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HUMAN EXPOSURE TO MICROPLASTICS: TOXICOLOGICAL MECHANISMS AND POTENTIAL HEALTH IMPLICATIONS - A NARRATIVE REVIEW

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ABSTRACT

Introduction: Microplastics represent an increasingly widespread environmental contaminant detected in water, food, and ambient air. In recent years, microplastic particles have also been identified in human biological materials, raising concerns about their potential impact on human health.

Objective: The aim of this review was to summarize current evidence regarding the main routes of human exposure to microplastics, the mechanisms underlying their potential toxicity, and the possible health consequences associated with exposure.

Materials and Methods: A review of the most recent scientific literature available in major medical databases was conducted, with particular emphasis on experimental studies, systematic reviews, and publications reporting the presence of microplastics in the human body.

Results: The primary exposure routes include ingestion of contaminated food and water, as well as inhalation of airborne particles and household dust. Experimental studies suggest that micro- and nanoplastics may induce oxidative stress, inflammatory responses, and cellular dysfunction. Plastic particles may also act as vectors for other chemical contaminants and potentially interfere with endocrine regulation. The most frequently discussed health effects involve the gastrointestinal, respiratory, and reproductive systems, as well as metabolic disturbances. However, the majority of currently available evidence is derived from in vitro studies and animal models.

Conclusions: Despite the presence of biologically plausible mechanisms of toxicity, there is currently no conclusive clinical evidence establishing a direct association between microplastic exposure and specific human diseases. Further epidemiological studies and standardization of analytical methods for detecting microplastics in biological samples are required to enable a reliable assessment of health risks.

KEYWORDS

Microplastics, Nanoplastics, Toxicology, Environmental Pollution

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1. Introduction

Although plastic production began only in the late nineteenth century, plastics are now ubiquitous serving as packaging materials, construction components, textiles, and even ingredients in cosmetics and food-related applications. Global plastic production is currently estimated at approximately 500 million tons annually and continues to increase, with projections suggesting that microplastic pollution may double by 2040 (Thompson et al., 2024).

The exceptional durability of plastic materials, their widespread use, and the lack of effective environmental mitigation strategies have significant implications for living organisms. Microplastics have been detected in food products, drinking water, household dust, and human biological materials. Their widespread presence raises serious concerns regarding potential long-term health consequences (Zhang et al., 2025; Leslie et al., 2022).

Current evidence suggests that micro- and nanoplastics may induce inflammatory responses and oxidative stress. They have been shown to interfere with cellular function and may act as carriers of organic pollutants and heavy metals. However, most available data are derived from in vitro studies and animal models, and clinical evidence in humans remains limited and requires further investigation.

The aim of this review is to summarize current scientific evidence regarding the routes of human exposure to micro- and nanoplastics, their potential mechanisms of toxicity, and the associated health consequences.

2. What Are Microplastics?

Under the influence of environmental factors, plastic materials undergo fragmentation into smaller particles classified as microplastics (diameter <5 mm) and nanoplastics (diameter <1 µm). Microplastics are generally categorized as either primary intentionally manufactured small particles such as microbeads used in cosmetics or secondary, which result from the degradation of larger plastic items, including single-use packaging, under chemical and physical stressors.

The most common polymer types include polyethylene terephthalate (PET), polypropylene (PP), polyethylene (PE), and polystyrene (PS). Particle size is biologically relevant, as smaller particles are more capable of crossing biological barriers and penetrating tissues. Nanoplastics, in particular, may translocate across epithelial and endothelial barriers, potentially leading to systemic distribution.

3. Routes of Human Exposure

Plastics have become an integral component of modern daily life. Human exposure to micro- and nanoplastics occurs through several primary pathways.

Ingestion represents a major route of exposure. Individuals may consume microplastics through contaminated seafood, sea salt, inadequately filtered drinking water, and food products packaged, transported, or stored in plastic materials (Smith et al., 2018; Zhang et al., 2025).

Inhalation constitutes another important exposure pathway. Microplastic particles are present in household dust, urban air pollution (Leslie et al., 2022), and airborne synthetic fibers released from textiles (Zhao et al., 2024).

Microplastics have been identified in numerous human biological samples, including blood, placenta, feces, semen, lungs, bone tissue, and sputum (Huang et al., 2022). Lifestyle factors and certain medical procedures may also contribute to exposure.

It is important to emphasize that the term micro- and nanoplastics encompasses a highly heterogeneous group of materials that differ in chemical composition, size, shape, and surface characteristics. These differences may influence their biological interactions and toxicity profiles. Importantly, the mere presence of microplastic particles in human tissues does not necessarily imply confirmed toxic effects.

Children may represent a particularly vulnerable population due to increased environmental contact (e.g., plastic toys and consumer products), immature biological barriers, and higher ventilation rates relative to body mass (Luo et al., 2025). Nevertheless, based on current evidence, it cannot yet be conclusively stated that microplastic exposure causes specific developmental disorders in children (Zurub et al., 2024).

4. Fate of Microplastics in the Human Body (Toxicokinetics) and Mechanisms of Potential Toxicity

4.1. Oxidative Stress

Micro- and nanoplastics detected in tissues such as the lungs and intestinal epithelium may induce oxidative stress by increasing the production of reactive oxygen species (ROS). Excessive ROS generation can lead to secondary damage to proteins, DNA, membrane lipids, and intracellular structures, ultimately resulting in cellular dysfunction and activation of apoptotic pathways (Deng et al., 2017; Zhang et al., 2025).

These effects have been observed across various cell types, suggesting a potentially common pathophysiological mechanism underlying the adverse effects of microplastics, particularly smaller particles, on multiple organs. Given their high surface area and reactivity, nanoplastics may be especially prone to inducing oxidative imbalance at the cellular level.

4.2. Inflammatory Response

Particles recognized as foreign by the immune system are typically phagocytosed by macrophages. As exogenous materials, micro- and nanoplastics may trigger immune activation and promote the release of pro-inflammatory mediators, including interleukin-1β (IL-1β), tumor necrosis factor-α (TNF-α), and interleukin-6 (IL-6), as well as activation of the nuclear factor kappa B (NF-κB) signaling pathway (Leslie et al., 2022; Zhao et al., 2024).

Chronic stimulation of the immune system may contribute to secondary tissue damage and impaired organ function. In vitro studies have demonstrated associations between microplastic exposure and activation of NF-κB-dependent signaling pathways, accompanied by increased expression of genes involved in inflammatory responses.

A study conducted in 2024 reported that higher levels of microplastics detected in human blood were associated with an increased prevalence of myocardial infarction and greater complexity of atherosclerotic lesions, as well as elevated cardiovascular risk (Yang et al., 2024). These findings suggest that microplastics may act as a risk factor or biomarker of inflammation in certain cardiovascular conditions; however, a direct causal relationship between microplastic exposure and cardiovascular disease has not yet been established.

Similarly, experimental studies in rodents have demonstrated increased renal injury markers and inflammatory parameters following microplastic exposure (Tan et al., 2025). Animal models examining pulmonary and intestinal tissues have also revealed inflammatory infiltrates in response to chronic microplastic exposure, potentially linking long-term exposure to the development of inflammation-driven pathological changes (Leslie et al., 2022; Sun et al., 2023).

Despite these findings, clinical evidence in humans remains limited, and further research is necessary to clarify the long-term health implications of chronic exposure.

4.3. Microplastics as Carriers of Chemical Pollutants and Heavy Metals: The “Trojan Horse” Effect

The adverse effects of microplastics may also be related to their ability to adsorb environmental contaminants. Due to their physicochemical properties including large surface area and hydrophobic characteristics plastic particles can bind a wide range of potentially toxic substances, such as pesticides, heavy metals, persistent organic pollutants (POPs), and polycyclic aromatic hydrocarbons (PAHs).

In this context, contaminated microplastics may function as “Trojan horses,” facilitating the transport of co-adsorbed pollutants into biological systems and potentially enhancing their bioavailability and toxicity (Barboza et al., 2018; Khu et al., 2025). Once internalized and translocated across biological barriers, these substances may be released within tissues, potentially increasing local concentrations and promoting accumulation, including that of heavy metals.

However, the magnitude and real-world relevance of this phenomenon remain uncertain. Most available studies have been conducted under laboratory conditions, often using contaminant concentrations higher than those typically observed in environmental settings. Future research should aim to determine whether microplastics play a significant role in the transport and bioaccumulation of environmental toxicants in humans.

4.4. Potential Endocrine-Disrupting Effects

Plastics are composed of complex mixtures of polymers and chemical additives. Materials that subsequently fragment into micro- and nanoplastics may contain additional compounds such as bisphenol A (BPA), flame retardants, and phthalates. Many of these substances are classified as endocrine-disrupting chemicals (EDCs), which may interfere with hormone receptor signaling and consequently alter hormone synthesis, metabolism, and transport, thereby disrupting endocrine axis regulation.

Exposure to such compounds has been associated with metabolic disturbances, alterations in sex hormone balance (estrogenic and androgenic pathways), reproductive dysfunction, and precocious puberty. Indirect effects may also occur through mechanisms involving oxidative stress and chronic inflammation, which can further interfere with hormonal homeostasis (Zhang et al., 2025; Rahman et al., 2021).

As with other proposed mechanisms of microplastic toxicity, robust epidemiological and clinical studies in human populations are needed to establish the extent and clinical significance of these potential endocrine effects.

5. Potential Health Effects and Organ-Specific Toxicity

Despite the growing number of publications documenting the presence of microplastics in human tissues, robust clinical evidence regarding their biological effects remains limited. Most currently available data are derived from *in vitro* experiments and animal models. Nevertheless, based on existing evidence, several organ systems appear to be particularly susceptible to the potential adverse effects of micro- and nanoplastic exposure.

5.1. Gastrointestinal System

Given that ingestion represents the primary route of exposure, the gastrointestinal tract is likely the most directly affected system. Microplastics consumed with contaminated food and drinking water may interact with the intestinal epithelium and disrupt its integrity.

Experimental studies indicate that microplastics can increase ROS production, impair epithelial barrier function, and enhance activation of inflammatory and apoptotic pathways (Zhang et al., 2025; Thin et al.,

2025). Animal studies further suggest that exposure may alter gut microbiota composition (Gao et al., 2024), potentially contributing to dysbiosis.

Such alterations may promote metabolic disturbances and impair nutrient absorption. However, although mechanistically plausible, the translation of these findings to clinically significant gastrointestinal disease in humans remains uncertain.

5.2. Respiratory System

Inhalation constitutes another important exposure pathway. Airborne microplastic fibers, household dust, and urban particulate pollution may accumulate in the respiratory tract. Similar to effects observed in the gastrointestinal system, inhaled particles may induce oxidative stress and localized chronic inflammation.

Animal studies have demonstrated inflammatory changes in lung tissue following prolonged exposure; however, human data remain scarce. It has been hypothesized that chronic inhalation exposure may contribute to the development or exacerbation of respiratory diseases (Leslie et al., 2022; Zhao et al., 2024).

In addition, alterations in the nasal microbiome have been observed in exposed individuals, including increased abundance of potentially pathogenic bacteria and decreased levels of beneficial commensal species (Zhang et al., 2022). Some authors have suggested possible associations between microplastic exposure and chronic pulmonary inflammation or even lung cancer, although such relationships remain speculative and insufficiently supported by clinical evidence (Winiarska et al., 2024; Zhao et al., 2024).

5.3. Reproductive System

The potential impact of microplastics on reproductive function is of particular concern due to the presence of endocrine-disrupting chemicals (EDCs) in many plastic materials. Animal models have demonstrated associations between microplastic exposure and impaired spermatogenesis, reduced semen quality, ovarian dysfunction, and disturbances in hormonal axis regulation (Chartres et al., 2024).

However, clinical data in humans remain insufficient to establish a definitive long-term relationship between microplastic exposure and reduced fertility. A 2023 study reported differences in the composition of microplastics detected in testicular tissue and semen samples, suggesting potential variability in exposure routes or internal distribution mechanisms (Zhao et al., 2023). The clinical implications of these findings remain to be determined.

5.4. Metabolic Disorders and Other Potential Outcomes

Experimental evidence suggests a possible association between microplastic exposure and metabolic dysregulation. Reported effects include impaired glucose tolerance, altered lipid metabolism, and insulin resistance (Zhao et al., 2024). Chronic inflammation and oxidative stress may disrupt systemic homeostasis, thereby contributing to the development of chronic diseases.

Some researchers have also hypothesized a potential carcinogenic role of microplastics, given their capacity to induce intracellular DNA damage and sustain inflammatory signaling pathways. Additionally, a 2024 study detected microplastics in all examined gallstone samples, raising questions about their potential involvement in lipid metabolism and gallstone formation (Zhang et al., 2024).

Although adverse cellular-level effects have been repeatedly demonstrated, conclusive clinical evidence confirming specific disease mechanisms or direct causality in humans is still lacking (Mittal et al., 2023). Large-scale, long-term epidemiological studies are urgently needed to clarify the true health implications of chronic microplastic exposure (Zhang et al., 2025; Leslie et al., 2022).

6. Study Limitations and Methodological Challenges

The current state of knowledge regarding microplastics is substantially limited by methodological constraints that significantly hinder and in some cases prevent a reliable assessment of health risks associated with long-term exposure. One of the primary challenges is the lack of standardized methods for the detection and quantification of microplastics in tissues and biological samples (Lee et al., 2025). Different analytical techniques are employed across studies, which complicates the comparison and interpretation of results.

Additionally, there is a considerable risk of sample contamination by microplastics originating from the laboratory environment, including airborne dust, plastic packaging materials, laboratory equipment, and even researchers' clothing (Zhao et al., 2024). These sources of contamination are difficult to eliminate entirely and may lead to overestimation of actual exposure levels.

Another important issue is the substantial heterogeneity of microplastic particles. They differ in chemical composition, size, shape, surface properties, and the presence of adsorbed additives or environmental contaminants. Experimental studies typically use homogeneous, standardized particles, which do not adequately reflect the complexity of real-world environmental exposure (Thin et al., 2025).

Furthermore, significant uncertainty remains regarding the actual bioavailability of microplastics specifically, what proportion of particles enters the human body, the mechanisms governing their translocation, and their persistence in tissues (Blackburn et al., 2022).

An additional limitation concerns the discrepancy between exposure doses used in laboratory settings and those encountered in the natural environment. Animal and in vitro studies frequently employ concentrations substantially higher than those relevant to human environmental exposure, potentially leading to an overestimation of toxic effects (Leslie et al., 2022). Moreover, most experimental studies are short-term, whereas human environmental exposure is typically chronic and occurs at low doses.

Current clinical data are derived from relatively small study populations and remain fragmented, allowing only limited conclusions to be drawn. These constraints make it difficult to establish a clear relationship between exposure levels and specific health outcomes within defined populations (Zhang et al., 2025).

In light of these limitations, the interpretation of available findings requires caution. Future research should prioritize methodological standardization, accurate exposure assessment, and the implementation of long-term population-based studies.

7. Conclusions

Microplastics represent a widespread environmental contaminant that primarily enters the human body through ingestion and inhalation (Zhang et al., 2025). Numerous studies conducted in recent years have demonstrated their ability to penetrate biological systems and distribute across various tissues.

Available evidence suggests several plausible mechanisms of toxicity, including activation of inflammatory pathways, induction of oxidative stress, disruption of cellular function, and the capacity to act as carriers of chemical pollutants and toxic compounds. These mechanisms may contribute to disturbances affecting the gastrointestinal, respiratory, reproductive, and metabolic systems (Zhao et al., 2024; Leslie et al., 2022; Thin et al., 2025). The literature also proposes a possible association between microplastic exposure and carcinogenic processes, although the underlying mechanisms remain insufficiently understood (Goswami et al., 2024).

It should be emphasized, however, that current knowledge is based predominantly on animal models and in vitro studies, while robust clinical evidence in humans remains limited. Numerous methodological challenges further complicate the accurate assessment of health risks.

In conclusion, microplastics should presently be regarded as a potential, yet incompletely understood, health risk factor that warrants further well-designed clinical and epidemiological investigations to determine its true impact on human health.

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