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Breathing plastics: Influence of airborne microplastics on the respiratory microbiome and health of human lungs (Review)

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Abstract. The issue of airborne microplastic pollution is a significant environmental challenge due to its widespread and rapid distribution and potential health hazards. One of these issues is the potential impact of microplastics on the lung microbiome and its potential effects on respiratory health. Evidence suggests that microplastic fibers may contribute to an increased incidence of respiratory conditions, including asthma, by affecting the lung microbiome and triggering inflammation. The lung microbiome is essential for regulating innate and adaptive immune mechanisms. The microbiome contributes to maintaining lung homeostasis by inhibiting the growth of pathogenic microbial colonies and supporting tissue repair processes. It has been demonstrated that changes in the lung microbiome due to microplastic pollution are associated with increased airway inflammation, increased mucus production and fibrosis in asthmatic mice, suggesting that microplastics may compromise respiratory health by affecting the lung microbiome. The present review aimed to integrate available research on the impact of inhaled microplastics on the lung microbiome, resulting in changes in microbial composition and function, and to assess the potential implications of microplastics for inflammation, immune dysfunction and respiratory disease. Previous research has validated the detection of microplastics in pulmonary tissues and bronchoalveolar lavage samples, further indicating that inhalation serves as a major means of exposure. Concurrently, academic investigations indicate that these foreign particles may interfere with the respiratory microbiome by engaging in physical interactions with microbial communities, eliciting inflammatory responses, and transporting chemical additives and environmental toxins.

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Contents

1. Introduction

Microplastics are types of plastic pollution that have piqued increased academic curiosity due to their widespread presence across different environmental landscapes and their possible adverse effects on human health. Microplastic particles have a diameter of <5 mm (1). These entities can be intentionally manufactured for specific applications in the cosmetics and textile industries, or they may be generated inadvertently through the degradation of larger plastic items (2). The detection of microplastics has been noted across a range of environmental locations, comprising water systems, soil landscapes and air conditioners. It has been reported that they accumulate in ecological frameworks, seeping into the food web and creating possible hazards to human health from consuming unsafe food and water sources (1,3). These tiny plastic fragments can carry hazardous substances, such as bisphenol A and phthalates, which can affect the endocrine system. Microplastic identification can be carried out through a physical approach based on characteristics, size and color. Furthermore, identification can be combined with chemical methods to distinguish them from natural materials. Spectroscopy is one method often used for comprehensive analysis (4).

Apart from microplastics, there are other small plastic particles known as nanoplastics. Nanoplastics are particles measuring between 1 nm and 1 μm (2). Nanoplastics have smaller dimensions, higher reactivity and mobility compared to microplastics. Nanoplastics more easily pass through cell membranes than microplastics, which can affect cellular processes (5). Nanoplastics exhibit unique interactions with light and natural colloidal substances, thus affecting their environmental durability and bioavailability (6). Some potential effects of nanoplastics on humans are the possibility of triggering inflammation, oxidative stress and various other health issues (3). In detecting and identifying nanoplastics, sophisticated methodologies are still required due to their very large size (6). The Microplastics and Nanoplastics dataset is one of the innovations that has been created for the automatic identification and classification of microplastics and nanoplastics through the application of deep learning algorithms (7). Although both forms constitute plastic pollution, the present review primarily focuses on microplastics. Although nanoplastics may exhibit similar mechanisms, current detection evidence is predominantly for microplastics in human tissues.

Several studies have detected the presence of microplastics in the human body, including the respiratory system. Small particles are easily absorbed through inhalation. The discovery of microplastics in lung tissue has also revealed that microfibers are more common in neoplastic lesions than in non-neoplastic tissue (8). This observation suggests a link between microplastic inhalation and the development of ground glass nodules (9). The detection of microplastics in ambient air and their possible role in triggering inflammatory responses and tissue injury in the lungs has been documented, although research into the mechanisms of their impact on health remains limited (10).

Previously considered to be free of microbial inhabitants, microbial communities have been found in respiratory organs. The lung microbiota, as a complex and adaptive ecosystem, plays a role in maintaining respiratory health. The respiratory microbiota originates from the upper respiratory tract. The lung microbiome consists of bacteria, fungi and viruses, with *Firmicutes*, *Bacteroidetes* and *Proteobacteria* being particularly abundant in the lungs (11). The microbial population in the lungs is lower than that found in other anatomical locations, such as the gastrointestinal tract, due to the unique lung environment, which includes a lipid-rich surfactant that inhibits microbial proliferation. The lung microbiome is generally transient and is influenced by microaspiration from the oropharynx, which then undergoes a clearance process facilitated by mechanisms such as coughing and ciliary movement (12).

The lung microbiome plays a role in the adjustment of innate and adaptive immune mechanisms. The microbiome functions to enhance immune tolerance and reduce excessive inflammatory responses (13). The microbiome contributes to maintaining lung homeostasis by preventing colonization by pathogenic microbes and supporting tissue repair processes (14). Metabolites produced by microbes have the capacity to influence human host metabolism and immune responses, but further research is needed (15). The lung microbiome also plays a role related to the gut and oropharyngeal microbiomes. One such function is the influence of changes in the composition of the lung microbiome on the immune response and the potential for health problems such as asthma and chronic obstructive pulmonary disease (COPD) (11).

The growing concern about the disruption of the lung microbiome by microplastics and their contribution to respiratory health risks is attracting increasing academic attention due to their potential consequences for public health. Evidence suggests that microplastics may contribute to the increased incidence of respiratory conditions, including asthma, by affecting the lung microbiome and triggering inflammation (16). Exposure to fibrous microplastics has been shown to significantly alter the bacterial composition of the lungs (16). Research on ovalbumin-induced asthma in mice has demonstrated that exposure to microplastics causes significant changes in the relative prevalence of various bacterial genera, characterized by a decrease in the amount of beneficial bacteria, such as *Escherichia-Shigella* and unidentified strains, alongside an increase in potentially harmful bacteria, including *Prevotella* (16). It has been shown that changes in the lung microbiome coincide with increased airway inflammation, mucus buildup and fibrosis in asthmatic mice, suggesting that microplastics may compromise respiratory health by affecting the lung microbiome (16).

While the detrimental implications of microplastics on the lung microbiome and respiratory health are increasingly being elucidated, it remains critical to consider the broader scope of environmental pollution and its complex impacts on health. Data suggest that air pollution, particularly fine particulate matter, can alter the lung microbiota and increase the rates of respiratory infections, highlighting the need for comprehensive strategies to mitigate environmental health hazards (17). The present review aimed to integrate research on the effects of inhaled microplastics on the lung microbiome, resulting in changes in microbial composition and function, and to assess the potential implications of these modifications for inflammation, immune dysfunction and respiratory disease. Furthermore, the present review aimed to describe the broader health consequences of microplastic exposure and outline key priorities for future research.

2. Sources and characteristics of airborne microplastics

Microplastics have been detected in both indoor and outdoor environments, originating from a variety of sources and exhibiting varying characteristics. The main contributors to indoor airborne microplastics are synthetic fabric residues, degradation of plastic items and the use of plastic polyphenylene ether. Furthermore, activities such as drying clothes and the presence of household furniture substantially increase indoor microplastic concentrations (18). Outdoor sources of microplastics are diverse, including urban material particles, emissions associated with vehicle traffic, and industrial operations. Furthermore, wastewater treatment facilities and the use of biosolids in agricultural methods further accelerate the release of microplastics into the atmosphere (18). In coastal areas, maritime transport can increase airborne microplastic concentrations, although land-based sources of pollutants also have a more pronounced impact (19). Airborne microplastics possess the capacity for long-distance transport, thereby reaching isolated locales and exacerbating global pollution levels.

Airborne microplastics are primarily characterized by their fibrous morphology, with fibers constituting the predominant manifestation observed in both indoor and outdoor settings (20). The dimensions of these particulates are heterogeneous; however, they are frequently aggregated within diminutive size categories, typically measuring <1 mm (19). Common polymers found in airborne microplastics include

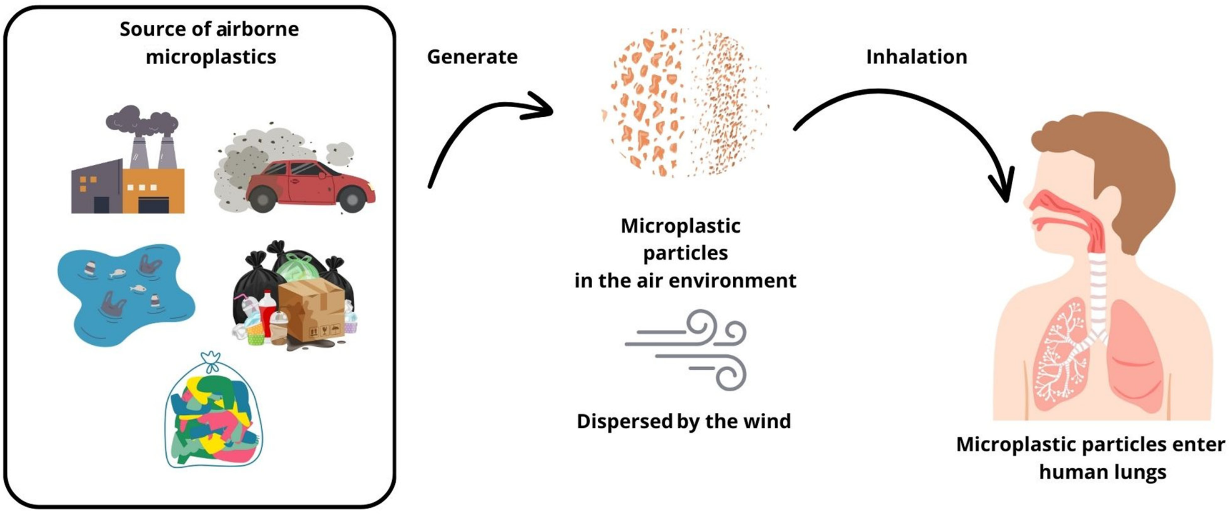

Figure 1. Schematic diagram illustrating the primary sources and pathways of airborne microplastics entering the human lungs.

polyethylene terephthalate, polyethylene, polypropylene and polystyrene (20). The chemical composition is subject to variations contingent upon the source, with disparate environments displaying unique polymeric profiles (19). Airborne microplastics are manifested in a spectrum of colors, with transparent and white hues being particularly prevalent across numerous studies (18). Morphological forms include spherules, films, fragments and granules, with fibers representing the most dominant morphological category (21).

Airborne microplastics are increasingly acknowledged as a critical environmental and public health issue, with numerous sources and pathways contributing to their infiltration into the human pulmonary system (Fig. 1). Urbanization and industrial activities serve as primary determinants of airborne microplastics. Particulates associated with vehicular traffic, synthetic textiles and emissions from industrial operations are significant contributors within urban locales (21). Indoor air is frequently characterized by elevated concentrations of microplastics relative to outdoor air. Textiles, including garments and domestic fabrics, represent principal sources of indoor microplastics, with fibers constituting the predominant form (22). Roadside dust and household refuse further augment the prevalence of microplastics within the atmosphere. These particulates can be reintroduced into the air via aeolian processes (23). Agricultural methodologies, such as the application of plastic mulch, in conjunction with maritime activities, also facilitate the release of microplastics into the atmosphere, thereby exacerbating outdoor pollution levels (24). The leading method through which microplastics reach human lungs is by means of inhalation. Tiny plastic particles may be lifted into the air and are subsequently inhaled, reaching the lungs where they could possibly move into the alveolar space and enter the bloodstream (22). The distribution and transport dynamics of microplastics within the atmosphere are influenced by environmental parameters, including wind velocity, directional flow and precipitation, which can promote their long-range transport and eventual deposition (24).

Microplastics have been identified within human pulmonary tissues, with one investigation reporting the presence of 108 microplastic particles in samples derived from non-smoking individuals. The major types observed were polypropylene, polyethylene terephthalate, and polystyrene, presenting a median level of 2.19 particles/g (25). In a previous study, the presence of microplastics was confirmed in bronchoalveolar lavage fluid (BAL). There were 13 unique forms of microplastics in samples from those who had not smoked. The predominant substance identified was polyethylene, succeeded by polyethylene terephthalate and polypropylene (26). This body of evidence implies that microplastics can penetrate deeply into the respiratory apparatus, potentially resulting in detrimental health consequences. A comprehensive overview of the evidence pertaining to human exposure to microplastics via inhalation and their identification in respiratory tissues is presented in Table I (25-33).

There are several methods that can be used to identify microplastics in human respiratory tissues. FTIR/Raman spectroscopy, the gold standard for microplastic identification, has limited efficacy due to diffraction-limited resolution (~1-5 μm), systematically underdetecting nanoplastics (<1 μm) due to poor signal-to-noise ratios. Complementary SEM/TEM provides nanoscale visualization, but requires EDX coupling for chemical confirmation, as tissue debris frequently generates false positives (34).

BAL sampling introduces critical contamination risks from airborne microplastics shed by laboratory plastics, synthetic clothing and polyethylene bronchoscopes. Jenner *et al* (28) detected 1.42±1.50 microplastics/g in 11/13 uncontaminated lung tissues, underscoring procedural blanks as mandatory controls. Best practices mitigating >90% contamination include: ISO Class 5+ cleanroom processing; silicone (not polyethylene) bronchoscopy tubing; ≥3 procedural blanks per batch; 100% cotton lab attire excluding synthetic fabrics; and HEPA H14-filtered air during dissection. These protocols described by Jenner *et al* (28) enable the reliable quantification of lung burdens reported separately in the studies presented in Table I (25-33), such as 2.19 particles/g tissue and 0.14-12.8 particles/100 ml BAL.

While the identification of microplastics within human respiratory tissues and environmental air samples provides substantial evidence of inhalation exposure, the comprehensive

Table I. Reports of detection microplastics in human lung tissues.

Microplastics and nanoplastic materials	Reported samples	(Refs.)
Plant and plastic fibers	Detected in 13 histopathology slides of lung tissue from 17 slides that were examined	(27)
Microplastics sized between 20-100 μm consisted of PP (34.26%), PET (21.30%), and PS (8.33%)	Lung tissue samples from 12 non-smoking patients	(25)
12 polymer types such as PP (23%), PET (18%) and resin (15%)	Detected in 11 of the 13 lung tissue samples with an average of 1.42±1.50 microplastics/g of tissue	(28)
PP (35.1%), PET (24.3%); cotton (16.2%); polyvinyl chloride and cellulose acetate (5.4%); and polyamide, polyethylene co-polypropylene, polystyrene, polystyrene-co-polyvinyl chloride and polyurethane (2.7%)	Observed in 13 of the 20 autopsied decedents	(29)
Various microplastics/nanoplastics in the interval of 0.14-12.8 particles per 100 ml of BALF	BALF samples of 10 patients undergoing diagnostic bronchoscopy	(30)
PE (86.1%), PET (7.5%) and PP (1.9%)	A total of 18 never-smokers aged 32-74 years who underwent fiberoptic bronchoscopy with BALF	(26)
Microplastics (25.86 particles/g), PE (11.34 particles/g), and silicone (1.15 particles/g)	Smokers	(31)
Microfibers, polyester fiber, acrylic fiber, rayon fiber, wool fiber, cotton fiber and cellulose fiber.	BALF of 44 adult European citizens.	(32)
Microplastics composed of PP (41.9%), PE (19.4%) and PS (13.6%)	BALF of children with pulmonary diseases	(33)

PET, polyethylene terephthalate; PE, polyethylene; PP, polypropylene; PS, polystyrene; BALF, bronchoalveolar lavage fluid.

ramifications of such exposure on health remain ambiguous. Contemporary investigations underscore the necessity for the establishment of standardized protocols to detect smaller-sized microplastics and the implementation of longitudinal studies to evaluate their prospective health hazards. Comprehending the routes through which microplastics impact human well-being, particularly regarding the respiratory system, is essential for formulating successful approaches to lessen exposure and prevent possible health effects.

3. The respiratory microbiome: Composition, functions and vulnerabilities

The respiratory microbiome functions as a vital and multifaceted ecosystem that is key to upholding respiratory health and supporting disease development. A recent bibliometric analysis confirmed microplastics as an emerging respiratory research priority (35). The lung microbiome predominantly originates from the upper respiratory tract yet possesses a distinct microbial flora. It is defined by a transient and mobile characteristic, attributable to mechanisms, such as coughing, pulmonary macrophages and alveolar surfactant (11). The collection of microorganisms in the respiratory system includes a wide variety of beneficial, cooperative and harmful entities that play a crucial role in supporting human wellness. Its composition is modulated by variables, such as age and dietary habits, which in turn influence its stability and functionality. The microbiome engages with the host immune system and external pathogens, thus underscoring its inherent vulnerabilities (36). The microbiome composition of the respiratory tract and related organs as documented in prior studies is presented in Table II (11,36-42).

Geographic location exerts a profound influence on the foundational microbial architecture of healthy pulmonary systems. In addition, variables such as chronological age, external factors and sequencing methodology also influence the results of microbial composition analysis (38). The lung microbiome exhibits a dynamic nature, initiated by the influx of microbes from the upper respiratory tract and environmental exposures (43). The lung microbiome plays a crucial role in immune balance and provides resistance to pathogen colonization (39). Microbiome stability is a phenomenon regulated by factors such as host characteristics, environmental context, and microbial interrelationships. Such stability is essential for maintaining health, as disruptions can trigger dysbiosis and associated diseases. Determinants affecting microbiome stability can be broadly classified into host-related factors, environmental variables, microbial interrelationships and external interventions (44-48).

Host-related factors include biological sex, chronological age, health conditions and the immune system, in addition to host genetic predisposition. Biological sex has emerged as an key determinant of microbiome composition, with females typically exhibiting greater stability. Chronological age also influences microbiome dynamics, as changes in bacterial populations are evident across life stages (45). Health conditions such as metabolic liver disease and diabetes mellitus are associated with microbiome instability, often resulting in reduced diversity and increased abundance of facultative pathogens, which can exacerbate dysbiosis (46). The immune

Table II. Microbiome composition of respiratory organs.

Respiratory organ	Microbiome composition	(Refs.)
Healthy lung	*Firmicutes, Proteobacteria* and *Bacteroidetes*.	(11)
Healthy lung	Bacteria: *Firmicutes, Proteobacteria, Bacteroidetes, Actinobacteria, Fusobacteria* Fungi: *Ascomycota, Basidiomycota* Virus: Bacteriophages	(36)
Healthy lung	*Bacteroidetes, Firmicutes, Proteobacteria* and *Fusobacteria*	(37)
Nasopharyngeal samples (healthy)	The anterior nare and nostril included *Staphylococcaceae, Propionbibacteriaceae* and *Corynebacteriaceae*. Also found some oral cavity microorganism, *Streptococcaceae, Veillonellaceae*, and *Prevotellaceae*	(38)
Oropharynx and oral wash (healthy)	*Streptococcaceae, Veillonellaceae, Fusobacteriaceae* and *Neisseriaceae*	(38)
Bronchoalveolar lavage (healthy)	*Sphingomonadaceae, Pseudomonaceae, Burkholderiaceae, Staphylococcaceae, Propionibacteriaceae* and *Staphylococcaceae*	(38)
Lungs of patients with COPD	*Haemophilus, Moraxella, Klebsiella* and *Pseudomonas*. *Streptococcus* and *Pseudomonas* is associated with different immune responses and exacerbation frequencies in COPD	(39,40)
Lungs of patients with cystic fibrosis	*Staphylococcus aureus* and *Prevotella shahii*	(41)
Lungs of smokers	*Ralstonia*	(42)

COPD, chronic obstructive pulmonary disease.

system of the host and genetic makeup can also shape microbiome stability by influencing microbial interactions and responses to environmental stimuli (47).

The diversity of microbial taxa present in the environment can drive temporal fluctuations in the host microbiome. Environments with high microbial diversity can foster a more dynamic but potentially less stable microbiome (49). Increased biodiversity and complex microbial networks can enhance microbiome resilience. However, excessive complexity can also lead to instability; thus, a balance between diversity and stability is necessary (50). Microbial competition has the potential to enhance stability by limiting positive feedback mechanisms and weakening ecological interactions. Conversely, cooperative networks, while exhibiting efficiency, may paradoxically possess reduced stability (47). Antibiotic interventions can induce substantial disturbances within the microbiome, with the recovery trajectory being contingent upon prior exposures and the presence of restorative dynamics within the microbial consortium (48). The strategic design of microbial communities, characterized by specific initial parameters and inoculation ratios, can optimize metabolic stability, particularly within regulated environments such as fermentation processes (51).

4. Mechanisms: How airborne microplastics affect the respiratory microbiome

Microplastics, characterized as diminutive particulates, possess the capability to be inhaled and subsequently accumulate within the respiratory tract, where they may engage with the pulmonary microbiota, potentially giving rise to deleterious health implications. The pathways through which microplastics influence the respiratory microbiome are illustrated in Fig. 2.

Microplastics exhibit considerable variability in dimensions, morphology and chemical makeup, which significantly affects their interactions with pulmonary tissues and associated microbiota. Smaller particulates, particularly those exhibiting positive charges, possess an elevated propensity for translocation and engagement with systemic circulation, thereby impacting the equilibrium of the respiratory microbiome (52). The surfaces of microplastics provide an ecological niche for a variety of microbial communities, encompassing bacteria and fungi, which collectively form biofilms referred to as the 'plastisphere'. This microbial niche is enriched with bacteria, fungi and protists via extracellular polymeric substances (EPS) that enhance adhesion and horizontal gene transfer. For example, Laboratory and field studies confirm rapid colonization on polyethylene, polypropylene and polystyrene in marine/freshwater systems, with biofilms altering microplastic buoyancy, sorption and pathogen harboring within days to weeks (53). Microplastics of smaller dimensions stimulate the development of microbes, where resilient forms such as polyvinyl chloride and polystyrene display greater biomass and diversity within biofilms (54). Microplastics, mainly those measuring <5 μm, can cross epithelial barriers and gather in lung tissues, possibly leading to irritation and a disruption of mucosal integrity (55).

Pulmonary plastisphere formation, however, lacks direct human evidence and requires validation beyond environmental analogs. In murine models of ovalbumin-induced asthma, fibrous microplastics (e.g., polyester) exposure significantly enriched *Prevotella* spp. in BALF, which is associated with airway inflammation, mucus hypersecretion

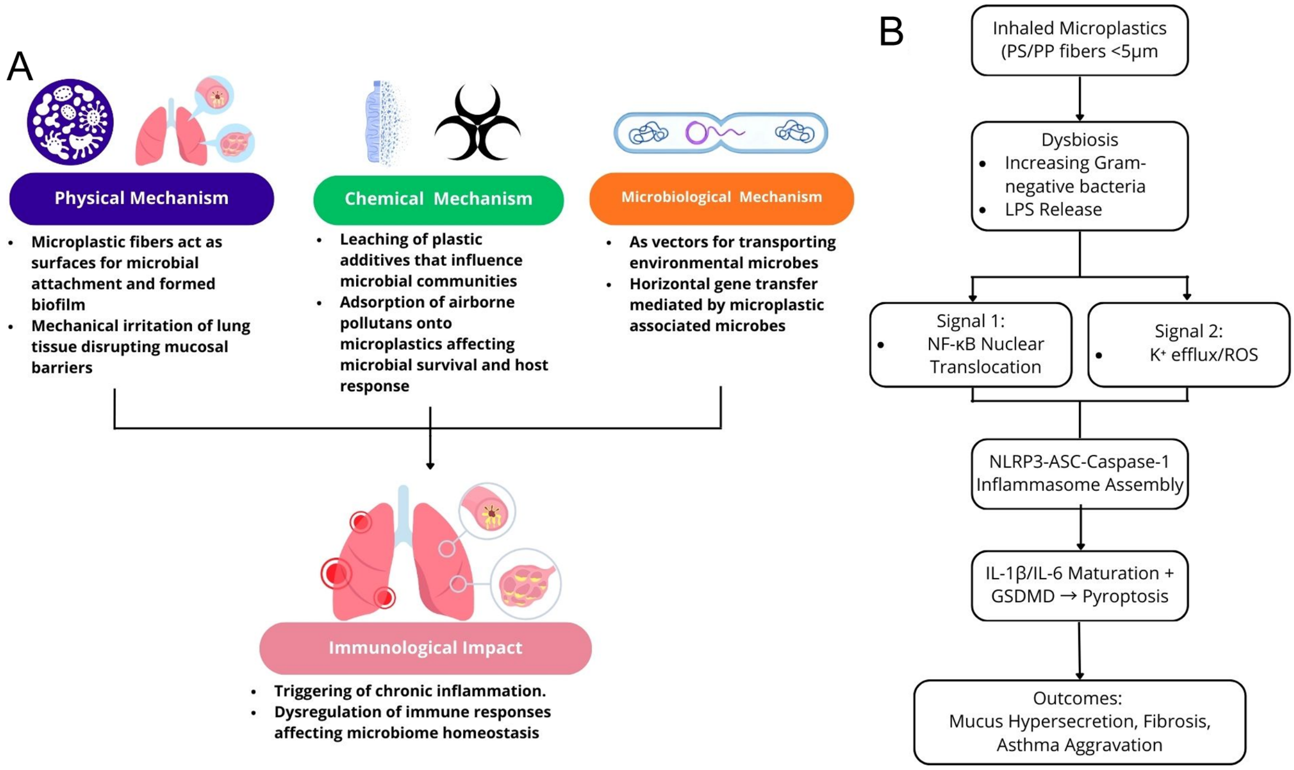

Figure 2. (A) Illustration of the mechanisms by which airborne microplastics affect the respiratory microbiome. (B) Schematic diagram of microplastic-induced pulmonary inflammatory cascade. Inhaled microplastics adhere to alveolar epithelium, promoting Gram-negative dysbiosis and the release of LPS. LPS activates TLR4 → NF-κB priming → NLRP3 inflammasome assembly → IL-1β/IL-6/pyroptosis, driving microbiome shifts (↑*Prevotella*) and asthma exacerbation. LPS, lipopolysaccharides; ROS, reactive oxygen species; PS, polystyrene; PP, polypropylene; GSDMD, gasdermin D.

and fibrosis; *Prevotella* was found to increase 2-5-fold vs. the controls, alongside reduced levels of *Lactobacillus* (16). This suggests fiber surfaces may selectively promote anaerobic Gram-negatives in asthmatic lungs, although true biofilm (EPS-matrix) confirmation requires scanning electron microscopy/transmission electron microscopy analysis.

The process of microplastic formation can instigate the leaching of plastic additives. Phthalates and bisphenol A (BPA) are expelled from plastics via several environmental methods, like photodegradation and mechanical fragmentation. This leaching phenomenon is intensified by the aging of plastics, which augments the release of dissolved organic carbon (DOC) into the ecological milieu, with aged plastics emitting substantially greater quantities of DOC compared to their virgin counterparts (56). The movement of plastic derivatives such as phthalates and BPA extensively modifies microbial environments. The leaching of these additives can precipitate a decline in microbial diversity and induce a shift in community composition. For example, exposure to leachates from plastics has been demonstrated to adversely affect marine picophytoplankton and modify the taxonomic and functional diversity of marine microbial assemblages (57).

As explained in the previous section, microplastics potentially release phthalates and BPA. BPA exposure has been shown to affect fetal lung development by altering the expression of genes and proteins involved in steroid synthesis and metabolism (58). It increases the expression of GPR30 and ERβ, and activates the NF-κB signaling pathway, which leads to the decreased release of immune modulators such as IL-6 and ET-1, and increases oxidative stress and DNA damage in lung fibroblasts. Additionally, BPA suppresses glucocorticoid receptor (GR) activity through ERβ/NF-κB signaling, leading to the reduced expression of ENaCγ, a GR target gene crucial for lung function (58). *In vitro* research suggests that BPA affects lung fibroblasts by altering gene expression and immune signaling pathways, which could affect lung development and increase susceptibility to respiratory diseases (58). BPA exposure has been linked to changes in gut microbial composition, increasing certain bacterial species that may influence systemic immune responses and respiratory health via the gut-lung axis (59).

Phthalate exposure is associated with lung microbiota dysbiosis, which can mediate adverse respiratory effects. This dysbiosis is linked to conditions, such as asthma and COPD. Phthalates, such as di-(2-ethylhexyl) phthalate (DEHP), have been shown to alter the gut microbiome. DEHP exposure increases the diversity of gut microbiota and alters the abundance of specific bacterial families, which may influence immune responses and respiratory health (59). Phthalates have been linked to an increased risk of developing asthma and respiratory symptoms. The potential mechanism involves the activation of peroxisome proliferator-activated receptors, which are implicated in inflammatory responses in the lungs (60). Additionally, phthalate exposure is associated with changes in the nasal microbiome, such as an increased abundance of *Moraxella*, which is linked to the development of asthma (61).

The extensive surface area and natural chemical features of microplastics enhance their ability to capture pollutants.

Various living organisms can ingest these pollutants that are rapidly taken up by microplastics, leading to their entry into the food chain. Microplastics containing pollutants can trigger an imbalance in microbial health, mainly within the respiratory and gastrointestinal systems, by altering the stability of microbial communities. This dysbiosis has been shown to be associated with the enhanced generation of reactive oxygen species and systemic inflammation, which may jeopardize microbial viability and host health (62).

Microplastics establish a uniform, water-resistant base that aids in the attachment and increase of microorganisms, encompassing bacteria, fungi and viruses. The microbial composition of these biofilms is modulated by the type of plastic utilized, prevailing environmental conditions and the 'weathering age' of microplastics (63). Microplastics found in aquatic environments can promote the growth and transmission of diseases, thus raising the chances of human exposure, notably in scenarios such as wastewater management facilities and aquaculture practices (64). The introduction of microplastics into the food system raises alarming issues regarding food safety, since these particles can hold foodborne bacteria and toxic agents. In addition, the existence of microplastics enhances the lateral transfer of genes among bacterial communities, possibly aiding in the spread of genes related to antibiotic resistance. The role of the plastisphere in disseminating antibiotic-resistant bacteria represents a novel challenge for mitigating antibiotic resistance within the environmental context (65).

5. Health implications of respiratory microbiome dysbiosis induced by microplastics

The impact on health linked to respiratory microbiome disturbances, resulting from microplastic contact, is becoming more widely recognized as a critical issue, given how widespread microplastics are in the ecosystem and their ability to harm human health. Microplastics possess the capability to be inhaled and subsequently deposited within the respiratory tract, penetrating deeply into the pulmonary system, including the alveolar regions where gas exchange transpires. Such deposition may contribute to respiratory microbiome dysbiosis by triggering oxidative stress and inflammatory reactions (66,67).

The presence of microplastics in the respiratory system may affect the composition of the lung microbiota, leading to an increased prevalence of Gram-negative bacterial species. This disruption can trigger immunological responses, such as the release of lipopolysaccharides (LPS), which trigger Toll-like receptor 4 (TLR4) signaling pathways, leading to increased inflammation and potential lung damage (67). LPS binds TLR4/MD-2 complexes on alveolar macrophages and epithelial cells, inducing receptor dimerization and the recruitment of adaptor proteins such as MyD88. This activates downstream NF-κB translocation to the nucleus, promoting the transcription of pro-inflammatory genes, while simultaneously triggering NLRP3 inflammasome assembly via K^+ efflux and the generation of reactive oxygen species. NLRP3 activation leads to caspase-1 autocleavage, which processes pro-IL-1β and pro-IL-18 into mature cytokines, alongside gasdermin D cleavage driving pyroptotic cell death and IL-6 release (68).

In polystyrene microplastic inhalation models, the upregulation of TLR4 is associated with lung ferroptosis and fibrosis, with burdens (e.g., 2.19 particles/g tissue) matching inflammatory thresholds. This cascade exacerbates mucus hypersecretion and airway remodeling in mice with ovalbumin-induced asthma. Furthermore, the capacity of microplastics to accumulate toxic chemicals and heavy metals may increase their toxicological effects on the respiratory microbiome. These types of interactions have the potential to perturb microbial ecosystems and exacerbate dysbiosis (55).

Current evidence indicates inflammatory responses to inhaled microplastics with dose in experimental animals/ cell culture. A number of studies have demonstrated dose-dependent increases in oxidative stress, pro-inflammatory cytokines (e.g., IL-6 and TNF-α), epithelial barrier disruption and an aggravated pathology with greater exposure to microplastics (66,67). Examples include controlled inhalation or instillation studies where higher doses or concentrations produced greater inflammatory readouts and functional impairment (67). Human studies have quantified internal microplastics burdens, but were only observational and did not provide controlled dose-response data (25-33). Mechanistically, immune/inflammatory systems often display graded responses rather than a single hard threshold; low exposures may prime immune signaling, whereas greater exposures provoke overt inflammation (67). Apparently, a study quantitative human threshold has not yet been established.

Chronic respiratory conditions such as asthma and COPD may result from the effects of microplastics on the gut flora. Microplastic-induced inflammation and oxidative stress are key contributors to the development and course of these disorders (69). Systemic health depends on the respiratory microbiome, and a dysbiosis there can have far-reaching effects. According to the gut-lung association, hypothetical changes in the respiratory microbiome may have effects on gut health and vice versa, which may lead to chronic health issues and widespread inflammation (70). A microplastic-induced immune response can lead to chronic inflammation, a known risk factor for a number of diseases, including metabolic and cardiovascular conditions (69).

6. Research gaps and future directions

The possible health risks linked to airborne microplastics are clarified by current research, particularly as regards their effects on lung structure, function and the respiratory microbiome (16,25-26,67). However, further research is urgently required to fully elucidate these effects and develop effective mitigation measures. It is clear that there are no established procedures for assessing the exact harmful effects of microplastics on pulmonary tissues. To facilitate comparisons between studies and fully determine the extent of health effects attributed to microplastics, this standardization is essential (52). There is a gap in the current knowledge of the long-term health effects and possible chronic conditions that may arise from prolonged exposure to airborne microplastics, as the majority of studies focus on acute exposure (16,33,66-67). The interactions of the human respiratory microbiome with airborne microplastics are not yet fully understood. Further investigations are warranted to assess the mechanisms through which microplastics affect microbial diversity and the risk of dysbiosis, which can lead to respiratory illnesses. The health hazards that airborne microplastics pose to human populations have not been extensively studied. *In vitro* and animal studies provide the majority of the evidence, highlighting the need for human epidemiological research to assess effects in the real world (71).

The development of novel techniques is essential for measuring exposure to microplastics. This includes creating effective, low-cost instruments for airborne particle sampling and analysis. To establish a clear link between exposure to airborne microplastics and the consequences for respiratory health, comprehensive epidemiological studies are necessary. Quantifying exposure levels and relating them to related health effects should be the goals of such studies (72). Future studies are required to focus on elucidating the ways in which airborne microplastics impact respiratory health, with a particular emphasis on pathways associated with oxidative stress, inflammatory reactions and immune regulation. Targeted therapeutic interventions may be made easier with a thorough understanding of these mechanisms (67).

The most up-to-date data are derived from *in vitro* or short-term animal experiments, leaving marked uncertainty as regards the long-term and dose-specific health consequences following the inhalation of microplastics. To address this knowledge gap, prospective cohort studies of occupational exposure workers (e.g., textile producers and waste handlers) and general population cohorts are urgently required. Such studies would integrate quantitative exposure quantifications of inhaled microplastics and nanoplastics with 16S rRNA sequencing or shotgun metagenomics of respiratory microbiota together with clinical and immunological characterization. Such an integrative approach would enable correlation analyses between microplastics burdens, microbial dysbiosis and disease symptoms, such as asthma, COPD, or subclinical inflammation. Furthermore, follow-up studies need to firmly control for confounding environmental and lifestyle variables, particularly fine particulate matter exposure, cigarette smoking, occupational pollutant exposures and the level of urbanization, to assess the independent impact of microplastics on alterations in respiratory microbiome and lung disease.

Examining the health effects of changes in the respiratory microbiome brought on by exposure to airborne microplastics may lead to the development of novel treatment strategies. Developing methods to increase beneficial microbial populations and investigating the possible protective roles of a diverse microbiome are two examples of this (73).

7. Conclusion

The evidence provided in the present review demonstrates that airborne microplastics are omnipresent environmental contaminants capable of entering the human respiratory system through inhalation. There are numerous studies that have confirmed the presence of microplastics in pulmonary tissues and BAL fluid, establishing inhalation as the primary route of exposure (25-33). Apart from their physical presence, microplastics have been shown to invade and transform the respiratory microbiome, an essential regulation of pulmonary immune homeostasis and resistance to disease. By physical contact with microbial communities, induction of inflammatory reactions and the carriage of chemical additives or environmental pollutants, such particles have the potential to contribute to microbial dysbiosis, oxidative stress, immune dysregulation and increased susceptibility to chronic respiratory illness, such as asthma and COPD. Their potential use as vehicles for pathogenic microorganisms and antibiotic resistance genes merely serves to enhance their broader public health risk.

In contrast to previous reviews that have had centrally a focus on exposure quantification, the present synthesis provides a mechanistic, microbiome-oriented perspective, timing microplastic exposure with the specific biological pathways underlying inflammation and microbial dysbiosis (8,29,32,35). The resolution of this complex issue requires an array of interdisciplinary synergies that extend from environmental science, molecular microbiology, through pulmonary medicine, to toxicology. Key future areas of research include standardization of exposure assessment methods, long-term cohort studies correlating inhaled microplastic burdens with microbiome alterations and clinical outcomes and mechanistic explorations of host-microbe-plastic interactions at the molecular level.

Finally, effective mitigation strategies are necessary to reduce exposure to humans and its corresponding health effect. These include practical interventions such as indoor air filtration, increased ventilation systems, and policy measures to reduce synthetic fiber pollution from clothing and manufacturing units. Together, these scientific and policy efforts can further elucidate the respiratory effects of microplastics while advocating evidence-based solutions to safeguard public health.

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Authors' contributions

JTW and RHH contributed equally to the conception, design and writing of the review. JTW conducted the literature search, organized the references and prepared the initial draft of the manuscript. RHH refined the thematic structure, contributed to the interpretation and synthesis of the literature, and revised the manuscript critically for intellectual content. Both authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work. Data authentication is not applicable.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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