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The Plastic Burden: How Microplastic Exposure Shapes Gut Microbiota, Immune Function, and Reproductive Outcomes – A Review

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ABSTRACT

Microplastic pollution has emerged as a ubiquitous environmental contaminant with profound implications for human health. These particles, defined as plastic fragments smaller than 5 mm, have been detected throughout the human body, including in blood, brain tissue, placenta, and reproductive organs. This comprehensive review examines the multifaceted impacts of microplastic exposure on three interconnected biological systems: gastrointestinal health and gut microbiota, immune function, and reproductive outcomes. Evidence demonstrates that microplastics disrupt gut microbiome composition, trigger chronic inflammatory responses, compromise intestinal barrier integrity, and interfere with endocrine pathways essential for reproductive health. Mechanistically, microplastics induce oxidative stress, activate pro-inflammatory cascades, and serve as vectors for endocrine-disrupting chemicals. Recent studies reveal concerning bioaccumulation patterns, with brain tissue showing 50% higher microplastic concentrations in 2024 compared to 2016 samples. The detection of microplastics in placental tissues and umbilical cord blood raises critical concerns about transgenerational health impacts. This review synthesizes current evidence on exposure pathways, biological mechanisms, and health consequences while identifying critical research gaps and potential therapeutic interventions.

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1. Introduction

The proliferation of plastic pollution represents one of the most significant environmental challenges of the 21st century, with global plastic production reaching unprecedented levels and continuing to escalate annually (Thin et al., 2025). Plastic particles smaller than 5 mm in diameter are known as microplastics (MPs), and they have been found in terrestrial, marine, and atmospheric environments all over the world (Enyoh et al., 2020). These particles come from a variety of sources, such as tire wear, direct release from synthetic fabrics, weathering processes that break up bigger plastic waste, and industrial operations (Li et al., 2023).

Since microplastic particles have been found in almost every environmental matrix that people come into contact with on a daily basis, including the air, water, and food sources, the widespread nature of microplastic pollution has sparked serious worries about the potential effects on human health (Jahedi and Jaafarzadeh Haghighi Fard, 2025). Microplastics in human tissues may now be found and measured owing to recent analytical developments, revealing their presence in blood, lungs, liver, brain, placenta, and reproductive organs (Nihart et al., 2025; Ragusa et al., 2021).The concentration of microplastics in human brain tissue has shown a concerning upward trend, with approximately 50% higher levels detected in 2024 compared to 2016 samples (Nihart et al., 2025).

There are several ways that humans might be exposed to microplastics, but the primary method is by ingestion. Current estimates imply that humans ingest between 39,000 to 52,000 microplastic particles yearly through food and beverages, with an additional 74,000 particles potentially inhaled from air sources (Nihart et al., 2025). The gastrointestinal

tract serves as the initial point of contact for ingested microplastics, where these particles can accumulate and interact with the complex gut microbiome ecosystem, potentially disrupting microbial balance and compromising intestinal barrier function (Zhang et al., 2025).The biological effects of microplastic exposure extend beyond localized gastrointestinal impacts, with evidence demonstrating systemic consequences affecting immune function and reproductive health (Tamargo et al., 2022). The persistence of these particles in biological systems, combined with their capacity to serve as vectors for endocrine-disrupting chemicals and other toxic compounds, creates conditions for chronic exposure effects that may manifest across multiple organ systems. Emerging evidence suggests that nanoplastics may bypass biological barriers more efficiently than larger microplastics, increasing risks of systemic bioaccumulation and neurotoxicity (Bora et al., 2024; Mahmud et al., 2024).

This thorough analysis looks at the existing understanding of the effects of microplastics on three important biological systems: immune system function, reproductive outcomes, and gastrointestinal health and gut microbiota composition. This study attempts to give a comprehensive knowledge of the processes by which microplastics damage human health and recommend priority topics for future research and intervention methods by incorporating data from recent studies carried out between 2020 and 2025.

2. Environmental sources and human exposure pathways

Microplastics enter the environment through diverse pathways, creating ubiquitous exposure opportunities for human populations. Primary sources include the breakdown of larger plastic debris through ultraviolet radiation, mechanical weathering, and biological processes, as well as direct release from consumer products such as synthetic textiles, personal care products containing microbeads, and tire wear particles (Zurub et al., 2024). Through both deliberate and in advertent releases, industrial methods such as the production of plastic and waste management operations significantly increase the amounts of microplastics in the environment (Zhang et al., 2025).

The global distribution of microplastics varies geographically, with Asia contributing approximately 81% of global microplastic emissions, primarily driven by activities in China (Sofield et al., 2024). Surface water concentrations in major river systems, such as the Yangtze River, have been reported to range from 0.48 to 21.52 items/L, while sediment concentrations can reach 3,185 particles/kg (Sofield et al., 2024). These high environmental concentrations create multiple exposure pathways for human populations through contaminated drinking water, food webs, and atmospheric deposition.

Ingestion, inhalation, and skin contact are the three main ways that humans are exposed to microplastics. Ingestion represents the dominant exposure pathway, with contaminated food and beverages serving as major sources. Seafood consumption is particularly significant, as marine organisms bioaccumulate microplastics throughout their tissues, with shellfish consumption alone contributing approximately 11,000 microplastic particles per person annually (Dubey et al., 2022). Drinking water represents another critical exposure source, with both tap and bottled water containing variable concentrations of microplastic particles (Cowley et al., 2025).

Inhalation exposure occurs through atmospheric microplastics, with concentrations often higher in indoor environments compared to outdoor air due to synthetic textiles, carpets, and furniture releasing particles (Mahmud et al., 2024). Occupational exposure in industries handling plastic materials can result insignificantly elevated inhalation levels, particularly affecting workers exposed to synthetic fibers and plastic dust (Soltani et al., 2021). The small size of air borne microplastics enables deep lung penetration, potentially leading to pulmonary deposition and subsequent systemic circulation (Soltani et al., 2021).

2.1. Gastrointestinal accumulation and microbiome disruption

The human gastro intestinal tract represents the primary site of microplastic accumulation following ingestion, where the persistent particles resist normal digestive processes and can remain for extended periods (Sofield et al., 2024). Unlike organic compounds subject to enzymatic breakdown, microplastics demonstrate remarkable resistance to digestive enzymes, acids, and mechanical forces, allowing them to interact directly within testinal epithelium and resident microbiota (Sofield et al., 2024).

The gut microbiome, comprised of trillions of bacteria crucial or digestion, metabolism, and im-

munological function, displays substantial susceptibility to microplastic exposure. Studies using human colonic microbiota models have demonstrated that PET microplastics alter microbial community composition during simulated gastrointestinal digestion (Tamargo et al., 2022). These changes include decreased abundance of beneficial Bacteroidetes species and increased populations of potentially pathogenic bacteria, particularly within the Firmicutes phylum. Experimental evidence from multiple animal models consistently demonstrates microplastic-induced gut microbiome dysbiosis. Exposure to polystyrene microplastics results in significant reductions in microbial diversity and species richness, with particularly pronounced effects on anti-inflammatory bacterial populations (Mahmud et al., 2024). The Bacteroidetes phylum, which plays crucial roles in maintaining gut barrier function and modulating immune responses, shows consistent decreases following microplastic exposure across different study models (Dubey et al., 2022).

Microplastic-induced microbiome changes extend beyond taxonomic shifts to encompass functional alterations in microbial metabolism. Key metabolic processes, such as the synthesis of bile acids, the metabolism of amino acids, and the formation of short-chain fatty acids, are disrupted by metagenomic investigations (Soltani et al., 2021). These functional changes compromise the gut's capacity to maintain metabolic homeostasis and support immune function. Particularly concerning is the observed increase in genes encoding antibiotic resistance and virulence factors in microplastic-exposed gut microbiomes, suggesting potential implications for antimicrobial resistance development (Sofield et al., 2024). The interaction between microplastics and gut microbiota appears to be bidirectional, with certain bacterial species demonstrating the capacity to adhere to microplastic surfaces and form biofilms. This biofilm formation may facilitate the transport of pathogenic bacteria across intestinal barriers and contribute to the persistence of microplastics within the gastrointestinal tract (Sofield et al., 2024). Recent research has identified probiotic strains, including Lacticaseibacillus paracasei and Lactiplantibacillus plantarum, that can bind microplastics and facilitate their excretion, suggesting potential therapeutic applications for microplastic-induced gut dysfunction (Nihart et al., 2025).

2.2. Intestinal barrier dysfunction and systemic translocation

Recent multi-omics investigations demonstrate that microplastic-induced dysbiosis may contribute to metabolic disorders through altered microbial signaling and host metabolic pathways. Microplastic accumulation in the gastrointestinal tract leads to significant structural and functional alterations in the intestinal epithelium, compromising the critical barrier function that separates the intestinal lumen from systemic circulation. Physical interactions between microplastic particles and the gut lining can cause mechanical injuries, including micro abrasions and epithelial damage that disrupts normal barrier integrity (Teng et al., 2025).

Experimental studies demonstrate that microplastic exposure induces intestinal inflammation, epithelial injury, oxidative stress, and impairment of intestinal barrier integrity (Mahmud et al., 2024). These structural alterations are accompanied by functional impairments in nutrient absorption, hormone production, and immune surveillance. Particularly significant is the impact on goblet cells responsible for mucus production, as microplastic exposure alters both the quantity and quality of protective mucus layers that normally prevent bacterial translocation and maintain barrier function. Increased intestinal permeability, sometimes known as "leaky gut syndrome," is a symptom of intestinal barrier integrity breakdown. This syndrome permits hazardous particles, bacterial endotoxins, and other potentially toxic chemical to enter systemic circulation, producing broad inflammatory reactions (Zhu et al., 2024). Despite a variety of processes, including the disruption of tight junction proteins like occludin and zonulin, which are crucial for preserving these selective barrier function between intestinal contents and blood circulation, microplastics increase intestinal permeability.

Once intestinal barrier integrity is disrupted, microplastics can translocate from the gastrointestinal tract to systemic circulation, permitting dispersion throughout the body. Numerous organs, including the liver, kidneys, lungs, brain, and reproductive tissues, have been shown to contain microplastics, indicating effective systemic translocation after gastrointestinal exposure, according to studies (Zurub et al., 2024). The blood-brain barrier, normally protective against foreign particles, appears particularly vulnerable to microplastic penetration, with concerning implications for neurological health.

2.3. Immune system activation and chronic inflammation

Microplastic exposure triggers robust immune responses that can evolve into chronic inflammatory states, representing a primary mechanism of microplastic-induced toxicity. The immune system recognizes these foreign particles as potential threats, initiating inflammatory cascades designed to neutralize and eliminate perceived invaders (Du et al., 2025). However, the persistence and non-biodegradable nature of microplastics prevent normal. Immune clearance mechanisms from effectively removing these particles, leading to sustained immune activation and chronic inflammation.

Microplastic exposure induces inflammatory responses characterized by increased production of proinflammatory cytokines and oxidative stress pathways (Mahmud et al., 2024). These inflammatory mediators create systemic inflammatory conditions that contribute to the pathogenesis of autoimmune disorders, metabolic dysfunction, and cardiovascular disease (Thin et al., 2025). The chronic nature of microplastic-induced inflammation distinguishes it from acute inflammatory responses and may contribute to age-related disease development. Macrophages represent the primary immune cells responsible for microplastic phagocytosis, but these cells often become overwhelmed by persistent particle loads. When macrophages attempt to engulf microplastics, the non-degradable nature of these particles leads to frustrated phagocytosis, condition where immune cells become chronically activated without successfully clearing foreign material. This process contributes to the formation of foreign body granulomas and persistent inflammatory foci throughout affected tissues. Experimental studies using various immune cell lines, includingTHP-1macrophages and peripheral blood mononuclear cells, consistently demonstrate dose-dependent inflammatory responses to microplastic exposure. At low doses (3ng/mL), inflammatory markers are activated, whereas higher concentrations (300ng/mL) additionally stimulate autophagy and apoptotic pathways (Hoang et al., 2025). T cells are least influenced by microplastic exposure, but phagocytic dendritic cells and macrophages exhibit great sensitivity to these particles.

Microplastics activate immune signalling pathways, including pattern-recognition receptors and oxidative stress mechanisms, contributing to persistent inflammation (Hoang et al., 2025).

2.4. Oxidative Stress and Cellular Damage Mechanisms

Microplastic exposure generates considerable oxidative stress through many cellular pathways, constituting a key route of biological damage. Both extracellular and intracellular activities produce reactive oxygen species (ROS), and weathered microplastics have a particularly high oxidative potential because of surface changes that increase their ability to produce free radicals. (Parthipan et al., 2025).

The primary source of microplastic-induced oxidative stress stems from mitochondrial dysfunction, as these particles can directly interact with mitochondrial membranes and disrupt normal electron transport chains. This disturbance leads to increased ROS generation, including superoxide radicals, hydrogen peroxide, and hydroxyl radicals, which can cause significant cellular damage (Zhu et al., 2024). Mitochondrial damage is particularly worrying since these organelles are necessary for cellular energy generation and control of apoptotic pathways. Cellular antioxidant defense systems, including superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase, become overwhelmed by chronic microplastic exposure. Studies across various experimental models demonstrate consistent decreases in antioxidant enzyme activity following microplastic exposure, indicating that cellular protective mechanisms are insufficient to neutralize ongoing oxidative challenges (Mahmud et al., 2024). This oxidative imbalance leads to lipid peroxidation, protein oxidation, and DNA damage that can contribute to cellular dysfunction and death.

The extent of oxidative stress appears to be size-dependent, with smaller microplastic particles generally inducing greater ROS production due to their larger surface area-to-volume ratios and enhanced cellular uptake. Nanoscale plastic particles demonstrate particularly potent oxidative effects, as they can more readily penetrate cellular membranes and interact with intracellular components (Mahmud et al., 2024). This size-dependent toxicity has important implications for human health, as environmental degradation processes tend to produce increasingly smaller plastic fragments over time. In vitro studies using human cell lines, including intestinal Caco-2cells, lung A549 cells, and kidney HEK-293 cells, consistently demonstrate microplastic-induced oxidative stress responses. Polystyrene nanoplastics exposure leads to irreversible lysosomal internalization and alterations

in mitochondrial and cellular structures within 24 hours, associated with increased expression of protective genes against oxidative stress, including SOD2 and heme oxygenase 1 (HO1) (Enyoh et al., 2020).

2.5. Reproductive health impacts and endocrine disruption

Microplastics pose significant threats to reproductive health through their capacity to disrupt endocrine function and interfere with hormonal signaling pathways essential for normal reproductive physiology. These particles serve as vectors for endocrine-disrupting chemicals (EDCs), including bisphenol A (BPA), phthalates, and polybrominated diphenyl ethers (PBDEs), which can leach from plastic matrices and exert hormonal effects throughout the body (Zurub et al., 2024).

The identification of microplastics in human reproductive organs has generated major concerns about fertility and reproductive consequences. Recent studies have detected microplastics in human follicular fluid, indicating that plastic particles can reach the ovarian microenvironment surrounding developing oocytes. Their presence raises concerns regarding oxidative stress, altered follicular function, and potential adverse effects on female reproductive health. (Zurub et al., 2024). Exposure of bovine oocytes to the semicroplastic concentrations in vitro resulted in compromise oocyte maturation and significant proteomic alterations affecting oxidative stress responses and DNA damage pathways. Male reproductive health faces particular vulnerability to microplastic exposure, with documented effects including impaired spermatogenesis, reduced sperm count, decreased motility, and increased production of morphologically abnormal sperm (Liu et al., 2024). These consequences occur from oxidative stress-induced damage to germinal epithelium, disturbance of the blood-testis barrier, and hormonal abnormalities influencing testosterone synthesis. Chronic exposure to polystyrene microplastics has been demonstrated to produce male reproductive damage through LH-mediated LHR/cAMP pathways, resulting in lower testosterone levels (Bora et al., 2024). Female reproductive health is similarly affected by microplastic exposure, with documented impacts including disrupted menstrual cycles, reduced fertility, and increased risks of pregnancy complications. Animal studies demonstrate that microplastic exposure leads to ovarian accumulation, where particles induce oxidative stress, promote granulosa cell apoptosis, and disrupt normal folliculogenesis (Li et al., 2023). These effects are mediated through activation of inflammatory pathways and direct toxic effects on ovarian cell populations essential for reproductive function. In particular being concerned was the finding of microplastics in placental tissues and umbilical cord blood, indicating direct fetal exposure during crucial developmental phases. Studies have revealed microplastics in all investigated placental samples, with amounts ranging from 6.5 to 790 micrograms per gram of tissue (Jahedi and Jaafarzadeh Haghighi Fard, 2025). The presence of these particles in maternal-fetal interface raises questions about potential transgenerational effects and developmental consequences for exposed offspring (Ragusa et al., 2021).

Recent research has established a placenta-cord blood-meconium pathway for microplastic transfer, providing evidence of direct neonatal exposure. Placentas from preterm deliveries exhibit higher amounts of microplastics than those from full-term pregnancies, indicating potential links between plastic exposure and unfavorable pregnancy outcomes (L. Zhu et al., 2023). Prenatal exposure to microplastics may have transgenerational consequences, affecting not only directly exposed foetuses but potentially subsequent generations through epigenetic mechanisms.

2.6. Bioaccumulation patterns and tissue distribution

Recent comprehensive analyses of human autopsy tissues have revealed distinct bioaccumulation patterns for microplastics across different organ systems, with concerning implications for long-term health effects. Recent human studies demonstrate significant accumulation of microplastics in brain tissue, highlighting potential neurological health concerns. Recent human autopsy studies have demonstrated that polyethylene is the predominant polymer detected in brain tissue, where microplastics are primarily present as nanoscale fragments. Individuals with documented dementia exhibited significantly higher concentrations of brain microplastics than neurologically normal controls, although current evidence does not establish a causal relationship between microplastic accumulation and neurodegeneration (Nihart et al.,

2025). This pattern suggests potential causal relationships between microplastic accumulation and neurodegenerative processes. Temporal analysis of tissue microplastic concentrations reveals alarming increases over recent years. Comparative studies of samples collected in 2016 versus 2024 show significant increases in both liver and brain microplastic concentrations, with brain tissue demonstrating approximately 50% higher levels in more recent samples (Dzierżyński et al., 2025).This temporal trend indicates that environmental microplastic exposure levels continue to increase, with corresponding increases in human tissue burden. Liver and kidney tissues how moderate microplastic accumulation, with concentrations averaging 8.2 and 6.8 particles per gram respectively. In hepatic tissues, microplastics aggregate within lipid-rich regions and along sinusoidal spaces, while renal accumulation occurs primarily in glomerular structures and tubular pathways (Wang et al., 2024). Post-mortem analyses have demonstrated widespread systemic bioaccumulation of microplastics in multiple human organs, including the brain, liver, kidneys, and selected musculoskeletal tissues, indicating that these particles can be transported and retained beyond the gastrointestinal tract following chronic exposure (Dzierżyński et al., 2025).

2.7. Gut-brain axis disruption and neurological consequences

The gut-brain axis, which includes neuronal, hormonal, and immunological signaling pathways, is a bidirectional network of communication between the central nervous system and the gastrointestinal tract. Microplastic exposure disrupts gut–brain communication through alterations in gut microbiota, intestinal permeability, and neuroinflammatory pathways (Grodzicki et al., 2021; Sofield et al., 2024).

Microplastic exposure disrupts the gut-brain axis through multiple interconnected mechanisms. Primary disruption occurs through gut microbiome alterations that affect the production of neurotransmitter precursors and neuromodulatory compounds. The gut microbiota performs critical functions in producing neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), and dopamine, which are crucial for mood control and cognitive function (Hettiarachchi and Meegoda, 2023). Microplastic-induced dysbiosis therefore directly impacts the availability of these critical signaling molecules.

Bacterial toxins and inflammatory mediators can reach the systemic circulation through compromised intestinal barrier function linked to microplastic exposure. From there, they can pass across the blood-brain barrier and cause neuroinflammation. Lipopolysaccharides (LPS) from gram-negative bacteria are particularly worrying neurotoxic chemicals that might activate microglia and induce inflammatory responses in brain tissue (Karak et al., 2025). This neuroinflammation has been implicated in the pathogenesis of neurodegenerative diseases, including Alzheimer's disease and Parkinson's disease.

Direct microplastic accumulation in brain tissue represents an additional mechanism of neurological toxicity. The detection of increasing microplastic concentrations in human brain tissue overtime, with preferential accumulation in areas associated with dementia, suggests possible causal relationships between plastic pollution and cognitive decline (Ghosh et al., 2023). Real-time imaging studies in animal models demonstrate microplastics moving through brain vasculature and blocking blood vessels, with potential long-term effects on neurological disorders and cardiovascular health.

3. Therapeutic interventions and mitigation strategies

The recognition of microplastics as significant biological threats has prompted research into potential therapeutic interventions and exposure reduction strategies. Recent breakthroughs in probiotic-based approaches offer promising avenues for reducing microplastic burden in the gastrointestinal tract. Comprehensive screening of bacterial strains has identified specific probiotic species capable of binding and facilitating excretion of ingested microplastics.

Lacticaseibacillus paracasei DT66 and Lactiplantibacillus plantarum DT88 demonstrate optimal microplastic adsorption capabilities across various plastic polymer types. Treatment with selected probiotic strains significantly increased microplastic excretion and reduced intestinal microplastic burden in experimental models (Teng et al., 2025). Additionally, these probiotic interventions mitigate microplastic-induced intestinal inflammation, providing dual benefits for both particle clearance and tissue protection.

Dietary interventions focused on supporting cellular antioxidant defenses may provide additional protective benefits against microplastic-induced ox-

idative stress. Compounds with proven antioxidant properties, including polyphenols, vitamin C, vitamin E, and selenium-containing compounds, may help counteract ROS generation associated with microplastic exposure (Deng et al., 2017). However, the chronic nature of microplastic exposure suggests that dietary interventions alone may be insufficient without concurrent efforts to reduce exposure levels.

Technological approaches to reducing microplastic contamination in drinking water and food systems represent essential components of exposure mitigation strategies. Advanced membrane-based treatment technologies, including reverse osmosis, nanofiltration, ultrafiltration, and membrane bioreactors, have demonstrated high efficiencies in removing microplastic particles from drinking water and wastewater. The effectiveness of these technologies depends on particle size, membrane characteristics, and operational conditions, with reverse osmosis generally providing the highest removal efficiencies for fine microplastics (Shen et al., 2020). However, because microplastics are widely distributed throughout aquatic and terrestrial ecosystems, technological treatment should be complemented by source-control measures, improved plastic waste management, and environmental pollution prevention to achieve long-term reductions in human exposure (Li et al., 2023). However, the widespread contamination of food webs necessitates broader environmental remediation efforts to address the source of contamination rather than merely treating symptoms.

4. Public health implications and future research directions

The emerging evidence of microplastic impacts on human health presents unprecedented challenges for public health policy and environmental regulation. The ubiquitous nature of these pollutants, combined with their persistence in biological systems, suggests that current and future generations will face chronic exposure scenarios with potentially cumulative health effects (Wang et al., 2024). The lack of established safe exposure limits and standardized measurement protocols further complicates efforts to assess and manage health risks. Epidemiological studies linking microplastic exposure to specific health outcomes remain limited, largely due to the recent recognition of this exposure pathway and technical challenges in quantifying tissue microplastic burdens. However, available evidence from animal studies and in vitro research consistently demonstrates concerning biological effects across multiple organ systems (Grodzicki et al., 2021). The dose-response relationships for microplastic toxicity remain poorly characterized, particularly for chronic low-level exposures typical of environmental conditions. The potential for transgenerational effects represents a particularly concerning aspect of microplastic exposure, as EDCs associated with plastic particles can induce epigenetic modifications that may be inherited by subsequent generations (Akhbarizadeh et al., 2019). This possibility suggests that current exposure patterns could have health consequences extending far beyond directly exposed individuals (Thin et al., 2025). Understanding these transgenerational effects requires long-term prospective studies tracking health outcomes across multiple generations. Recent multi-omics investigations demonstrate that microplastic-induced dysbiosis may contribute to metabolic disorders through altered microbial signalling and host metabolic pathways (Bora et al., 2024; Chokshi et al., 2026).

Future research priorities must include development of standardized analytical methods for quantifying microplastics in biological samples, establishment of dose-response relationships for various health end points, and identification of vulnerable populations at highest risk for adverse effects (Teng et al., 2025).Additionally, research into effective intervention strategies, including both individual-level protective measures and population-level exposure reduction approaches, remains critical for developing comprehensive public health responses.

5. Conclusion

The mounting evidence regarding microplastic impacts on human health reveals a complex web of biological disruptions affecting fundamental physiological processes across multiple organ systems. The gastrointestinal tract serves as the primary interface between humans and these ubiquitous pollutants, with microplastic-induced gut microbiome disruption creating cascading effects throughout the body. The chronic inflammatory responses triggered by persistent microplastic accumulation contribute to immune dysfunction and may predispose individuals to autoimmune disorders and metabolic diseases. The endocrine-disrupting properties of microplastics and their associated chemicals pose significant threats

to reproductive health through hormonal interference and direct toxic effects on reproductive tissues. The detection of microplastics in placental tissues and umbilical cord blood rises particularly concerning questions about transgenerational health impacts and potential developmental consequences for future generations. The preferential accumulation of microplastics in brain tissue, with concentrations increasing over time, raises critical questions about neurological health impacts and potential contributions to neurodegenerative diseases. The disruption of the gut-brain axis through microplastic-induced intestinal dysfunction provides additional pathways for neurological consequences, highlighting the interconnected nature of these health impacts. While therapeutic interventions such as specialized probiotics offer hope for mitigating microplastic burdens, the scale and persistence of environmental plastic pollution necessitate comprehensive approaches combining exposure reduction, technological innovation, and public health policy responses. The transgenerational potential of microplastic-associated health effects underscores the urgency of addressing this emerging health crisis before irreversible consequences become apparent in future generations.

As global plastic production continues to increase and environmental microplastic concentrations rise correspondingly, the scientific community faces the critical challenge of rapidly advancing understanding of these health impacts while simultaneously developing effective intervention strategies. The evidence presented in this review demonstrates that microplastics represent far more than an aesthetic environmental problem, they constitute a fundamental threat to human health that demands immediate and sustained attention from researchers, policymakers, and public health officials worldwide.

CRediT statement

SRB: Investigation, Formal Analysis, Writing Original draft, Finalization of the manuscript AD: Editing, Supervision and Visualisation.

MRD: Validation, Data curation, Original draft preparation

Declaration of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could influence the work reported in this paper.

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During the preparation of this manuscript, the authors used AI tools to improve the language and readability. After using this tool, the authors reviewed the content and made changes wherever necessary. The authors take full responsibility for the contents of this publication.

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