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Original Research

Systematic Review: Impact of Microplastic Exposure on Human Health

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Abstract
Microplastics, defined as plastic particles smaller than 5mm, have emerged as a significant environmental and public health concern. With their widespread presence in food, water, air, and consumer products, human exposure to microplastics is unavoidable. This systematic review examines the potential health impacts of microplastic exposure, focusing on ingestion, inhalation, and dermal absorption pathways. The review synthesizes evidence from epidemiological studies, in vivo and in vitro research, and highlights the biological effects of microplastics, including inflammation, oxidative stress, cytotoxicity, endocrine disruption, and their potential role in chronic diseases. Additionally, we discuss current knowledge gaps and the need for standardized risk assessment methodologies to better understand the long-term implications of microplastic exposure on human health.

Keywords: Microplastics, human health, toxicity, inflammation, endocrine disruption, oxidative stress

Introduction

Microplastics have become pervasive contaminants in various environmental compartments, including oceans, rivers, soil, and air. These plastic fragments, typically smaller than 5 millimeters in diameter, originate from a variety of sources such as the breakdown of larger plastic waste, synthetic clothing fibers, personal care products, and industrial processes [1–3]. Their small size allows them to be ingested or inhaled easily by humans, leading to potential accumulation in bodily systems. Recent research has shown microplastics present in human feces, blood, placenta, and even lung tissues, highlighting the systemic nature of this exposure [4–6].

Vector potential of plastics, which is one of the most worrying aspects of microplastics. Heavy metals, persistent organic pollutants (POPs), and endocrine-disrupting chemicals (EDCs) from surrounding environments could be adsorbed to microplastics because they are highly chemically active complex organic compounds with a large surface area. Upon inhibit ingestion or inhalation of microplastics these potentially harmful factors could be secreted back into the human body, with simultaneous complexity effects on the toxicology [7–9]).

Exposure routes include ingesting contaminated food and water, inhalation of airborne fiber, and (less frequently) dermal absorption. Seafood (especially filter-feeding organisms such as mussels and oysters) is often highly contaminated with microplastics, penetrating the human food chain [10–12]. Microplastics have also been identified in bottled water and other processed consumables, contributing to exposure levels [13-15].

Research on the health effects of microplastics is still in its early stages. In vitro and in vivo studies indicate various biological effects such as oxidative stress, inflammation, cytotoxicity, and disruption of the endocrine and immune systems [16–18]. Little is known about the potential consequences of chronic, low-dose exposure to microplastics but they could be substantial, especially for sensitive groups like

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infants, expectant mothers and people with underlying health problems.

This systematic review aims to synthesize existing evidence regarding microplastics' existence in the human environment and their potential health effects. We review the route of exposure, how microplastics may induce toxicological effects, and physiological systems and processes that are sensitive to these chemicals, as well as gaps in the current body of knowledge. The review also delineates important knowledge gaps and suggests future avenues for research and public health action.

Methods

This systematic review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [18].

Search Strategy and Study Selection: We conducted a comprehensive search across four major electronic databases: Pub Med, Scopus, Web of Science, and Google Scholar. The search period spanned from January 2000 to December 2023. Search terms included combinations of keywords such as "microplastics,""human exposure,""toxicity,""endocrine disruption,""oxidative stress," and "health impact."

Inclusion and Exclusion Criteria Inclusion criteria:

- Peer-reviewed studies published in English.
- Original research articles, systematic reviews, and meta-analyses.
- Studies involving microplastic exposure in humans or relevant animal models.
- Studies reporting health outcomes such as inflammation, immune response, or organ-specific toxicity.

Exclusion criteria:

- Studies not related to human health.
- Duplicates or articles lacking sufficient data.
- Non-peer-reviewed reports or editorial pieces.

Data Extraction and Quality Assessment: Two independent reviewers screened titles and abstracts, followed by full-text assessment. Data were extracted regarding study design, exposure route, population/sample, key outcomes, and major findings. Disagreements were resolved by a third reviewer. Risk of bias was assessed using a modified version of the Cochrane Risk of Bias Tool.

PRISMA Flowchart

The PRISMA flow diagram below illustrates the study selection process:

Stage	Number of Articles
Identified through database search	820
Duplicates removed	140
Titles and abstracts screened	680
Full-text articles assessed	220
Studies included in review	75

Study Classification

Among the 75 included studies:

- 28 were review articles.
- 22 were randomized controlled trials (RCTs).
- 25 were original research articles including in vivo and in vitro studies [19–21].

Sources and Pathways of Human Exposure

Human exposure to microplastics occurs through three primary routes: ingestion, inhalation, and dermal absorption. Each route has unique pathways and contributes to cumulative health risks.

Ingestion

Ingestion is the most important and well-studied route of human exposure to microplastics. Microplastics have been found in food sources such as seafood, salt, and sugar, as well as drinking water, fruit, and vegetables. Microplastics accumulate especially in filter-feeding organisms like mussels and oysters due to their feeding mechanisms [10]. The consumption of contaminated seafood can cause measurable concentrations of microplastics to enter the human gastrointestinal tract [11–12]. Apart from marine foods, bottled water has been detected to contain hundreds of microplastic particles per liter and thus delivering considerable contributions to dietary exposure [13–14]. In addition, processed and packaged foods can be contaminated through contact with the packaging, such as plastic, or during the food manufacturing process [15].

Inhalation

Inhalation of airborne microplastics is a growing concern for exposure, particularly in urban and indoor environments. Sources are synthetic textiles, upholstery, industrial emissions, and atmospheric deposition. Microplastic fibers (MPFs) released during laundry drying or inhaled fibers from household dust can be inhaled and settled into the respiratory system [16]. Occupational environments such as plastic manufacturing and recycling industries expose workers to high levels of atmospheric microplastics. Microplastic fragments have been detected in human lung tissue using spectroscopic imaging [16], lending support that inhaled particles can translocate to alveolar regions [17]. These particles can trigger inflammatory reactions and are a potential factor for chronic respiratory diseases.

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Dermal Absorption

Although less studied, dermal absorption represents a potential route for microplastic exposure. Personal care products like facial scrubs, body washes, and toothpaste may contain microbeads, which can interact with the skin during regular use. While intact human skin provides an effective barrier against large particles, nanoplastic-sized microplastics (<100 nm) have been shown to penetrate the stratum corneum under certain conditions [18]. Additionally, skin exposure may be heightened by the presence of surfactants or abrasions that compromise the skin barrier. Though the extent of systemic absorption via dermal routes is still under investigation, it remains a plausible contributor to total exposure, especially among individuals using plastic-containing cosmetic products daily.

Mechanisms of Toxicity

Microplastics exert toxicity through several biological mechanisms that influence cellular, immunological, and endocrine systems. Their small size enables them to enter and accumulate within tissues, while their surface chemistry facilitates the adsorption of hazardous chemicals, contributing to cumulative biological effects.

Oxidative Stress

Oxidative stress is one of the most well-documented effects of microplastic exposure. Studies have shown that microplastics induce the generation of reactive oxygen species (ROS) in both in vitro and in vivo models, which leads to lipid peroxidation, mitochondrial dysfunction, and DNA damage. This oxidative imbalance can contribute to chronic inflammatory states and has been linked to diseases such as cancer, cardiovascular dysfunction, and neurodegenerative disorders.

Inflammatory Responses

Innate immune recognition of the microplastics can activate the inflammatory signaling pathway. Microplastic particles internalized by phagocytes may elicit release of pro-inflammatory cytokines IL-6, TNF-α, and IL-1β. Chronic inflammation is a general feature of many non-communicable diseases and can worsen underlying health conditions, especially among vulnerable populations.

Endocrine Disruption

Microplastics is a carrier of endocrine-disrupting chemicals (EDCs) such as bisphenol A (BPA), phthalates, flame retardants, and other chemicals. These compounds disrupt hormonal regulation by acting like hormones or antagonizing hormone receptors. Animal studies have indicated that such disruptions can affect reproductive function, thyroid hormone homeostasis, and metabolic syndrome, and raise serious concerns on reproductive health in the long-term and developmental toxicity.

Cytotoxicity and Genotoxicity

Microplastics have cytotoxic effects at the cellular level, including decreased cell viability, increased apoptosis, and compromised membrane integrity. Nanoplastic particles are of particular interest due to their potential to cross cell membranes and consequently interact with intracellular organelles. Genotoxic effects such as chromosomal aberrations and DNA fragmentation have been reported in multiple in vitro assays, indicating a potential association between exposure to microplastics and mutagenesis.

These mechanisms highlight the complex biological impact of microplastics and underscore the urgent need for further mechanistic studies to quantify exposure thresholds and understand long-term risks.

Human Health Implications

Microplastic exposure has been linked to adverse effects across multiple physiological systems, with the severity and nature of these impacts often depending on the route of exposure and particle size.

Respiratory System

Inhaled microplastics can lodge in the respiratory tract, reaching bronchioles and alveoli. Chronic exposure, particularly in occupational settings, may trigger inflammatory responses in lung tissue, leading to symptoms such as persistent coughing, wheezing, and even pulmonary fibrosis. Studies have reported elevated levels of cytokines and other inflammatory markers in exposed individuals, indicating immune system activation.

Gastrointestinal Tract

Ingested microplastics may disrupt gut homeostasis by altering the gut microbiota composition, increasing gut permeability, and promoting inflammation. Such disruptions have been associated with symptoms like bloating, abdominal pain, and potentially long-term consequences such as irritable bowel syndrome (IBS) or inflammatory bowel disease (IBD). Animal studies have also suggested that chronic ingestion may lead to structural damage of intestinal villi, reducing nutrient absorption.

Nervous System

Nanoplastic particles have been shown in experimental models to cross the blood-brain barrier, raising concerns about their neurotoxic potential. These particles may initiate neuroinflammatory responses and oxidative damage, possibly contributing to neurodegenerative conditions such as Alzheimer's and Parkinson's disease. Preliminary data suggest a correlation between high environmental microplastic exposure and increased neurobehavioral symptoms, particularly in developing children.

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Immune and Endocrine Systems

Systemic exposure to microplastics can activate immune cells, triggering persistent low-grade inflammation. Moreover, microplastics and their adsorbed toxicants (e.g., bisphenol A, phthalates) may act as endocrine disruptors, interfering with hormonal balance and affecting reproductive health. Endocrine disruption has been implicated in reduced fertility, thyroid dysfunction, and metabolic disorders including obesity and type 2 diabetes.

These implications underscore the urgency of further research to assess long-term risks and the need for regulatory interventions to mitigate human exposure.

Results

This section presents findings from epidemiological studies, in vitro and in vivo experiments, and meta-analyses regarding the health effects of microplastic exposure. The findings are categorized by exposure pathways and their respective physiological impacts.

Microplastic Ingestion and Gastrointestinal Effects

Microplastics have been detected in human fecal samples, confirming their ingestion and gastrointestinal passage [19]. Studies have shown that prolonged microplastic ingestion alters gut microbiota composition, leading to dysbiosis and increased intestinal permeability [20]. This disruption can contribute to irritable bowel syndrome (IBS), inflammatory bowel disease (IBD), and chronic inflammation [21].

In vitro studies suggest that microplastic particles adhere to the intestinal epithelium, triggering oxidative stress and inflammatory responses. Animal models demonstrate histological changes in intestinal villi, which may impair nutrient absorption and lead to gastrointestinal distress [22]. Additionally, microplastics act as carriers for heavy metals and persistent organic pollutants (POPs), which can bioaccumulate and exacerbate toxicity in the digestive system [23].

Microplastic Inhalation and Respiratory Toxicity

Airborne microplastics pose a significant risk due to their ability to penetrate deep into the respiratory system, reaching bronchial and alveolar tissues. Occupational exposure in plastic manufacturing facilities has been associated with higher incidences of pulmonary inflammation, fibrosis, and lung dysfunction [24]. Studies indicate that inhaled microplastic fibers induce pro-inflammatory cytokine release (IL-6, TNF-α), leading to chronic lung diseases [25].

Experimental evidence suggests that small-sized microplastics (≤10 μm) may translocate from lung tissue into the bloodstream, raising concerns over systemic circulation and cardiovascular toxicity [26]. Moreover, individuals with pre-existing conditions such as asthma and chronic obstructive pulmonary disease (COPD) may experience exacerbated symptoms upon prolonged inhalation exposure [27].

Dermal Absorption and Toxicity

Although microplastic penetration through intact human skin is considered minimal, nanoplastic particles (<100 nm) exhibit transdermal absorption potential. Studies using human skin models have identified the ability of nanoplastics to permeate the stratum corneum, particularly when assisted by surfactants in cosmetic products [28].

Emerging research indicates that skin exposure to microplastics may lead to oxidative stress, immune sensitization, and dermatitis-like reactions, especially in individuals with compromised skin barriers (e.g., eczema, psoriasis) [29]. Further studies are required to determine whether microplastics, once absorbed through the skin, contribute to systemic toxicity [30].

Systemic and Neurotoxic Effects

The ability of microplastics to cross biological barriers raises concerns about their long-term accumulation in critical organs. Animal studies indicate that microplastics may cross the blood-brain barrier (BBB), leading to neuroinflammation, cognitive dysfunction, and potential links to neurodegenerative disorders [31].

Recent studies have detected microplastics in human placental tissue, suggesting maternal-fetal transfer during pregnancy. This raises concerns about potential effects on fetal development, immune dysregulation, and endocrine disruption in neonates [32]. Furthermore, microplastic exposure has been linked to systemic inflammation and metabolic disorders, further implicating their role in chronic diseases [33].

Summary of Key Findings The following table summarizes the key findings from reviewed studies on the impact of microplastic exposure on different physiological systems:

Exposure Route	Health Impact	Key Findings
Ingestion	Gastrointestinal distress	Altered gut microbiota, increased permeability [19–23]
Inhalation	Respiratory inflammation	Chronic lung inflammation, fibrosis [24–27]
Dermal Absorption	Skin irritation and immune sensitization	Oxidative stress, dermatitis [28–30]
Systemic Circulation	Endocrine disruption, neurotoxicity	Neuroinflammation, fetal effects [31–33]

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Discussion

The discussion highlights the long-term implications of microplastic exposure on human health by analyzing data from the reviewed studies. While microplastics have been primarily studied in environmental contexts, mounting evidence reveals that they can have profound biological effects in humans. The diversity of microplastic shapes, sizes, and polymer compositions complicates risk assessment but also reveals a wide spectrum of toxicological mechanisms.

Chronic Inflammation and Systemic Impact

Several studies report a consistent association between microplastic exposure and chronic inflammation [34]. Inflammatory markers such as C-reactive protein (CRP) and IL-6 are elevated in individuals with higher environmental microplastic exposure [35]. Chronic inflammation, in turn, is a known contributor to metabolic syndrome, atherosclerosis, and cancer development [36].

Endocrine Disruption and Reproductive Effects

Plasticizers such as phthalates and bisphenol A (BPA), often adsorbed onto microplastics, interfere with hormone signaling [37]. Research links BPA exposure to decreased fertility, polycystic ovarian syndrome (PCOS), and disrupted testosterone levels in males. Animal studies confirm disrupted spermatogenesis and altered estrous cycles following microplastic ingestion [38].

Neurotoxicity and Behavioral Impairment

Microplastics under 100 nm in size can cross the blood-brain barrier (BBB), posing risks to neural tissues. Rodent models show that polystyrene nanoplastics accumulate in brain regions like the hippocampus, triggering oxidative damage and neuroinflammation [39]. In zebrafish embryos, microplastic exposure correlates with reduced motor activity and impaired cognitive behavior [40].

Immune System Dysregulation

Microplastics activate macrophages and dendritic cells, initiating immune cascades that lead to either heightened immune responses or immunosuppression [41]. Persistent immune activation may aggravate autoimmune conditions such as rheumatoid arthritis or lupus, particularly in genetically predisposed individuals [42].

Cardiovascular and Metabolic Disorders

Circulating microplastics have been linked to vascular dysfunction and altered lipid metabolism. Histological analysis reveals arterial inflammation and increased plaque formation in animal models exposed to polystyrene microplastics [43]. These findings align with human studies showing elevated blood pressure and cholesterol in communities with high microplastic exposure [44].

Prenatal and Developmental Toxicity

Microplastics have been detected in human placental tissue, indicating potential fetal exposure [45]. Developmental toxicity studies demonstrate that microplastics disrupt endocrine pathways during organogenesis, which can lead to growth restriction, birth defects, or neurodevelopmental delays [46].

Combined Exposure and Co-toxicity

An emerging area of research focuses on the role of microplastics as vectors for other environmental contaminants. Co-exposure with pesticides, heavy metals, or pharmaceuticals may amplify toxic effects [47]. The bioavailability of these chemicals is often increased when bound to microplastic surfaces, leading to more potent systemic impacts than when encountered alone [48].

Policy Implications and Future Research Directions

Given the ubiquity and persistence of microplastics, proactive regulatory frameworks are critical. Policymakers must enforce limitations on plastic production, encourage biodegradable alternatives, and improve wastewater treatment infrastructure to reduce environmental loading [49]. Simultaneously, future research should focus on longitudinal human studies, validated biomarkers of exposure, and standardized methods for measuring microplastics in biological tissues [50].

In summary, microplastic exposure affects multiple organ systems through mechanisms involving inflammation, endocrine disruption, oxidative stress, and bioaccumulation. These findings call for urgent interdisciplinary collaboration to quantify exposure, mitigate risks, and develop global health strategies.

Conclusion

This systematic review underscores the growing evidence that microplastic exposure poses significant risks to human health through diverse biological mechanisms, including inflammation, oxidative stress, endocrine disruption, and potential neurotoxicity. As microplastics are increasingly detected in various human tissues and exposure pathways expand through food, air, and personal care products, the urgency for more standardized research and effective policy interventions becomes clear. Strengthening global regulations on plastic use and investing in long-term human health studies will be essential steps toward mitigating the pervasive health threats posed by microplastic contamination.

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