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Airborne Particulate Matter and Systemic Inflammation: Mechanisms, Health Impacts, and Public Health Implications

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ABSTRACT

Airborne particulate matter (PM) is a major component of air pollution and represents a significant global public health concern. Fine and ultrafine particles derived from traffic emissions, industrial processes, biomass burning, and natural sources can penetrate deep into the respiratory tract and, in some cases, enter the systemic circulation. Increasing evidence links exposure to particulate matter, particularly PM2.5 and ultrafine particles, with systemic inflammation, a key underlying mechanism in the development of cardiovascular disease, metabolic disorders, respiratory conditions, and neurodegenerative diseases. This review examines the sources and characteristics of airborne particulate matter, the biological mechanisms that connect inhaled particles to systemic inflammatory responses, and the clinical and epidemiological evidence supporting these associations. Key pathways include oxidative stress, activation of innate immune responses, endothelial dysfunction, and dysregulation of autonomic balance. The role of vulnerable populations and long-term health consequences is discussed, along with emerging biomarkers of exposure and inflammation. Finally, the review highlights policy and public health interventions aimed at reducing exposure and mitigating inflammatory health effects. Understanding the relationship between airborne particulate matter and systemic inflammation is critical for developing effective preventive strategies and reducing the global burden of pollution-related diseases.

Keywords: Particulate matter, Systemic inflammation, Oxidative stress, Cardiovascular disease, Air pollution

INTRODUCTION

Air pollution is one of the leading environmental risk factors for morbidity and mortality worldwide, contributing substantially to cardiovascular, respiratory, and metabolic diseases[1]. Among its various components, airborne particulate matter has attracted considerable scientific and regulatory attention due to its pervasive distribution and strong association with adverse health outcomes across diverse populations. Particulate matter consists of a heterogeneous mixture of solid particles and liquid droplets suspended in air[2]. These particles vary in size, chemical composition, surface characteristics, and source of origin, all of which influence their deposition within the respiratory tract and their subsequent biological effects. Particles are commonly classified according to their aerodynamic diameter. Coarse particles (PM10) have diameters of 10 micrometers or less and tend to deposit in the upper airways. Fine particles (PM2.5), measuring 2.5 micrometers or smaller, can penetrate deeper into the bronchioles and alveolar regions[3]. Ultrafine particles, smaller than 0.1 micrometers, are of particular concern because of their ability to cross the alveolar-capillary barrier and potentially enter systemic circulation. Their small size and large surface area enable them to interact efficiently with cellular structures and biological molecules[4]. Systemic inflammation refers to a body-wide inflammatory response characterized by elevated circulating mediators such as cytokines, chemokines, and acute-phase proteins[5]. Chronic low-grade systemic inflammation is increasingly recognized as a unifying mechanism underlying many non-communicable diseases, including atherosclerosis, diabetes, and neurodegeneration. Growing experimental and epidemiological evidence

demonstrates that inhalation of particulate matter can initiate pulmonary oxidative stress and immune activation, which may extend beyond the lungs and sustain systemic inflammatory processes. This mechanistic link provides a critical framework for understanding how environmental exposures translate into long-term chronic disease risk.

2. Sources and Composition of Particulate Matter

Airborne particulate matter originates from both natural and anthropogenic sources, often interacting in complex atmospheric processes[6]. Natural sources include desert dust storms, sea salt aerosols, volcanic eruptions, pollen, and wildfires. Anthropogenic sources, which dominate in urban and industrialized regions, include vehicle exhaust emissions, industrial manufacturing, coal-fired power plants, biomass burning for cooking or heating, agricultural activities, and construction-related dust[7]. The chemical composition of particulate matter is highly variable and depends on its source and atmospheric transformations[8]. It may contain transition metals such as iron, nickel, copper, and vanadium; organic compounds including polycyclic aromatic hydrocarbons; sulfates and nitrates formed from gaseous precursors; elemental carbon; and secondary organic aerosols generated through photochemical reactions. These constituents influence particle reactivity and toxicity. Fine and ultrafine particles generated from combustion processes are particularly harmful[9]. Their high surface area-to-mass ratio allows them to adsorb toxic chemicals and generate reactive oxygen species. This enhances their ability to induce oxidative stress, activate inflammatory signaling pathways, and contribute to systemic inflammatory responses following inhalation.

3. Mechanisms Linking Particulate Matter to Systemic Inflammation

3.1 Pulmonary Oxidative Stress

Upon inhalation, particulate matter deposits along the respiratory tract, with fine and ultrafine particles reaching the alveolar regions where gas exchange occurs[10-14]. Reactive components within these particles, including transition metals and organic compounds such as polycyclic aromatic hydrocarbons, catalyze the formation of reactive oxygen species[11-16]. Excessive generation of these species overwhelms endogenous antioxidant defenses, leading to oxidative stress. This state results in lipid peroxidation, protein oxidation, mitochondrial dysfunction, and DNA damage in airway epithelial cells and alveolar macrophages[12,17-23]. Oxidative stress also serves as a signaling mechanism. It activates redox-sensitive transcription factors such as nuclear factor kappa B (NF-κB) and activator protein-1, which regulate the expression of numerous pro-inflammatory genes[13,24-28]. Consequently, inflammatory mediators including interleukin-6, tumor necrosis factor-alpha, and interleukin-1 beta are synthesized and released, initiating a cascade of immune activation[14,29-34].

3.2 Activation of Innate Immune Responses

Particulate matter interacts with pattern recognition receptors, including toll-like receptors, expressed on epithelial cells and immune cells[15,35-40]. Engagement of these receptors triggers intracellular signaling pathways that amplify inflammatory gene expression. Alveolar macrophages and recruited neutrophils release cytokines and chemokines, sustaining local pulmonary inflammation[16,41-46]. Importantly, inflammatory mediators can enter the systemic circulation. Circulating cytokines stimulate hepatic production of acute-phase proteins such as C-reactive protein, reflecting a transition from localized lung inflammation to systemic inflammatory activation.

3.3 Translocation into the Circulation

Ultrafine particles may cross the alveolar-capillary barrier and directly enter the bloodstream. Once systemic, they interact with vascular endothelial cells and circulating leukocytes, enhancing oxidative stress, promoting adhesion molecule expression, and contributing to endothelial dysfunction, an early step in atherosclerosis[17,47-52].

3.4 Autonomic Nervous System Imbalance

Exposure to particulate matter can disrupt autonomic regulation, decreasing heart rate variability and increasing sympathetic activity[18,53-60]. This imbalance influences inflammatory signaling pathways and may exacerbate cardiovascular risk through combined neural and inflammatory mechanisms.

4. Cardiovascular Consequences

The cardiovascular system is particularly vulnerable to systemic inflammation induced by particulate matter exposure[19,61-64]. A substantial body of epidemiological evidence demonstrates that short-term increases in PM2.5 concentrations are associated with higher rates of hospital admissions for myocardial infarction, stroke, arrhythmias, and heart failure[20,65-68]. Even brief exposure to elevated pollution levels can trigger acute cardiovascular events, particularly in individuals with pre-existing conditions. Chronic exposure to particulate matter contributes to the initiation and progression of atherosclerosis. Persistent systemic inflammation promotes endothelial dysfunction, characterized by reduced nitric oxide bioavailability and impaired vascular relaxation[21,68-70]. Oxidative stress and inflammatory cytokines stimulate the expression of adhesion molecules, facilitating the recruitment of monocytes into the vascular wall and the formation of fatty streaks. Over time, these processes accelerate plaque development. Elevated circulating biomarkers such as C-reactive protein, fibrinogen, and interleukin-6 are frequently observed in populations exposed to higher levels of air pollution, reflecting sustained inflammatory activation[22]. Inflammation also destabilizes established atherosclerotic

plaques by weakening fibrous caps and increasing enzymatic degradation of extracellular matrix components[23]. This increases the risk of plaque rupture and subsequent thrombosis. In addition, systemic inflammation enhances platelet activation and alters coagulation pathways, promoting a prothrombotic state that further elevates cardiovascular risk.

5. Respiratory Effects and Beyond

Although particulate matter primarily affects the respiratory system, its impact extends beyond localized lung pathology[24]. Chronic exposure is associated with increased frequency and severity of asthma attacks, progression of chronic obstructive pulmonary disease, and accelerated decline in lung function. Inflammatory responses within the lungs can spill over into the systemic circulation, influencing distant organs[25]. Systemic inflammation triggered by pulmonary exposure may disrupt metabolic homeostasis. Increasing evidence links long-term air pollution exposure with insulin resistance, type 2 diabetes, and obesity[26]. Pro-inflammatory cytokines interfere with insulin signaling pathways, contributing to impaired glucose uptake and metabolic imbalance[27]. Emerging research also implicates particulate matter in neuroinflammation and cognitive decline. Circulating inflammatory mediators may cross the blood-brain barrier or activate neural pathways, promoting microglial activation and potentially accelerating neurodegenerative processes[28].

8. Long-Term and Chronic Implications

Chronic low-level exposure to particulate matter may sustain persistent systemic inflammation, even when acute symptoms are not evident[29]. Continuous inhalation of fine and ultrafine particles can maintain a state of immune activation characterized by elevated cytokines, oxidative stress markers, and acute-phase proteins[30]. Over time, this persistent inflammatory milieu contributes to cumulative tissue injury, vascular remodeling, and metabolic dysregulation. Such long-term inflammatory burden is strongly associated with increased risks of chronic diseases, including hypertension, type 2 diabetes, ischemic heart disease, neurodegenerative disorders such as Alzheimer's disease, and certain malignancies[31]. Repeated exposure may also alter immune homeostasis, leading to impaired host defense and dysregulated inflammatory resolution. The concept of inflammaging, defined as age-related chronic low-grade inflammation, may be exacerbated by lifelong exposure to air pollution[32]. Persistent immune stimulation accelerates biological aging processes, including telomere shortening, mitochondrial dysfunction, and epigenetic alterations[33]. Consequently, populations residing in highly polluted environments may experience earlier onset of age-associated diseases and reduced life expectancy.

9. Public Health and Policy Implications

Reducing exposure to particulate matter is critical for mitigating systemic inflammatory health effects at the population level[34]. Effective regulatory standards for ambient air quality, stringent emission controls for vehicles and industries, transition to cleaner and renewable energy sources, and sustainable urban planning are central strategies[35]. Promoting public transportation, green spaces, and low-emission zones can significantly decrease pollution levels in densely populated areas. At the individual level, measures such as indoor air filtration, use of protective masks during severe pollution episodes, and limiting outdoor activities when air quality is poor may reduce personal exposure[36]. However, structural interventions implemented at community, national, and international levels are more impactful and equitable[37]. Global cooperation is essential to address transboundary air pollution and the interaction between climate change and particulate matter formation. Incorporating health impact assessments into environmental and energy policies can help prioritize actions that reduce inflammation-related disease burdens and improve long-term public health outcomes[38].

10. Conclusion

Airborne particulate matter is a potent environmental trigger of systemic inflammation. Through mechanisms involving oxidative stress, immune activation, endothelial dysfunction, and autonomic imbalance, inhaled particles exert effects far beyond the respiratory tract. Systemic inflammation serves as a central pathway linking particulate matter exposure to cardiovascular, metabolic, respiratory, and neurological diseases. Given the pervasive nature of air pollution and its profound health implications, comprehensive strategies are required to reduce exposure and protect vulnerable populations. Continued research into mechanistic pathways, biomarkers, and long-term outcomes will enhance understanding and support evidence-based policy decisions. Addressing airborne particulate matter is not only an environmental priority but also a crucial step toward reducing the global burden of inflammation-related chronic diseases.

REFERENCES

1. Adeoye M, Rahimzadeh S, Taylor S, Shrikhande S, Perel P, Shah A, Di Cesare M, Miller MR. The Impact of Air Pollution on Cardiovascular Health Outcomes in African Populations: A Scoping Review. JACC Adv. 2024 Nov 13;3(12):101371. doi: 10.1016/j.jacadv.2024.101371. PMID: 39817083; PMCID: PMC11733974.
2. Y Tanko, ED Eze, A Jimoh, M Yusuf, KA Mohammed, F Balarabe, MG Magaji, A Mohammed (2010). Cardio-respiratory changes among farmers and non-farmers in Rafi Local Government Area, Niger State, Nigeria.*Pulse, 93, (1.46), 92.36-1.23ns.www.iosrjournals.org*

3. Palacio LC, Pachajoa DC, Echeverri-Londoño CA, Saiz J, Tobón C. Air Pollution and Cardiac Diseases: A Review of Experimental Studies. Dose Response. 2023 Nov 4;21(4):15593258231212793. doi: 10.1177/15593258231212793. PMID: 37933269; PMCID: PMC10625734.
4. Mu Q, Jiang G, Chen L, Zhou H, Fourches D, Tropsha A, Yan B. Chemical basis of interactions between engineered nanoparticles and biological systems. Chem Rev. 2014 Aug 13;114(15):7740-81. doi: 10.1021/cr400295a. Epub 2014 Jun 13. PMID: 24927254; PMCID: PMC4578874.
5. Hetta HF, Ramadan YN, Al-Harbi AI, A. Ahmed E, Battah B, Abd Ellah NH, Zanetti S, Donadu MG. Nanotechnology as a Promising Approach to Combat Multidrug Resistant Bacteria: A Comprehensive Review and Future Perspectives. Biomedicines. 2023; 11(2):413. https://doi.org/10.3390/biomedicines11020413
6. Damiati S, AlMashrea BA, Rabiei N, Sujatha AP, Sabir DK, Alhosani M, Kodzius R. Aerosol Pollutants and Health: Role of Size and Chemical Composition. Public Health Chall. 2025 Sep 26;4(4):e70134. doi: 10.1002/puh2.70134. PMID: 41020050; PMCID: PMC12466196.
7. Andriulė A, Suzdalev S, Vasiliauskienė E, Dailidienė I. Air Pollution and Dispersion of Airborne Chemical Elements in Klaipėda Seaport-City. Sustainability. 2025; 17(9):3834. https://doi.org/10.3390/su17093834
8. Jandacka D, Durcanska D. Differentiation of Particulate Matter Sources Based on the Chemical Composition of PM10 in Functional Urban Areas. Atmosphere. 2019; 10(10):583. https://doi.org/10.3390/atmos10100583
9. Schraufnagel DE. The health effects of ultrafine particles. Exp Mol Med. 2020 Mar;52(3):311-317. doi: 10.1038/s12276-020-0403-3. Epub 2020 Mar 17. PMID: 32203102; PMCID: PMC7156741.
10. Morakinyo OM, Mokgobu MI, Mukhola MS, Hunter RP. Health Outcomes of Exposure to Biological and Chemical Components of Inhalable and Respirable Particulate Matter. International Journal of Environmental Research and Public Health. 2016; 13(6):592. https://doi.org/10.3390/ijerph13060592
11. UO Suleiman, ED Eze, YM TsauRI, JB Adewale, Y Abdullahi, OU Olasunkanmi, MD Adams (2018). Comparison of physical fitness of rural, semi-urban and urban of primary school children in their abdominal strength, flexibility and cardio-respiratory endurance in federal …*MOJ Sports Med. 2, (1), 37-42.*
12. Liu X, Chen Z. The pathophysiological role of mitochondrial oxidative stress in lung diseases. J Transl Med. 2017 Oct 13;15(1):207. doi: 10.1186/s12967-017-1306-5. PMID: 29029603; PMCID: PMC5640915.
13. Kim S-J, Cheresh P, Jablonski RP, Williams DB, Kamp DW. The Role of Mitochondrial DNA in Mediating Alveolar Epithelial Cell Apoptosis and Pulmonary Fibrosis. International Journal of Molecular Sciences. 2015; 16(9):21486-21519. https://doi.org/10.3390/ijms160921486
14. Ayala A, Muñoz MF, Argüelles S. Lipid peroxidation: production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. Oxid Med Cell Longev. 2014;2014:360438. doi: 10.1155/2014/360438. Epub 2014 May 8. PMID: 24999379; PMCID: PMC4066722.
15. Bauer RN, Diaz-Sanchez D, Jaspers I. Effects of air pollutants on innate immunity: the role of Toll-like receptors and nucleotide-binding oligomerization domain-like receptors. J Allergy Clin Immunol. 2012 Jan;129(1):14-24; quiz 25-6. doi: 10.1016/j.jaci.2011.11.004. PMID: 22196521; PMCID: PMC4341993.
16. Nagappan A, Park SB, Lee S-J, Moon Y. Mechanistic Implications of Biomass-Derived Particulate Matter for Immunity and Immune Disorders. Toxics. 2021; 9(2):18. https://doi.org/10.3390/toxics9020018
17. Ogbonna OA., Egba, SI., Uhuo EN., Omeoga HC., Obeagu EI. Toxic outcomes of ciprofloxacin and gentamicin co-administration and possible ameliorating role for antioxidant vitamins C and E in Wistar Rats. Elite Journal of Medicine, 2024; 2(3): 1-14.
18. Park SK, O'Neill MS, Vokonas PS, Sparrow D, Schwartz J. Effects of air pollution on heart rate variability: the VA normative aging study. Environ Health Perspect. 2005 Mar;113(3):304-9. doi: 10.1289/ehp.7447. PMID: 15743719; PMCID: PMC1253756.
19. Ejemot-Nwadiaro R. I, Ofili D. F. C, Ogbodo S. C, Okoroiwu H. U, Ukah U. V .Risk of Cardiovascular Disease Comorbidity in People Living with Diabetes in Africa. *Kampala International University Western Campus, Ishaka, Available at SSRN 4901142.*
20. Krittanawong, C., Qadeer, Y. K., Hayes, R. B., Wang, Z., Thurston, G. D., Virani, S. S., … & Lavie, C. J. (2023). PM2.5 and cardiovascular diseases: State-of-the-Art review. International Journal of Cardiology Cardiovascular Risk and Prevention, 19, 200217. https://doi.org/10.1016/j.ijcrp.2023.200217
21. Robert I U., Amarachi A., Egba S I., Sangodare RSA.,Oluomachi N U., Nwuke CP and Osisioma K N (2020) Heavy Metal contents in commercial fishes consumed in Umuahia and their associated human health risk, EQA-International Journal of Environmental Quality 39(2020): 11-19
22. Rückerl R, Greven S, Ljungman P, Aalto P, Antoniades C, Bellander T, Berglind N, Chrysohoou C,

Forastiere F, Jacquemin B, von Klot S, Koenig W, Küchenhoff H, Lanki T, Pekkanen J, Perucci CA, Schneider A, Sunyer J, Peters A; AIRGENE Study Group. Air pollution and inflammation (interleukin-6, C-reactive protein, fibrinogen) in myocardial infarction survivors. Environ Health Perspect. 2007 Jul;115(7):1072-80. doi: 10.1289/ehp.10021. PMID: 17637925; PMCID: PMC1913563.

23. Kim JH, Woo HD, Choi S, Song DS, Lee JH, Lee K. Long-Term Effects of Ambient Particulate and Gaseous Pollutants on Serum High-Sensitivity C-Reactive Protein Levels: A Cross-Sectional Study Using KoGES-HEXA Data. International Journal of Environmental Research and Public Health. 2022; 19(18):11585. https://doi.org/10.3390/ijerph191811585
24. Tavella RA, da Silva Júnior FMR, Santos MA, Miraglia SGEK, Pereira Filho RD. A Review of Air Pollution from Petroleum Refining and Petrochemical Industrial Complexes: Sources, Key Pollutants, Health Impacts, and Challenges. ChemEngineering. 2025; 9(1):13. https://doi.org/10.3390/chemengineering9010013
25. Tkacova R. Systemic inflammation in chronic obstructive pulmonary disease: may adipose tissue play a role? Review of the literature and future perspectives. Mediators Inflamm. 2010;2010:585989. doi: 10.1155/2010/585989. Epub 2010 Apr 20. PMID: 20414465; PMCID: PMC2857618.
26. Marzec JM, Nadadur SS. Inflammation resolution in environmental pulmonary health and morbidity. Toxicol Appl Pharmacol. 2022 Aug 15;449:116070. doi: 10.1016/j.taap.2022.116070. Epub 2022 May 23. PMID: 35618031; PMCID: PMC9872158.
27. Berbudi A, Khairani S, Tjahjadi AI. Interplay Between Insulin Resistance and Immune Dysregulation in Type 2 Diabetes Mellitus: Implications for Therapeutic Interventions. Immunotargets Ther. 2025 Apr 3;14:359-382. doi: 10.2147/ITT.S499605. PMID: 40196377; PMCID: PMC11974557.
28. Ogbodo John Onyebuchi, Chinazom Precious Agbo, Ugoci Olivia Njoku, Martins Obinna Ogugofor, Egba Simeon Ikechukwu, Stella Amarachi Ihim, Adaeze Chidiebere Echezona Kenneth Chibuike Brendan, Aman Babanrao Upaganlawar, and ChandrashekarDevidas Upasani (2021) Alzheimer's Disease: Pathogenesis and Therapeutic Interventions, Current Aging Science, 21:1-25.
29. Pope CA 3rd, Bhatnagar A, McCracken JP, Abplanalp W, Conklin DJ, O'Toole T. Exposure to Fine Particulate Air Pollution Is Associated With Endothelial Injury and Systemic Inflammation. Circ Res. 2016 Nov 11;119(11):1204-1214. doi: 10.1161/CIRCRESAHA.116.309279. Epub 2016 Oct 25. PMID: 27780829; PMCID: PMC5215745.
30. Orji, U. O., Ibiam, U. A., Awoke, J. N., Atoki, A. V., Eze, E. D., Obasi, O. D., … Aja, P. M. (2025). Ethanolic extract of *Cnidoscolus aconitifolius* leaves alleviates insulin resistance by modulating oxidative stress and inflammation in albino rats exposed to heavy metal mixture. *Natural Product Research*, 1–14. https://doi.org/10.1080/14786419.2025.2478654
31. Alum, E. U., Nwali, B. U., Aniokete, U. C., Emeruwa, A. P., Obasi, D. C., Okoroh, P. N., … & Aja, P. M. (2025). Gut microbiota at the crossroads of food additives, pollutants, and chronic disease risk. Toxicology and Environmental Health Sciences. https://doi.org/10.1007/s13530-025-00295-3
32. Khan MSH, Hegde V. Obesity and Diabetes Mediated Chronic Inflammation: A Potential Biomarker in Alzheimer's Disease. Journal of Personalized Medicine. 2020; 10(2):42. https://doi.org/10.3390/jpm10020042
33. Ferrucci L, Fabbri E. Inflammageing: chronic inflammation in ageing, cardiovascular disease, and frailty. Nat Rev Cardiol. 2018 Sep;15(9):505-522. doi: 10.1038/s41569-018-0064-2. PMID: 30065258; PMCID: PMC6146930.
34. Janjua S, Powell P, Atkinson R, Stovold E, Fortescue R. Individual-level interventions to reduce personal exposure to outdoor air pollution and their effects on people with long-term respiratory conditions. Cochrane Database Syst Rev. 2021 Aug 9;8(8):CD013441. doi: 10.1002/14651858.CD013441.pub2. PMID: 34368949; PMCID: PMC8407478.
35. Wu W, Jin Y, Carlsten C. Inflammatory health effects of indoor and outdoor particulate matter. J Allergy Clin Immunol. 2018 Mar;141(3):833-844. doi: 10.1016/j.jaci.2017.12.981. PMID: 29519450.
36. Laumbach RJ, Cromar KR, Adamkiewicz G, Carlsten C, Charpin D, Chan WR, de Nazelle A, Forastiere F, Goldstein J, Gumy S, Hallman WK, Jerrett M, Kipen HM, Pirozzi CS, Polivka BJ, Radbel J, Shaffer RE, Sin DD, Viegi G. Personal Interventions for Reducing Exposure and Risk for Outdoor Air Pollution: An Official American Thoracic Society Workshop Report. Ann Am Thorac Soc. 2021 Sep;18(9):1435-1443. doi: 10.1513/AnnalsATS.202104-421ST. PMID: 34468284; PMCID: PMC8489863.
37. Hassan S, Kagwanja N, Diallo B, Oyando R, Perel P, Etyang A, Tsofa B, Nolte E; IHCoR-Africa Collaborators. Implementing community-based interventions for the management of chronic conditions in low- and middle-income countries: A scoping review of qualitative evidence. PLOS Glob Public Health. 2025 Jul 8;5(7):e0004860. doi: 10.1371/journal.pgph.0004860. PMID: 40627592; PMCID: PMC12237052.

38. Brown AF, Ma GX, Miranda J, Eng E, Castille D, Brockie T, Jones P, Airhihenbuwa CO, Farhat T, Zhu L, Trinh-Shevrin C. Structural Interventions to Reduce and Eliminate Health Disparities. Am J Public Health. 2019 Jan;109(S1):S72-S78. doi: 10.2105/AJPH.2018.304844. PMID: 30699019; PMCID: PMC6356131.
39. Ugwu OPC, Offor CE, Ibiam UA, Ezugwu AL, Uraku AJ, Igwe CN. The effect of ethanol extract of Jatropha curcas on renal markers of chloroform intoxicated albino Wistar rats. Eur J Biol Sci. 2015;7(1):21–5.
40. Ugwu OPC, Nzubechukwu E. The effect of ethanol leaf extract of Jatropha curcas on chloroform-induced hepatotoxicity in albino rats. Glob J Biotechnol Biochem. 2015;10:11–5.
41. Ugwu OPC, Onwe SC, Okon MB. The effect of methanol extract of Rauwolfia vomitoria on lipid profile of chloroform-intoxicated Wistar albino rats. IAA J Sci Res. 2022;8(1):73–82.
42. Ugwu OPC. The effect of ethanol leaf extract of Rauwolfia vomitoria on hepatic markers of chloroform-intoxicated albino Wistar rats. IAA J Appl Sci. 2022;8(1):87–97.
43. Ugwu OPC. The effect of methanol leaf extract of Rauwolfia vomitoria on hepatic markers of chloroform-intoxicated albino Wistar rats. IAA J Appl Sci. 2022;8(1):76–86.
44. Aja PM, Ezeudeh NL, Umahi BG, Ugwu OPC, Enechi OC, Edwin N, et al. Hepato-protective effect of ethanol extract of Pterocarpus santalinoides leaf on carbon tetrachloride (CCl4)-induced albino rats. Caribb J Sci Technol. 2016;4(1):882–95.
45. Enechi OC, Okpe CC, Ibe GN, Omeje KO, Ugwu OPC. Effect of Buchholzia coriacea methanol extract on haematological indices and liver function parameters in Plasmodium berghei-infected mice. Global Vet. 2016;16(1):57–66.
46. Offor CE, Ibiam UA, Ugwu PC, Nwankwo H. The effect of ethanol leaf-extract of Gmelina arborea on liver enzymes. Eur J Biol Sci. 2015;7(1):38–40.
47. Offor CE, Nwankwegu NJ, Ugwu OPC, Aja PM. The effects of ethanol leaf-extract of Pterocarpus santalinoides on liver enzymes of albino rats. 2015.
48. Aja PM, Udeh SMC, Opajobi AO, Uzuegbu UE, Alum EU, Edwin N, et al. Hepato-protective effect of aqueous leaf-extract of Talinum triangulare in monosodium glutamate (MSG)-induced hepatic damage in albino rats. Indo Am J Pharm Sci. 2017;4(2):464–70.
49. Alum EU, Umoru GU, Uti DE, Aja PM, Ugwu OPC, Orji OU, et al. Hepato-protective effect of ethanol leaf extract of Datura stramonium in alloxan-induced diabetic albino rats. J Chem Soc Nigeria. 2022;47(5).
50. Orji OU, Ibiam UA, Aja PM, Uraku AJ, Inya-Agha OR, Ugwu OPC, et al. Hepatoprotective activity of ethanol extract of Vernonia ambigua against carbon tetrachloride-induced hepatotoxicity in albino rats. IOSR J Dent Med Sci. 2015;14:22–9.
51. Orji OU, Ibiam UA, Aloke C, Awoke J, Aja PM, Ugwu OPC, et al. Biochemical assessment of ethanol leaf extract of Cnidoscolus aconitifolius on liver integrity of albino rat treated with lead. Glob J Pharmacol. 2016;10(4):108–13.
52. Ezekwe CI, Ada AC, Ugwu OPC. Effects of methanol extract of Parkia biglobosa stem bark on the liver and kidney functions of albino rats. 2013.
53. Ezekwe CI, Uzomba CR, Ugwu OPC. The effect of methanol extract of Talinum triangulare (water leaf) on the hematology and some liver parameters of experimental rats. Glob J Biotechnol Biochem. 2013;8(2):51–60.
54. Ugwu OPC, Amasiorah VI. Review on health implications, benefits and biochemistry of alcohol intoxication. INOSR Exp Sci. 2020;6(1):62–74.
55. Ezeonwumelu JOC, Uhama KC, Ugwu OPC, Alum EU. Comprehensive overview of alcohol intoxication: mechanisms, effects, and interventions. Res Invent J Sci Exp Sci. 2024;3(1):12–20.
56. Nwaka AC, Ikechi-Agba MC, Ugwu OPC, Igwenyi IO, Agbafor KN, Ibiam UA, et al. The effects of ethanol extracts of Jatropha curcas on some hematological parameters of chloroform intoxicated rats. Am-Eur J Sci Res. 2015;10(1):45–9.

57. Ibiam UA, Ama-Udu, Egwu OA, Ugwu OPC. The protective effect of Gongronema latifolium leaf extract against hepatotoxicity of Rambo and Baygon mosquito coil smoke respectively in albino rats. World Appl Sci J. 2015;33(12):1915–22.
58. Odo CE, Nwodo OFC, Joshua PE, Ubani CS, Etim OE, Ugwu OPC. Effects of Bonny light crude oil on anti-oxidative enzymes and total proteins in Wistar rats. Afr J Biotechnol. 2012;11(98):16455–60.
59. Ozioko PC, Ugwu OPC, Chilaka FC, Ude S. Purification and characterization of beta-D-glucosidase from the digestive tracts of an African Giant Snail (Achatina achatina). 2013.
60. Ozioko PC, Ikeyi AP, Ugwu OPC. Review article: cellulases, their substrates, activity and assay methods. Exp. 2013;12(2):778–85.
61. Ezekwe CI, Okorie A, Ugwu OPC, Nwodo OFC, Egba SC. Blood pressure lowering effect of extract of Gongronema latifolium. Res J Pharm Biol Chem Sci. 2014;5(2):952.
62. Alum EU, Ejemot-Nwadiaro RI, Basajja M, Ugwu OPC, Aja PM. Epitranscriptomic alterations induced by environmental toxins: implications for RNA modifications and disease. Genes Environ. 2025;47:14.
63. Okon MB, Olorunnisola SO, Ifie JE, Ugwu OPC, Alum EU. Transgenerational reproductive risks of BPA: epigenetic mechanisms and biomarker applications. A critical review. Environ Epigenet. 2026:dvag010.
64. Okon MB, Ugwu OPC, Ugwu CN, Ogenyi FC, Swase DT, Alum EU, et al. Endocrine disruption rewards: bisphenol-A-induced reproductive toxicity and the precision ameliorative potential of flavonoids in preclinical studies. A systematic review and meta-analysis. Front Toxicol. 2025;7:1687862.
65. Aja PM, Chukwu CA, Ugwu OPC, Ale BA, Agu PC, Deusdedi T, et al. Cucumeropsis mannii seed oil protects against bisphenol A-induced hepatotoxicity by mitigating inflammation and oxidative stress in rats. RPS Pharm Pharmacol Rep. 2024;3(1):rqad033.
66. Udeogu CH, Ugwu MC, Okwara J, Ohale AC, Ike OC, Amadi NC, et al. Levels of maternal serum alpha-fetoprotein and beta human chorionic gonadotropin in HIV seropositive pregnant women attending antenatal care at Nnamdi Azikiwe University Teaching Hospital. J Adv Med Med Res. 2021:32–8.
67. Udeogu CH, Ogbu ISI, Mbachu AN, Ugwu MC, Odo OF, Ugwu O. Apolipoprotein B-48 and tumour necrosis factor-alpha levels in male welders in Nnewi, south-eastern Nigeria. Asian J Res Cardiovasc Dis. 2020;1(1):16–24.
68. Udeogu CH, Ogbu ISI, Mbachu AN, Ugwu MC, Odo OF, Ugwu O. Sex hormones levels in male welders in Nnewi, south-eastern Nigeria. Asian J Biochem Genet Mol Biol. 2020;3(3):22–32.
69. Ogbanshi ME, Agbafor KN, Ominyi CM, Ugwu OPC, Nwali BU. Changes in reproductive functions of adult male rats administered water and salt samples from Okposi and Uburu Nigerian salt lakes. Am-Eur J Toxicol Sci. 2015;7(2):55–62.
70. Ugwu OPC. Pharmacogenomics and toxicogenomics in personalized public health. Curr Pharmacogenomics Pers Med. 2026. doi:10.2174

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