REVIEW ARTICLE

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Multiple effects of microplastic particles on human internal organs – narrative review

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Abstract

Introduction and Objective. Microplastics are tiny plastic particles less than 5 millimeters in diameter. Their omnipresence in the environment has raised significant concerns about their potential impacts on human health. The aim of the review is to examine the current state of knowledge regarding the effects of microplastics on the human body.

Review Methods. The review was conducted in accordance with PRISMA guidelines, and based on a structured PubMed search of peer-reviewed human studies published in English throughout 2025.

Brief description of the state of knowledge. Human exposure to microplastics primarily occurs through ingestion, inhalation, and dermal contact. The potential health impacts of microplastics on the human body include both physical and chemical effects. Microplastics can induce inflammation and cellular damage in the respiratory and gastrointestinal tracts. They can also carry hazardous substances which are capable of leaching into the body and cause endocrine disruption, carcinogenicity, and reproductive toxicity. Studies have shown that microplastics can induce oxidative stress and inflammatory responses, compromise cellular functions, and potentially lead to immune dysregulation and endocrine disruption. However, up-to-date studies were performed on a small number of human subjects, making it difficult to draw definitive conclusions. The extant knowledge about the pathogenicity of microplastics still requires extensive studies and elucidation.

Summary. Current research underscores the potential risks of microplastics to human health via physical and chemical pathways. Laboratory studies offer insights into health impacts, but human research remains nascent. Addressing these gaps through comprehensive, interdisciplinary studies is crucial to fully understand the impact of microplastics on health, shaping effective regulatory and mitigation strategies.

Key words

microplastics, human health, plastic, inflammation, organs destruction, oxidative stress

INTRODUCTION

In recent years there have been increasing concerns regarding the presence of microplastic particles in the environment and the human body. Plastic, a crucial material in modern life, is being produced on a massive scale, with the United States alone manufacturing 35.7 million tons in 2018 [1].

Microplastics (MPs) – plastic particles smaller than 5 millimeters – are ubiquitous contaminants detected in marine and freshwater environments, drinking water, agricultural soils, atmosphere, food, and inside living organisms, including plants and animals [2, 3]. Although well studied in the environment and animal models, their effects on human organs remain poorly understood.

This narrative review synthesizes data on microplastics in human tissues, their toxicological mechanisms, and health risks, highlighting key research gaps as well as the most recent studies on the effects of MPs on human organs.

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MATERIALS AND METHODS

The review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A structured literature search was carried out using the PubMed database (National Library of Medicine, Centre for Biotechnology Information, https://www.ncbi.nlm.nih.gov/) to find relevant studies published throughout 2025.

The search strategy used Boolean operators ('AND', 'OR') to combine key terms such as microplastic, human health, absorption, distribution, metabolism, cancer, and organs. These terms were arranged in different combinations to cover the topic thoroughly and reduce irrelevant results. All references retrieved were imported into a reference management system, and duplicates were removed before screening.

Studies were eligible for inclusion if they met the following criteria: (i) peer-reviewed, full-text articles published in English; (ii) *in vitro* or *in vivo* studies involving human subjects; (iii) published up to and including 2025. Exclusion criteria were as follows: (i) studies involving animal models or experiments conducted on animal tissues, (ii) commentaries, opinion papers, editorials, conference abstracts, and non-peer-reviewed reports.

The final set of studies that met the inclusion criteria was included in the qualitative synthesis.The overall selection process, consisting of identification, screening, eligibility assessment, and final inclusion, is summarized in the PRISMA flow diagram (Fig. 1).

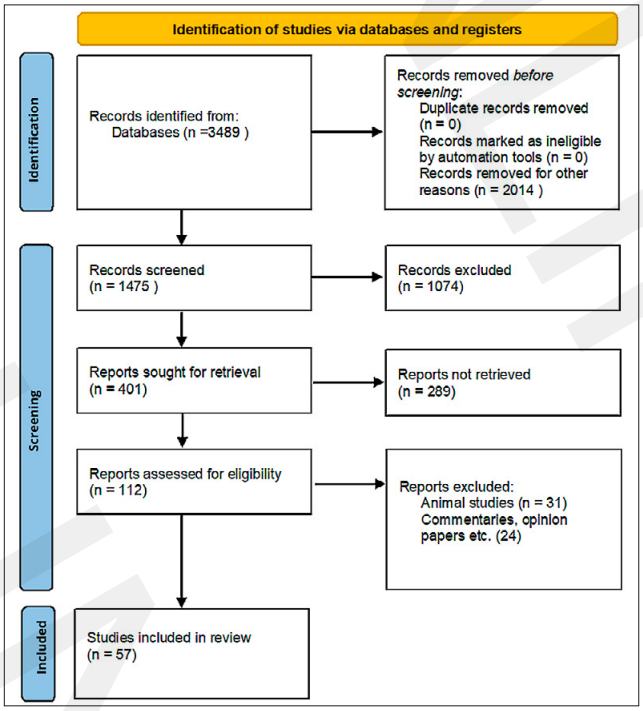

Figure 1. Flow diagram illustrating the process of study identification, screening, eligibility assessment, and final inclusion in the review, along with the outcomes described in the review [4]

Routes of uptake of microplastics by the human body. There are three main ways microplastics can enter the human body: ingestion through food or drink, inhalation, and direct skin contact [5, 6].

Ingestion via the alimentary tract. The primary way humans are exposed to microplastics is typically through eating. Based on food consumption data, microplastic intake ranges from 39,000 – 52,000 particles per person each year [7]. How these particles behave in the gastrointestinal tract, however, remains unclear. Microplastics probably cannot pass through tight junctions (~1.5 nm pores) [8], but they might be absorbed via immune-related processes such as phagocytosis by M cells in Peyer's patches [9].

Nanoplastics may be absorbed more efficiently because of their smaller size (50 nm), with polystyrene nanoparticles showing bioavailability rates of 2–7% [10]. In the gastrointestinal tract, nanoplastics interact with biomolecules to form a protein corona, which can influence uptake, and is affected by digestion [11]. Environmental factors, such as organic matter in water, may also impact nanoparticle behavior.

Uptake via airways. Inhalation is the second most important route of nanoplastic exposure in humans. Microplastics are released into the atmosphere from various sources, e.g. as dust, synthetic textiles, wear and tear of such materials as car tires and buildings, as well as resuspension of microplastics from surfaces. Studies have estimated that individual inhalation can lead to exposure to about 26 – 130 airborne microplastic particles per day [12]. The alveolar surface area of the lungs (~150 m^2) has a thin barrier (<1 µm), which allows nanoparticles to pass into the bloodstream and circulate throughout the body [13].

Inhaled micro- and nanoplastics pose health risks, including physical and chemical toxicity, as well as the potential transport of pathogens. These particles may lodge in the alveoli or migrate to other organs. Absorption depends on factors such as particle size, hydrophobicity, surface charge and formation of protein coronas [14].

Transdermal uptake. Nanoplastics are notably common in health and beauty products, especially in body and facial scrubs intended for topical application on the skin [15]. However, it should be noted that the transdermal uptake of plastic particles is more likely in the case of skin injury, and injuries caused directly by microplastics can lead to local toxicity or mechanical trauma. Health and beauty products, particularly exfoliating scrubs, are a significant source of nanoplastics through skin contact [16]. Another potential exposure route is the use of nanocarriers in topical drug delivery.

Some agents can enhance the transdermal passage of microplastics by compromising the integrity of the skin barrier. Mechanical wear of plastic microbeads in cosmetics increases the risk of generating nanoplastics. A study on polyethylene microbeads in scrubs showed that they break down into nanoparticles between 24–52 nm in size, confirmed by electron microscopy and spectroscopy [17]. Skin damage from UV exposure also weakens the barrier function, allowing more nanoparticles to pass through [18].

Microplastics size. The route of penetration depends primarily on the size of the plastic microparticles. Examples of the most common microparticles, classified according to their size, origin, and route of uptake, are shown in Figure 2 and Table 1.

Table 1. Absorption rate of microplastics varies depending on the food type and environment [22]

Source of microplastics	Microplastic absorption
Food	(4.88–5.77) × 10^5 MPs/year
Salt	(5.00–7.00) × 10^3 MPs/year
Fish	(0.50–1.20)×10^4 MPs/year
Fruits	(4.48–4.62) × 10^5 MPs/year
Vegetables	(2.96–9.55)×10^4 MPs/year
Water	(0.22–1.2)×10^6 MPs/year
Inhalation or atmospheric environment	(0.21–2.51) × 10^6 MPs/year
Indoor inhalation	(0.16–2.30) × 10^6 MPs/year
Outdoor inhalation	(0.46–2.10) ×10^5 MPs/year

Microplastic accumulation in human organs. Numerous studies have found microplastics in various parts of the human body, such as the liver, colon, lungs, placenta, hand washes,

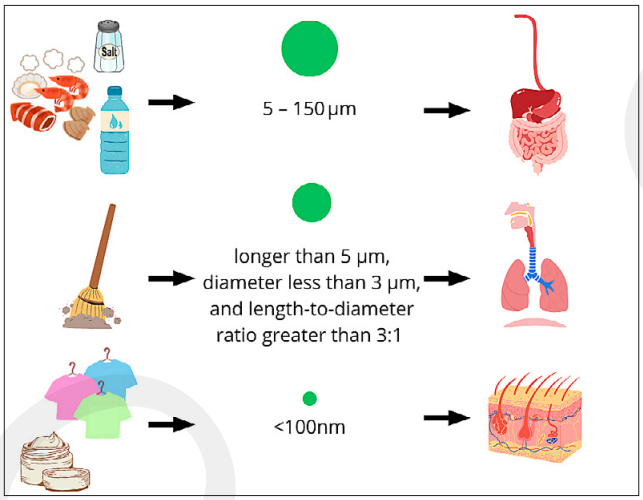

Figure 2. Differential uptake of microparticles by different organs depending on their size and origin [19, 20, 21]

face skin, and head hair. Additionally, microplastics have been detected in physiological fluids such as faeces, blood, saliva, sputum, and breast milk, as well as bronchoalveolar lavage fluid. The main results of these studies are summarized in Table 2.

Table 2. Summary of studies on the presence of plastic microparticles in human tissues and body fluids

Reference	No. of patients (N)	Biological sample	Particle size (range) (µm)
[23]	N=2,000	Hand, Face Washes, Hair, Saliva	< 100 µm
[24]	N=6	Urine	4–15 µm
[25]	N = 44	Bronchoalveolar Lavage Fluid (BALF)	< 500 µm
[26]	N = 22	Sputum	< 500 µm
[27]	N = 34	Breast Milk	2–12 µm
[28]	N = 22	Blood	≥700 nm
[29, 30]	N = 18	Placenta, Meconium, Infant Stool	20–50 µm
[31]	N = 50	Faeces	50–500 µm
[32]	N = 54	Placental Tissues	5 – 10 µm
[33]	N = 20	Pulmonary Tissues	< 5.5 µm
[34]	N = 11	Gastrointestinal Tissues	1.1 ±0.3 mm
[35]	N = 34	Liver, kidney, spleen	4 – 30 µm
[36]	N = 5	Vascular Tissues (Saphenous Vein)	< 5 µm

As shown in Table 2, many studies involved a small number of subjects, making it difficult to draw definitive conclusions. In particular, the absence of detectable microplastics in some stool samples raises important questions whether these particles are being absorbed systemically, or broken down within the gastrointestinal tract into sizes beyond current detection limits. Therefore, further comprehensive studies are needed to understand the uptake and accumulation of microparticles in different parts and specific organs of the human body.

Toxic impact. Numerous *in vitro* and *in vivo* studies have shown that both micro- and nanoplastics can cause significant adverse effects on the human body, such as physical stress and damage, apoptosis, necrosis, inflammation, oxidative stress, and immune responses.

Alimentary tract. The alimentary tract is the primary route of microplastic entry into the human body. Polypropylene and polyethylene terephthalate were detected in all eight human stool samples, comprising nearly 80% of the total microplastic load, with a median of 20 particles per 10 g of faeces [37].

The intestine, a key organ for digestion, absorption, and immune defense via gut-associated lymphoid tissue (GALT), can accumulate microplastics from ingestion and inhalation. Larger particles are typically excreted, while smaller ones may translocate into tissues through active uptake and phagocytosis by intestinal epithelial cells [38]. Ibrahim et al. (2020) found microplastics in all 11 human colectomy samples (90% polycarbonate, 50% polyamide, 40% polypropylene) [39]. Lin et al. (2022) demonstrated that 80 nm nanoplastics could enter the human liver and lung cells, inducing mitochondrial damage and metabolic disturbances without causing widespread cell death [40].

These findings highlight the gastrointestinal tract as a major site for microplastic accumulation. However, evidence is limited by the small cohorts, varying polymer profiles across tissues, and a lack of standardized analytical approaches. Larger, well-controlled studies are needed to determine size- and polymer-specific uptake, tissue distribution, and health impacts.

Nervous system. The potential toxicity of micro- and nanoplastics (MNPs) to human health remains incompletely understood. Marfella et al. (2024) reported the presence of MNPs in carotid atherosclerotic plaques, associated with increased inflammatory responses and a higher risk of subsequent cardiovascular events [41]. This may suggest that MNPs may play a part in the development of cardiovascular disorders. This however, needs extensive investigations.

Accumulation of MNPs in the brain has also been reported, primarily in the form of nanoscale polyethylene (PE) fragments or flakes. MNP concentrations in the brain tissue from individuals without neurological disease were 7–30 times higher than in liver or kidney, with even greater levels in dementia cases [42]. Although these findings suggest a potential link, causality remains unproven. Amato-Lourenço et al. (2024) detected microplastics in the olfactory bulbs of 8/15 individuals, mainly polypropylene (43.8%) measuring 5.5–26.4 µm, indicating a possible translocation pathway via the olfactory route [43].

Research on MNPs in cardiovascular and neural tissues is currently limited by small cohorts, methodological variability, and largely correlative data. Considering the putative pathogenicity of MNPs in these tissues, larger, contamination-controlled studies with standardized protocols and mechanistic endpoints are needed to clarify their biological effects.

Reproductive system. The accumulation of microplastics in the reproductive organs may lead to reproductive toxicity and impaired fertility. Although numerous animal studies describe the effects of plastic microparticles on the female reproductive tract, no reports on human female organs were found, highlighting the urgent need for research into their role in the pathogenesis of reproductive disorders.

The estimated minimum human equivalent dose of micro- and nanoplastics (MNPs) associated with abnormal semen quality is 0.016 mg/kg/day [44]. Montano et al. (2023)

detected 16 pigmented microplastic fragments (2–6 µm) in 6 of 10 human semen samples [45]. Zhao et al. (2023) reported microplastics in human testicular tissue (11.60 ± 15.52 particles/g) and semen (0.23 ± 0.45 particles/mL) [46]. While these findings confirm the presence of MNPs, their effects on spermatogenesis, sperm motility, and male fertility remain unclear.

Grafmueller et al. (2015) demonstrated that 50 nm MNPs can cross the placental barrier in an *ex vivo* human model, accumulating in the syncytiotrophoblast layer through active, energy-dependent transport, with more efficient transfer from the foetal to the maternal circulation [47]. Ragusa et al. (2021) verified the presence of microplastics in the human placenta, indicating possible prenatal exposure [48]. MNPs have also been found to change anabolic steroid hormone gene expression in cultured human chorionic trophoblast cells [49]. Proposed mechanisms of embryotoxicity include oxidative stress, abnormal energy metabolism, and immune disturbance, but further mechanistic *in vitro* and *in vivo* studies are necessary to clarify these effects.

Respiratory tract diseases. It has been demonstrated that inhaling or ingesting microplastics can lead to respiratory problems, including airway inflammation, worsening of asthma symptoms, and impaired lung function [50]. Lauren C. Jenner et al. (2022) analyzed human lung tissue and detected microplastics in 11 out of 13 samples, with an average concentration of 1.42 ± 1.50 particles per gram of tissue [51]. These findings strongly suggest the need for further extensive research into the mechanisms of action of microplastics on the human body and their impact on health.

Although detecting microplastics in human lung tissue raises concerns about respiratory exposure, current evidence is limited and mainly descriptive. Sample sizes are small, and studies often lack control for airborne contamination and standardized particle characterization. Data on how microplastics cause respiratory issues, especially oxidative stress and inflammation, are scarce. Future research should use rigorous methods, include functional respiratory tests, and examine dose-response relationships to better understand health risks.

Effects of MPs on microbiota. Recent studies suggest that microplastics (MPs) may influence human microbiota, a complex microbial community essential for host health. Due to their small size and environmental ubiquity, MPs can interact with microbiota via microbial colonization of MP surfaces, or through leached chemicals that alter microbial growth and diversity.

Actinobacteria, a minor gut microbiota component, exhibit notable biodegradation potential toward polypropylene, polylactic acid polymer, polyurethane, and polyethylene. Within the Actinomycetota phylum, genera such as *Actinomadura*, *Amycolatopsis*, *Kibdelosporangium*, *Micromonospora*, *Nonomuraea*, *Pseudonocardia*, *Saccharothrix*, *Streptoalloteichus*, *Streptomyces*, *Thermomonospora*, and *Thermopolyspora,* have been identified as capable of degrading polylactic acid microplastics [52]. This suggests a possible dual role for Actinobacteria in maintaining gut health and contributing to MP biodegradation, although *in vivo* relevance remains unconfirmed.

Liu et al. (2023) analyzed placental and meconium samples from 18 mother-infant pairs, detecting 16 MP types in all samples, predominantly polyamide (PA) and polyurethane (PU). Microbiota in both matrices were dominated by *Proteobacteria*, *Bacteroidota*, and *Firmicutes* [53].

Current evidence on MP-microbiota interactions in humans is limited, fragmented, and largely derived from *in vitro* or animal models, with no confirmed causal links. The biodegradation potential of gut microbes, particularly Actinobacteria, is of great interest but requires validation in controlled, long-term human studies using high-resolution metagenomics to assess functional impacts.

MPs as vectors of toxic substances. Plastics are synthetic polymers made from monomers with added chemicals to improve their properties. Interestingly, over 13,000 chemicals used in plastics and their additives have been identified worldwide, with more than 3,200 classified as substances of potential concern due to their persistence, bioaccumulation, or toxicity [54]. It is therefore important to note that these additives may pose health risks as potential carcinogens or endocrine disruptors. Many of these chemicals have not been studied, therefore their toxicity remains unknown. Among these substances phthalanes and bisphenol A (BPA) are of particular interest. BPA is a commonly used plasticizer and is considered a pollutant of particular concern because of its widespread use and potential health effects [55]. BPA is classified as an endocrine-disrupting compound because of its hormone-mimicking properties [56]. Microplastics can also serve as carriers for heavy metals such as Pb, Zn, Cd, Fe, Ag, As, Co, Al, and Cu, aiding their transport and potential buildup in biological tissues [57].

Additionally, the synergistic effects between microplastics and co-transported contaminants, such as heavy metals, are not yet fully understood. Future research should focus on comprehensive chemical screening, mixture toxicity assessments, and studies on bioavailability and accumulation in human tissues. This is an important and emerging problem; however, it goes beyond the scope of the present review.

Potential intervention and mitigation strategies. Given the increasing evidence of micro- and nanoplastics in human tissues and their potential health effects, the implementation of coordinated mitigation strategies is urgently required. Regulations should focus on restricting the use of the most hazardous plastic additives, enhancing labelling and transparency in plastic manufacturing, and establishing limits on microplastic content in consumer products and food packaging. In this context, robust national and international laws are vital to limit the use of toxic plastic additives and hold producers responsible. Meanwhile, raising public awareness through education campaigns can encourage behavioural changes – such as reducing single-use plastics and improving waste sorting – that collectively help decrease environmental and human exposure. Investments in innovation, including biodegradable materials and advanced filtration technologies, also provide promising options for long-term risk reduction.

SUMMARY AND CONCLUSIONS

This narrative review covered the current knowledge about the presence, uptake, tissue distribution, and possible health effects of micro- and nanoplastics (MNPs) in the human body. The main effects of MPs on human organs

Table 3. Summary of reported pathogenic effects of MPs on human systems and organs

System	Primary Exposure Route	Reported Health Effects	Level of Evidence	References
Gastrointestinal tract	Ingestion	Inflammation, oxidative stress, epithelial uptake, mitochondrial dysfunction, MP accumulation in gut and liver, gut dysbiosis	Moderate – based on human samples and *ex vivo* studies	[37–40, 52]
Nervous system	Inhalation, olfactory nerve	Brain accumulation, neuroinflammation, possible cognitive effects, olfactory translocation	Emerging – limited observational data and *ex vivo* models	[55–57]
Reproductive system	Ingestion, systemic circulation	MPs in testis and semen, potential effects on spermatogenesis and fertility, placental translocation, hormonal disruption, possible embryotoxicity	Limited – early human data without confirmed functional outcomes	[44–49, 53]
Respiratory system	Inhalation	Airway inflammation, asthma exacerbation, impaired lung function	Moderate – detection in human lung tissue with supportive clinical associations	[50, 51]
Systemic circulation	Multiple (ingestion, inhalation)	MPs detected in blood, urine, breast milk, BALF, sputum, faeces, and vascular tissue; potential immune activation and inflammation	Moderate – widespread detection but limited clinical correlations	[23–36]

are summarized in Table 3. MNPs mainly enter the body through ingestion, inhalation, and, to a lesser extent, skin contact, accumulating in organs, such as the gastrointestinal tract, lungs, liver, brain, placenta, and reproductive tissues. Their presence has been confirmed in many human biological samples, including blood, faeces, urine, breast milk, and placental tissues. Evidence indicates that MNPs may cause inflammatory responses, oxidative stress, immune activation, and hormonal disruption, potentially leading to reproductive toxicity, neuroinflammation, and respiratory problems. Additionally, MPs build up in organs like the liver, gut, kidneys, lungs, brain, placenta, spleen, blood vessels, and testis.

While the detection of MNPs in human tissues is increasingly well-documented, most studies are limited by small sample sizes, methodological inconsistencies, and lack of causal data. Furthermore, MNPs can act as carriers of hazardous additives and environmental contaminants, such as bisphenol A, phthalates, and heavy metals, compounding their potential toxicity. Emerging findings also indicate that MNPs may disrupt the human microbiota, with possible downstream effects on immune and neurological health.

Given the widespread exposure and the preliminary evidence of harm caused by nano- and microplastics, there is an urgent need for standardized research protocols, longitudinal human studies and mechanistic investigations. In parallel, policy actions – including stricter regulations on plastic additives, improved product labeling, and public education – are essential to mitigate exposure. Innovation in sustainable materials and filtration technologies may further help reduce future health risks associated with microplastic pollution.

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