetuses and infants. Evidence linking MP exposure to adverse reproductive outcomes, including intrauterine growth

restriction and declining semen quality, suggests possible long-term implications for fertility, pregnancy outcomes, and early-life development.

Given the exponential increase in plastic waste, coordinated intervention is imperative to prevent irreversible environmental and biological damage. Medical professionals can contribute through patient education, community engagement, and advocacy for evidence-based policies aimed at reducing exposure to MPs. Policymakers and health agencies should prioritize regulation of sources of MPs, monitoring of MPs in human biological matrices, and targeted awareness campaigns, especially for pregnant women.

In the interim, precautionary measures, including reduced use of plastic food packaging, avoidance of plastic cookware, preference for natural textiles and personal care products, as well as minimization of single-use plastics in healthcare settings and regular air ventilation, may help lower exposure. Strengthening both research and preventive strategies is essential to safeguard human health throughout one's lifespan.

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