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Pathophysiological effects of inhaled airborne microplastics on the respiratory epithelial barrier: A review

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ABSTRACT

Airborne microplastics (MPs) are increasingly recognised as a prominent environmental and public health concern, with evidence of human inhalation. However, their behaviour and fate within the respiratory system remain poorly understood. This review synthesises current knowledge on the mechanisms of MP inhalation and deposition in the respiratory tract, with particular emphasis on their interactions with the respiratory epithelial barrier. Physiochemical properties influencing MP deposition such as size, shape, density, and surface charge are discussed, alongside host-related factors including airway geometry, breathing patterns, and the influence of environmental humidity. The contributions of mucociliary clearance and macrophages as frontline defence against these pollutants are also reviewed. Critical evaluation of *in vitro* and *in vivo* experiments indicates that MPs cause epithelial barrier dysfunction, highlighting instances of endoplasmic reticulum stress, oxidative stress, mitochondrial dysfunction, inflammation, and disturbances to tissue repair and development that could potentially aggravate respiratory diseases. Despite growing evidence of adverse effects, significant deficiencies remain in the existing scope of research, particularly on the chronic health consequences of airborne MP exposure. This review necessitates the need for long-term inhalation studies, more diverse representative particle models, and mechanistic investigations to clarify the long-term implications of airborne MP exposure on human health.

Keywords: *Airborne microplastics; inhalation exposure; microplastic toxicity; respiratory epithelial barrier dysfunction and particle deposition*

INTRODUCTION

The invention of plastics in the last few decades has revolutionised multiple sectors by providing highly producible, cost-effective, and versatile materials, with applications spanning from automotive, textile, and packaging industries. Nonetheless, the extensive and uncontrolled use of plastic products has led to unprecedented waste accumulation (Akber Abbasi et al., 2020; Bengalli et al., 2022). An estimated 8-1 million tonnes of plastics are released in the ocean every year, harbouring over 75-99 tonnes of plastic burden in total (Donelly, 2025). Moreover, two-thirds of all mass-produced plastics have been estimated to enter the environment at various stages of their lifecycle, encompassing plastics in diverse forms from finished products to degraded fragments (Fadare & Okoffo, 2020).

Although valued for their durability, plastics are highly resistant to biodegradation, which enables them to persist in the environment for extended periods. Eventually, they will degrade into smaller particles known as microplastics (MPs). MPs are defined as plastic particles less than 5 mm in size, which are generally classified into two categories. Primary MPs are intentionally manufactured at microscopic scales for consumer and industrial products, including

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cosmetics and personal care items. Conversely, secondary MPs are generated through the degradation of larger plastic debris, driven by environmental processes such as photodegradation, mechanical abrasion, and chemical weathering (Ahmad
et al., 2023; Danopoulos et al., 2021; Fava, 2022; Koner et al., 2024). Morphologically, MPs are present in a variety of forms such as fibres, fragments, films, foams, and spheres. They are produced from a wide range of polymer types, most notably polyethylene (PE), polypropylene (PP), polystyrene (PS), and polyethylene terephthalate (PET). These materials frequently incorporate chemical additives such as plasticisers, flame retardants, and stabilisers (Bengalli et al., 2022; Mason et al., 2018; Rosal, 2021). MPs' surface properties, including charge, roughness, and ageing state, further influence their environmental fate by enhancing the adsorption of metals, persistent organic pollutants, and microbial communities. Given their persistence and mobility, MPs are now recognised as ubiquitous contaminants across terrestrial, aquatic, and atmospheric environments (Ahmed et al., 2021; Y. Chen et al., 2021; Q. Zhang et al., 2020).

Among the different routes through which MPs can disperse, airborne MPs are a critical concern due to their persistence and widespread distribution. Primary studies found that fibrous MPs are dominant in the outdoor air environment, largely attributed to the shedding of textile products (Dris et al., 2017). Dris et al. (2016) were the first to report the presence of MPs in atmospheric fallout, evaluating dry and wet deposition of MPs on rooftops in urban and suburban areas of Paris, France. Fibrous MPs dominated the samples, with higher concentrations in urban areas, likely due to greater population density and human activities. Similarly, Liu et al. (2019) estimated approximately 120.72 kg of suspended atmospheric MPs annually in Shanghai, with fibrous MPs comprising 67% of the total load, followed by 30% fragments and 3% beads. Studies across the globe corroborate the observed trend, consistently identifying fibrous MPs as the most prevalent type in urban atmospheric fallout (Ahmad et al., 2023; K. Liu et al., 2019; X. Wang et al., 2020; Zhou et al., 2017). Common polymers identified across these studies include PET, PE, and PP.

Airborne MPs have been found not only in densely populated areas but also in remote locations, including the mountain catchments of New Zealand and France, where human activity is minimal (Allen et al., 2019; Aves et al., 2024). In these studies, fibrous MPs were often dominant, with Aves et al. (2024) identifying PET as the most prevalent polymer, while Allen et al. (2019) reported a higher concentration of PS fragments. This variability likely results from the complex transport mechanisms of MPs, influenced by differing wind trajectories in various regions. The capacity of MPs to be transported long distances via wind was also demonstrated, having been found in the atmospheric fallout of Antarctica and across the South China Sea and Indian Ocean (González-Pleiter et al., 2021; Marina-Montes et al., 2022; X. Wang et al., 2020). These studies collectively demonstrate the widespread distribution and diverse composition of airborne MPs in both urban and remote environments.

Indoor environments pose significant levels of airborne MPs due to the various sources of plastic products. Dris et al. (2017) reported higher concentrations of fibrous MPs found in office settings and households compared to the outdoor environment. The study also revealed that the concentration of fibrous MPs is higher in offices than in households, suggesting that a larger scale of human activities in larger spaces contributes to more MPs generation. The abundance of PP in this study aligns with the observation that homes and offices contain various potential sources of this polymer, such as carpets, sofas, and chairs. Additionally, MPs were detected in hospital surgical theatres, which are typically considered a sterile environment (Field et al., 2022). Primary sources of airborne MPs include the wear and tear of synthetic fabrics, synthetic rubber tires, and urban and household dust. Other significant sources are construction materials, waste incineration, landfill sites, industrial emissions, and synthetic particles (Dris et al., 2016, 2017; Prata et al., 2019, 2020). These studies illustrate that MPs are prevalent in both outdoor and indoor settings, generated from a range of sources.

METHODOLOGY

Inhalation and presence of MPs in the respiratory system

The presence of airborne MPs found in indoor and outdoor environments has raised concerns about the potential health risks of inhaling these particles. Inhalation of MPs in an indoor environment was demonstrated by Vianello et al. (2019), revealing that a mannequin simulating human exposure in such environments inhaled up to 272 MPs within 24 hours. The most prevalent polymer types of MPs inhaled by the mannequin in this experiment were polyester (81%), PE (5%), and nylon (3%), with 87% of the MPs shaped as fragments and 13% as fibres. To date, inhalation of airborne MPs has been acknowledged as one of the major routes through which MPs enter the body (Blackburn & Green, 2021).

Notably, MPs inhalation rates differ across regions, with developing countries experiencing higher levels than developed nations. For instance, individuals in East and Southeast Asia might inhale up to 2.8 million MPs particles daily, while in Northwestern Europe, including Sweden and Norway, the rate is as low as 0.3 million particles per day (Zhao & You, 2024). Higher MPs levels in Asia are linked to substantial plastic waste from maritime activities and rapid industrialisation, with polyester and PET being the most common types (X. Wang et al., 2020; Zhou et al., 2017). Malaysia has one of the highest inhalation rates, with an estimated daily intake of 494,000 particles per person (Zhao and You, 2024). Additionally, Atis et al. (2005) reported a 3.6-fold increase in respiratory issues among workers exposed to PP flocking, highlighting an elevated risk of MPs inhalation due to occupational exposure. Although these findings

highlight the significant presence and risk of airborne MPs inhalation, the magnitude and implication of inhaled MPs in the body are still not completely understood.

MPs have been consistently detected throughout the human respiratory system, from the upper airways to deep lung tissues. Pauly et al. (1998) were the first to report the presence of MPs in human lungs, with plastic fibres up to 250 µm found in 87% of lung tissue specimens collected during tumour removal surgeries. It was shown that MPs concentrations in the lung tissues found increase with age, indicating long-term accumulation (Q. Chen et al., 2022; Pauly et al., 1998). Studies in Brazil and the United Kingdom corroborated this finding, reporting MPs, including fibres, fragments, and beads, found in both live and deceased human lung tissue samples (Amato-Lourenço et al., 2021; Jenner et al., 2022). These studies identified diverse types of polymers, including PP, PET, PS, and resin, with PP being the highest reported in both studies. Fibres and fragments were the most prevalent types of MPs found, with dimensions spanning from 11.23 µm to 2475 µm.

In addition, MPs were found in respiratory fluids collected from healthy and diseased individuals (S. Huang et al., 2022; Jiang et al., 2022; L. Qiu et al., 2023). MPs were identified in sputum and nasal lavage fluid located in the upper airways, with common polymers found including polycarbonate, polyamide, and PE (Jiang et al., 2022). Studies on the bronchoalveolar lavage fluid (BALF) collected from lung cancer patients have also detected MPs, with higher concentrations found in smokers compared to non-smokers (W. Lu et al., 2023; L. Qiu et al., 2023). Collectively, these findings provided concrete evidence of MPs' infiltration into the body, highlighting their widespread distribution in the respiratory tract from the upper airways to the deepest lung regions with varying types, shapes, and sizes.

It is apparent that MPs, once inhaled, can be deposited in varying locations in the respiratory pathway. However, the exact mechanisms or factors that affect its deposition have yet to be sufficiently explored. Once inside the body, MPs can translocate and accumulate in various tissues and organs, raising concerns about potential cytotoxicity, hypersensitivity, and immune responses (Banerjee & Shelver, 2021; Danopoulos et al., 2021; Stock et al., 2021). Understanding these factors is crucial to determine the implications of inhaled MPs on human health. Therefore, research on the deposition patterns of particles with similar characteristics to MPs may provide valuable comparisons.

RESULTS

Factors affecting MPs deposition mechanisms

Airway anatomy and breathing patterns affect MPs deposition

The respiratory tract comprises the nasal passages, pharynx, trachea, bronchi, bronchioles, and alveoli, functioning collectively to filter and deliver air to the lungs. Mucous membranes and cilia lining the pathway trap and remove inhaled particles, offering protection against contaminants (Atsmon, 1988; Berglund et al., 2020). The anatomical complexity from the upper airways to the alveoli creates varied deposition sites for particles, with individual breathing patterns further influencing airflow and particle distribution. Using computational fluid dynamics (CFD), Islam et al. (2023) demonstrated that MPs predominantly deposit in the nasal cavity, a hotspot driven by its asymmetrical structure and airflow. Larger, fibre-like MPs exhibit greater deposition efficiency than smaller forms, consistent with reports identifying fibres as the most frequent MPs in the respiratory tract (Jenner et al., 2022; Jiang et al., 2022).

Islam et al. (2023) further explored that lower airflow rates (7.5 L/min) prolong MPs' residence time in the airways compared to higher rates (30 L/min), suggesting greater retention in individuals with slower breathing rate, such as children or patients with chronic obstructive pulmonary disease (COPD) and sleep apnoea. Deposition is influenced by gravitational sedimentation and Brownian diffusion, the latter being more prominent at low flow rates but decreasing as airflow increases (Ou et al., 2020). Using CFD, Dang Khoa et al. (2022) showed that man-made fibres (3.66 µm) and asbestos fibres (1 µm) concentrated in the anterior nasal cavity, consistent with Islam et al. (2023). Moreover, MPs were more abundant in male lung tissues than in females, likely due to narrower female airways, highlighting the influence of anatomical differences across age, sex, and health status on MPs deposition.

Effects of MPs' physical dimensions on deposition

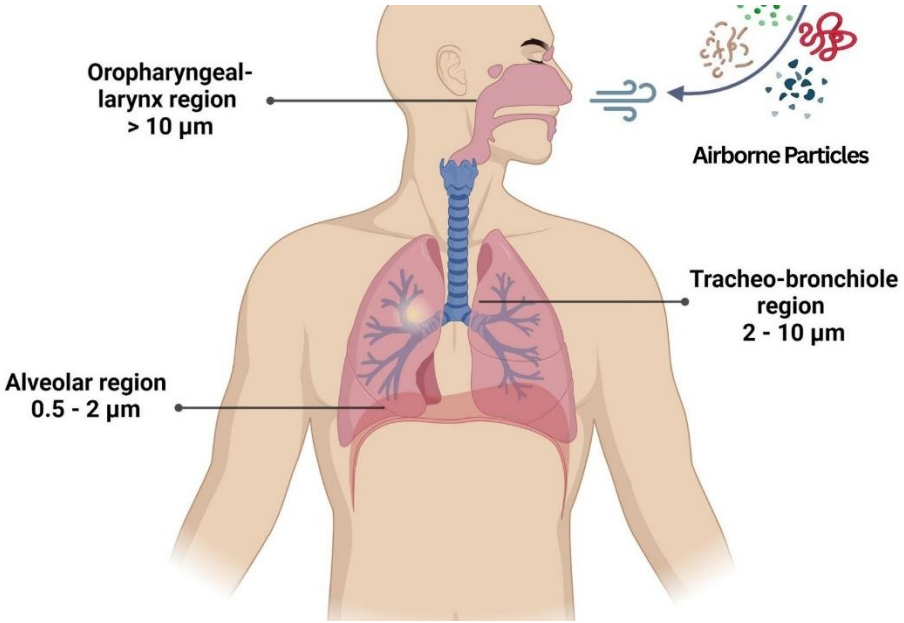
The deposition sites of inhaled particles are influenced by their physical characteristics, including size, shape, airflow dynamics, air humidity, and their affinity to adhere to pulmonary walls or receptors (Clarà et al., 2023; Ou et al., 2020). Particles larger in size (>10 µm) are mainly retained in the oropharynx and larynx via inertial impaction, while those between 2–10 µm tend to deposit in the tracheobronchial region through impaction and sedimentation. Fine particles (0.5–2 µm) can reach the small airways and alveoli via gravitational sedimentation, while ultrafine particles (<0.5 µm) are able to penetrate the alveoli, predominantly by diffusion (Hinds & Zhu, 2022; K. Lin et al., 2023). Particles propelled with specific velocities may exhibit differential deposition patterns, directing them towards specific areas within the respiratory tract. Smaller particles can reach the deeper lung regions in regard to their aerodynamic properties, posing risks of deeper tissue exposure (Clarà et al., 2023). Meanwhile, larger particles accumulate in the upper airways, creating localised deposition. These distinct patterns highlight that both fine and large particles pose health risks, depending on their site of retention in the respiratory tract. Figure 1 shows the proposed size range of particles deposited at different sites of the respiratory tract.

Despite the deposited particle size range established, several studies reported findings that challenge this current knowledge. Pauly et al. (1998) detected MP fibres in human lung tissues which exceed 250 µm in width and 50 µm in length, found in both the upper and lower lung regions. The sizes of MPs found were considered too large to reach the lungs, as noted in an earlier study by Donaldson et al. (1993). Similarly, Jenner et al. (2022) corroborated the finding by detecting fibres up to 2475 µm long and 88 µm wide across all lung regions. Both authors reported fibres as the dominant shape of MP found compared to fragments and beads. Conversely, Amato-Lourenço et al. (2021) reported fragments to be more abundant in upper lung tissues of human cadavers, with average dimensions of 16.80 µm × 5.56 µm, though lower regions were not assessed.

These reported sizes often exceed the conventional respirable fraction (Figure 1), highlighting the influence of particle shape in deposition. Large MP fragments may preferentially settle in the upper airways, whereas MP fibres with higher aspect ratios may penetrate deeper into the lower regions. Indeed, the shape of MP fibres resembles asbestos, with an elongated form that enhances its aerodynamic penetration, intensifying toxicity (Dang Khoa et al., 2022). Fibres may also become entangled in mucus, with their biopersistence further enabling evasion of clearance mechanisms, leading to prolonged retention and accumulation in lung tissue (Clarà et al., 2023; Saha & Saha, 2024; Sturm, 2017). In contrast, spherical particles can move more freely within the airways and are more readily cleared (Dang Khoa et al., 2022). Collectively, these findings suggest that larger MPs, once considered non-respirable, can indeed deposit within the lungs, highlighting the need to re-evaluate inhalable size ranges across different particle shapes (Jenner et al., 2022). While most studies emphasise particles within the respirable range (5–6 µm), recent evidence indicates the importance of also considering larger particles.

Figure 1

Reported particle size ranges capable of depositing in different regions of the respiratory tract (Hinds & Zhu, 2022; K. Lin et al., 2023).



Note: Illustration depicting reported particle size ranges associated with deposition across different regions of the respiratory system (Hinds & Zhu, 2022). Image created with BioRender.com.

Air humidity influences MPs' deposition

Environmental conditions such as air humidity also influence particle deposition in the airways through hygroscopicity, the capacity of particles to absorb or release water. Particles may expand at high humidity levels and conversely shrink at low humidity, altering the particle size and shape (J. Qiu et al., 2024; Yuan et al., 2023). Humidity also modulates particle-to-surface interactions in which increased moisture may enhance particle adhesion to bronchial walls, whereas excessive humidity may induce aggregation or excess mucus, hindering clearance (Rajaraman et al., 2020). These effects can be applied to MPs and their deposition patterns, especially those that are environmentally weathered and are heterogenous in nature (Thakur et al., 2020).

Mucociliary and macrophage phagocytosis clearance mechanism

Mucociliary clearance

Inhaled MPs interact with the respiratory epithelial barrier primarily through mucociliary clearance and phagocytosis by macrophages. The mucociliary clearance system acts as the first line of defence, featuring a coordinated network of ciliated cells and mucus-producing goblet cells (Celebi Sözener et al., 2020; Sparrow et al., 1995). The distinct feature of ciliated cells is cilia, which are hair-like structures that facilitate the movement of mucus containing contaminants out of the airways. This ensures efficient transportation of inhaled pollutants to the pharynx, where they are either expelled through exhalation or directed to the digestive tract via swallowing (Bustamante-Marin & Ostrowski, 2017). This mechanism prevents airborne pollutants and contaminants from reaching the deeper regions of the lungs. Apart from that, circulating macrophages complement the mucociliary system by patrolling the airways and phagocytosing foreign particles to be removed (Hirota et al., 2007; Saradna et al., 2018).

Clearance mechanisms can be compromised by certain factors. This includes chronic exposure to pollutants, harmful chemicals, and underlying respiratory conditions (Cecchi et al., 2022; J.-W. Lee et al., 2021; Thakur et al., 2020). It was hypothesised that MPs, once inhaled, may travel through the respiratory tract and bypass these clearance mechanisms (Amato-Lourenço et al., 2021). Due to their resistance to degradation, MPs may retain and accumulate over time, with the potential to translocate to other organs through systemic circulation or lymphatic pathways (Liang et al., 2021; Pauly et al., 1998; Saha & Saha, 2024). Suboptimal mucociliary clearance is a hallmark feature of respiratory disorders including asthma, acute respiratory distress syndrome (ARDS), and COPD, often due to compromised ciliary function (Bustamante-Marin & Ostrowski, 2017; Gasperi et al., 2018). Exposure to airborne MPs may aggravate these challenges, further jeopardising respiratory and systemic health (Shamsul Anuar et al., 2022; Huang et al., 2021).

Beyond their transport to the lower respiratory tract, the mucociliary system may also inadvertently transport MPs to the gastrointestinal system (Danopoulos et al., 2021; Fournier et al., 2020). Once inhaled, MPs may be transported upward by ciliary action towards the throat, where they are swallowed and introduced into the digestive system (Bustamante-Marin & Ostrowski, 2017). This may allow MPs to interact with both the respiratory and gastrointestinal epithelial barrier. It is important to note, however, that most MPs in the gastrointestinal system are ingested rather than inhaled, originating from sources such as contaminated food, leaching from food containers, and MPs present in drinking water (Banerjee & Shelver, 2021; Vasse & Melgert, 2024; Q. Zhang et al., 2020). These ingested particles further expose the gastrointestinal system to MPs, exacerbating their impact on both the lungs and gut.

Macrophage activity

Macrophages play a crucial role in removing MPs from the airways. Dysfunctional macrophage activity can impair their phagocytic efficiency, leading to unregulated inflammatory responses (Hirota et al., 2007; Kwon et al., 2022). This can result in maladaptive immune functions that exacerbate disease progression of respiratory disorders, largely characterised by chronic inflammation (J.-W. Lee et al., 2021; Y.-C. Liu et al., 2014; Saradna et al., 2018). The efficiency of macrophages to engulf materials was found to be highly dependent on particle size and shape. As demonstrated by Champion et al. (2008), spherical PS MPs (2-3 µm) were shown to achieve optimal phagocytic efficiency in both NR8383 rat alveolar macrophages and MH-S mouse alveolar macrophages. This is attributed to the shape and size of the PS MPs, which complement the structural features of macrophages, such as membrane ruffles, assisting in optimal uptake (Champion et al., 2008; Sadhu et al., 2022).

In contrast, fibres were found to be more challenging for macrophages to remove. Evidently, Makino et al. (2023) and Hirota et al. (2007) reported incomplete engulfment of fibrous structures by macrophages, resulting in a less efficient removal of the particle. Longer fibres (15-20 µm) were reported to not be effectively eliminated by macrophages, being more likely to trigger biological effects compared to shorter fibres (Donaldson et al., 1993; Wright & Kelly, 2017). It was hypothesised that the high aspect ratio of fibres plays a significant role in it being less efficiently phagocytosed (Donaldson et al., 1993). Nevertheless, other studies have shown that macrophages failed to engulf smaller particles, such as titanium dioxide (TiO_2) NPs (200-1000 nm) (Allegri et al., 2016). This implies that other factors, such as particle surface charge, may also play a significant role in influencing macrophage uptake (Champion et al., 2008). Thus, the varied shapes and sizes of MPs, among other factors, may interrupt the normal functioning of macrophages, causing a deficiency in the mucociliary clearance pathway.

DISCUSSION

Effects of MPs and NPs exposure on the respiratory epithelial barrier

Although MPs are the primary focus of this review, it is crucial to address the growing body of evidence suggesting that NPs may also penetrate the respiratory system, reaching alveolar regions and causing epithelial disruptions (Annangi et al., 2023; Cary et al., 2023; Liang et al., 2021). NPs are classified as plastic particles lesser than 1 µm, formed as primary NPs, or through the further breakdown of MPs (Shahzadi et al., 2023). Structurally, MPs and NPs share similar morphologies and polymer types, and both may carry chemical additives and adsorb pollutants. The main distinction

between MPs and NPs is their dimensions, with NPs having a higher surface-area-to-volume ratio. This allows them greater mobility and an increased ability to cross biological barriers and interact at the cellular and subcellular level in comparison to MPs (Cary et al., 2023; J. Huang, Dong, Miaoting, et al., 2022; Matthews et al., 2021). To provide a more relevant assessment of plastic particle toxicities across size ranges, this section will also consider the impact of NPs alongside MPs on the respiratory epithelial barrier.

Characteristics of MPs and NPs and the effects on respiratory epithelial cell viability

Particle characteristics strongly influence MP and NP toxicity, with smaller particles often inducing stronger cellular effects. For example, exposure to PS MPs and NPs (80 nm-1 μm) was shown to decrease A549 and BEAS-2B cell viability in a dose-dependent manner. However, larger MPs (1–10 μm) did not markedly reduce cell viability but caused impaired proliferation and induced morphological alterations (Goodman et al., 2021; X. Shi et al., 2022). Smaller MPs also consistently demonstrated higher cellular uptake efficiency. Beyond size, surface morphology strongly influences biological responses. Weathered MPs with heterogenous structures were shown to induce greater cell damage compared to pristine MPs. This is likely via mechanical disruption of cell membrane and cellular components, due to the rough and jagged surfaces of weathered MPs compared to the smooth structure of pristine MPs (Choi et al., 2021; Dong et al., 2020). Surface charge is another critical factor, with positively and negatively charged MPs causing decreased cell viability in BEAS-2B cells and A549 cells in a dose-dependent manner, compared to neutral ones (Jeon et al., 2023; X. Shi et al., 2022). The toxicity of charged MPs is associated with endosomal acidification, causing osmotic swelling and rupture of endoplasmic vesicles, leading to cell damage. Electrostatic interactions of charged MPs and enhanced lipid binding exaggerated these effects, further facilitating cell uptake (Halimu et al., 2022; J. Huang, Dong, Liang, et al., 2022). Collectively, particle size, morphology, and surface charge act synergistically to shape the toxicological profile of MPs and NPs, highlighting the need to consider these interrelated features when evaluating their biological impact.

Corona formation affects MPs' and NPs' toxic effects

Corona formation fundamentally alters the identity and behaviour of MPs and NPs, thereby influencing their interactions with cells and tissues. Eco-coronas (ECs) are formed from the adsorption of environmental pollutants such as salts, organic compounds, and heavy metals. Upon entering biological systems, MPs and NPs may rapidly adsorb biomolecules including proteins, lipids, and nucleic acids, forming bio-coronas (BCs), which may coexist with ECs (Ali et al., 2024; Cao et al., 2022). These coronas are largely driven by hydrophobic and electrostatic interactions. Particle size is a key determinant of corona properties. For example, smaller PS NPs (100 nm) develop thicker protein coronas (25 nm) compared with larger PS NPs (400–500 nm), due to their greater surface area and adsorption capacity (Y. Chen et al., 2022). The toxicological consequences of corona formation are influenced by the physicochemical properties of MPs and NPs, the composition of biomolecules, and the involvement of co-pollutants. Both ECs and BCs can modulate toxicity, either mitigating or exacerbating adverse effects (Kihara et al., 2021; Tan et al., 2020).

In the respiratory tract, inhaled MPs/NPs encounter lung surfactants, phospholipids, and mucins, which critically shape corona composition and subsequent cellular interactions. Ji et al. (2021) reported that PS–benzopyrene nanoparticles formed a mucin corona upon contact with the lung epithelial mucus layer. This enhanced cellular uptake but delayed intracellular trafficking, thereby reducing ROS production, mitochondrial dysfunction, and apoptosis. Likewise, Theodorou et al. (2016) observed that phospholipid coronas on zinc oxide nanowires markedly increased uptake by human alveolar epithelial type I-like cells, shifting cell death pathways. Other studies suggest that lipid- and mucin-rich coronas may contribute to respiratory and digestive tract injuries (Ji et al., 2021, Tan et al., 2020). Despite these advances, the mechanisms by which corona formation governs the biological responses to MPs/NPs remain poorly characterised, particularly in the context of inhalation exposure.

Mitochondrial dysfunction and oxidative stress: activation of redox-sensitive signalling pathways

Upon contact with epithelial cells, MPs and NPs may adhere to the lipid bilayer and gain entry through multiple pathways, including passive diffusion, transport proteins, and endocytosis. The adherence of MPs and NPs to the lipid bilayer induces stretching and structural perturbations of the plasma membrane, compromising its integrity and facilitating further entry (Fleury & Baulin, 2021). This disruption is closely associated with excessive production of reactive oxygen species (ROS), which trigger lipid peroxidation (LPO) through the oxidation of polyunsaturated fatty acids (PUFAs) and damage other lipid-containing structures, including cellular and organelle membranes (Nam, 2011). Once internalised, MPs are trafficked to lysosomes and subsequently transported via the biofilm system to mitochondria, where they alter mitochondrial membrane potential and disrupt intracellular homeostasis (Deng et al., 2022; Geys et al., 2006).

Mitochondria are essential eukaryotic organelles that govern cellular bioenergetics, biosynthesis, and signalling. They metabolise nutrients to produce ATP and maintain cellular homeostasis through redox regulation and calcium

storage. MP exposure has been shown to disrupt mitochondrial integrity, particularly through NADPH oxidase 4 (NOX4)-mediated pathways, leading to impaired ATP synthesis, membrane potential collapse, and electron leakage (S. E. Lee et al., 2022; Rudolph et al., 2021; Woo et al., 2023). These changes collectively drive excessive mitochondrial-ROS production, leading to redox imbalances and oxidative stress, which compromises respiratory epithelial cell metabolism. Lin et al. 2022 demonstrated that human lung cells exposed to MPs displayed marked mitochondrial damage, including elevated mitochondrial-ROS, altered membrane potential, and suppressed respiration, further supporting mitochondrial dysfunction as a pivotal mechanism underlying MP-induced oxidative stress and cell dysfunction.

Excessive ROS production activates multiple redox-sensitive signalling pathways, including mitogen-activated protein kinases (MAPKs), nuclear factor-kappa B (NF-κB), and nuclear factor erythroid 2–related factor 2 (Nrf2). These cascades are reflected in bronchial and alveolar epithelial cells, presented in both *in vivo* and *in vitro* studies (Brzicova et al., 2019; Y. Wu et al., 2023; T. Zhang et al., 2022). ROS-mediated phosphorylation of MAPKs, particularly p38, enhances NF-κB signalling and promotes transcription of pro-inflammatory cytokines including IL-6, IL-8, and TNF-α (Jeong et al., 2022; Woo et al., 2023; D. Xu et al., 2021). Additionally, Nrf2 activation induces the expression of cytoprotective enzymes such as heme oxygenase-1 (HO-1), catalase, superoxide dismutases (SOD1/2), and glutathione peroxidase 1 (GPx1), constituting a compensatory antioxidant defence (K C et al., 2023; Kadac-Czapska et al., 2024; Li et al., 2022). The balance between NF-κB–driven inflammation and Nrf2-mediated antioxidant protection ultimately determines cellular fate under MPs and NPs exposure. Dysregulation of this balance shifts the epithelial response toward oxidative injury, barrier dysfunction, and chronic inflammatory remodelling. ROS-mediated damage extends to lipids, proteins, and DNA, promoting lipid peroxidation, protein carbonylation, and genotoxicity. This is shown from the elevated levels of biomarkers including malondialdehyde (MDA) and 4-hydroxynonenal (HNE), indicating membrane damage, and elevated 8-hydroxy-2′-deoxyguanosine (8-OHdG), reflecting oxidative DNA damage (Khan & Jia, 2023; Z. Liu & You, 2023; Z. Wang et al., 2025). This disruption of cellular homeostasis amplifies pro-inflammatory signalling and perpetuates an oxidative-inflammatory cycle.

Endoplasmic reticulum stress and cell death mechanisms

MP-induced oxidative stress has also been implicated in triggering protein misfolding within the endoplasmic reticulum (ER), initiating ER stress and activation of the unfolded protein response (UPR). In respiratory epithelial cells, exposure to amino-modified PS NPs has been shown to activate the PERK–eIF2α–ATF4 signalling cascade while phosphorylation of IRE1 increases in a dose-dependent manner, further reinforcing UPR activation (Wiseman et al., 2022; Q. Wu et al., 2024). In response, autophagic processes are often employed to clear damaged proteins and organelles. However, ER stress under persistent MP exposure can overwhelm this protective mechanism, causing impaired or dysregulated autophagy (Jeon et al., 2023; Y.-Y. Lu et al., 2022; Y.-L. Wang et al., 2021). Annangi et al. (2023) demonstrated that PS NPs disrupt autophagic clearance in HNEpCs, evidenced by p62 accumulation and LC3-II conversion, exacerbating redox imbalance. Positively charged PS NPs were also shown to induce excessive autophagic flux via ROS generation, ER stress, and lysosomal overload, accompanied by nuclear deformities (Chiu et al., 2014; Jeon et al., 2023).

The dysregulation and ER stress further shift the mechanism towards other cell death pathways, most notably apoptosis. Sustained activation of the ATF4–CHOP axis promotes pro-apoptotic signalling, alongside molecular damage from lipid peroxidation, oxidised biomolecules, DNA mutations, and excess ROS. In A549 cells, PS NPs elicited classical apoptotic responses, including upregulation of BAX, CASP3, and BCL2 (Milillo et al., 2024). Similarly, mitochondria-mediated apoptosis has been demonstrated via cleavage of caspase-9 and caspase-3 and a shift in the BCL2/BAX ratio towards a pro-apoptotic profile (Y.-L. Wang et al., 2021). Ferroptosis, an iron-dependent cell death mechanism marked by lipid peroxidation and mitochondrial damage is now widely associated with MP and NP toxicity. This involves GPX4 inhibition, NCOA4 upregulation, and ferritin degradation, driving iron-dependent oxidative injury (Guo et al., 2023; Q. Wu et al., 2024). Recent findings link ferroptosis in A549 cells to activation of the cGAS/STING pathway, while ROS-mediated PI3K-Akt and HIF-1 signalling exhibit dual roles, balancing antioxidant defences with promotion of apoptosis or ferroptosis under chronic stress (Cao et al., 2023; Xuan et al., 2023).

Inflammatory responses following MPs and NP exposure

Alongside direct cytotoxic effects, exposure to MPs elicits inflammatory responses, largely mediated by dysregulated cytokine signalling. The most frequently reported cytokines are IL-6, IL-8, TNF-α, and IL-1β, though their expression patterns differ between studies (Table 1). IL-6 and TNF-α are the most consistently elevated, with both showing dose-dependent upregulation at the gene and protein levels across a range of particle sizes, polymer types, and experimental models (Bengalli et al., 2022; da Silva Brito et al., 2023; K C et al., 2023; Lim et al., 2019). TNF-α is elevated in A549 epithelial cells, in culture supernatants, and in BALF from *in vivo* exposures, underscoring its involvement in both local airway inflammation and systemic immune activation (Donkers et al., 2022; Woo et al., 2023; D. Xu et al., 2021).

Mechanistically, TNF-α activates NF-κB and MAPK signalling cascades, promoting the transcription of genes related to inflammation and apoptosis and contributing to epithelial barrier dysfunction and chronic tissue remodelling (Cao et al., 2023; Devarapu et al., 2017). It also intersects with IL-6/STAT3 signalling reinforcing ferroptosis, linking cytotoxic and immunomodulatory effects. IL-6 plays a central role in activating the JAK/STAT3 pathway, which regulates inflammation, cell survival, and tissue repair (Guo et al., 2023; Hong et al., 2022). Sustained STAT3 activation has been associated with mitochondrial dysfunction, oxidative stress, and suppression of apoptosis. Together with TGF-β, IL-6 promotes epithelial–mesenchymal transition (EMT), implicating these pathways in fibrosis development. MPs and NPs further amplify these processes through activation of the TLR4/NF-κB axis and the NLRP3 inflammasome, leading to intensified oxidative stress, mitochondrial damage, and apoptotic signalling, consistent with observations of MP-induced airway inflammation and fibrotic changes (Rout-Pitt et al., 2018; J. Shi et al., 2016).

Table 1

Summary of cytokine levels from included studies.

Polymer	Size	Experimental Model	Inflammatory Mediator	Author
PE, PP fragments	< 50 µm	A549	IL-6 (↑), IL-8 (↑ at high conc.)	(Bengalli et al., 2022)
PS beads	64 nm, 202 nm, 535 nm	A549	IL-8 (⇑),	(Brown et al., 2001)
PS beads	1–5 µm, 10–20 µm	A549, Mice	TNF-α (↑), IL-1B (↑), IL-6 (↑), IL-8 (↑)	(Cao et al., 2023)
PS beads	50 nm, 200 nm, 1 µm	A549	IL-6 (↑), IL-8 (↑), TNF-α (↑)	(da Silva Brito et al., 2023)
PS beads	1.72 ± 0.26 µm	BEAS-2B	IL-6 (↑), IL-8 (↑ at high conc.)	(Dong et al., 2020)
Nylon fibre, PS beads, car tyre fragment,	0.05–100 µm	Primary 3D Lung Epithelial Cells	IL-6 (↑), IL-8 (—), IFN-γ (↑), TNF-α (↑), IL-10 (↑)	(Donkers et al., 2022)
Polytetrafluoroethylene (PTFE) beads	6.0 µm, 31.7 µm	A549	IL-6 (↑), TNF-α (↑)	(K C et al., 2023)
PS beads	100 nm	Mice	TGB (↑), TNF-α (↑)	(Lim et al., 2019)
PS beads	800 nm	A549	IL-1A (⇑), IL-1B (⇑), IL-6 (⇑), IL-8 (⇑)	(Milillo et al., 2024)
PP fragments, PP beads	0.66 ± 0.27 µm	Mice	TNF-α (⇑), IL-1B (⇑), IL-6 (⇑)	(Woo et al., 2023)
PS beads	25 nm, 70 nm	A549	TNF-α (↑), IL-1B (—), IL-6 (↑), IL-8 (↑)	(M. Xu et al., 2019)

*Note: *A single arrow (↑) denotes an increase in protein release, whereas a double arrow (⇑) indicates upregulated gene transcription.*

By contrast, IL-8 and IL-1β responses are more variable. Smaller MPs and NPs (<1 µm) generally elicit stronger induction of IL-8 and IL-1β, whereas larger pristine MPs tend to produce weaker or negligible effects unless present at high concentrations (Brown et al., 2001; Donkers et al., 2022; M. Xu et al., 2019). Inconsistencies are especially evident for IL-1β. For instance, Milillo et al. (2024) reported increased transcription following exposure to submicron PS particles (800 nm), while Xu et al. (2019) observed no change with ultrafine PS NPs (25–70 nm). Woo et al. (2023), however, detected elevated IL-1β secretion in BALF from mice exposed to irregular PP MPs (0.66 µm), accompanied by

macrophage and neutrophil infiltration. These discrepancies suggest that inflammatory outcomes may be shaped not only by particle size but also by shape, polymer composition, and surface chemistry, as well as by differences between *in vitro* and *in vivo* models. Overall, the evidence points to a robust induction of IL-6 and TNF-α as hallmark responses, while IL-8 and IL-1β remain context dependent, reflecting the interplay of particle characteristics and model systems. The resulting inflammatory response may aggravate the toxicity, leading to a progressive loop of cellular stress and chronic inflammation that exacerbates inflammatory respiratory diseases such as asthma, ARDS, and COPD (Atsmon, 1988; Sethi et al., 2019).

MPs and NPs disrupt respiratory barrier permeability

Disruption of epithelial barrier integrity is a critical pathological event in respiratory diseases and has been consistently reported following MP and NP exposure (Y Cao et al., 2024; J. Huang et al., 2022). Barrier function is primarily maintained by tight junctions (e.g., ZO-1, occludin, claudins) and adherens junctions (e.g., E-cadherin), which regulate epithelial cohesion and permeability. Transepithelial electrical resistance (TEER) is widely used as a marker of tight junction integrity, with reductions indicating compromised barrier function (Srinivasan et al., 2015). Multiple studies have shown significant TEER reduction in response to MPs and NPs, irrespective of particle size or polymer type. For example, PS MPs reduced TEER in BEAS-2B cells, accompanied by tight junction disorganisation and translocation of particles across the alveolar–capillary barrier into circulation. Similar reductions have been reported in organoid models, where MP fibres impaired barrier integrity. Oxidative stress is known to exacerbate this dysfunction by inducing ZO-1 phosphorylation and disrupting tight junction architecture (Sethi et al., 2019).

Loss of cell adhesion, regulated via adherens junctions, also triggers epithelial–mesenchymal transition (EMT), characterised by reduced E-cadherin and acquisition of mesenchymal traits such as enhanced proliferation, migration, and extracellular matrix (ECM) production (Rout-Pitt et al., 2018). EMT is often regulated by TGF-β, which has shown elevation in response to MP and NP exposure. TGF-β activates transcription factors such as Snail/Slug, repressing epithelial genes and increasing mesenchymal characteristics (Naber et al., 2013). Downstream effects include the activation of matrix metalloproteinases (MMPs), which degrade ECM and promote cell migration and invasion. EMT contributes directly to fibrosis by promoting tissue remodelling, collagen accumulation, and hyperplasia (Di Gregorio et al., 2020). *In vitro*, PS NP exposure has induced EMT-like changes in A549 cells, including cytoskeletal reorganisation and increased fibrotic marker expression (Halimu et al., 2022; Cao et al., 2023; Goodman et al., 2021). Complementary *in vivo* studies in murine models revealed collagen deposition, alveolar wall thickening, and fibrotic remodelling following prolonged MP and NP (J. Huang, Dong, Liang, et al., 2022; Li et al., 2022; Yang et al., 2021).

Effects of MPs and NPs on epithelial cell growth and repair, progression to fibrosis

Ultimately, impairment of barrier integrity and persistent epithelial cell injury due to MPs and NPs led to abnormal tissue remodelling, as demonstrated in organoid models. MPs markedly reduced airway organoid growth, particularly during early developmental stages, indicating impaired epithelial proliferation and differentiation, which is a critical phase for cell renewal (Winkler et al., 2022). Polarised growth patterns and encapsulation of MPs within epithelial barrier further suggest a foreign body-like response resembling excessive ECM interactions during tissue repair (van Dijk et al., 2020). These changes disrupt normal regenerative processes and predispose tissues to fibrotic outcomes (Li et al., 2022; Yang et al., 2024).

Indeed, fibrotic alterations have been confirmed *in vivo*. Li et al. (2022) reported pulmonary fibrosis in mice exposed to PS NPs (40 nm), with collagen deposition and fibrotic regions worsening with prolonged exposure. Yang et al. (2024). similarly observed alveolar thickening, hyperplasia, and marked collagen accumulation, with severity increasing over time. Beyond the lungs, upper airway damage has also been documented where S. Huang et al. (2022) found significant thinning of the nasal mucosa in exposed mice. Mechanistically, persistent cell death, combined with the chronic activation of NF-κB and MAPK pathways, perpetuates inflammation and elevates the secretion of pro-fibrotic cytokines such as TGF-β. These mediators stimulate fibroblast activation and ECM remodelling, which are hallmarks of fibrosis progression. Collectively, MP exposure not only compromises epithelial integrity but also initiates signalling cascades that underpin chronic inflammation, tissue remodelling, and long-term fibrotic progression. Figure 2 illustrates the signalling cascade triggered by MPs and NPs and the resulting effects on the respiratory epithelial barrier.

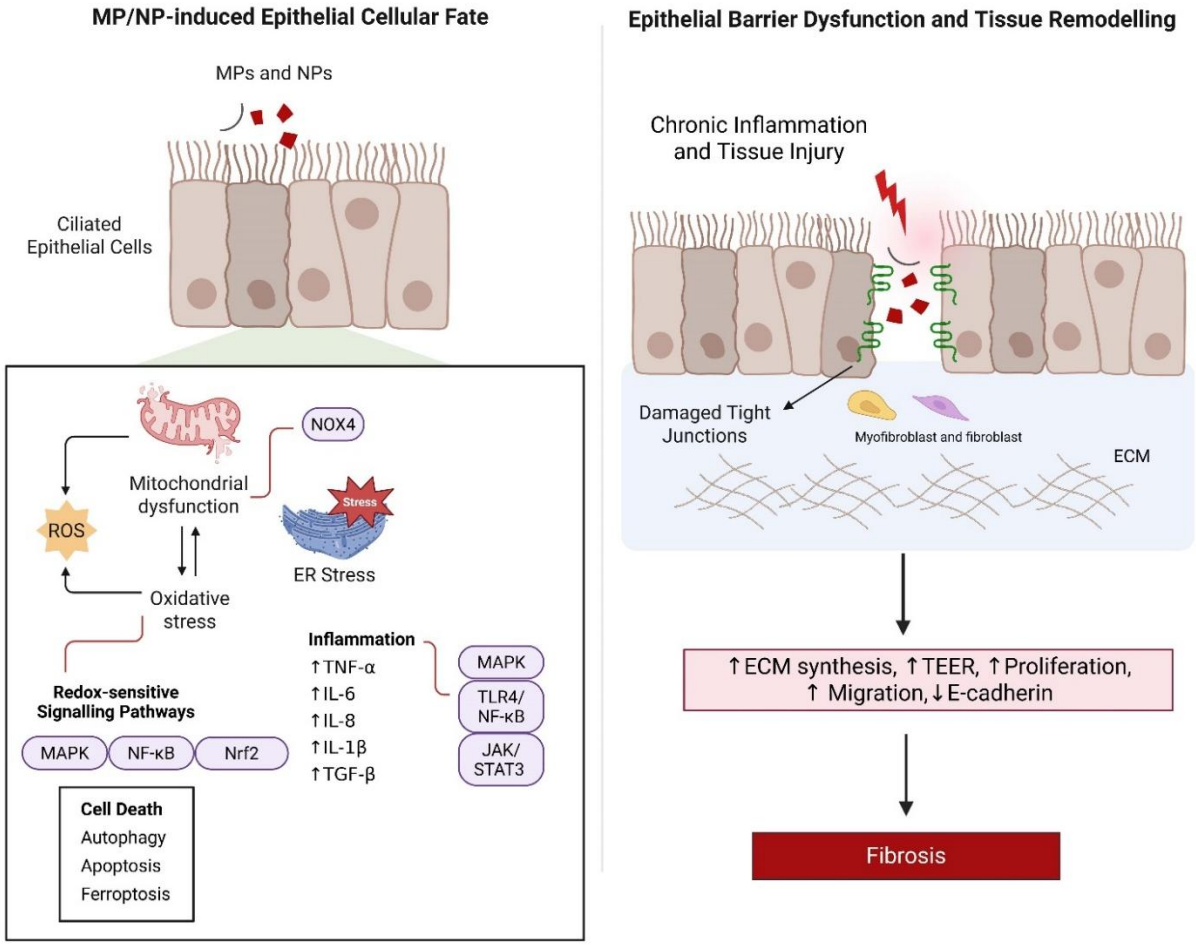
CONCLUSION

This review highlights the growing concern that airborne MPs and NPs compromise respiratory epithelial health through multiple mechanisms, including oxidative stress, immune activation, direct cell damage, and impaired tissue repair. Particle size and morphology appear to dictate toxicity pathways, with smaller NPs readily internalised and activating biochemical stress responses, while larger fibrous MPs exert mechanical damage and interfere with epithelial remodelling. Physicochemical properties including charge, shape, polymer composition, associated additives and corona formation further modulate these effects, with fibres emerging as particularly hazardous due to their

persistence, abundance in lung tissues, and ability to evade clearance mechanisms. Despite growing evidence of MPs' impact on respiratory health, several critical knowledge gaps and discrepancies remain. There is no consensus on the exact range of inhalable MPs, as emerging studies suggest that particles larger than the traditionally defined respirable range can still enter and deposit in the lungs. Experimental inconsistencies, such as variations in MPs' size, shape, concentration, and exposure duration, limit the comparability of studies. Furthermore, most toxicity studies rely on pristine MPs, which may not fully reflect real-world exposure to weathered, chemically altered, or mixed MPs from daily environments. From a biochemical perspective, few studies address chronic low-dose MP exposure at physiologically relevant levels, with most *in vitro* work focusing on acute or high doses that may not reflect the cumulative responses of epithelial cells during long-term inhalation. Standardised protocols for MPs characterisation, exposure models, and health assessments are urgently needed to ensure reproducibility and accurate risk evaluation. A multidisciplinary approach integrating environmental science, toxicology, respiratory medicine, and epidemiology is crucial in bridging these gaps, allowing us a thorough understanding of airborne MPs inhalation and its impact on human health. Future mechanistic work can also include proteomic and metabolomic profiling of exposed epithelial cells to identify novel biochemical signatures of MP exposure.

Figure 2

Summary of the downstream effects of MPs and NPs on the epithelial barrier



Note: Illustration summarising the signalling cascades and downstream effects triggered by MPs and NP exposure on the respiratory epithelial barrier. This figure visualises findings from studies discussed in the section 'Effects of MPs and NPs exposure on the respiratory epithelial barrier'. The image is created in BioRender.com.

AUTHOR CONTRIBUTIONS

Ungku Maryam Jamilah binti Ungku Mohsin: Substantial contributions to the conception or design of the work, writing the original manuscript and revising it critically for important intellectual content. Ting Kang Nee: Substantial contributions to the conception of the work, drafting and revising the work critically for important intellectual content, supervision, and final approval of the version to be published. Fang Chee-Mun: Supervision, revising the work critically for important intellectual content, and final approval of the version to be published. Tshai Kim Yeow: Supervision, and

final approval of the version to be published. Sivathass Bannir Selvam: Supervision, and final approval of the version to be published. Christopher Gibbins: Supervision, and final approval of the version to be published. Georgina Marsh: Substantial contributions to the conception of the work, draft revisions, and final approval of the version to be published. Lee Mei Kee: Substantial contributions to the conception of the work, drafting and revising the work critically for important intellectual content, supervision, and final approval of the version to be published.

CONFLICT OF INTEREST

The author declares no conflict of interest in the work produced.

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