ISSN Print: 2664-7591
ISSN Online: 2664-7605
Impact Factor: RJIF 8-16
IJPCR 2026; 8(1): 01-04
www.pharmaceuticaljournal.in
Received: 02-10-2025
Accepted: 05-11-2025

Uzowuru Daniel I
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Ebeh-Njoku Augusta U
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Eze Rita
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Nwankudu Chinyere J
Department of Botany, Faculty of Science, Lagos State University, Ojo, Nigeria

Raji Funmilayo K
Department of Botany, Faculty of Science, Lagos State University, Ojo, Nigeria

Nwanjo SO
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Onodu WU
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Corresponding Author:
Uzowuru Daniel I
Department of Animal and Environmental Biology (Zoology), Faculty of Biological Science, Imo State University, Owerri, Nigeria

Physiological impacts of aerosols on human health among adult individuals of Owerri, imo state Nigeria between January 2024 and October 2025

Uzowuru Daniel I, Ebeh-Njoku Augusta U, Eze Rita, Nwankudu Chinyere J, Raji Funmilayo K, Nwanjo SO and Onodu WU

DOI: https://doi.org/

Abstract
This study examined the physiological impacts of aerosols on adult individuals in Owerri, Imo State, Nigeria, between January 2024 and October 2025, using a cross-sectional field research design. Aerosol samples were collected across diverse urban environments to capture variations in particle size, chemical composition, and acidity. The characteristics of aerosols were systematically analyzed, and their potential physiological relevance, including deep lung deposition, oxidative stress induction, and tissue irritation, was assessed. Mechanistic pathways such as inflammatory responses, vascular dysregulation, genotoxic effects, and epigenetic modifications were evaluated, alongside health outcomes in respiratory, cardiovascular, metabolic, and neurological systems. Analysis of 150 samples indicated that fine particles ($PM_{2.5}$) were most prevalent (45.3%), followed by ultrafine particles (<0.1 µm, 21.3%), acidic aerosols (16.7%), coarse particles (PM_{10}, 10.0%), and high-metal aerosols (6.7%), with corresponding physiological effects including alveolar inflammation, oxidative stress, tissue irritation, and cardiovascular stress. Mechanistically, oxidative stress (46.7%) and inflammatory responses (23.3%) were the dominant pathways, while vascular dysregulation (14.7%), DNA/genotoxic damage (10.0%), and epigenetic modification (5.3%) were less frequent. Respiratory diseases were the most common health outcome (64.0%), followed by cardiovascular (22.0%), systemic/metabolic (8.0%), and neurological effects (6.0%). These findings highlight the significant burden of aerosol exposure on pulmonary and systemic health, underscoring the need for targeted interventions and public health strategies to mitigate exposure in urban environments.

Keywords: Aerosols, $PM_{2.5}$, ultrafine particles, oxidative stress, inflammation

Introduction
Atmospheric aerosols, defined as suspensions of fine solid or liquid particles in air, are ubiquitous and significantly influence environmental chemistry, climate regulation, and human health (Baron, 2004) [1]. These particles originate from natural sources, including volcanic eruptions, dust storms, and sea spray, as well as anthropogenic activities such as combustion, industrial emissions, and vehicular exhaust (Pöschl, 2005) [19]. The physicochemical properties of aerosols—size, composition, and acidity—determine both their atmospheric behavior and biological reactivity upon inhalation, making aerosol science a multidisciplinary field spanning atmospheric chemistry, toxicology, and public health (Kelly and Fussell, 2012) [13]. Globally, particulate air pollution is a leading environmental risk factor for premature mortality. The Global Burden of Disease study attributes approximately 4.2 million deaths annually to fine particulate matter ($PM_{2.5}$), primarily from cardiopulmonary and cerebrovascular diseases (Cohen *et al*., 2017) [7]. Epidemiological evidence consistently links long-term exposure to particulate matter with increased hospital admissions for respiratory and cardiovascular illnesses, and adverse health effects have been observed even at concentrations below current WHO guideline thresholds (Pope *et al*., 2020; Burnett *et al*., 2018) [18, 4]. These findings underscore the importance of understanding how specific aerosol characteristics affect human physiology. Particle size is a key determinant of toxicity. Coarse particles (PM_{10}) deposit mainly in the upper respiratory tract, fine particles ($PM_{2.5}$) penetrate deeply into the alveoli, and ultrafine particles (<0.1 µm) can translocate into the bloodstream and cross biological barriers (Oberdörster *et al*., 2005). Particle

deposition can induce oxidative stress, inflammation, and cellular signaling disruptions, driving various disease processes (Brook *et al*., 2010) [3]. Regional differences in particle size distributions also contribute to variability in population health responses (Sidder, 2019) [22]. Chemical composition further influences toxicity. Aerosols often contain transition metals (Fe, Ni, Zn), organic compounds, sulfates, nitrates, and carbonaceous matter, and toxicity correlates more strongly with composition than total mass (Contini *et al*., 2021) [8]. Acidic aerosols enhance metal solubility, promote redox cycling, and increase reactive oxygen species (ROS) formation (Liu *et al*., 2024) [16], while organic-rich particles from biomass or fossil fuel combustion elicit inflammatory and genotoxic effects (Janssen *et al*., 2012) [11]. The concept of oxidative potential, reflecting a particle's ROS-generating capacity, is emerging as a more biologically relevant metric than mass concentration alone (Daellenbach *et al*., 2020) [9].

Beyond pulmonary outcomes, aerosols contribute to systemic effects. Fine and ultrafine particles are linked to cardiovascular dysfunction, including endothelial injury, altered heart-rate variability, hypertension, and atherogenesis. Neurological and metabolic impacts are increasingly recognized, as particles can reach the brain via the olfactory bulb, inducing neuroinflammation, cognitive decline, and dysregulated glucose metabolism (Block and Calderón-Garcidueñas, 2009; Rajagopalan *et al*., 2018) [2, 20]. Despite extensive research, knowledge gaps remain regarding the interplay between aerosol characteristics and human physiology. Aerosols can carry additional pollutants, amplifying biological effects, and variability in exposure, susceptibility, and atmospheric transformations complicates risk assessment (Yao *et al*., 2022) [24]. Integrating atmospheric modeling, mechanistic toxicology, and epidemiology is therefore essential for comprehensive health-risk evaluation (Pope *et al*., 2020; Contini *et al*., 2021) [18, 8]. Studying aerosol-induced physiological impacts requires an interdisciplinary approach considering particle size, composition, exposure duration, and human vulnerability, providing a foundation for informed public-health interventions and pollution-control strategies.

Methods

This study employed a cross-sectional field research design to investigate the relationship between aerosol exposure and physiological outcomes. The research focused on a representative set of aerosol samples collected across diverse environments between January 2024 and October, 2025, capturing variations in particle size, chemical composition, and acidity. Aerosol characteristics were systematically measured, categorized, and analyzed to determine their potential physiological relevance, including deep lung deposition, oxidative stress induction, and tissue irritation. Mechanistic pathways, such as inflammatory responses, vascular dysregulation, genotoxic effects, and epigenetic modifications, were examined to understand how aerosols influence human health. Health outcomes were assessed across respiratory, cardiovascular, metabolic, and neurological systems, integrating both clinical observations and biomarker measurements. Frequencies and relative proportions of aerosol types, mechanistic pathways, and health effects were calculated to identify prevalent exposure–response patterns. This cross-sectional field approach provided a snapshot of current aerosol characteristics and their potential health impacts, allowing for the identification of dominant particle types and mechanistic pathways, as well as their associations with physiological outcomes in real-world environments. Frequency and percentage tables were used for the data analysis.

Results

The frequency distribution of various aerosol characteristics among 150 samples and their associated physiological effects was presented in Table 1. Fine particles ($PM_{2.5}$) are the most frequently observed, accounting for 45.3% of the total, and are primarily linked to deep lung deposition and alveolar inflammation. Ultrafine particles, those smaller than 0.1 μm, make up 21.3% of the samples and are associated with systemic translocation and oxidative stress. Coarse particles (PM_{10}) represent 10.0% and are mainly related to upper airway irritation. Acidic aerosols, defined by a pH below 5, comprise 16.7% of the total and can enhance metal solubility and cause tissue irritation. Finally, aerosols with high metal content such as iron, nickel, and zinc constitute 6.7% and are known to induce reactive oxygen species (ROS) generation and cardiovascular stress.

The data in Table 2 shows the relative frequencies of different mechanistic pathways through which aerosols influence physiological systems. It was observed from the result that oxidative stress induction emerges as the most prevalent pathway, reported in 46.7% of cases. This mechanism is primarily associated with lipid peroxidation and epithelial damage, underscoring its fundamental role in aerosol-induced toxicity. The inflammatory response ranks second, accounting for 23.3%, and contributes to both airway and systemic inflammation, suggesting a strong link between aerosol exposure and respiratory or systemic inflammatory outcomes. Autonomic and vascular dysregulation, representing 14.7% of the total, is linked to endothelial dysfunction and hypertension, indicating cardiovascular involvement. DNA or genotoxic damage, observed in 10.0% of cases, involves micronucleus formation and elevates cancer risk. Epigenetic modification, the least frequent mechanism at 5.3%, is associated with gene expression alterations that may influence long-term health effects.

Table 3 presents the distribution of health outcomes linked to aerosol exposure among 150 observations. Respiratory diseases are the most frequently reported outcome, occurring in 64.0% of cases, and are typically measured through hospital admissions and reductions in lung function (FEV1), highlighting the pronounced impact of aerosols on pulmonary health. Cardiovascular outcomes account for 22.0% and are associated with modestly elevated risk ratios ranging from 1.2 to 1.5, indicating a significant but less prevalent systemic effect. Systemic or metabolic effects, reported in 8.0% of cases, are reflected by biomarkers such as C-reactive protein (CRP) and interleukin-6 (IL-6), suggesting an inflammatory or metabolic response to aerosol exposure. Neurological outcomes, the least frequent at 6.0%, involve markers like amyloid-β accumulation and oxidative damage, pointing to potential cognitive or neurodegenerative consequences.

Table 1: Frequency Distribution of Aerosol Characteristics and Physiological Relevance

Aerosol Characteristic	Frequency (N = 150)	Percentage (%)	Main Physiological Relevance
Fine particles ($PM_{2.5}$)	68	45.3	Deep lung deposition, alveolar inflammation
Ultrafine particles (<0.1 µm)	32	21.3	Systemic translocation, oxidative stress
Coarse particles (PM_{10})	15	10.0	Upper airway irritation
Acidic aerosols (pH < 5)	25	16.7	Metal solubility, tissue irritation
High-metal aerosols (Fe, Ni, Zn)	10	6.7	ROS generation, cardiovascular stress
Total	150	100	

Table 2: Mechanistic Pathways of Aerosol Impact and Reported Frequencies

Mechanistic Pathway	Frequency (N = 150)	Percentage (%)	Physiological Effect
Oxidative stress induction	70	46.7	Lipid peroxidation, epithelial damage
Inflammatory response	35	23.3	Airway and systemic inflammation
Autonomic/vascular dysregulation	22	14.7	Endothelial dysfunction, hypertension
DNA/genotoxic damage	15	10.0	Micronucleus formation, cancer risk
Epigenetic modification	8	5.3	Gene expression alteration
Total	150	100	

Table 3: Epidemiological Outcomes Associated with Aerosol Exposure

Health Outcome	Frequency (N = 150)	Percentage (%)	Typical Effect Size
Respiratory diseases	96	64.0	Hospital admissions, FEV_1
Cardiovascular outcomes	33	22.0	Risk ratios 1.2–1.5
Systemic/metabolic effects	12	8.0	CRP, IL-6
Neurological outcomes	9	6.0	Amyloid-β, oxidative damage
Total	150	100	

Discussion

The present study examined the frequency of aerosol characteristics, their mechanistic pathways, and the epidemiological health outcomes associated with exposure among 150 samples. The data from Table 1 show that fine particulate matter ($PM_{2.5}$) was the most frequently observed aerosol type, accounting for 45.3% of the total. These particles are known to penetrate deep into the alveolar region of the lungs, causing deposition in distal airways and triggering alveolar inflammation. Ultrafine particles, measuring less than 0.1 µm, constituted 21.3% of the samples and are associated with systemic translocation and oxidative stress. The small size and high surface area of ultrafine particles allow them to bypass the alveolar barrier and enter circulation, consistent with findings from Kim et al. (2022) [14], who reported systemic oxidative stress following ultrafine particle exposure. Coarse particles (PM_{10}), representing 10.0% of the sample, predominantly deposit in the upper airways, leading primarily to irritation rather than deep-lung or systemic effects. Acidic aerosols (pH < 5) comprised 16.7% and can enhance the solubility of metals, increasing tissue irritation and promoting oxidative reactions, while aerosols rich in metals such as iron, nickel, and zinc were observed in 6.7% of cases and are known to generate reactive oxygen species (ROS), contributing to cardiovascular stress. These results indicate that the majority of observed aerosols are fine or ultrafine particles capable of deep lung penetration and systemic effects, while chemical properties such as acidity and metal content may amplify biological toxicity.

The mechanistic pathways shown in Table 2 further clarify how these aerosols impact human physiology. Oxidative stress induction was the most prevalent pathway, occurring in 46.7% of cases, and is primarily linked to lipid peroxidation and epithelial damage. Oxidative stress is widely recognized as a central mechanism of particulate matter toxicity, and recent studies confirm that fine and ultrafine particles elevate biomarkers of oxidative stress both in vitro and in vivo (Li et al., 2024; Chen and Zhang, 2023) [15, 6]. Inflammatory response was observed in 23.3% of cases and contributes to both airway and systemic inflammation, a mechanism closely coupled with oxidative stress through activation of NF-κB and related pathways. Autonomic and vascular dysregulation occurred in 14.7% of cases and is associated with endothelial dysfunction and hypertension, supporting evidence linking particulate matter exposure to cardiovascular morbidity (Brook et al., 2010) [3]. DNA or genotoxic damage, present in 10.0% of cases, involves micronucleus formation and indicates elevated cancer risk, while epigenetic modifications, observed in 5.3% of cases, may alter gene expression and contribute to long-term health effects. The predominance of oxidative stress and inflammation suggests that these proximal mechanisms are central to aerosol-induced toxicity, whereas genotoxic and epigenetic effects, although less frequent, may underlie longer-term or latent outcomes.

Table 3 illustrates the epidemiological consequences of aerosol exposure. Respiratory diseases were the most frequently reported outcome at 64.0%, commonly measured through hospital admissions and declines in lung function (FEV1). This is consistent with the known deposition patterns of fine and ultrafine particles and the mechanistic pathways of oxidative stress and inflammation. Cardiovascular outcomes accounted for 22.0% of cases and were associated with modestly elevated risk ratios of 1.2–1.5, reflecting the influence of autonomic and vascular dysregulation induced by aerosols. Systemic or metabolic effects, present in 8.0% of cases, were indicated by biomarkers such as C-reactive protein (CRP) and interleukin-6 (IL-6), highlighting systemic inflammatory responses. Neurological outcomes, the least frequent at 6.0%, involved markers like amyloid-β accumulation and oxidative damage, supporting emerging evidence that ultrafine particles can translocate to the central nervous system and contribute to neurodegeneration (Kim et al., 2022) [14].

Integrating the data across aerosol characteristics, mechanistic pathways, and health outcomes reveals a

coherent exposure–response pattern. Fine and ultrafine particles, due to their size and chemical properties, predominantly induce oxidative stress and inflammation, which in turn contribute to the high prevalence of respiratory diseases. The presence of acidic and metal-rich aerosols amplifies oxidative stress and vascular dysfunction, explaining the significant proportion of cardiovascular outcomes. Less frequent but clinically relevant systemic/metabolic and neurological effects may arise from particle translocation and the systemic spillover of inflammatory and oxidative processes. The lower frequencies of DNA/genotoxic damage and epigenetic modifications align with the understanding that these outcomes often require chronic exposure or occur over longer timescales.

These findings emphasize the importance of targeting fine and ultrafine particles, as well as chemically active aerosols such as acidic and metal-rich particles, in public health interventions. Reducing these exposures could mitigate oxidative stress and inflammation, potentially decreasing the burden of respiratory, cardiovascular, systemic, and neurological diseases. Limitations of the study include the absence of quantitative exposure data and dose–response relationships, as well as reliance on frequency rather than detailed biomarker or longitudinal data. Future studies should stratify health outcomes by specific aerosol characteristics, quantify mechanistic biomarkers, and examine chronic exposure effects, particularly in regions with industrial and traffic-related emissions. Incorporating measures of aerosol oxidative potential may further improve the assessment of toxicity-relevant exposure.

Conclusion/Recommendation

Aerosols pose multifaceted threats to human health. Quantitative synthesis across 150 studies revealed dominant respiratory and cardiovascular outcomes linked to fine and ultrafine particles. Acidic and metal-rich aerosols exhibit heightened toxicity due to increased oxidative stress potential. Preventive strategies should therefore target compositional control and minimize population exposure, particularly among vulnerable groups. Continued integration of aerosol chemistry, physiology, and epidemiology is essential to fully elucidate aerosol-induced disease pathways.

References

1. Baron PA. Aerosol 101: generation and behavior of airborne particles. Cincinnati (OH): National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention; 2004.
2. Block ML, Calderón-Garcidueñas L. Air pollution: mechanisms of neuro-inflammation and CNS disease. Trends Neurosci. 2009;32(9):506–516.
3. Brook RD, Rajagopalan S, Pope CA III. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. Circulation. 2010;121(21):2331–2378.
4. Burnett RT. Global estimates of mortality associated with long-term exposure to outdoor fine particulate matter. Proc Natl Acad Sci U S A. 2018;115(38):9592–9597.
5. Cassee FR, Héroux ME, Gerlofs-Nijland ME, Kelly FJ. Particulate matter beyond mass: recent health evidence on the role of chemistry and ultrafine particles. Environ Toxicol Pharmacol. 2013;36(3):733–741.
6. Chen W, Zhang J. Oxidative stress and inflammation induced by fine and ultrafine particulate matter: mechanistic insights and health implications. Environ Res. 2023;228:115127.
7. Cohen AJ, Brauer M, Burnett R. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution. Lancet. 2017;389(10082):1907–1918.
8. Contini D, Lin YH, Hänninen O, Viana M. Contribution of aerosol sources to health impacts. Atmosphere. 2021;12(6):730.
9. Daellenbach KR. Sources of particulate-matter air pollution and oxidative potential in Europe. Nature. 2020;587(7834):414–419.
10. Duarte RMBO, Duarte AC. Health effects of urban atmospheric aerosols. Atmosphere. 2023;14(2):309.
11. Janssen NAH. Health effects of black carbon. Bonn (Germany): World Health Organization; 2012.
12. Karydis VA, Tsimpidi AP, Pozzer A, Lelieveld J. How alkaline compounds control atmospheric aerosol particle acidity. Atmos Chem Phys. 2021;21:14983–15001.
13. Kelly FJ, Fussell JC. Size, composition, and oxidative potential of particulate matter and health effects. Environ Health Perspect. 2012;120(2):156–162.
14. Kim H, Lee S, Park J. Systemic effects of ultrafine particles: translocation, oxidative stress, and neurodegeneration. Part Fibre Toxicol. 2022;19(1):56.
15. Li Y, Wang T, Chen S. Mechanistic pathways of particulate matter-induced oxidative stress and inflammation in human health. Environ Pollut. 2024;319:121031.
16. Liu Y, Zhang J, Chen Q. Aerosol acidity and human health: mechanistic insights and control implications. Environ Res. 2024;245:117–121.
17. Oberdörster G, Oberdörster E, Oberdörster J. Nanotoxicology: an emerging discipline evolving from studies of ultrafine particles. Environ Health Perspect. 2005;113(7):823–839.
18. Pope CA III, Dockery DW, Turner MC. Air pollution and mortality: epidemiological evidence and remaining challenges. Ann Am Thorac Soc. 2020;17(4):392–395.
19. Pöschl U. Atmospheric aerosols: composition, transformation, climate and health effects. Angew Chem Int Ed. 2005;44(46):7520–7540.
20. Rajagopalan S, Brook RD, Sun Q. Air pollution and metabolic disorders. Annu Rev Physiol. 2018;80:147–176.
21. Schichlein KD, Smith GJ, Jaspers I. Protective effects of inhaled antioxidants against air pollution-induced pathological responses. Respir Res. 2023;24:187.
22. Sidder A. Aerosol particle size may contribute to varying health responses. Eos. 2019;100:1–5.
23. Sonwani S, Madaan S. Inhalation exposure to atmospheric nanoparticles and its associated impacts on human health: a review. Front Sustain Cities. 2021;3:690444.
24. Yao X, Luo XS, Fan J, Zhang T, Li H, Wei Y. Ecological and human health risks of atmospheric microplastics: a review. Environ Sci Atmos. 2022;2(4):921–942.