REVIEW ARTICLE

The Health Effect of Inhaled Microplastic in Vivo And In Vitro: A Systematic Review

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ABSTRACT

Airborne microplastic have recently been recognized as an emerging component of air pollution, as a component of particulate matter (PM). Due to their small size, they can be inhaled and deposited in the respiratory tract, yet the mechanisms underlying their health effects have not been fully elucidated. Previous research has primarily focused on in vivo and in vitro microplastic exposure. This study evaluates the impact of inhaled microplastics in vivo and in vitro from previous studies. **Method:** A systematic search was conducted under PRISMA guidelines using Boolean operators with the following keywords: "(inhaled OR respiratory exposure OR airborne exposure OR pulmonary exposure) AND (polystyrene OR polypropylene OR polyethylene) AND (in vivo OR in vitro) AND (toxicity OR inflammation OR lung injury OR adverse effects OR health effects)." Searches were conducted in Google Scholar, Scopus, and PubMed to identify peer-reviewed articles in English published between 2020 and 2024. Inclusion criteria required the use of in vivo or in vitro models, with randomized controlled trials for in vivo studies, while studies lacking statistical data were excluded. The search period, from July 16 to August 20, 2024, showed that most of the retrieved publications addressed polystyrene microplastics. **Result:** Most polymers in these studies are polystyrene, and all polymers indicated an increase in Reactive Oxygen Species (ROS) and inflammation. **Conclusion:** The polymer, size, dose, exposure time, endpoints, and cell types both in vivo and in vitro cause alterations in the target organ in results in these studies.

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INTRODUCTION

The global surge in plastic production and consumption has led to extensive environmental accumulation (1), with more than 60% of plastics already exist in the environment (2). J Only about 9% of plastic waste is recycled in developed countries, while the remaining 91% is discarded and persists for decades (3). Over time, plastics fragment into microplastics through a combination of physical, chemical, and environmental processes. Prolonged sunlight exposure, particularly ultraviolet (UV) radiation, triggers photodegradation, weakening the polymer structure and making it brittle (4). Once weakened, plastics are further broken down by mechanical forces such as wind, wave motion, and abrasion with surfaces like sand. Fluctuating weather conditions, including rainfall and temperature changes, accelerate this degradation. Although biological factors such as ingestion by animals or microbial activity may contribute, most plastics are highly resistant to biodegradation (5). As a result, microplastics remain widespread and persistent in the environment due to improper disposal and their slow degradation rates (6,7). Microplastics are typically defined as plastic particles smaller than 5 mm (8). Their presence in the environment originates from multiple sources and pathways. They are classified into two categories: primary and secondary. Primary microplastics are intentionally manufactured at the microscale for use in products such as biomedical materials and personal care items. These particles, often spherical and ranging from 1 to 5 μm in diameter, are commonly composed of polypropylene (PP),

polystyrene (PS), or polyethylene (PE) (9). In contrast, secondary microplastics are formed when larger plastic debris fragments over time. Sources of secondary particles include tire wear, synthetic textiles, industrial emissions, personal care products, and agricultural practices, while transport pathways include stormwater runoff, wastewater discharge, atmospheric deposition, and accidental spills (10). Because macroplastic debris gradually disintegrates into smaller particles, it is considered the primary contributor of secondary microplastics found in soils and marine systems (11). Their environmental behavior and distribution are strongly influenced by particle properties such as density, size, and shape, and by external factors including wind, air currents, and precipitation (4,12).

Evidence of the pervasive occurrence of microplastics in the air, water, and soil, along with their potential effects on human health is mounting. Some experts have even described the situation as a looming plastic health catastrophe (13). A 2024 study, using an advanced method capable of detecting extremely small plastic particles, found that 90% of the 110,000–370,000 plastic particles per liter of bottled water were nanoplastics. According to a 2024 research that used a testing method that could identify minuscule plastic particles, indoor concentrations of microplastics may also be up to 60 times higher than outdoor levels, and it is estimated that the average person inhales between 2,000 and 7,000 microplastic particles each day (14).

Humans are exposed to microplastics primarily through ingestion and inhalation (15). Even though microplastics are recognized as air pollutants rather than typical atmospheric particulates, their potential health effects warrant serious attention (16). Due to their small size, microplastics pose a significant risk of being inhaled by organisms (17–19). However, the mechanisms underlying their health effects remain unclear. Previous research has primarily investigated microplastic exposure through in vivo and in vitro models (16,17,19–21). In vitro studies are conducted in a laboratory settings, allowing for controlled experiments and high-throughput analysis, but they may not fully replicate the complexity of living organisms. Conversely, in vivo studies take place within living organisms, providing a more realistic perspective on whole-body responses, though they tend to be more expensive, time-consuming, and technically challenging (22). Many questions remain unanswered regarding inhaled microplastics, particularly their health effects and the most effective strategies for measuring and reducing contact with it. Further research is needed to examine the extent of inhalation, the effects on the respiratory system, and the potential long-term consequences, especially for individuals with pre-existing respiratory conditions. This study therefore, aims to evaluate the in vivo and in vitro effects of inhaled microplastics drawing on existing evidence.

METHODOLOGY

Source

A systematic search of all published, peer-reviewed journal articles on microplastics was conducted using Boolean operators, following PRISMA guidelines (23).

Formulation of the research question

The research question for this systematic review was formulated using the PICO framework (Population, Intervention, Comparison, and Outcome), which was adapted to Problems, Interests, and Context. The review focused on three main elements: inhaled microplastics (problems), in vivo and in vitro studies (interests), and their effects (context).

Eligibility Criteria

A comprehensive literature search was conducted to identify English-language articles that fulfilled the following inclusion criteria: published status, experimental study design, microplastic exposure, and outcomes related to changes in organ function.

Information Sources

Published studies on the in vivo and in vitro health impacts of inhaled microplastics from the previous five years (2020–2024) were retrieved using Google Scholar, Scopus, Web of Science, and PubMed.

Search Strategy

A systematic database search was performed using Boolean operators with the following keywords: (microplastics OR microplastic particles OR plastic microfibers OR airborne microplastics) AND (inhaled OR respiratory exposure OR airborne exposure OR pulmonary exposure) AND (polystyrene OR polypropylene OR polyethylene) AND (in vivo OR in vitro) AND (toxicity OR inflammation OR lung injury OR adverse effects OR health effects) yielding approximately 2,000 results in Google Scholar, 3 results in Scopus, 57 result in Web of Science, and 29 results in PubMed.

Selection Process

Duplicate records were initially removed, and the remaining articles underwent screening based on titles and abstracts. Studies on nanoplastics smaller than 1 µm and those involving oral exposure were excluded, with only full-text articles assessed. For in vivo studies, we selected original research articles published between 2020 and 2024 that employed a randomized controlled trial design. In vitro studies were examined if they had both negative and positive control groups, clearly specified microplastic dosages, identified the cell lines

used, and validated the methods employed. Studies that lacked statistical data were omitted. At least two independent reviewers were involved in the review process. Discrepancies between the two reviewers were resolved through discussion. If consensus could not be reached, a third reviewer made the final decision. Nineteen studies met the eligibility criteria, and data were extracted according to polymer type, particle size, dose, and mechanisms observed in both in vivo and in vitro studies. All retrieved articles were imported into Mendeley for reference management, where duplicates were identified, selected, and removed. Covidence is used as a collaborative workspace for all researchers. The screening process involved two stages: first, two reviewers evaluated titles and abstracts for relevance; second, full-text articles were reviewed to determine eligibility.

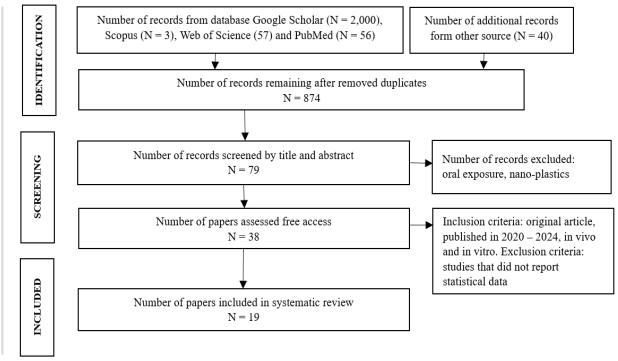

Figure 1: PRISMA flow chart

RESULTS

Polymer, Size, Dose

The polymers investigated in these studies were polypropylene (PP), polystyrene (PS), polyethylene (PE), polyvinyl chloride (PVC), and polyamide (PA), with sizes ranging from 100 nm to 5 mm. Four of 19 studies did not mention the polymer size (24–27). The dose variance was between 0.6 mg/kg – 100 mg/kg; 10 µg/µL– 2 mg/200mL, 0 – 100 µg/mL, 0.3 mg/mL – 10 mg/mL (24–42). Studies on similar types of polymers used varying dose ranges and assessed different endpoints. Particle size plays a crucial role in determining toxicity, absorption, and distribution; smaller microplastics or nanoplastics tend to be more biologically active, are capable of entering cells, and often trigger more intense systemic effects. The experiments were meticulously designed to maintain strict control over particle size, dosage, and exposure route in both in vitro and in vivo studies.

Table I: Characteristics of Microplastics Used in the Reviewed Studies

No	Polymers	Size	Dose	Ref
1	Polystyrene	5 µm	10 µg/µL	Zha et al., 2023
2	Polypropylene, Polystyrene, and Polyethylene	<20 µm	5 mg/kg	Danso et al., 2024
3	Polystyrene	5 µm	1.25 mg/kg and 6.25 mg/kg	Li et al., 2022
4	Polystyrene, Polypropylene, and Polyvinyl Chloride	N/A	5 mg/kg	Danso et al., 2022
5	Polyethylene, Polypropylene, Polystyrene, and Polyvinyl chloride	N/A	25 mg/kg and 100 mg/kg	Jin et al., 2024
6	High-Density Polyethylene	44.2–552.4 µm	0.3 mg/ml,1.0 mg/ml, 3.0 mg/ml, 5.0 mg/ml and 10.0 mg/ml	Anuar et al., 2022

CONTINUE

Table I: Characteristics of Microplastics Used in the Reviewed Studies (CONT.)

No	Polymers	Size	Dose	Ref
7	Polystyrene	1–5 µm and 10–20 µm	40 mg/kg	Cao et al., 2023
8	Polystyrene	5 µm	0.6 mg/kg, 3 mg/kg, and 15 mg/kg	Kang et al., 2024
9	Polyamide	3 µm	10 µg and 50 µg	Wu et al., 2024
10	Polystyrene	100 nm	0.5 mg/200 µL, 1 mg/200 µL, and 2 mg/200 µL.	Fan et al., 2022
11	Polystyrene	100 nm	0.5mg/m^3 ± 20%, 1.0mg/m^3± 20%, and 2.0mg/m^3± 20%	Luo et al., 2023
12	Polystyrene	0.10 µm	0.75x10^5 particle/cm^3, 1.50x10^5 particle/cm^3, and 3.00x10^5 particle/cm^3	Lim et al., 2021
13	Polyethylene	10–45 µm	0, 6 µg, 60 µg	Han et., al, 2021
14	Polystyrene	1 µm	025 µg and 50 µg	Zhou et al., 2023
15	Polystyrene	N/A	1.6 µg/ml	da Silva Brito et al., 2023
16	Polystyrene	1 mm, 5 mm	1–30 mg/l	Hayek et al., 2023
17	Polystyrene	2 µm and 80 nm	0, 50, 100 µg/ mL	Shi et al., 2022
18	Polyethylene	30.5±10.5 and 6.2±2.0 µm	N/A	Gautam et al, 2022
19	Polystyrene	N/A	N/A	Dong et al., 2020

In Vivo Mechanisms

All studies used mice as the experimental model, and findings were presented based on polymer type, affected organ system, and primary mechanism. Four polymers were examined: PS in the nasal cavity, lungs, and cardiac; PP in the lungs; PE in the lungs, liver, and intestines; and PA in the lungs. The mechanisms underlying disease occurrence reported in the studies included increased ROS production and the induction of inflammation (24,28–33,37–42). One study (37)

observed nasal dysbiosis, while two studies reported elevated oxidative stress with reported changes in superoxide dismutase (SOD) and glutathione (GSH) levels, apoptosis, and accelerated fibrosis (39,41). Eight studies (24,28,29,31,33,38,41,42) observed increased infiltration of inflammatory cells, collagen deposition, impaired lung function, and disruption of pulmonary barrier permeability. One study (39) reported lung fibrosis associated with activation of the Wnt/β-catenin signaling pathway. Additionally, one study (29) demonstrated bronchial and alveolar damage along with altered expression of 109 lncRNAs and 269 circRNAs. Another study (30), found an increased numbers of senescent cells, as indicated by β-galactosidase staining. One study (40) showed enhanced tissue contractile responses to carbachol, a muscarinic agonist.

Table II: Summary of In Vivo Effects of Microplastics by Polymer Type

Type Polymer	Organ System	Primary Mechanism	Reference
Polystyrene	Nasal	**Microbiota Dysbiosis:** dysbiosis of the nasal microbiota	Zha et al., 2023
Polystyrene	Lung	**Oxidative Stress, Cellular Damage, Fibrosis/Remodeling:** oxidative stress (SOD and GSH), apoptosis, and hastened the fibrosis process	Cao et al., 2023; Li et al., 2022
Polystyrene	Lung	**Inflammation & Immune Dysregulation, Fibrosis/ Remodeling, Cellular Damage, Barrier Dysfunction:** increased inflammatory cells macrophages, neutrophils, and eosinophils, IL-1, IL-1β, IL-6, TNF-α, TGF-β, activating the NLRP3 and NF-κB inflammasomes through the TLR4 pathway, apoptosis and collagen deposition, reduced lung function and permeability of the pulmonary barrier in lung tissue	Danso et al., 2022; Cao et al., 2023; Kang et al., 2024; Danso et al., 2024; Fan et al., 2022; Lim et al., 2021
Polystyrene	Lung	**Fibrosis/Remodeling (Signaling Pathway–Mediated):** causes lung fibrosis by triggering the Wnt/β-catenin signaling pathway	Li et al., 2022
Polystyrene	Lung	**Cellular/Tissue Damage:** demonstrates bronchial epithelium and alveolar damage	Fan et al., 2022
Polystyrene	Lung	**Molecular/Epigenetic Alteration:** differential expression of 109 lncRNAs and 269 circRNAs	Fan et al., 2022
Polystyrene	Lung	**Cellular Senescence:** β-galactosidase staining revealed a higher number of senescent cells	Luo et al., 2023
Polystyrene	Cardiac	**Oxidative Stress, Inflammation, Fibrosis, Cellular Damage:** elevated levels of apoptosis, oxidative stress, inflammatory response, and collagen buildup	Zhou et al., 2023
Poly-propylene	Lung	**Inflammation & Immune Dysregulation:** elevated inflammatory cell counts, an inflammatory cytokine, chemokine levels, lung tissue NLRP3, ASC, caspase-1 levels, and IL-1 levels	Danso et al., 2024
Poly-propylene		**Physiological/Functional Alteration:** enhanced the contractile responses of the tissues to carbachol (muscarinic agonist)	Anuar et al., 2022
Polyethylene	Lung	**Bioaccumulation/Deposition:** detected PE-MPs	Han et., al, 2021
	Liver	**Bioaccumulation/Deposition:** detected PE-MPs	Han et., al, 2021
	Intestine	**Bioaccumulation/Deposition:** detected PE-MPs	Han et., al, 2021
Polyamide	Lung	**Inflammation, Fibrosis/ Remodeling, Barrier Dysfunction:** in asthma, airway inflammation, mucus hypersecretion, and fibrosis were exacerbated	Wu et al., 2024
	Lung	**Immune Dysregulation / Allergic Response:** raised the amounts of major Th2 and Th1 pro-inflammatory cytokines as well as overall Ig E	Wu et al., 2024
		Cellular Damage, Barrier Dysfunction: caused apoptosis in lung epithelial cells and compromised the integrity of the epithelial barrier	Wu et al., 2024

In Vitro Mechanism

The in vitro studies employed various human cell lines, consisting of alveolar cells (A549), bronchial cells (BEAS-2B), embryonic kidney cells (HEK293), cervical cancer cells (HeLa), colorectal adenocarcinoma cells (Caco-2), myeloid leukemia cells (U937), monocytic leukemia cells (THP-1), T lymphocytes (Jurkat), and epidermal keratinocytes (HaCaT). Three polymers were investigated: PS, polyvinyl chloride (PVC), and PE. Particle concentration and size were found to be tightly connected to particle uptake in one study (26). The primary mechanisms involved increased ROS production and stimulating inflammatory responses (25,27,36). Specific cellular responses alterations in gene or protein expression in the human A549 cells (25); cytotoxicity in BEAS-2B (27), and specific cytokines in HaCaT, THP-1, and U937 (36).

DISCUSSIONS

Polymer, Size, Dose

This review synthesizes evidence from in vivo and in vitro studies on the biological effects of microplastic exposure, emphasizing the role of polymer type, particle size, and dose in determining biological outcomes. The findings demonstrate consistent patterns while

Table III: Summary of In Vitro Effects of Microplastics by Polymer Type

Type Polymer	Organ System	Primary Mechanism	Reference
Polystyrene	A549, HEK293, and HeLa	**Particle Characteristics & Uptake Dynamics (Exposure–Dose Relationship):** particle concentration and size showed a strong correlation with particle uptake	da Silva Brito et al., 2023
Polystyrene	BEAS-2B	**Oxidative stress (ROS)–mediated cytotoxicity and inflammation:** cytotoxic and inflammatory effects in cells by inducing reactive oxygen species	Dong et al., 2020
Polystyrene	BEAS-2B	**Oxidative Stress & Impaired Protective Mechanisms:** leading to lower levels of α1-antitrypsin in cells and reactive oxygen species	Dong et al., 2020
Polystyrene	A549 and BEAS-2B	**Cellular Senescence:** induced senescence of human lung-derived	Jin et al., 2024
Polyvinyl chloride (PVC)	A549 and BEAS-2B	**Oxidative Stress dan Inflammation/Immune Dysregulation:** raised the amount of ROS induce an increased systemic inflammation level	Jin et al., 2024
Polyethylene	Caco-2 and A549	**Oxidative Stress:** increased ROS and increased NO in all of the cell lines	Gautam et al, 2022
Polyethylene	HaCaT, THP-1, and U937	**Inflammation:** altered levels of pro-inflammatory cytokines IL-6 levels post-treatment with PE-MPs.	Gautam et al, 2022

also expose important gaps and inconsistencies that warrant critical reflection. Polystyrene (PS) was the most frequently studied polymer, reflecting its widespread use and environmental prevalence. PS microplastics consistently exhibited toxicity in both in vitro and in vivo studies, often triggering oxidative stress, inflammatory responses, and disruption of epithelial barriers (43,44). These effects were particularly evident in immune and lung epithelial cells such as THP-1 and A549. In contrast, polyethylene (PE) and polypropylene (PP) produced milder or more variable effects, although certain formulations (e.g., irregularly shaped particles, higher doses) could still provoke inflammatory responses (44). In some cases, the combination of optimal particle size and concentration can lead to highly efficient uptake. For instance, nanoparticles of an ideal size range (e.g., 50-200 nm) at moderate concentrations may exhibit the highest cellular uptake. Balancing both particle size and concentration is crucial for maximizing internalization. Very small particles at high concentrations may aggregate or induce toxicity, while larger particles at high concentrations may not be internalized as effectively (45,46).

Nanoplastics (<100 nm) were capable of crossing biological barriers and were internalized by cells, triggering reactive oxygen species (ROS) production and cytokine release. In contrast, larger particles (>1 μm) exhibited lower uptake and required higher doses to elicit cellular responses (47). Interestingly, optimal uptake occurred at intermediate sizes (50-200 nm) and moderate concentrations, while very small or large particles at high doses risked aggregation or poor internalization (48).

Dose-dependent responses were observed across most studies. Doses above 100 μg/mL consistently reduced cell viability and promoted inflammation and oxidative damage, especially in PS exposures. However, some studies observed effects even at lower concentrations, suggesting that both particle characteristics and the biological context modulated toxicity (27,44).

In Vivo Mechanisms

Across 14 in vivo studies, PS exposure was consistently linked to respiratory effects such as ROS accumulation, inflammation, apoptosis, and fibrosis (24,29,31,38,39,41,42). Lung histopathology frequently revealed alveolar septal thickening, inflammatory infiltrates, and collagen deposition. Several studies also reported a dose– and time–dependent decline in pulmonary function. Moreover, exposure to 1–5 μm PS particles at ambient levels was sufficient to induce structural damage and cytokine dysregulation. However, discrepancies emerged in the literature: one study reported no histopathological fibrosis, yet it detected upregulation of inflammatory markers TNF-α and TGF-β (31).

A novel but under-investigated finding was the connection between microplastic exposure and respiratory tract dysbiosis. PS exposure altered nasal and pulmonary microbiota, increasing the abundance of taxa such as Staphylococcus, Roseburia, and Eggerthella, which may serve as microbial biomarkers of microplastic-induced inflammation (36).

Other polymers, such as PP and PA also triggered lung inflammation and immune cell recruitment. PE exposure led to particle adhesion in the airway and enhanced bronchial contractility, while PVC increased ROS and systemic inflammatory responses (40). Collectively, these findings supported the hypothesis that chemical composition, size, and delivery route significantly shape microplastic toxicity.

In Vitro Mechanism

In contrast to in vivo studies, in vitro models (n=3) produced less consistent results. The most commonly used cell lines were A549 and BEAS-2B. In contrast to in vivo studies, in vitro models (n=3) yielded less consistent results. The most common cell lines used

were A549 and BEAS-2B. Some studies observed minimal cytotoxicity, whereas others demonstrated ROS production, senescence, and mild cytokine release. This divergence reflected the absence of systemic factors in vitro, including immune modulation, microbiome interaction, and particle clearance mechanisms.

While certain cell types and conditions showed minimal effects, others, particularly under PS nanoparticle exposure, exhibited significant inflammatory and oxidative responses (43,49). Thus, the impact of microplastics in vitro is highly context-dependent.

Compared to previous reviews, this study underscored existing concerns regarding PS microplastics while providing new insight into polymer-specific and size-related effects in respiratory models. The findings aligned with global reports highlighting oxidative stress and immune dysregulation as key mechanisms of microplastic toxicity (50,51). Notably, the inclusion of respiratory microbiota dysbiosis provided a novel perspective rarely addressed in earlier reviews.

This review had several methodological limitations. Only full-text articles that were freely accessible and published within the past five years were selected, and the search relied primarily on Google Scholar, which may have omitted indexed but paywalled studies. The small number of in vitro studies limited cross-model comparisons, and outcome heterogeneity restricted meta-analysis. Additionally, numerous studies in the dataset presented data in tabular rather than mechanistic form, which constrained interpretation.

The evidence suggests that respiratory exposure to PS nanoplastics, even at environmentally relevant concentrations, could impair lung function and provoke immune responses. These findings highlight significant public health concerns, especially for indoor air quality, occupational settings, and vulnerable populations such as children and asthmatics. Future studies should focus on standardizing particle characterization (e.g., size, surface chemistry) and establishing chronic low-dose inhalation models.

CONCLUSION

This review demonstrates that various polymeric microplastics, including PS, PE, and PP have the potential to induce adverse respiratory effects, particularly ROS, inflammation, apoptosis, and fibrosis, as evidenced in both in vivo and in vitro models. While the specific responses varied according to polymer type, particle size, dose, exposure route, and cell type, a general pattern emerged: nanoplastics and higher concentrations consistently provoked more pronounced biological effects.

However, it is important to note that the studies analyzed in this review varied considerably in terms of study design, experimental endpoints, exposure protocols, and biological models, which limits direct comparability. The apparent similarity of outcomes across studies may obscure subtle but important variations in mechanistic pathways or long-term implications, especially under low-dose, chronic exposure scenarios that are more relevant to environmental health. Limitations of this review should be acknowledged. While major databases were systematically searched, relevant studies may still have been missed, particularly non-English publications or those outside the indexed databases

Future research should prioritize polymer prevalent in daily-use items, particularly PE and PP, which belong to the polyolefin class of thermoplastics and are extensively used in packaging and consumer goods. These polymers may degrade under environmental conditions such as UV exposure, potentially altering their toxicity profiles over time. Despite being underrepresented in mechanistic toxicity studies, their ubiquity in real-world settings makes them critical targets for future risk assessments.

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