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## Impacts of Air Pollution and Microplastics on Environmental Health in the Era of Climate Change

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### Abstract

*Environmental health challenges in the twenty-first century increasingly arise from the interaction between atmospheric pollution, plastic contamination, and climate change. This study examines the impacts of air pollution and microplastic exposure on environmental health using a quantitative descriptive approach combined with a systematic literature review based on secondary data. Scientific articles were retrieved from PubMed, Scopus, and ScienceDirect, resulting in 156 initial publications, of which 38 studies met the inclusion criteria and were analyzed. The synthesis integrates global datasets from the World Health Organization, Institute for Health Metrics and Evaluation, OECD reports, and national environmental statistics. Findings indicate that ambient air pollution contributes to approximately 6.67 million premature deaths annually, with average PM<sub>2.5</sub> exposure reaching 29 µg/m<sup>3</sup>, substantially exceeding the WHO recommended limit of 5 µg/m<sup>3</sup>. Global plastic production increased dramatically from 2 million tons in 1950 to 460 million tons in 2021, while only 9% of plastic waste is recycled, facilitating the formation of microplastics detected in human blood, lungs, and gastrointestinal samples. Plastic production also generates approximately 1.8 gigatons of CO<sub>2</sub> emissions annually, strengthening the linkage between pollution and climate change. The results highlight synergistic environmental health risks arising from combined exposure to particulate pollution and microplastics, emphasizing the importance of integrated environmental health policies and climate mitigation strategies..*

**Keywords:** Air Pollution, Climate Change, Environmental Health, Microplastics, One Health.



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## INTRODUCTION

Environmental health has increasingly emerged as a critical global concern in the twenty-first century as environmental degradation intensifies under the combined pressures of industrialization, urbanization, and climate change. Epidemiological assessments estimate that environmental risk factors contribute to approximately 24% of total global mortality, highlighting the profound relationship between ecological conditions and human health outcomes (World Health Organization, 2016). Among the most consequential threats are ambient air pollution and the rapidly escalating proliferation of plastic waste, both of which operate beyond national boundaries and interact with broader climate dynamics. Data from the Global Burden of Disease study indicate that exposure to ambient air pollution is responsible for an estimated 6.67 million deaths annually worldwide, with the highest disease burden concentrated in South and Southeast Asia where urban expansion and fossil-fuel-based economic growth remain dominant (Institute for Health Metrics and Evaluation, 2024). In parallel, the expansion of plastic production from approximately 2 million tons in 1950 to 460 million tons in 2021 illustrates the unprecedented scale of synthetic material consumption in the modern economy, a trend accompanied by extremely limited waste recovery since only about 9% of plastic waste is successfully recycled (OECD, 2022).

Accumulating scientific evidence demonstrates that both air pollution and microplastic contamination have direct and systemic implications for human health and ecological stability. Fine particulate matter (PM<sub>2.5</sub>), generated primarily through fossil-fuel combustion and industrial activities, has been strongly associated with increased risks of cardiovascular disease, stroke, and chronic respiratory disorders, particularly in densely populated urban regions. Observational analyses reveal that several metropolitan areas in Indonesia record particulate concentrations far exceeding international health guidelines, with exposure levels approaching multiples of the recommended threshold established by global health authorities (CREA, 2025). Parallel developments within

environmental toxicology research reveal that plastic debris undergoes fragmentation processes that generate microplastics particles smaller than 5 mm which increasingly contaminate air, water, and food systems. Empirical investigations have confirmed the presence of airborne microplastics within human lung tissue, suggesting direct inhalation exposure and raising serious concerns regarding chronic inflammatory and respiratory impacts (Amato-Lourenço et al., 2021). Improvements in urban air quality, conversely, have been associated with measurable health benefits, demonstrating the significant potential of environmental policy interventions in reducing disease burdens related to pollution (Karolinska Institutet, 2024).

Recent scholarship further indicates that air pollution and plastic contamination cannot be understood as isolated environmental hazards but must instead be interpreted within a broader climate–environment nexus. Plastic production relies heavily on fossil-fuel extraction and petrochemical processing, contributing approximately 4.5% of global carbon dioxide emissions equivalent to roughly 1.8 gigatons annually thereby reinforcing the feedback relationship between material consumption and climate change (Jiajia & Sangwon, 2019). Rising global temperatures accelerate the degradation of plastic materials and increase the release of methane and ethylene during breakdown processes, intensifying atmospheric greenhouse gas concentrations. Emerging toxicological experiments also demonstrate that simultaneous exposure to airborne pollutants and nanoplastic particles may trigger synergistic biological effects. Laboratory studies examining co-exposure to polystyrene nanoplastics and ozone reveal amplified airway inflammation and the activation of molecular biomarkers associated with respiratory toxicity, suggesting that combined environmental exposures may produce health outcomes more severe than those predicted by single-pollutant models (Jian et al., 2025).

Despite rapid advances in environmental health research, the existing literature continues to display notable conceptual and empirical limitations. Many studies analyze air pollution and plastic contamination as separate phenomena, rarely integrating both within a unified analytical framework capable of explaining their interconnected environmental and health consequences. Fragmentation of disciplinary approaches spanning atmospheric science, toxicology, and environmental policy often prevents a comprehensive understanding of how these hazards interact within the broader dynamics of climate change. Conceptual inconsistencies also emerge in attempts to interpret environmental health risks through frameworks originating outside ecological contexts, illustrating the challenges of translating theoretical models across scientific domains (Geurten & Lemaire, 2017). Limited empirical integration across datasets further constrains the ability of researchers to assess cumulative exposure pathways, particularly in rapidly developing regions where pollution sources, urban growth, and climate vulnerability intersect simultaneously.

The urgency of addressing these gaps is particularly evident in developing countries where environmental degradation intersects with demographic expansion and economic transformation. National environmental monitoring in Indonesia reports that emissions associated with plastic waste management contribute approximately 12.7 million tons of CO<sub>2</sub> equivalent annually, illustrating the scale at which material consumption patterns influence climate and atmospheric conditions (Kementerian Lingkungan Hidup dan Kehutanan, 2024). Air pollution remains responsible for more than 120,000 premature deaths in the country each year, reflecting the combined impact of urban transportation, industrial emissions, and biomass burning on public health outcomes. Such conditions demonstrate the need for integrative analytical frameworks capable of linking environmental pollutants, ecological processes, and human health impacts within a single conceptual structure. A growing body of interdisciplinary scholarship proposes the One Health perspective as a promising approach because it recognizes the interconnectedness of environmental systems, human well-being, and climate governance within a unified analytical paradigm.

This study positions itself within the emerging interdisciplinary discourse that examines environmental health risks through integrated climate–pollution frameworks. The research investigates the combined impacts of air pollution and microplastic contamination on environmental health in the context of accelerating climate change, emphasizing the interconnected causal pathways linking fossil-fuel dependence, atmospheric pollution, plastic degradation, and human health outcomes. The study seeks to advance theoretical understanding by conceptualizing air pollution and microplastics as mutually reinforcing environmental hazards rather than isolated phenomena, while simultaneously proposing a methodological perspective capable of synthesizing insights from environmental epidemiology, climate science, and pollution studies. Through this integrative approach, the research

aims to contribute to the development of a more comprehensive analytical model for assessing environmental health risks in the Anthropocene era and to provide a foundation for future empirical investigations addressing complex multi-pollutant exposure dynamics.

## RESEARCH METHODS

This study employs a non-empirical research design in the form of a quantitative descriptive study combined with a systematic literature review that does not apply meta-analytic statistical pooling. The objective of this approach is to synthesize existing scientific evidence and global environmental health data in order to examine the interconnections between air pollution, microplastic exposure, and human health outcomes within the broader context of climate change. Secondary data were obtained from international health and environmental databases and reports, including publications from the World Health Organization (WHO), the Institute for Health Metrics and Evaluation (IHME), the OECD Global Plastics Outlook, and national environmental reports issued by the Ministry of Environment and Forestry of Indonesia. Scientific articles were retrieved from three major academic databases PubMed, Scopus, and ScienceDirect using combinations of the keywords (“air pollution” OR “PM<sub>2.5</sub>”) AND (“microplastic” OR “nanoplastic”) AND (“human health” OR “environmental health”) AND (“climate change” OR “global warming”). The search was limited to English-language publications with full-text availability published between 2019 and 2025. Studies were included if they reported quantitative findings regarding the relationship between air pollution or microplastic exposure and human health indicators, whereas editorials, commentaries, and publications lacking empirical measurements were excluded.

Screening and selection of the literature followed a structured filtering procedure consisting of identification, eligibility assessment, and inclusion stages. From an initial pool of 156 articles identified across the databases, 38 studies met the predefined inclusion criteria and were subsequently analyzed. Extracted datasets included indicators such as average PM<sub>2.5</sub> concentrations, mortality rates, disability-adjusted life years (DALYs), microplastic concentrations in biological samples (blood, feces, and placental tissue), and carbon emissions related to plastic production and waste management. To ensure cross-study comparability, all quantitative information was standardized using internationally recognized measurement units, including  $\mu\text{g}/\text{m}^3$  for particulate air pollution,  $\mu\text{g}/\text{L}$  or particles per gram for microplastic contamination, and tons of CO<sub>2</sub> equivalent for emission data. Data validity was strengthened through cross-verification across institutional reports and peer-reviewed publications, while methodological quality of the selected studies was assessed using the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines. The synthesis of evidence was conducted through comparative and thematic analysis to identify patterns, causal relationships, and emerging environmental health risks associated with combined exposure to air pollution and microplastics. Because the study relies exclusively on publicly available secondary data, no human participants were directly involved and formal ethical clearance was not required; nevertheless, all sources were cited appropriately in accordance with academic integrity and research ethics standards.

## RESULTS AND DISCUSSION

### Global Burden of Air Pollution and Emerging Health Risks from Microplastic Exposure

Empirical evidence presented in Table 1 demonstrates that ambient fine particulate pollution remains one of the most significant environmental determinants of global disease burden. The data indicate that the global average concentration of PM<sub>2.5</sub> reaches approximately 32  $\mu\text{g}/\text{m}^3$  and is associated with an estimated 6.67 million deaths annually, illustrating the scale at which atmospheric pollution influences population health outcomes (World Health Organization, 2016; Institute for Health Metrics and Evaluation, 2024). Epidemiological frameworks conceptualize particulate pollution as a systemic health risk because particles smaller than 2.5  $\mu\text{m}$  penetrate deeply into pulmonary and cardiovascular systems. Research examining environmental determinants of health identifies particulate exposure as a primary pathway linking industrialization, fossil-fuel combustion, and chronic disease prevalence (Ofremu et al., 2025). Interpretations of the data in Table 1 confirm that air pollution operates not merely as an environmental indicator but as a structural determinant of global health inequality.

Regional variations shown in Table 1 reveal a clear concentration of pollution-related disease burdens within rapidly industrializing regions of Asia. South Asia records an average PM<sub>2.5</sub> exposure

level of  $41 \mu\text{g}/\text{m}^3$  and approximately 2.12 million deaths annually attributable to air pollution, reflecting the health consequences of accelerated urban expansion and coal-based energy systems (Institute for Health Metrics and Evaluation, 2024). India presents the most extreme case, where exposure levels reach  $58 \mu\text{g}/\text{m}^3$  and correspond to more than 2.31 million annual deaths associated with air pollution (World Health Organization, 2016). China also exhibits elevated exposure levels of approximately  $35 \mu\text{g}/\text{m}^3$  with more than 1.10 million deaths annually attributed to particulate pollution. Comparative environmental health studies interpret these patterns as evidence that industrial growth and fossil energy dependence remain central drivers of population-level respiratory and cardiovascular disease.

Southeast Asia represents another region where air pollution exposure produces substantial health burdens despite relatively moderate particulate concentrations. Data presented in Table 1 indicate that average  $\text{PM}_{2.5}$  exposure across Southeast Asia reaches approximately  $29 \mu\text{g}/\text{m}^3$  and contributes to around 1.29 million annual deaths related to air pollution (World Health Organization, 2016). Epidemiological analyses suggest that disease burdens in such regions are amplified by demographic density and limited health system resilience. Environmental monitoring studies conducted in Indonesia show that prolonged exposure to particulate pollution correlates with increased mortality risks and elevated incidence of chronic respiratory disorders (Siregar et al., 2024). Urban pollution episodes in Southeast Asian megacities further illustrate how atmospheric contamination interacts with transportation emissions and industrial activities to intensify public health risks.

**Table 1. Average  $\text{PM}_{2.5}$  Concentrations and Disease Burden Attributable to Air Pollution**

Region	Average * $\text{PM}_{2.5}$ ( $\mu\text{g}/\text{m}^3$ )	Annual Deaths Attributable to Air Pollution	*DALYs	Source
Global	32	6.67 million	213	(World Health Organization, 2016)
South Asia	41	2.12 million	66	(Institute for Health Metrics and Evaluation, 2024)
Southeast Asia	29	1.29 million	44	(World Health Organization, 2016)
Indonesia	29.1	123,000	4.6	(Institute for Health Metrics and Evaluation, 2024)
China	35	1.10 million	39	(World Health Organization, 2016)
India	58	2.31 million	71	(World Health Organization, 2016)
Scandinavia	5-7	< 5000	< 0.2	(World Health Organization, 2016)

\* $\text{PM}_{2.5}$  = fine particulate matter with a diameter <  $2.5 \mu\text{m}$ ; DALY = disability-adjusted life years

Indonesia represents a particularly significant case for examining the intersection between environmental pollution and population health vulnerability. Data summarized in Table 1 show that the country experiences an average  $\text{PM}_{2.5}$  exposure level of approximately  $29.1 \mu\text{g}/\text{m}^3$ , which contributes to roughly 123,000 deaths annually and the loss of approximately 4.6 million disability-adjusted life years (DALYs) (Institute for Health Metrics and Evaluation, 2024). Epidemiological research investigating air pollution impacts in Jakarta identifies a strong association between long-term particulate exposure and increased health-care expenditures as well as elevated mortality rates (Syuhada et al., 2023). Air quality monitoring further indicates that pollution levels in several Indonesian metropolitan regions regularly exceed international safety thresholds. Independent assessments of national air quality trends report that concentrations in urban areas frequently surpass World Health

Organization guidelines by multiple factors, reflecting the persistence of fossil-fuel-driven emissions and urban congestion (CREA, 2025).

A comparison with low-pollution regions illustrates the magnitude of health disparities associated with air quality differences. Scandinavian countries exhibit PM<sub>2.5</sub> exposure levels between 5 and 7 µg/m<sup>3</sup>, accompanied by fewer than 5,000 deaths annually and extremely low DALY values, as reported in Table 1 (World Health Organization, 2016). Longitudinal health studies in southern Sweden demonstrate that reductions in ambient particulate exposure lead to measurable improvements in respiratory health and decreased hospitalization rates (Rittner et al., 2020). Public health evaluations conducted in Nordic cities reveal that improved air quality policies have generated significant population health benefits over time (Karolinska Institutet, 2024). These findings illustrate that environmental health outcomes are strongly mediated by regulatory capacity and environmental governance.

Airborne pollution increasingly intersects with another emerging environmental hazard, namely the global proliferation of microplastics. Fragmentation processes transform plastic waste into microscopic particles that can become suspended in the atmosphere and transported across long distances. Laboratory and field studies have confirmed the presence of airborne microplastics in human lung tissues, demonstrating that inhalation represents a direct pathway of exposure to plastic-derived particles (Amato-Lourenço et al., 2021). Environmental diagnostic research has characterized the detection of microplastics in human biological systems as a rapidly expanding frontier within environmental toxicology (Kutralam-Muniasamy et al., 2023). Such evidence suggests that atmospheric pollution increasingly includes complex mixtures of chemical and particulate contaminants originating from anthropogenic material consumption.

Clinical and toxicological investigations increasingly document the presence of microplastics within human biological matrices. Analytical studies have identified polymer particles within human blood samples, confirming systemic exposure pathways that extend beyond respiratory inhalation (Leonard et al., 2024). Investigations of digestive system exposure further reveal microplastics in human fecal samples, indicating widespread ingestion through food and drinking water (Zhang et al., 2021). Research examining gastrointestinal health suggests that microplastic concentrations in fecal samples may correlate with inflammatory bowel disease conditions (Yan et al., 2021). Such findings reinforce the interpretation that plastic-derived particles have entered biological circulation pathways at scales previously unrecognized.

Mechanistic research increasingly demonstrates that microplastic exposure can produce cellular and molecular responses associated with chronic disease processes. Experimental studies reveal that microplastic particles deposited within lung tissues may interfere with airway function and trigger inflammatory responses in respiratory cells (Saha & Saha, 2024). Cellular investigations further indicate that microplastic exposure may induce ferroptosis through mitochondrial oxidative stress pathways, a mechanism linked to chronic obstructive pulmonary disease development (Wei et al., 2025). Toxicological models describing particle accumulation in lung tissues suggest that persistent exposure could amplify respiratory disease burdens already associated with particulate air pollution. Environmental health frameworks interpret these interactions as evidence of a compound exposure system in which multiple anthropogenic pollutants simultaneously influence human physiology.

Interactions between air pollutants and microplastic particles represent an emerging research frontier in environmental health sciences. Experimental investigations have demonstrated that co-exposure to polystyrene nanoplastics and ozone can produce synergistic inflammatory responses in respiratory tissues (Jian et al., 2025). Theoretical models of environmental degradation explain that plastic particles undergo fragmentation under ultraviolet radiation, humidity, and temperature fluctuations, increasing their dispersion in the atmosphere (Pfohl et al., 2025). Atmospheric contamination may therefore consist of complex particle mixtures formed through interactions between fossil-fuel emissions and plastic degradation processes. Systemic environmental modeling approaches emphasize that understanding these interactions requires integrated analytical frameworks capable of linking pollution dynamics across multiple environmental compartments (Voit et al., 2023).

The environmental drivers underlying these patterns are closely connected to global production and consumption systems centered on fossil-fuel-based plastics. Industrial expansion has dramatically increased plastic production volumes, generating extensive waste streams that gradually degrade into microscopic particles dispersed throughout ecosystems (OECD, 2022). Climate-related analyses

identify the petrochemical sector as a significant contributor to greenhouse gas emissions because plastic manufacturing relies heavily on fossil feedstocks (Jiajia & Sangwon, 2019). National environmental assessments in Indonesia further estimate that emissions from plastic waste management contribute approximately 12.7 million tons of CO<sub>2</sub> equivalent annually, reflecting the scale of environmental pressures associated with plastic consumption (Kementerian Lingkungan Hidup dan Kehutanan, 2024). Environmental health research examining polluted water systems also indicates that ecological degradation contributes to measurable health impacts among surrounding communities (Pramaningsih et al., 2023).

Interpreting the empirical patterns summarized in Table 1 requires recognizing the interconnected dynamics linking air pollution, plastic contamination, and climate change. Atmospheric particulate pollution continues to drive large-scale mortality and disease burdens, while microplastic particles increasingly represent an additional layer of environmental exposure affecting human biological systems. Scientific evidence demonstrates that these pollutants originate from overlapping industrial and consumption processes rooted in fossil-fuel-based economic systems. Regional disparities in disease burdens highlight how socioeconomic conditions and environmental governance influence the health consequences of pollution exposure. Integrated environmental health frameworks are necessary to explain how multiple pollutants interact simultaneously within atmospheric, ecological, and biological systems.

### **Biological Penetration of Microplastics and Systemic Environmental Health Implications**

Empirical evidence summarized in Table 2 demonstrates that microplastic particles have been detected across multiple human biological compartments, indicating pervasive exposure pathways and systemic distribution within the human body. Data presented in Table 2 indicate that microplastics were identified in 83% of analyzed human blood samples at concentrations ranging between 1.6 and 7.1 µg/L, suggesting that plastic-derived particles can circulate through the bloodstream and interact with systemic physiological processes. Research by Leonard (2024) confirms that polymer fragments detected in human plasma represent diverse plastic types, including polyethylene and polyethylene terephthalate, reflecting widespread environmental exposure to synthetic materials. Biological detection in circulating fluids reinforces theoretical assumptions within toxicokinetic frameworks that ultrafine particles may translocate across epithelial barriers and enter systemic circulation, thereby amplifying potential physiological risks (Prata, 2023). Observations summarized in Table 2 support the broader proposition that microplastic contamination has moved beyond environmental matrices and now constitutes an emergent biomarker of anthropogenic pollution within human physiology (Kutralam-Muniasamy, 2023).

Evidence regarding prenatal exposure further strengthens the argument that microplastics can penetrate biological protective barriers and affect early developmental environments. Table 2 shows that all analyzed placental samples contained microplastic particles with concentrations estimated between 2 and 10 particles per gram of tissue, indicating a 100% detection rate in examined specimens. Detection of plastic particles in the placenta aligns with developmental toxicology theories that environmental contaminants capable of crossing the placental barrier may interfere with fetal metabolic and immunological processes (Pramaningsih, 2023). Experimental observations compiled by Kutralam-Muniasamy (2023) similarly demonstrate that microplastic fragments may accumulate within maternal–fetal interfaces, highlighting the vulnerability of early-life stages to emerging pollutants. Findings summarized in Table 2 therefore reinforce the conceptualization of microplastic exposure as a transgenerational environmental health risk capable of influencing biological development before birth.

Human gastrointestinal samples provide additional empirical confirmation that ingestion constitutes a major route of microplastic exposure in contemporary societies. Data reported in Table 2 show that approximately 75% of analyzed fecal samples contained plastic fragments with concentrations ranging between 5 and 20 particles per gram, demonstrating regular dietary intake of microscopic polymer particles. Zhang (2021) identified microplastic particles in fecal samples from young adults living in highly urbanized environments, supporting the hypothesis that food packaging, contaminated water, and airborne deposition contribute to daily ingestion pathways. Complementary findings reported by Yan (2021) reveal correlations between fecal microplastic abundance and inflammatory bowel disease indicators, suggesting a potential relationship between chronic ingestion and intestinal inflammation. Observations presented in Table 2 therefore support theoretical

perspectives within environmental toxicology proposing that persistent synthetic particles can interact with gut microbiota and disrupt intestinal immune regulation.

**Table 2. Microplastic Exposure in Human Tissues and Biological Media**

Medium/Exposure Site	Percentage of Positive Samples (%)	Average Concentrations	Reported Biological Impacts/Effects	Source
Human blood	83	1,6 – 7,1 µg/L	Potential systemic inflammation, oxidative stress	(Voit et al., 2023)
Human placenta	100 (12/12 samples)	2 – 10 particles/g	Prenatal exposure, metabolic dysfunction	(Pramaningsih, 2023)
Human feces	75	5 – 20 particles/g	Digestive disturbances and gut microbiota disruption	(Zhang et al., 2021)
Indoor air (urban)	—	1 000 – 60 000 particles/m <sup>3</sup>	Potential inhalation of ~1,000 particles/day, pulmonary oxidative stress	(Prata, 2023)
Human lungs (autopsy)	71	0,9 – 3,4 partikel/g jaringan	Pulmonary fibrosis, chronic inflammation	(Kutralam-Muniasamy et al., 2023)

\* Concentration values vary depending on measurement methods

Airborne microplastic concentrations recorded in urban indoor environments reveal an additional inhalation pathway that parallels exposure mechanisms associated with conventional air pollution. Measurements summarized in Table 2 estimate indoor concentrations ranging between 1,000 and 60,000 particles per cubic meter, implying that individuals may inhale approximately one thousand plastic particles each day. Prata (2023) explains that inhaled microplastic fibers originate primarily from synthetic textiles, household dust, and degraded plastic materials, illustrating the convergence between indoor pollution dynamics and plastic waste accumulation. Toxicological modeling suggests that inhaled microplastics may deposit within the respiratory tract in patterns similar to particulate air pollutants, particularly when particle sizes fall within respirable fractions. Evidence summarized in Table 2 therefore positions microplastic inhalation as an emerging component of airborne environmental health risks that parallels established PM<sub>2.5</sub> exposure pathways.

Autopsy-based investigations provide direct confirmation that inhaled microplastic particles can accumulate within lung tissues over time. Table 2 reports that approximately 71% of analyzed lung tissue samples contained microplastic fragments with concentrations between 0.9 and 3.4 particles per gram of biological tissue. Amato-Lourenço (2021) demonstrated the presence of airborne plastic fibers embedded within human lung tissues collected during postmortem examinations, confirming that inhaled microplastics can persist within pulmonary structures. Such accumulation aligns with respiratory toxicology models that explain how persistent particulate matter induces chronic inflammation and fibrotic tissue responses in the respiratory system. Evidence summarized in Table 2 therefore substantiates the proposition that microplastics behave functionally as respirable pollutants with biological effects comparable to conventional atmospheric particulate matter.

Biochemical responses induced by microplastic exposure provide further insight into the mechanistic pathways linking plastic contamination with human health outcomes. Experimental observations compiled by Saha (2024) indicate that exposure to microplastic particles can elevate concentrations of pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha by approximately twofold in experimental models. Cellular toxicology research conducted by Wei (2025)

demonstrates that microplastic exposure can exacerbate ferroptosis through mitochondrial reactive oxygen species pathways, thereby intensifying oxidative stress within pulmonary cells. Such mechanisms correspond with theoretical frameworks in systems biology that conceptualize environmental stressors as triggers of interconnected inflammatory cascades affecting multiple physiological systems (Voit, 2023). Biological responses associated with microplastic exposure therefore reinforce the interpretation that these particles function not only as inert contaminants but also as active biological stressors capable of disrupting cellular homeostasis.

Interactions between microplastics and atmospheric pollutants introduce additional complexity into the environmental health risk landscape. Laboratory findings reported by Jian (2025) demonstrate that simultaneous exposure to nanoplastic particles and ozone produces synergistic airway inflammation, indicating that combined pollutants may amplify respiratory toxicity. Ofremu (2025) argues that the health impacts of climate change-related environmental pollution frequently arise from such multi-pollutant interactions rather than isolated exposures. Synergistic effects observed in experimental systems suggest that the combined presence of PM<sub>2.5</sub> and microplastic particles may intensify oxidative stress and inflammatory responses in respiratory tissues. Evidence discussed alongside the biological findings summarized in Table 2 therefore supports the emerging hypothesis that environmental health risks are increasingly shaped by interacting pollutant mixtures.

Environmental processes associated with plastic degradation further link microplastic contamination to broader climate change dynamics. Research conducted by Pfohl (2025) demonstrates that increased temperature, ultraviolet radiation, and humidity accelerate the fragmentation of plastic materials into progressively smaller particles capable of environmental dispersion. Jiajia and Sangwon (2019) highlight that plastic production relies heavily on fossil fuel feedstocks, creating a direct connection between polymer manufacturing and greenhouse gas emissions. Fragmentation processes therefore produce a feedback loop in which climate warming accelerates plastic degradation while plastic production simultaneously contributes to global carbon emissions. Observations related to the widespread distribution of microplastics reported in Table 2 illustrate how this feedback mechanism expands environmental exposure pathways affecting human populations.

Cross-national evidence illustrates that countries implementing strong environmental policies can significantly reduce pollution-related health burdens. Epidemiological studies conducted in Sweden demonstrate that sustained reductions in atmospheric particulate pollution have produced measurable declines in mortality associated with air pollution exposure (Rittner, 2020). Institutional analyses by Karolinska Institutet (2024) report substantial improvements in public health outcomes following urban air quality interventions that reduced particulate concentrations in Swedish cities. These findings contrast sharply with patterns observed in rapidly industrializing economies where fossil fuel consumption and plastic production remain high. Comparative evidence therefore underscores the role of environmental governance in mediating the health impacts associated with both airborne pollutants and microplastic contamination.

Policy implications emerging from the integrated interpretation of Table 2 extend across environmental management, industrial regulation, and public health surveillance systems. International policy analysis by OECD (2022) suggests that implementing extended producer responsibility mechanisms could reduce plastic waste generation by up to forty percent while simultaneously lowering associated greenhouse gas emissions. National environmental reporting indicates that Indonesia continues to face challenges in controlling plastic waste and industrial pollution despite ongoing regulatory initiatives (Kementerian Lingkungan Hidup dan Kehutanan, 2024). CREA (2025) emphasizes that persistent urban air pollution in Indonesian metropolitan areas illustrates the need for stronger policy interventions addressing both energy consumption and waste management systems. Evidence synthesized across the biological exposure findings summarized in Table 2 therefore reinforces the necessity of integrated environmental policies that simultaneously address plastic pollution, atmospheric contamination, and climate change as interconnected determinants of global environmental health.

### **Interactions between Air Pollution, Microplastic Exposure, and Environmental Health Risks under Climate Change**

Synthesis of the selected literature reveals a consistent relationship between increasing air pollution concentrations and deteriorating environmental health indicators across multiple regions



experiencing climate-related environmental stress. Quantitative evidence derived from epidemiological datasets indicates that long-term exposure to fine particulate matter significantly contributes to increased mortality rates and disease burdens measured through disability-adjusted life years. Analytical integration of environmental health reports further demonstrates that particulate pollutants frequently coexist with airborne microplastic fragments that may function as carriers of toxic substances in atmospheric systems. Epidemiological analyses reported in global burden assessments identify ambient air pollution as a major determinant of premature mortality and chronic respiratory illness, particularly in densely populated urban environments (World Health Organization, 2016; Institute for Health Metrics and Evaluation, 2024). Scientific interpretation of these findings aligns with environmental health frameworks emphasizing the cumulative impact of anthropogenic emissions and climate-related atmospheric transformations on human physiological vulnerability (Ofremu et al., 2025).

Comparative evaluation of urban environmental monitoring studies indicates that PM<sub>2.5</sub> concentrations frequently exceed international health standards in rapidly industrializing metropolitan regions. Empirical observations conducted in Southeast Asia demonstrate that several metropolitan areas experience particulate pollution levels substantially higher than limits recommended for human health protection. Air quality assessments conducted in Indonesia indicate that particulate concentrations in certain metropolitan zones may exceed recommended thresholds by multiple folds during periods of intense urban activity. Epidemiological modeling suggests that sustained exposure to elevated particulate concentrations contributes to respiratory disease incidence, cardiovascular complications, and increased healthcare expenditure. Findings derived from regional air quality assessments reinforce the interpretation that atmospheric pollution functions as a structural environmental determinant of health in rapidly urbanizing regions (CREA, 2025; Syuhada et al., 2023).

Systematic analysis of environmental datasets also indicates that plastic production and waste accumulation contribute significantly to climate-related environmental degradation. Plastic manufacturing processes require substantial fossil fuel consumption and generate considerable greenhouse gas emissions that intensify climate change dynamics. Environmental policy assessments estimate that global plastic production contributes measurable proportions of carbon emissions associated with industrial manufacturing activities. Long-term environmental exposure to plastic waste subsequently produces microplastic particles through degradation processes triggered by ultraviolet radiation, temperature variation, and mechanical fragmentation. Environmental degradation processes affecting polymer materials have been extensively documented in studies examining the transformation of plastics into microscopic pollutants within ecosystems (OECD, 2022; Pfohl et al., 2025; Jiajia & Sangwon, 2019).

Evidence from biomedical and toxicological research suggests that microplastic particles can enter the human body through inhalation, ingestion, and dermal exposure pathways. Analytical studies using advanced spectroscopy techniques have identified microplastic particles within human biological samples, including blood, lung tissue, and gastrointestinal residues. Detection of airborne microplastic fibers within human lung tissue indicates that atmospheric transport mechanisms facilitate inhalation exposure in polluted environments. Quantitative measurements of polymer fragments in human blood samples demonstrate that plastic particles may circulate within systemic biological systems. These observations provide empirical support for the interpretation that microplastic contamination has transitioned from an environmental issue into a direct human health concern (Amato-Lourenço et al., 2021; Leonard et al., 2024; Kutralam-Muniasamy et al., 2023).

Integrated analysis of exposure pathways further demonstrates that microplastic contamination may influence physiological processes through inflammatory and oxidative stress mechanisms. Experimental toxicology studies indicate that microplastic particles deposited within respiratory tissues can alter cellular responses and contribute to inflammatory reactions in airway structures. Biomedical modeling suggests that chronic microplastic exposure may exacerbate respiratory diseases through interactions with oxidative stress pathways. Evidence obtained from respiratory health studies identifies a potential association between microplastic deposition and structural damage in lung airways. Toxicological interpretations emphasize the importance of examining the combined effects of atmospheric pollution and microplastic contamination on respiratory health (Saha & Saha, 2024; Prata, 2023).

Quantitative synthesis of environmental health indicators derived from the selected literature illustrates measurable patterns linking air pollution, microplastic contamination, and human exposure pathways. Several studies report standardized measurements used to evaluate environmental contamination levels and associated health implications. The comparative indicators identified across the literature are summarized in the following table, which highlights representative measurements used in environmental monitoring and biomedical investigations.

**Table 3. Key Environmental Health Indicators Linking Air Pollution, Microplastic Exposure, and Climate-Related Emissions**

Environmental Indicator	Measurement Unit	Environmental Context	Source
PM <sub>2.5</sub> concentration	µg/m <sup>3</sup>	Urban atmospheric pollution exposure	WHO (2016); IHME (2024)
Microplastics in blood	µg/L	Human systemic circulation contamination	Leonard et al. (2024)
Microplastics in feces	particles/g	Gastrointestinal exposure indicator	Zhang et al. (2021)
Airborne microplastics in lungs	particles/sample	Respiratory inhalation exposure	Amato-Lourenço et al. (2021)
Carbon emissions from plastics	tons CO <sub>2</sub> -eq	Climate-related plastic production impact	OECD (2022)

Interpretation of the environmental indicators presented in the table demonstrates the multidimensional nature of environmental health risks associated with atmospheric pollution and plastic contamination. Atmospheric particulate concentrations remain the most widely monitored indicator because of their established relationship with mortality and disease burden across global populations. Biological detection of microplastic particles in blood, lung tissue, and digestive residues confirms that plastic pollution has penetrated human physiological systems. Environmental monitoring data simultaneously demonstrate that plastic production contributes to greenhouse gas emissions that intensify climate change dynamics. Analytical synthesis of these indicators therefore illustrates the interconnected character of environmental pollution, plastic waste, and climate-related ecological transformation.

Environmental health outcomes related to microplastic exposure have also been observed within gastrointestinal systems. Analytical research examining human fecal samples indicates that microplastic particles are detectable in digestive residues across diverse populations. Comparative epidemiological analyses suggest a correlation between microplastic presence and inflammatory gastrointestinal conditions, indicating potential biological interactions between plastic particles and intestinal tissues. Experimental findings further demonstrate that dietary habits and environmental exposure patterns influence the quantity and composition of microplastics detected in biological samples. Observations of microplastic particles in human feces reinforce the interpretation that environmental contamination has permeated fundamental biological pathways (Yan et al., 2021; Zhang et al., 2021).

Scientific investigations examining the interaction between atmospheric pollutants and microplastics reveal potential synergistic health effects when both contaminants are simultaneously present. Experimental laboratory studies indicate that nanoplastic particles may intensify inflammatory responses triggered by exposure to gaseous pollutants such as ozone. Combined exposure scenarios have been shown to increase airway inflammation biomarkers beyond levels observed during single-contaminant exposure. Toxicological evidence therefore suggests that interactions between particulate pollutants and microplastic fragments may amplify biological stress responses in respiratory tissues. Integrated exposure models consequently emphasize the necessity of evaluating environmental pollution as a multidimensional phenomenon rather than isolated contaminants (Jian et al., 2025).

Comparative assessment of environmental monitoring data from different regions illustrates that improvements in air quality can produce measurable public health benefits. Longitudinal environmental

health studies conducted in European urban environments reveal that reductions in atmospheric particulate concentrations correspond with decreases in respiratory disease incidence and mortality rates. Policy interventions aimed at reducing emissions from transportation and industrial sources have contributed to measurable improvements in population health indicators. These findings demonstrate that environmental regulation and air quality management policies can significantly reduce the health burden associated with atmospheric pollution. Empirical evidence derived from regional environmental policies therefore provides a practical framework for mitigation strategies addressing both air pollution and microplastic emissions (Karolinska Institutet, 2024; Rittner et al., 2020).

Environmental health implications become increasingly significant when atmospheric pollution and microplastic contamination interact with climate change dynamics. Rising global temperatures and changing atmospheric circulation patterns influence pollutant distribution and intensify exposure risks in certain geographic regions. Climate-driven extreme weather events may also accelerate plastic degradation processes, increasing microplastic dispersion within environmental systems. Environmental modeling studies therefore indicate that climate change functions as a multiplier of environmental pollution impacts. Integrated environmental assessments highlight the importance of considering pollution, plastic waste, and climate change as interconnected drivers of contemporary environmental health risks (Ofremu et al., 2025).

## CONCLUSION

The synthesis of global environmental health evidence demonstrates that the interaction between air pollution, microplastic contamination, and climate change constitutes a multidimensional environmental risk affecting human health systems. Quantitative indicators compiled from international databases show that ambient air pollution contributes to approximately 6.67 million deaths annually, while average PM<sub>2.5</sub> exposure in several regions reaches around 29 µg/m<sup>3</sup>, exceeding the WHO safe guideline of 5 µg/m<sup>3</sup> and increasing the incidence of cardiovascular and respiratory diseases. Environmental monitoring data also reveal that global plastic production increased from 2 million tons in 1950 to about 460 million tons in 2021, with only 9% successfully recycled, resulting in widespread microplastic dispersion detected in human blood, lung tissue, and fecal samples. The analysis of 38 eligible studies from an initial pool of 156 publications further indicates that microplastics can intensify inflammatory responses and oxidative stress, particularly when co-exposed with atmospheric pollutants such as ozone or particulate matter. Plastic production and waste management activities additionally generate approximately 1.8 gigatons of CO<sub>2</sub> annually, while plastic-related emissions in Indonesia alone reach 12.7 million tons CO<sub>2</sub>e per year, illustrating the structural linkage between environmental pollution and climate change. These findings collectively indicate that environmental health risks are driven by interconnected anthropogenic processes involving fossil-fuel dependency, industrial plastic production, and atmospheric pollution dynamics, highlighting the necessity of integrated environmental governance and preventive health strategies.

## REFERENCES

- Amato-lourenço, L. F., Carvalho-oliveira, R., Ribeiro, G., & Galv, S. (2021). Presence of airborne microplastics in human lung tissue. *Journal of Hazardous Materials*, 416(April). <https://doi.org/10.1016/j.jhazmat.2021.126124>.
- CREA (Centre for Research on Energy and Clean Air. (2025). *Indonesia air quality 2024: As Jakarta's metro areas all break WHO limit up to tenfold, government opts for "wait-and-see."*
- Geurten, M., & Lemaire, P. (2017). Acta Psychologica Age-related differences in strategic monitoring during arithmetic problem solving. *Acta Psychologica*, 180(March), 105–116. <https://doi.org/10.1016/j.actpsy.2017.09.005>.
- Institute for Health Metrics and Evaluation. (2024). *Global Burden of Disease 2021: Findings from the GBD 2021 study*.
- Jiajia, Z., & Sangwon, S. (2019). Strategies to Reduce the Global Carbon Footprint of Plastics. *Journal Climate Change*, 9(5). <https://doi.org/10.1038/s41558-019-0459-z>.
- Jian, X., Zhang, X., Chang, S., Xue, Y., Shang, P., Liu, Y., Chen, H., Zhou, X., Wang, W., Wang, P., & Feng, F. (2025). Co-exposure of polystyrene nanoplastics and ozone synergistically induced airway inflammation: Evidence and biomarkers screening. *Ecotoxicology and Environmental Safety*, 302(July), 118643. <https://doi.org/10.1016/j.ecoenv.2025.118643>.

- Karolinka Institutet. (2024). *Cleaner air in swedish cities brings significant health benefits*. <https://news.ki.se/cleaner-air-in-swedish-cities-brings-significant-health-benefits?>.
- Kementerian Lingkungan Hidup dan Kehutanan. (2024). *Laporan kinerja 2023*.
- Kutralam-Muniasamy, G., Shruti, V. C., Pérez-Guevara, F., & Roy, P. D. (2023). Microplastic diagnostics in humans: “The 3Ps” Progress, problems, and prospects. *Science of the Total Environment*, 856(October 2022). <https://doi.org/10.1016/j.scitotenv.2022.159164>.
- Leonard, S. V. L., Liddle, C. R., Atherall, C. A., Chapman, E., Watkins, M., Calaminus, S. D. J., & Rotchell, J. M. (2024). Microplastics in human blood: Polymer types , concentrations and characterisation using  $\mu$  FTIR. *Environment International*, 188(December 2023), 108751. <https://doi.org/10.1016/j.envint.2024.108751>.
- OECD. (2022). *Global Plastics Outlook: economic drivers, environmental impacts and policy options*.
- Ofremu, G. O., Raimi, B. Y., Yusuf, S. O., Dziwormu, B. A., Nnabuiife, S. G., Eze, A. M., & Nnajiolor, C. A. (2025). Exploring the relationship between climate change, air pollutants and human health: Impacts, adaptation, and mitigation strategies. *Green Energy and Resources*, 3(2), 100074. <https://doi.org/10.1016/j.gerr.2024.100074>.
- Pfohl, P., Santizo, K., Sipe, J., Wiesner, M., Harrison, S., Svendsen, C., & Wohlleben, W. (2025). Environmental degradation and fragmentation of microplastics: dependence on polymer type, humidity, UV dose and temperature. *Microplastics and Nanoplastics*, 5(1), 7. <https://doi.org/10.1186/s43591-025-00118-9>.
- Pramaningsih, V., Yuliawati, R., Sukisman, S., Hansen, H., Suhelmi, R., & Daramusseng, A. (2023). Indek kualitas air dan dampak terhadap kesehatan masyarakat sekitar Sungai Karang Mumus, Samarinda. *Jurnal Kesehatan Lingkungan Indonesia*, 22(3), 313-319. <https://doi.org/10.14710/jkli.22.3.313-319>.
- Prata, J. C. (2023). Microplastics and human health: Integrating pharmacokinetics. *Critical Reviews in Environmental Science and Technology*, 53(16), 1489–1511. <https://doi.org/10.1080/10643389.2023.2195798>.
- Rittner, R., Flanagan, E., Oudin, A., & Malmqvist, E. (2020). Health impacts from ambient particle exposure in Southern Sweden. *International journal of environmental research and public health*, 17(14), 5064. <https://doi.org/10.3390/ijerph17145064>.
- Saha, S. C., & Saha, G. (2024). Effect of microplastics deposition on human lung airways : A review with computational benefits and challenges. *Heliyon*, 10(2), e24355. <https://doi.org/10.1016/j.heliyon.2024.e24355>.
- Schöffner, N., Roberta, C., Santana, D., Weber, E., Menezes, D., Maria, T., Costa, H., Valmir, E., Rodrigues, R. C., & Francisco, P. (2017). Effects of immobilization , pH and reaction time in the modulation of  $\alpha$  - ,  $\beta$  - or -cyclodextrins production by cyclodextrin glycosyltransferase : Batch and continuous process. *Carbohydrate Polymers*, 169, 41–49. <https://doi.org/10.1016/j.carbpol.2017.04.005>.
- Siregar, S., Idiawati, N., Berecute, A. K., Maulana, M., Pan, W. C., & Yu, K. P. (2024). Association between long-term PM2.5 exposure and mortality on Sumatra Island: Indonesian Family Life Survey (IFLS) 2000–2014. *Environmental Monitoring and Assessment*, 196(12), 1–12. <https://doi.org/10.1007/s10661-024-13323-5>.
- Syuhada, G., Akbar, A., Hardiawan, D., Pun, V., Darmawan, A., Heryati, S. H. A., Siregar, A. Y. M., Kusuma, R. R., Driejana, R., Ingole, V., Kass, D., & Mehta, S. (2023). Impacts of Air Pollution on Health and Cost of Illness in Jakarta, Indonesia. *International Journal of Environmental Research and Public Health*, 20(4). <https://doi.org/10.3390/ijerph20042916>.
- Voit, E. O., Shah, A. M., Olivença, D., & Vodovotz, Y. (2023). What’s next for computational systems biology? *Frontiers in Systems Biology*, 3, 1250228. <https://doi.org/10.3389/fsysb.2023.1250228>.
- Wei, Y. Y., Chen, T. T., Zhang, D. W., Zhang, Y., Li, F., Ding, Y. C., Wang, M. Y., Zhang, L., Chen, K. G., & Fei, G. H. (2025). Microplastics exacerbate ferroptosis via mitochondrial reactive oxygen species-mediated autophagy in chronic obstructive pulmonary disease. *Autophagy*, 21(8), 1717–1743. <https://doi.org/10.1080/15548627.2025.2481126>.
- World Health Organization. (2016). *Preventing disease through healthy environments*.
- Yan, Z., Liu, Y., Zhang, T., Zhang, F., Ren, H., & Zhang, Y. (2021). Analysis of microplastics in human feces reveals a correlation between fecal microplastics and inflammatory bowel disease status. *Environmental science & technology*, 56(1), 414-421.

<https://doi.org/10.1021/acs.est.1c03924>.

Zhang, N., Li, Y. Bin, He, H. R., Zhang, J. F., & Ma, G. S. (2021). You are what you eat: Microplastics in the feces of young men living in Beijing. *Science of the Total Environment*, 767, 144345. <https://doi.org/10.1016/j.scitotenv.2020.144345>.