Airborne microplastics: a trojan horse for respiratory dysfunction and multiorgan damage

Domiziana Picone,[1*] Adelaide Carista,[1*] Giulia Maria Giorgia Lo Piano,[1] Olga Maria Manna,[2,3] Stefano Burgio,[3,4] Fabio Bucchieri,[1] Giuseppa D'Amico,[1] Alessandro Pitruzzella,[1#] Alberto Fucarino[5#]

[1]Department of Biomedicine, Neurosciences and Advanced Diagnostics (BiND), University of Palermo, Palermo; [2]Pathologic Anatomy Unit, Department of Health Promotion, Mother and Child Care, Internal Medicine and Medical Specialties, University of Palermo, Palermo; [3]Euro-Mediterranean Institute of Science and Technology (IEMEST), Palermo; [4]Department of Medicine and Surgery, Kore University of Enna, Enna; [5]Department of Theoretical and Applied Sciences, eCampus University, Novedrate (CO), Italy

[*]These authors contributed equally to this work.

[#]These authors contributed equally to this work.

Abstract

Microplastics (MPs), a class of pollutants, have emerged as a global challenge impacting both ecosystems and human health. Over recent decades, extensive research has been conducted to assess their presence across aquatic, terrestrial, and atmospheric environments. The pervasive accumulation of MPs, resulting from both excessive plastic consumption and inefficient waste management, has established ingestion (via the food chain) and inhalation (via ambient air) as primary routes of human exposure. While numerous studies have investigated the effects of ingested MPs, research on inhaled particles and their respiratory system impacts remains comparatively limited. Owing to their diminutive size, with MPs ranging from 1 µm to 5 mm and Nanoplastics (NPs) being smaller than 1 µm, they can penetrate bronchioles and pulmonary alveoli, eliciting both localized effects (e.g., inflammatory responses, oxidative stress) and systemic consequences. Notably, studies demonstrate that MPs can traverse the Blood-Brain Barrier (BBB), inducing neurotoxic effects. This review provides an overview of MPs' environmental impact and their documented effects on major human organs and tissues, with a focused analysis on the respiratory system. Specifically, we evaluate epidemiological studies and *in vitro* experimental models employed to elucidate the mechanisms by which MPs may contribute to chronic respiratory disease pathogenesis.

Correspondence: Giuseppa D'Amico, Department of Biomedicine, Neurosciences and Advanced Diagnostics (BiND), University of Palermo, Palermo, Italy.
E-mail: giuseppa.damico01@unipa.it

Key words: microplastics, inhalation exposure, respiratory system, oxidative stress, inflammation.

Contributions: AF, AP, and FB, conceptualization; DP, AC, GMGLP, OMM, SB, GD writing—original draft preparation; DP, GD, AF, writing—review and editing; FB, AF, AP supervision. All authors have read and agreed to the published version of the manuscript.

Funding: this research received no external funding.

Conflicts of interest: the authors declare no conflicts of interest.

Ethics approval and conset to participate: not applicable.

Availability of data and materials: all data generated or analyzed during this study are included in this published article.

Received: 4 May 2025.
Accepted: 14 October 2025.
Early view: 22 October 2025.

Publisher's note: all claims expressed in this article are solely those of the authors and do not necessarily represent those of their affiliated organizations, or those of the publisher, the editors and the reviewers. Any product that may be evaluated in this article or claim that may be made by its manufacturer is not guaranteed or endorsed by the publisher.

©Copyright: the Author(s), 2025
Licensee PAGEPress, Italy
Journal of Biological Research 2025; 98(s3):13945
doi:10.4081/jbr.2025.13945

This article is distributed under the terms of the Creative Commons Attribution-NonCommercial International License (CC BY-NC 4.0) which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author(s) and source are credited.

Introduction

Over the past decades, the widespread production and improper disposal of plastic waste have led to growing concerns about plastic pollution and its potential consequences for both human health and the environment. Plastics are synthetic polymers synthesized from monomers derived from petrochemical sources, such as oil or gas, often incorporating a range of chemical additives to modify their characteristics.[1] Polymers commonly found in commercial products include materials like Polypropylene (PP), Polyethylene (PE), Polyethylene Terephthalate (PET), Polystyrene (PS), and Polyvinyl Chloride (PVC).[2] Once released into the environment, these materials can be fragmented through physical or chemical processes. In recent years, Microplastics (MPs) and Nanoplastics (NPs) have emerged as pollutants of particular concern. MPs are typically defined as plastic particles ranging from 1 µm to 5 mm, while NPs are generally considered to be smaller than 1 µm.[3] Nonetheless, the absence of a universally accepted size classification (some sources define NPs as smaller than 100 nm, others extend the boundary to 1000 nm) has introduced a degree of ambiguity in the literature, making it harder to compare studies, develop accurate detection methods, and establish clear regulations.[4] MPs can also be catego-

rized based on their origin: primary microplastics are intentionally produced at microscopic sizes, whereas secondary microplastics are those originating from the fragmentation of larger plastic wastes, which can happen over time in both aquatic or terrestrial environment.[5] The detection of these particles presents significant challenges due to their small sizes, complicating monitoring efforts. Moreover, these tiny particles are highly mobile and can be distributed throughout different compartments of the environment, such as the air, soil, and water. Approximately 65 million microplastic particles are estimated to be released into water on a daily basis as a result of activities taking place at wastewater treatment facilities.[6] Despite ongoing efforts to improve wastewater filtration and management, a substantial number of MPs originating from domestic, industrial, and personal care products continues to pass through treatment facilities and enter aquatic environments, exacerbating the contamination of rivers, lakes, and oceans.[7] The COVID-19 pandemic significantly worsened the already critical issue of plastic pollution due to the widespread use of Personal Protective Equipment (PPE), including single-use masks and gloves. In addition, the predominance of plastic in medical devices and equipment further increased the amount of plastic waste generated during the global health crisis.[8] In this context, it has been estimated that during the COVID-19 outbreak in Wuhan, China, medical waste sharply increased from 40 tons/day to nearly 240 tons/day at its peak, exceeding the incineration limit of 49 tons/day and challenging the capacity of waste management systems.[9] Indeed, globally, poor management of plastic waste remains one of the major environmental challenges, contributing significantly to plastic pollution and bringing waste disposal systems under increasing pressure.[9]

Plastic constitutes the largest portion of marine debris; for example, it was seen that in 2017 more than eight million tons of plastic waste were released into the oceans, an amount that is 33 times greater than the total plastic accumulated in the oceans by 2015.[10] Moreover, recent estimates indicate that by 2020, approximately 3 to 11 million metric tonnes of plastic had accumulated on the seafloor, highlighting the critical role of the deep ocean as a long-term reservoir for plastic pollution.[11] Studies have shown that MPs are nearly ubiquitous in marine environments, and due to their ability to easily move across trophic levels in the food chain, they have a significant impact also on the health of marine animals.[10-13] In addition, it has been reported that they can act as carriers of antibiotic resistance genes, presenting additional significant risks to both humans and aquatic ecosystems.[14] Microplastic impact on terrestrial ecosystems has also been extensively studied and documented, with agricultural practices identified as one of the primary sources of soil contamination.[2,15] As demonstrated by De Souza Machado *et al.*,[16] MPs in soil can alter crucial physical properties such as bulk density and water retention. Lastly, the atmosphere has a pivotal role in the dispersion of MPs. Processes such as atmospheric circulation and both wet and dry deposition are mechanisms through which plastic particles originating from land-based sources can disperse into other environmental compartments.[2] Although the presence and abundance of MPs have been extensively investigated in many environmental settings, especially in aquatic ecosystems, studies focusing specifically on the occurrence and characterization of MPs in the atmosphere remain limited. In 2015, Dris *et al.* identified MPs (mainly fibers) in atmospheric fallout for the first time. Their study also provided insights into the types and sizes of MPs, specifically within the 100–5000 µm range.[17] Recently, interest in atmospheric MPs has increased, with studies identifying airborne MPs across diverse environments. These include urban areas globally,[18-20] indoor spaces,[21,22] and even remote terrestrial regions.[23] However, the characterization of airborne MPs and NPs, particularly in aerosol samples, represents a challenging task due to their small size and the limitations of current analytical instruments. As a result, there is a lack of comprehensive studies focusing on the chemical composition and distribution of airborne MPs.

In 2022, Yao and colleagues conducted a study in northern New Jersey where they characterized microplastic particles in indoor and outdoor air samples. They identified diverse MPs forms, such as fibers and fragments, and attributed their major sources to textiles and other household products. These results indicate that microplastic particles are readily abundant in urban air, posing potential risks to both human health and the environment.[24] A 2023 study by Tatsii *et al.* further explored the role of MPs shape in atmospheric transport, revealing that MPs fibers can travel longer distances and remain airborne longer than other shapes of MPs. This property significantly enhances their ability to disperse across various environments.[25] Most concerning is the potential impact of MPs on human health since these particles can enter the human body through various routes, including inhalation, ingestion, and dermal contact.[26] Thus far, most studies on human exposure to MPs have primarily focused on the gastrointestinal effects resulting from ingesting these particles through food and water.[27,28] However, recent studies indicate that the level of human exposure through inhalation may be two to three times greater than that through ingestion.[29] The presence of plastic fibers in human lungs was first documented by Pauly *et al.* in 1998.[30] Later, in 2021, Amato *et al.* identified and characterized environmental MPs in human lung tissue obtained from autopsies.[31] Their findings revealed that degraded particles from commonly used plastics, such as PP and PE, were most prevalent in lung tissue samples. This provided strong evidence of the impact of MPs, present in the air we breathe, on the respiratory system where the lungs serve as a site for the accumulation of these air pollutants.[31] Despite the growing attention to airborne MPs, progress in this research field has been significantly constrained by methodological inconsistencies. As Zhang *et al*. pointed out, the lack of standardized protocols, combined with the technical challenges of analyzing microscopic plastic particles, has led to differences in sample collection approaches (e.g., active air sampling, passive sampling, or analysis of settled dust), handling, and pretreatment, complicating the comparability of data across studies.[32] To date, standard analytical tools employed for MPs detection and characterization include optical and fluorescence microscopy, Fourier-Transform Infrared (FTIR) and Raman spectroscopy, alongside thermal analytical methods such as pyrolysis-Gas Chromatography/Mass Spectrometry (pyrolysis-GC/MS).[33]

Due to the urgent need for standardized protocols, Kadija *et al.* developed a reproducible method for analyzing airborne MPs. Their approach combines sonication, oxidative digestion with 15% H_2O_2 at 70°C, and density separation using $ZnCl_2$. This method preserves particle integrity and completes sample processing within two days, offering a promising step toward greater methodological consistency and improved data comparability in the field.[34]

Although several *in vitro* models have been used to study the effects of MPs on the respiratory tract, such as immortalized cell lines (e.g., A549 and BEAS-2B), human airway organoids, and primary human respiratory epithelial cells, the main problem lies in the significant gap in understanding the molecular mechanisms and the real implications of MPs on human health.[35] Although their effects are still under investigation, studies indicate that plastic particles may negatively impact cells by causing oxidative damage, triggering inflammation, potentially damaging genetic material, and disrupting the endocrine system.[36-38] Notably, increased production of Reactive Oxygen Species (ROS) can lead to inflammation, mitochondrial dysfunction, and even programmed cell death, potentially increasing the risk of cancer.[36] Despite growing evidence of the adverse effects of MPs on cellular structures and functions, and considering that the respiratory system is a direct and easily accessible pathway for air-

borne MPs, research regarding the respiratory system remains limited. To address this knowledge gap, this review aims to analyze the documented health risks associated with MPs, with a particular focus on the respiratory system, given the ease with which airborne MPs can be inhaled. Therefore, a better understanding of the interaction of MPs with the respiratory system is critical to the investigation of their long-term impact on human health, which in turn paves the way for more effective mitigation strategies and public health interventions.

Microplastics effects on human health

The ubiquitous presence of MPs in the environment has raised significant concerns regarding their potential effects on human health. Although MPs primarily enter the human body through the intestinal, dermal, and respiratory tracts, they have been detected in various anatomical sites and biological fluids, suggesting their translocation to organs and tissues beyond the initial entry points. This translocation can trigger oxidative stress, inflammatory responses, and cytotoxic processes.[39,40] Specifically, MPs ingested daily with food accumulate in the gut, where they can compromise the integrity of the intestinal barrier by altering metabolic functions and triggering chronic inflammatory processes. Notably, only a small fraction of ingested MPs is absorbed, while the majority is excreted through feces.[41] Studies in murine models have demonstrated that exposure to polystyrene MPs (PS-MPs) disrupts intestinal epithelial tight junctions, leading to dysregulated substance passage. In particular, Su and colleagues demonstrated that 4-week administration of MPs-PS alters gene expression of tight junctions in the jejunum and duodenum and the composition of the intestinal flora.[42] Similar results were obtained in another study in which it was shown that administering PS-MPs for 28 days causes increased intestinal permeability and reduced mucus production due to the downregulation of tight junction proteins such as zona occludens-1 (ZO-1), occludin (OCLN) and claudin-1 (CLDN-1) at the colon level.[43] The same study revealed that MPs can induce oxidative stress by impairing the function of the intestinal barrier via the ROS-dependent Nuclear Factor kappa-light-chain-enhancer of activated B cells (NF-κB)/Nucleotide-Binding Oligomerization Domain (NOD)-like receptor family pyrin domain containing 3 (NLRP3)/Interleukin-1 beta (IL-1β)/Myosin Light Chain Kinase (MLCK) pathway.

The damaging effects of MPs are also associated with their chemical components. Histological studies in mouse models have shown that exposure to MPs in polyethylene at high concentrations (600 μg/mL) is associated with disintegration and atrophy of the intestinal villi. This condition was also associated with severe weight loss in mice due to reduced absorption of essential nutrients such as vitamins and folates.[44] MPs with a diameter between 1 and 10 μm can penetrate the intestinal barrier, impair colic function and alter the composition of the microbiota.[45,46] In particular, Chen and colleagues demonstrated that the accumulation of MPs is associated with a reduction in Actinobacteria and an increase in the genera *Muribaculum, Akkermansia, Anaerostipes* and *Prevotella*, microorganisms related to intestinal barrier dysfunction and the triggering of inflammatory processes. The same study shows that the accumulation of MPs causes a reduction in the expression of genes associated with mucus production, such as *Muc1*, *Muc2* and *Kfl4*.[46] Similar results were obtained with *in vitro* studies, which demonstrated a correlation between MPs hyperaccumulation and dysfunction of the gut microbiota.[47] MPs accumulation seems to be associated with chronic inflammatory diseases such as Chron's disease.[48] Yan and colleagues[48] found a positive correlation between MPs concentration in faecal samples from patients with Inflammatory Bowel Disease (IBD) compared to control patients, while Wu *et al.* demonstrated a positive correlation between intestinal fibrosis in patients with Chron's disease and MPs concentration in the ileum emphasising the close correlation between MPs and disease progression.[49]

Given the close correlation between the gut and liver, it is clear how intestinal imbalances can affect also liver function. Indeed, numerous studies document the hepatocytotoxic role of ingested MPs and their ability to induce both disturbances in lipid metabolism and in the circulation of biochemical markers relating to liver function and fibrosis-related proteins.[50] In a study conducted by Chen and colleagues, the effect of chronic exposure to PVC-MPs of a size of 0.2 μm and a concentration of 0.5 mg/day was evaluated.[46]

Furthermore, biochemical investigations showed a significant increase in circulating Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) levels indicative of liver damage and activation of the Phosphatidylinositol 3-Kinase (PIK3K)/Protein Kinase B (PKB) signalling pathway associated with apoptosis and oxidative stress. The same study showed alterations in lipid metabolism with reductions in triglycerides and bile acids, and downregulation in Peroxisome Proliferator-Activated Receptor gamma (PPARγ) was observed.[51]

The role of MPs in liver fibrosis was further confirmed in a study using both mouse models and stabilized liver cell lines. This study showed that the accumulation of MPs less than 1μm (1 mg/L) for 60 days could activate the Cyclic GMP–AMP synthase (cGAS)/Stimulator of Interferon Genes (STING) pathway and trigger a chronic inflammatory response leading to the synthesis of fibrosis-associated proteins such as Alpha-smooth muscle actin (α-SMA) and fibronectin.[52]

Additionally, MPs have been shown to activate the immune system, leading to the formation of Macrophage Extracellular Traps (METs), which exacerbate liver inflammation and damage.[53]

An interesting study conducted in 2022 correlates the dysfunction of liver metabolism with the triggering of ferroptosis and apoptotic mechanisms in the cerebellum. The study was conducted on chickens exposed to PS-MPs for 6 weeks. PS-MPs alter hepatic glutamine metabolism, increasing the synthesis of glutamate and other oxidising compounds. The latter can cross the Blood-Brain Barrier (BBB) causing neurotoxicity and reducing Purkinje cell numbers. Importantly, MPs that cross the BBB appear to be capable of impairing learning.[54] This was demonstrated in a study in which MPs-induced oxidative stress was associated with increased Acetylcholinesterase (AchE) activity and a decrease in acetylcholine and Choline Acetyltransferase (ChAT), impairing cognitive function.[55] Furthermore, MP exposure led to elevated levels of ROS and Malondialdehyde (MDA), alongside reduced Glutathione (GSH) levels, resulting in brain damage. The cAMP Response Element-Binding Protein (CREB)/Brain-Derived Neurotrophic Factor (BDNF) pathway, crucial for long-term memory, was also significantly impaired.[55]

The second route of entry for MPs is by inhalation. The toxic effects of MPs in the brain have also been shown following inhalation. A recent study on cadavers showed that MPs could accumulate in the olfactory bulb, suggesting that this may be a direct route of entry into the brain.[56] Another study on subjects exposed to PS-MPs found a high concentration in the blood with the presence of neurological symptoms, suggesting a possible correlation with MPs inhalation.[57] Also related to MPs-PS inhalation, a study conducted on a cohort of employees of a plastics factory showed a very high concentration of their metabolites, Maleic Acid (MA) and Polyglutamic Acid (PGA), in urine, as well as mutations in DNA associated with genotoxic damage.[58]

Focus on the effects of MPs in the respiratory system

In recent years, increasing attention has been directed toward the respiratory consequences of inhaling MPs and NPs from contaminated air. Due to their reduced size, inhaled NPs can accumulate in the lungs and become internalized by alveolar epithelial cells, posing a potential risk factor for respiratory diseases.[59] The alveolar surface, spanning approximately 150 m² with a thin tissue barrier, facilitates NP penetration into capillaries, enabling systemic dissemination.[60] Upon inhalation, plastic particles >10 μm predominantly deposit in the upper airways, while those <10 μm reach the bronchioles, and ultrafine particles (<0.1 μm) penetrate the alveoli. Absorption of smaller MPs via the alveolar epithelium can induce systemic inflammation through the production of pro-inflammatory factors.[61] In 2023, the detection of MPs in human bloodstreams raised concerns about the long-term safety of airborne plastic exposure and underscored the urgency of regulatory measures to mitigate atmospheric plastic emissions.[61] Human biomonitoring studies have demonstrated that MPs in lung tissue induce chronic inflammation, mirroring pathological features observed in asthma and Chronic Obstructive Pulmonary Disease (COPD).[62] Prolonged MPs exposure has also been linked to pulmonary fibrosis.[63] A 2020 clinical case reported hypersensitivity pneumonitis in an individual employed in PET production from 1992 to 2013, with occupational exposure to terephthalic acid and dimethyl terephthalate, precursors in PET synthesis, identified as the causative agents. This marked the first documented case of Occupational Hypersensitivity Pneumonitis (OHP) associated with PET production.[64]

Numerous clinical reports further corroborate the development of respiratory pathologies following prolonged plastic exposure. The role of MNPs in asthma pathogenesis remains unclear. Chen *et al.*[65] addressed this gap by comparing Micro- and Nano-plastics (MNP) levels in Bronchoalveolar Lavage Fluid (BALF) between children with Community-Acquired Pneumonia (CAP) and asthma. While severe CAP cases exhibited higher MNP prevalence, no significant differences were observed between CAP and asthma cohorts. Notably, microfiber abundance correlated positively with patient's age.[65] Chronic MPs exposure is associated with long-term respiratory sequelae, including pulmonary interstitial fibrosis, chronic pneumonia, and lung cancer, particularly in occupationally exposed or susceptible populations.[56] A recent cohort study analyzing sputum, BALF, and pleural fluid confirmed inhalation as the primary MPs entry route into the respiratory system, with deposition patterns influenced by particle size, shape, occupational exposure, and smoking status.[66] *In vitro* studies have elucidated MNP mechanisms in airway toxicity. Traversa *et al.*[59] exposed human bronchial (BEAS-2B) and alveolar (A549) epithelial cells to polyethylene MNPs (25–100 μg/mL) for 24 hours, observing dose-dependent morphological changes and elevated oxidative stress via immune defense suppression. Similarly, polystyrene particles disrupted barrier integrity in BEAS2B cells, exhibiting pro-inflammatory and cytotoxic effects.[67] Although no studies have directly compared MNP levels in lung tissue between healthy individuals and COPD patients, smoking, a primary COPD risk factor, has been linked to elevated MPs exposure. Lu *et al.*[68] detected MPs (20–500 μm) in cigarette smoke and found higher concentrations in smokers' BALF versus non-smokers. Huang *et al.*[69] similarly reported increased MPs in sputum from smokers and COPD patients compared to controls. *In vitro* models further implicate MNPs in COPD pathogenesis: polystyrene microparticles reduced α1-antitrypsin expression in bronchial epithelial cells,[70] while nanoparticles replicated this effect in a lung-on-a-chip system, highlighting MNPs' role in compromising protease-antiprotease balance.[71] Finally, a schematic representation of the major adverse effects of MPs on the respiratory system are summarized in Figure 1.

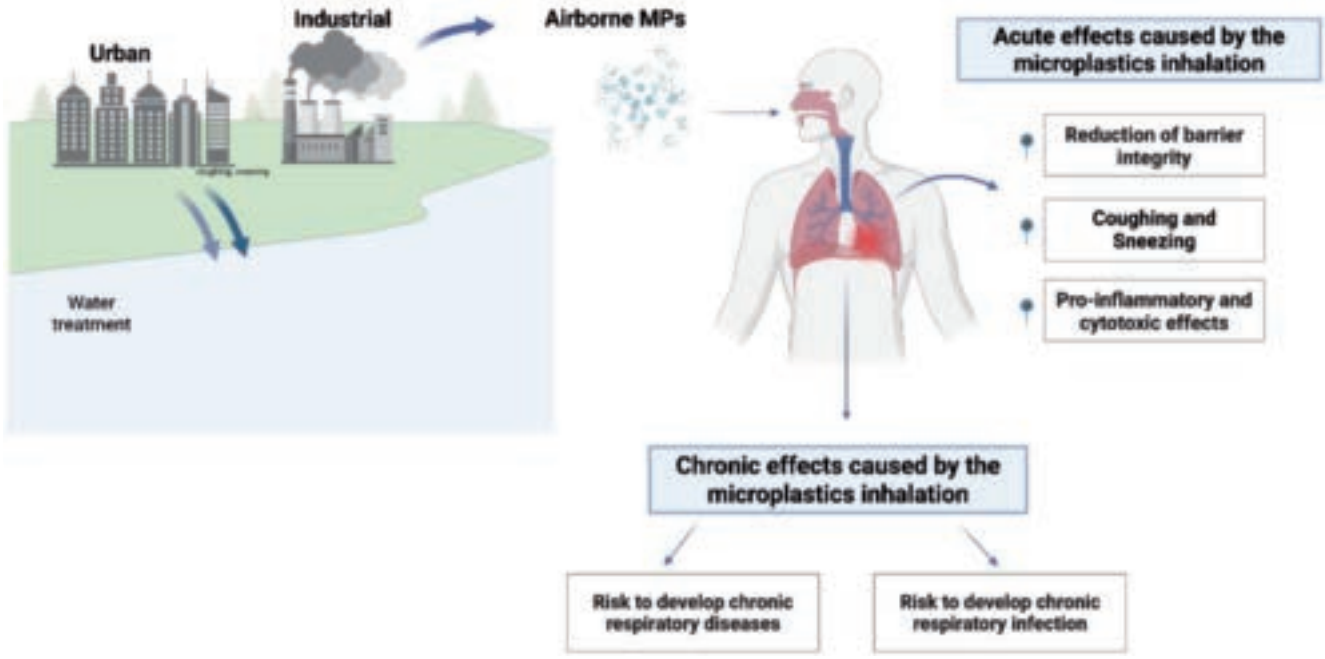

Figure 1. Schematic representation of the effects of microplastics (MPs) in the respiratory system. Urban and industrial emissions contribute to the presence of MPs in the air, which once inhaled can cause inflammation, oxidative stress, lung fibrosis and pro-inflammatory and cytotoxic effects. (Created with Biorender).

Conclusions

Only in recent decades the scientific community has focused attention on the problem of plastic pollution for human health. The aim of this review was to summarize the current evidence on the environmental distribution of MPs, their routes of human exposure and, most importantly, their effects on respiratory health since, while ingestion has long been recognized as a major route of exposure, inhalation of airborne MPs has only recently begun to receive the attention of the scientific community. Airborne MPs, due to their small size, can reach deeper regions of the respiratory tract, where they interact with epithelial and immune cells, triggering inflammatory responses, oxidative stress and potential long-term pathological changes. In particular, their ability to translocate from the lungs to the systemic circulation raises additional concerns about their multiorgan toxicity, including their impact on the gut, liver, and central nervous system. Despite growing awareness, several challenges limit our understanding of the true extent of MPs toxicity. Methodological inconsistencies in sampling, detection, and analysis, along with the predominant use of *in vitro* and animal models, limit the current understanding of MPs' negative effects. To overcome these challenges, future research should adopt standardized analytical protocols and more advanced biological systems, such as 3D airway models and organ-on-chip platforms, that better reflect human physiology. A deeper understanding of the molecular and cellular effects of MPs will be essential to develop effective public health interventions.

References

1. Thompson RC, Swan SH, Moore CJ, vom Saal FS. Our plastic age. Philos Trans R Soc Lond B Biol Sci 2009;364:1973-6.
2. Wang L, Zu B, Yang Q, et al. Distribution and environmental effects of microplastics: a systematic review. RSC Adv 2023;13:15566-15574.
3. Arif Y, Mir AR, Zieliński P, et al. Microplastics and nanoplastics: source, behavior, remediation, and multi-level environmental impact. J Environ Manage 2024;356:120618.
4. Hartmann NB, Hüffer T, Thompson RC, et al. Are we speaking the same language? Recommendations for a definition and categorization framework for plastic debris. Environ Sci Technol 2019;53:1039-47.
5. Jing S, Wang C, Li G. Defining primary and secondary microplastics: a connotation analysis. ACS ES&T Water 2024;4:2330-2.
6. Murphy F, Ewins C, Carbonnier F, Quinn B. Wastewater treatment works (WwTW) as a source of microplastics in the aquatic environment. Environ Sci Technol 2016;50:5800-8.
7. Talukdar A, Kundu P, Bhattacharya S, Dutta N. Microplastic contamination in wastewater: sources, distribution, detection and remediation through physical and chemical-biological methods. Sci Total Environ 2024;916:170254.
. Wang Q, Zhang M, Li R. The COVID-19 pandemic reshapes the plastic pollution research - A comparative analysis of plastic pollution research before and during the pandemic. Environ Res 2022;208:112634.
9. Klemeš JJ, Fan YV, Tan RR, Jiang P. Minimising the present and future plastic waste, energy and environmental footprints related to COVID-19. Renew Sustain Energy Rev 2020;127:109883.
10. Peng L, Fu D, Qi H, et al. Micro- and nano-plastics in marine environment: source, distribution and threats - A review. Sci Total Environ 2020;698:134254.
11. Kushwaha M, Shankar S, Goel D, et al. Microplastics pollution in the marine environment: a review of sources, impacts and mitigation. Mar Pollut Bull 2024;209:117109.
12. Duis K, Coors A. Microplastics in the aquatic and terrestrial environment: sources (with a specific focus on personal care products), fate and effects. Environ Sci Eur 2016;28:2.
13. He B, Wijesiri B, Ayoko GA, et al. Influential factors on microplastics occurrence in river sediments. Sci Total Environ 2020;738:139901.
14. Jaafarzadeh N, Talepour N. Microplastics as carriers of antibiotic resistance genes and pathogens in municipal solid waste (MSW) landfill leachate and soil: a review. J Environ Health Sci Eng 2024;22:1–12.
15. Hasan MM, Tarannum MN, Rafiqul Bari GAKM et al. Impact of microplastics on terrestrial ecosystems: a plant-centric perspective. Environ Pollut Manag 2024;1:223-34.
16. De Souza Machado AA, Kloas W, Zarfl C, et al. Microplastics as an emerging threat to terrestrial ecosystems. Glob Chang Biol 2018;24:1405-16.
17. Dris R, Gasperi J, Rocher V, et al. Microplastic contamination in an urban area: a case study in Greater Paris. Environ Chem 2015;12:592-9.
18. Nandi S, Naresh Kumar R, Dhandapani A, Iqbal J. Characterization of microplastics in outdoor and indoor air in Ranchi, Jharkhand, India: First insights from the region. Environ Pollut 2024;346:123543.
19. Wright SL, Ulke J, Font A, et al. Atmospheric microplastic deposition in an urban environment and an evaluation of transport. Environ Int 2020;136:105411.
20. Liu K, Wang X, Wei N, et al. Accurate quantification and transport estimation of suspended atmospheric microplastics in megacities: implications for human health. Environ Int 2019;132:105127.
21. Sheraz M, Kim J, Kim J. Nano/microplastics in indoor air: a critical review of synthesis routes for toxicity testing and preventative measure strategies. Process Saf Environ Prot 2023;180:274-304.
22. Dris R, Gasperi J, Saad M, et al. Synthetic fibers in atmospheric fallout: a source of microplastics in the environment? Mar Pollut Bull 2016;104:290-3.
23. Aves AR, Revell LE, Gaw S, et. al First evidence of microplastics in Antarctic snow. The Cryosphere 2022;16:2127–45.
24. Yao Y, Glamoclija M, Murphy A, Gao Y. Characterization of microplastics in indoor and ambient air in northern New Jersey. Environ Res 2022;207:112142.
25. Tatsii D, Bucci S, Bhowmick T, et al. Shape matters: long-range transport of microplastic fibers in the atmosphere. Environ Sci Technol 2023;58.1:671-82.
26. Li Y, Chen L, Zhou N, et al. Microplastics in the human body: a comprehensive review of exposure, distribution, migration mechanisms, and toxicity. Sci Total Environ 2024;946:174215.
27. Verla AW, Enyoh CE, Verla EN, et al. Microplastic–toxic chemical interaction: a review study on quantified levels, mechanism and implication. SN Appl Sci 2019;1:1400.
28. Prata J, da Costa JP, Lopes I, et al. Environmental exposure to microplastics: an overview on possible human health effects. Sci Total Environ 2020;702:134455.
29. Akanyange SN, Lyu X, Zhao X, et al. Does microplastic really represent a threat? A review of the atmospheric contamination sources and potential impacts. Sci Total Environ 2021;777:146020.

30. Pauly JL, Stegmeier SJ, Allaart HA, et al. Inhaled cellulosic and plastic fibers found in human lung tissue. Cancer Epidemiol Biomarkers Prev 1998;7:419-28.
31. Amato-Lourenço LF, Carvalho-Oliveira R, Ribeiro Júnior G, et al. Presence of airborne microplastics in human lung tissue. J Hazard Mater 2021;416:126124.
32. Zhang Y, Kang S, Allen S, et al. Atmospheric microplastics: a review on current status and perspectives. Earth-Sci Rev 2020;203:103118.
33. Sharma P, Sharma P, Abhishek K. Sampling, separation, and characterization methodology for quantification of microplastic from the environment. J Hazard Mater Adv 2024;14: 100416.
34. Sharaf Din K, Khokhar MF, Butt SI, et al. Exploration of microplastic concentration in indoor and outdoor air samples: morphological, polymeric, and elemental analysis. Sci Total Environ 2024;908:168398.
35. Vattanasit U, Kongpran J, Ikeda A. Airborne microplastics: a narrative review of potential effects on the human respiratory system. Sci Total Environ 2023;904:166745.
36. Nawab A, Ahmad M, Khan MT, et al. Human exposure to microplastics: a review on exposure routes and public health impacts. J Hazard Mater Adv 2024;16:100487.
37. Lei L, Wu S, Lu S, et al. Microplastic particles cause intestinal damage and other adverse effects in zebrafish *Danio rerio* and nematode *Caenorhabditis elegans*. Sci Total Environ 2018; 619-620:1-8.
38. Kadac-Czapska K, Jutrzenka Trzebiatowska P, Mazurkiewicz M, et al. Isolation and identification of microplastics in infant formulas - a potential health risk for children. Food Chem 2024;440:138246.
39. Han Y, Song Y, et al. No prominent toxicity of polyethylene microplastics observed in neonatal mice following intratracheal instillation to dams during gestational and neonatal period. Toxicol Res 2021;37:443-50.
40. Zhu L, Ma M, Sun X, et al. Microplastics entry into the blood by infusion therapy: few but a direct pathway. Environ Sci Technol Lett 2023;11:67-72.
41. Zhang N, Li YB, He HR, et al. You are what you eat: microplastics in the feces of young men living in Beijing. Sci Total Environ 2021;767:144345.
42. Su QL, Wu J, Tan SW, et al. The impact of microplastics polystyrene on the microscopic structure of mouse intestine, tight junction genes and gut microbiota. PLoS One 2024;19: e0304686.
43. Zeng G, Li J, Wang Y, et al. Polystyrene microplastic-induced oxidative stress triggers intestinal barrier dysfunction via the NF-κB/NLRP3/IL-1β/MCLK pathway. Environ Pollut 2024; 345:123473.
44. Saleh SMM, Abdel-Zaher S, Mohamed MS, et al. Microplastics induced ileum damage: morphological and immunohistochemical study. Microsc Res Tech 2024;88:251-69.
45. Donkers JM, Höppener EM, Grigoriev I, et al. Advanced epithelial lung and gut barrier models demonstrate passage of microplastic particles. Microplast Nanoplast 2022;2:6.
46. Chen X, Zhuang J, Chen Q, et al. Polyvinyl chloride microplastics induced gut barrier dysfunction, microbiota dysbiosis and metabolism disorder in adult mice. Ecotoxicol Environ Saf 2022;241:113809.
47. Fournier E, Leveque M, Ruiz P, et al. Microplastics: what happens in the human digestive tract? First evidences in adults using in vitro gut models. J Hazard Mater 2023;442:130010.
48. Yan Z, Liu Y, Zhang T, et al. Analysis of microplastics in human feces reveals a correlation between fecal microplastics and inflammatory bowel disease status. Environ Sci Technol 2022;56:414-21.
49. Wu F, Wu F, Liu X, et al. Microplastic accumulation in fibrotic intestinal tissue and mesenteric adipose tissue in Crohn's disease patients. Environ Res 2025;271:121077.
50. Xu R, Cao JW, Lv HL, et al. Polyethylene microplastics induced gut microbiota dysbiosis leading to liver injury via the TLR2/NF-κB/NLRP3 pathway in mice. Sci Total Environ 2024;917:170518.
51. Chen X, Zhuang J, Chen Q, et al. Chronic exposure to polyvinyl chloride microplastics induces liver injury and gut microbiota dysbiosis based on the integration of liver transcriptome profiles and full-length 16S rRNA sequencing data. Sci Total Environ 2022;839:155984.
52. Shen R, Yang K, Cheng X, et al. Accumulation of polystyrene microplastics induces liver fibrosis by activating cGAS/STING pathway. Environ Pollut 2022;300:118986.
53. Yin K, Wang D, Zhang Y, et al. Polystyrene microplastics promote liver inflammation by inducing the formation of macrophages extracellular traps. J Hazard Mater 2023;452: 131236.
54. Yin K, Wang D, Zhao H, et al. Polystyrene microplastics upregulates liver glutamine and glutamate synthesis and promotes autophagy-dependent ferroptosis and apoptosis in the cerebellum through the liver-brain axis. Environ Pollut 2022;307:119449.
55. Wang S, Han Q, Wei Z, et al. Polystyrene microplastics affect learning and memory in mice by inducing oxidative stress and decreasing the level of acetylcholine. Food Chem Toxicol 2022;162:112904.
56. Amato-Lourenço LF, Dantas KC, Ribeiro Júnior G, et al. Microplastics in the olfactory bulb of the human brain. JAMA Netw Open 2024;7:e2440018.
57. Werder EJ, Engel LS, Richardson DB, et al. Environmental styrene exposure and neurologic symptoms in U. S. Gulf coast residents. Environ Int 2018;121:480-90.
58. Teixeira JP, Gaspar J, Coelho P, et al. Cytogenetic and DNA damage on workers exposed to styrene. Mutagenesis 2010;25: 617-21.
59. Traversa A, Mari E, Pontecorvi P, et al. Polyethylene micro/nanoplastics exposure induces epithelial-mesenchymal transition in human bronchial and alveolar epithelial cells. Int J Mol Sci 2024;25:10168.
60. Lehner F, Wood AW, Vano JA, et al. The potential to reduce uncertainty in regional runoff projections from climate models. Nat Clim Chang 2019;9:926–33.
61. Khan A, Jia Z. Recent insights into uptake, toxicity, and molecular targets of microplastics and nanoplastics relevant to human health impacts. iScience 2023;26:106061.
62. Saha SC, Saha G. Effect of microplastics deposition on human lung airways: a review with computational benefits and challenges. Heliyon 2024;10:e24355.
63. Wright SL, Kelly FJ. Plastic and human health: a micro issue? Environ Sci Technol 2017;51:6634-47.
64. Sartorelli P, d'Hauw G, Spina D, et. Al. A case of hypersensitivity pneumonitis in a worker exposed to terephthalic acid in the production of polyethylene terephthalate. Int J Occup Med Environ Health 2020;33:119-23.
65. Chen C, Liu F, Quan S, et al. Microplastics in the bronchoalveolar lavage fluid of chinese children: associations with age, city development, and disease features. Environ Sci Technol 2023;57:12594-601.

66. Momeni MK, Taghipour H, Ghayebzadeh M, et al. Isolation and characterization of microplastics from the human respiratory system: sputum, broncho-alveolar lavage fluid, and pleural fluid simultaneously. Environ Pollut 2025;365:125389.
67. Jeon MS, Kim JW, Han YB, et al. Polystyrene microplastic particles induce autophagic cell death in BEAS-2B human bronchial epithelial cells. Environ Toxicol 2023;38:359-67.
68. Lu W, Li X, Wang S, et al. New evidence of microplastics in the lower respiratory tract: inhalation through smoking. Environ Sci Technol 2023;57:23.
69. Huang S, Huang X, Bi R, et al. Detection and analysis of microplastics in human sputum. Environ Sci Technol 2022; 56:4.
70. Dong CD, Chen CW, Chen YC, et al. Polystyrene microplastic particles: *in vitro* pulmonary toxicity assessment. J Hazard Mater 2020;385:121575.
71. Yang S, Zhang T, Ge Y, et al. Sentinel supervised lung-on-a-chip: a new environmental toxicology platform for nanoplastic-induced lung injury. J Hazard Mater 2023;458: 131962.