



AIRBORNE MICROPLASTICS: EMERGING THREATS AND HEALTH IMPLICATIONS FOR HUMANS

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ABSTRACT

Airborne microplastics (MPs) have recently emerged as a pervasive environmental contaminant with growing concern for human health. These particles, derived from the degradation of plastics and synthetic fibers, are now detected in indoor and outdoor air, posing new challenges to environmental and biomedical sciences. This review synthesizes current findings on the sources, atmospheric transport, exposure routes, and toxicological impacts of airborne microplastics on humans. Evidence from recent studies indicates that inhalation and ingestion are the primary exposure pathways, allowing MPs to accumulate in the respiratory and gastrointestinal systems. The physicochemical properties of these particles—such as size, morphology, and polymer composition significantly influence their biological interactions and toxicity. Health effects include oxidative stress, inflammation, and potential interference with cellular metabolism, contributing to respiratory, cardiovascular, and neurological disorders. Despite increasing research attention, large-scale epidemiological data and standardized analytical protocols remain limited. Future work should emphasize advanced detection techniques, risk assessment frameworks, and policy measures to mitigate airborne plastic pollution and safeguard public health.

Keywords: Human health, Toxicity, Respiratory effects, Inhalation exposure, Atmospheric transport.

INTRODUCTION

Plastic pollution has become one of the most pressing environmental issues of the 21st century. The widespread use of synthetic polymers in packaging, textiles, and consumer products has resulted in extensive plastic waste accumulation in terrestrial and aquatic ecosystems. Over time, mechanical abrasion, ultraviolet radiation, and chemical weathering fragment these materials into smaller particles known as microplastics (MPs) typically defined as plastic debris less than 5 mm in size. Initially recognized as marine pollutants, microplastics are now detected in soil, freshwater, and even atmospheric environments, highlighting their pervasive and persistent nature. Recent evidence suggests that airborne microplastics represent an overlooked yet critical exposure route for humans. They originate from diverse sources, including tire abrasion, synthetic textiles, construction dust, and the breakdown of

urban waste. These microscopic particles can be suspended in the air, transported across long distances, and eventually inhaled or ingested by humans. Indoor environments, characterized by limited ventilation and high textile use, often contain microplastic concentrations several times higher than outdoor air. Consequently, humans are continuously exposed to microplastics not only through food and water but also via the air they breathe.

The health implications of such exposure remain an emerging research frontier. Studies have reported that microplastic particles can deposit deep within the respiratory tract, where they may induce oxidative stress, inflammatory responses, and cellular damage. Smaller nanoparticles can even cross epithelial barriers, entering the bloodstream and potentially accumulating in organs such as the liver, kidney, and brain. The polymer type, surface charge, and adsorbed toxic chemicals or pathogens can

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amplify their biological reactivity, posing complex toxicological challenges. In addition, microplastics may act as vectors for heavy metals, persistent organic pollutants (POPs), and pathogenic microorganisms, further compounding their potential risks. Despite growing concern, the mechanisms of toxicity, exposure assessment methods, and epidemiological evidence remain limited. Differences in sampling strategies, particle isolation techniques, and analytical instrumentation (e.g., FTIR, Raman spectroscopy, pyrolysis-GC/MS) have resulted in inconsistent data on environmental concentration and human exposure levels. These methodological variations hinder cross-study comparisons and the establishment of global monitoring frameworks. Moreover, while in-vitro and in-vivo studies demonstrate cytotoxic and inflammatory effects, direct links between airborne microplastic exposure and human diseases are still under investigation.

Airborne microplastics originate from diverse anthropogenic sources such as synthetic textiles, tire wear, packaging, and construction dust. Studies have documented MPs in both indoor and outdoor environments, with indoor concentrations typically higher due to textile shedding and limited ventilation. Gasperi *et al.* (2018) and Wright *et al.* (2020) provided early evidence that humans are continuously exposed through inhalation of atmospheric microplastics.

Prata *et al.* (2020) emphasized the importance of contamination control in air sampling procedures to prevent overestimation of concentrations. Zhang *et al.* (2020) demonstrated significant microplastic fallout in households, dominated by polyester and polypropylene fibers. O'Brien *et al.* (2020) and Bi *et al.* (2020) linked domestic laundry dryers and terrestrial plant surfaces to the global microplastic cycle. Microplastics can travel long distances via atmospheric currents and undergo vertical deposition influenced by humidity and wind speed. Vianello *et al.* (2019) simulated indoor exposure using a breathing thermal manikin, quantifying inhalable MP levels within residential environments.

Akanyange *et al.* (2021) reviewed atmospheric contamination pathways, highlighting urban sources and fiber morphology as key factors in transport potential. Mbachu *et al.* (2020) described the atmosphere as a "contaminant superhighway," integrating MPs from both marine and terrestrial systems. Stanton *et al.* (2019) added that natural fibers also dominate airborne particulates, complicating MP identification. Humans encounter microplastics primarily via inhalation and ingestion. Cox *et al.* (2019) estimated that an average adult consumes over 50,000 microplastic particles annually through food, water, and air. Schwabl *et al.* (2019) provided clinical confirmation by detecting MPs in human stool samples, evidencing ingestion and intestinal passage. Prata (2018) outlined potential respiratory deposition mechanisms, demonstrating that particles <10 µm can penetrate deep into pulmonary tissues. Li *et al.* (2023) reported evidence of MPs crossing biological barriers, potentially entering the circulatory system. Campanale *et al.* (2020) emphasized that polymer composition and surface additives influence

the bioavailability and retention of MPs in the human body. Wright and Kelly (2017) established that MPs can induce oxidative stress, inflammation, and genotoxic effects due to their small size and reactive surface. Hirt and Body-Malapel (2020) reviewed immunotoxic and gastrointestinal impacts, reporting that nano-sized plastics may disrupt epithelial integrity. Rist *et al.* (2018) critically assessed early studies and called for standardized protocols in health-impact research. Barboza *et al.* (2018) and Enyoh *et al.* (2019) summarized that MPs act as vectors for persistent organic pollutants (POPs) and heavy metals, intensifying toxicological effects. Wang *et al.* (2021) and Yee *et al.* (2021) highlighted potential links between microplastic exposure and respiratory and cardiovascular disorders, urging further epidemiological studies.

MATERIALS AND METHODS

This study adopts a systematic review approach in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure transparency and reproducibility. Inclusion Criteria relates Studies addressing the presence, characterization, or impact of airborne microplastics. Articles focused on human exposure pathways, including inhalation and ingestion. Research reporting quantitative or qualitative data on health effects or toxicity. Publications in English, available as full-text, between 2010–2024. Exclusion Criteria have Non-biomedical or non-environmental studies (e.g., industrial materials research). Information extracted included: (i) study location and sample type, (ii) particle characterization methods (e.g., FTIR, Raman spectroscopy, SEM), (iii) exposure estimation models, (iv) biological or toxicological endpoints, and (v) key health outcomes. A qualitative synthesis was performed to summarize findings across different exposure routes, toxic mechanisms, and risk factors. Quantitative comparisons of reported concentration ranges and polymer types were used to highlight trends in microplastic pollution and potential dose–response relationships.

RESULTS AND DISCUSSION

Analysis of the 25 reviewed studies reveals that airborne microplastic concentrations vary significantly across sampling environments, ranging from 0.3–60 particles m⁻³ in outdoor air to 5–300 particles m⁻³ in indoor environments. Indoor air consistently exhibited higher microplastic loads, attributed to synthetic textile abrasion, carpet wear, and dryer exhaust emissions. Morphological characterization using Scanning Electron Microscopy (SEM) and optical microscopy identified irregular, fibrous, and fragment-shaped particles. Typical dimensions ranged from 1–100 µm, with fiber lengths up to 5 mm observed in urban settings. Surface roughness analysis revealed increased microcracking and oxidation in aged particles, enhancing their potential to adsorb toxic pollutants such as polycyclic aromatic hydrocarbons (PAHs) and heavy metals (Zn, Pb, Cd). These physicochemical features amplify the reactive surface area, promoting secondary

pollutant interactions and oxidative stress once inhaled. Simulation and field data confirm that airborne MPs behave similarly to coarse PM₁₀ aerosols, with aerodynamic diameters between 0.3 and 10 µm allowing suspension times exceeding 72–96 hours. Atmospheric deposition fluxes were reported between 40 and 700 particles m⁻² day⁻¹, influenced by wind velocity, relative humidity, and precipitation frequency. Vianello *et al.* (2019) used a Breathing Thermal Manikin (BTM) to simulate indoor exposure, demonstrating direct deposition of synthetic fibers in the human breathing zone at rates up to 22 particles per 30 minutes under moderate airflow conditions. Such findings support the inhalation relevance of indoor microplastics as a persistent exposure route. Using the standard adult inhalation model with an air intake of ~20 m³ day⁻¹, the Estimated Daily Inhalation Dose (EDI) of airborne MPs can reach up to 4×10^4 particles day⁻¹ in heavily contaminated indoor settings. Particles smaller than 10 µm (PM₁₀ equivalent) penetrate deep into the respiratory tract, while those below 2.5 µm (PM_{2.5} equivalent) can reach the alveolar sacs. Microscopic and histopathological studies have reported MP fragments embedded in human lung tissues, confirming retention and biopersistence. Inhaled MPs may also undergo mucociliary clearance, leading to secondary ingestion and gastrointestinal exposure, further increasing total body burden. Hirt and Body-Malapel (2020) further demonstrated that microplastics disrupt tight-junction proteins (occludin and claudin-5) in gut epithelial cells, compromising intestinal barrier integrity. Collectively, these effects mimic fine particulate matter (PM_{2.5}) toxicity, though polymer-specific pathways (e.g., leaching of plasticizers, adsorption of persistent organic pollutants) introduce additional risks. Despite strong mechanistic evidence, quantitative human health risk assessment remains limited due to lack of standardized detection and exposure models. Hazard Quotients (HQ) for airborne MPs are currently estimated below unity (HQ < 1), suggesting low acute risk; however, cumulative exposure over decades could exceed physiological clearance capacity. Current toxicological databases (e.g., ECOTOX, ToxCast) lack reference values for polymer-based aerosols, underscoring the need for integrated toxicokinetic modeling.

CONCLUSION

Airborne microplastics (MPs) have become an emerging environmental and public-health concern. Evidence from multiple studies shows their presence in both indoor and outdoor air, with concentrations up to 300 particles m⁻³ indoors. The dominant polymers PET, PP, and PE exist mainly as fibers and fragments smaller than 100 µm, enabling deep respiratory penetration. Experimental and toxicological data confirm that MPs can trigger oxidative stress, inflammation, and cellular damage, similar to fine particulate matter. However, standardized detection methods, dose-response data, and long-term epidemiological studies remain limited. Overall, MPs represent a persistent and reactive airborne contaminant with potential chronic health implications.

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CONFLICT OF INTERESTS

The authors declare no conflict of interest

ETHICS APPROVAL

Not applicable

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AI TOOL DECLARATION

The authors declares that no AI and related tools are used to write the scientific content of this manuscript.

DATA AVAILABILITY

Data will be available on request

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