

Impact of air pollution on respiratory health: a clinical review of urban vs rural populations.

Sarah Mwangi*

Department of Medical Microbiology, University of Nairobi, Nairobi, Kenya

Introduction

Air pollution remains a critical public health issue worldwide, with profound impacts on respiratory health. This clinical review compares the effects of air pollution on respiratory health in urban and rural populations, emphasizing the variation in exposure and associated clinical outcomes [1, 2, 3, 4].

In urban areas, populations are frequently exposed to high levels of traffic-related pollutants such as nitrogen dioxide (NO₂), particulate matter (PM_{2.5} and PM₁₀), ozone (O₃), and sulfur dioxide (SO₂). These pollutants are linked to increased incidences of asthma, chronic obstructive pulmonary disease (COPD), and acute respiratory infections. Urban dwellers, especially children and the elderly, are at heightened risk due to sustained exposure to industrial emissions and vehicular exhaust. Clinical data show higher rates of hospital admissions and emergency visits for respiratory conditions in metropolitan regions [5,6, 7].

Conversely, rural populations often encounter air pollutants from biomass fuel combustion, agricultural activities, and dust exposure. Although ambient pollution levels may be lower, indoor air quality is significantly compromised due to the use of solid fuels for cooking and heating. Women and children are disproportionately affected, with a higher prevalence of chronic bronchitis and lower respiratory tract infections in these groups [8, 9, 10].

Clinically, urban residents more frequently present with allergic rhinitis, asthma exacerbations, and reduced lung function metrics. Rural patients, on the other hand, tend to show symptoms of chronic exposure such as persistent cough, dyspnea, and signs of obstructive airway diseases, often underdiagnosed due to limited healthcare access.

Conclusion

while both urban and rural populations are vulnerable to the respiratory effects of air pollution, the nature and source of pollutants differ, resulting in distinct clinical presentations. Public health policies must be tailored to address these specific risks, ensuring equitable healthcare interventions and pollution control strategies across different geographic settings.

References

1. Eamsobhana P, Yong HS. Immunological diagnosis of human angiostrongyliasis due to *Angiostrongylus cantonensis* (Nematoda: Angiostrongylidae). *Int. J. Infect. Dis.* 2009;13(4):425-31.
2. Ishida K, Yoshimura K. Eosinophil responses of permissive and no permissive hosts to the young adult worms of *Angiostrongylus cantonensis*. *Journal of Parasitics.* 1986;72(5):661-71.
3. Mace ML, Olgaard K, Lewin E. New aspects of the kidney in the regulation of fibroblast growth factor 23 (FGF23) and mineral homeostasis. *Int J Mol Sci.* 2020;21(22):8810.
4. Martin A, David V, Quarles LD. Regulation and function of the FGF23/klotho endocrine pathways. *Physiol Rev.* 2012.
5. Morassutti AL, Levert K, Pinto PM, et al. Characterization of *Angiostrongylus cantonensis* excretory–secretory proteins as potential diagnostic targets. *Exp Parasitol.* 2012;130(1):26-31.
6. Nabeshima YI, Imura H. α -Klotho: a regulator that integrates calcium homeostasis. *Am J Nephrol.* 2008;28(3):455-64.
7. Tseng YT, Tsai HC, Sy CL, et al. Clinical manifestations of eosinophilic meningitis caused by *Angiostrongylus cantonensis*: 18 years' experience in a medical center in southern Taiwan. *J Microbiol Immunol Infect.* 2011;44(5):382-9.
8. Urban Jr JF, Madden KB, Svetica A, et al. The importance of Th2 cytokines in protective immunity to nematodes. *Immunological reviews.* 1992;127(1):205-20.
9. Wu Y, Xie L, Wang M, Xiong Q, Guo Y, Liang Y, Li J, Sheng R, Deng P, Wang Y, Zheng R. Mettl3-mediated m6A RNA methylation regulates the fate of bone marrow mesenchymal stem cells and osteoporosis. *Nat Commun.* 2018;9(1):4772.
10. Yu B, Zhao X, Yang C, Crane J, Xian L, Lu W, Wan M, Cao X. Parathyroid hormone induces differentiation of mesenchymal stromal/stem cells by enhancing bone morphogenetic protein signaling. *JBMR.* 2012;27(9):2001-14.

*Correspondence to: Sarah Mwangi, Department of Medical Microbiology, University of Nairobi, Nairobi, Kenya, E-mail: s.mwangi@uonbi.ac.ke

Received: 25-Dec-2024, Manuscript No. AAJCRM-25-166822; Editor assigned: 28-Dec-2024, PreQC No. AAJCRM-25-166822 (PQ); Reviewed: 11-Jan-2025, QC No. AAJCRM-25-166822; Revised: 16-Jan-2025, Manuscript No. AAJCRM-25-166822 (R); Published: 22-Jan-2025, DOI:10.35841/AAJCRM-9.1.248