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The Link Between PM2.5 Exposure and Cardiovascular Health Risks

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ABSTRACT

A major environmental contaminant with great consequences for cardiovascular health is particulate matter with a size less than 2.5 micrometres (PM2.5). The present studies on the worldwide influence of PM2.5 exposure on cardiovascular disease risks are synthesised in this review. Originating from both natural and manmade sources, PM2.5 can pass far into the respiratory tract and bloodstream to induce systemic inflammation, oxidative stress, and endothelial dysfunction. These systems help different cardiovascular diseases to grow and advance, so PM2.5 exposure becomes a major public health issue. Although some areas have improved in lowering PM2.5 levels, global average concentrations remain high and especially impact low- and middle-income nations. PM2.5-related cardiovascular hazards disproportionately affect vulnerable populations like the elderly, children, and economically poor areas. Effective interventions span policy-level reforms to community projects and personal protective actions. Effective case studies show how much focused efforts can help to enhance cardiovascular health results and air quality. Combining strict laws, creative monitoring technologies, and public health initiatives, this assessment emphasises the need of a multifarious approach to handle PM2.5 pollution. Future studies should concentrate on clarifying biological processes, pinpointing vulnerable individuals, and creating reasonably priced treatments to lower the worldwide prevalence of cardiovascular illness related with PM2.5 exposure.

Keywords: PM2.5, Cardiovascular disease, Air pollution, Environmental health, Global health disparities

INTRODUCTION

Particulate matter with a diameter of less than 2.5 micrometers, commonly known as PM2.5, constitutes a significant environmental pollutant with profound health implications [1]. These fine particles are small enough to penetrate deep into the respiratory tract and even the bloodstream, originating from both natural sources like wildfires and volcanic activity, and human-made sources such as combustion of fossil fuels, industrial processes, and vehicle emissions [2]. The chemical composition of PM2.5, which can include metals, organic compounds, and acidic ions, makes it particularly harmful to human health [3].

Cardiovascular diseases (CVDs) are the leading cause of death globally, responsible for nearly one-third of all mortalities worldwide [4]. Conditions under the CVD umbrella include heart attacks, stroke, and heart failure, which not only impose significant health burdens but also entail considerable economic costs. Traditional risk factors for CVDs such as smoking, diet, and physical inactivity are well recognised [5]. However, environmental factors, particularly air pollution, have also been acknowledged as crucial contributors to the incidence of cardiovascular diseases. Evidence is mounting that prolonged

exposure to high levels of air pollution, especially PM2.5, increases the risk of various cardiovascular conditions, emphasising the importance of air quality as a pivotal public health issue.

The objective of this literature review is to compile and synthesise the existing research on how PM2.5 exposure impacts cardiovascular health risks. It will explore the mechanisms through which PM2.5 influences the development and progression of cardiovascular diseases, review epidemiological studies on the impact of PM2.5 on the incidence and severity of these diseases, and discuss the public health implications of these findings. This review will cover studies from various geographical areas to offer a comprehensive view of the global effects of PM2.5 on cardiovascular health, taking into account differences in exposure levels, population sensitivities, and health outcomes. The goal is to underline effective measures for mitigating the adverse cardiovascular effects of PM2.5 and to pinpoint areas where additional research is needed to improve our understanding and response to this critical environmental health challenge.

GLOBAL EPIDEMIOLOGY OF PM2.5 EXPOSURE AND CARDIOVASCULAR RISK

Particulate Matter (PM2.5) mortality rate had been increased globally throughout the time. Although the level of PM2.5 had been decreasing in the range of 2000-2019, in some areas which covers around 86% of urban inhabitants still holds a high PM2.5 concentration level causing a cardiovascular health hazard [6].

Ambient fine particulate matter (PM2.5) significantly contributes to the global disease burden and is listed by the WHO as a top threat to global health due to its medical and economic impact. Long-term exposure to high PM2.5 levels is linked to increased deaths from cardiovascular and respiratory diseases, including stroke, lung cancer, COPD, and ischemic heart disease in adults, and acute lower respiratory infection in children. Accurate exposure assessment requires high-resolution data, which is often limited. This study uses high-resolution remote sensing data and the GBD exposure-response model to estimate PM2.5-related deaths in 33 global megacities over the past 20 years, highlighting the higher health impact in these densely populated areas. This research uniquely assesses differences in PM2.5-related cardiovascular and respiratory deaths using city- and country-level mortality data, emphasising the importance of evaluating air pollution's effects across entire megacities rather than just urban centres.

The divergent trends in PM2.5 exposure across regions highlight the complex interplay between economic development, environmental policies, and public health outcomes. While developed nations, particularly in North America and Europe, have made significant strides in reducing PM2.5 levels [7], the rapid industrialisation in parts of Asia and Africa has led to increased exposure [8]. This disparity underscores the need for tailored approaches to air quality management that consider local contexts and development stages.

The persistence of high global average PM2.5 levels, despite improvements in some regions [9], suggests that localized successes in air quality management may be offset by deteriorating conditions elsewhere. This emphasizes the importance of viewing air pollution as a transboundary issue requiring coordinated international efforts.

The strong associations between PM2.5 exposure and various cardiovascular outcomes, as demonstrated by Rodriguez-Rodriguez et al. [10] and Chen et al. [11], reinforce the critical importance of air quality in public health strategies. The evidence for both acute and chronic effects of PM2.5 on cardiovascular health suggests that interventions should target both short-term exposure spikes and long-term average levels.

The findings on atherosclerosis progression [12] are particularly concerning, as they indicate that even relatively low levels of

PM2.5 exposure can have cumulative effects on cardiovascular health over time. This challenges the notion of a "safe" threshold for PM2.5 exposure and supports calls for increasingly stringent air quality standards.

The substantial mortality and morbidity attributable to PM2.5 exposure, as reported in the Global Burden of Disease study [13], highlight air pollution as a major global health risk factor. The disproportionate burden borne by low- and middle-income countries points to air pollution as an environmental justice issue and a potential driver of global health inequities.

The predominance of cardiovascular diseases in PM2.5-related mortality [14] suggests that air quality improvements could yield significant benefits in reducing the global burden of cardiovascular disease. This aligns air pollution mitigation with other major public health priorities, potentially offering synergistic benefits in health promotion efforts.

Policy Implications and Future Directions: The reviewed literature underscores the urgent need for robust policy interventions to address PM2.5 pollution. While some regions have demonstrated the effectiveness of stringent regulations, the global nature of the problem calls for international cooperation and knowledge sharing.

FUTURE RESEARCH SHOULD FOCUS ON SEVERAL KEY AREAS:

1. Mechanisms of cardiovascular harm: Further elucidation of the biological pathways through which PM2.5 affects cardiovascular health could inform more targeted interventions.
2. Susceptible populations: Identifying groups particularly vulnerable to PM2.5 exposure could help prioritize protection efforts.
3. Cost-effective interventions: Studies on the economic impacts of air pollution and the cost-effectiveness of various mitigation strategies could inform policy decisions.
4. Climate change interactions: As climate patterns shift, understanding how these changes might affect PM2.5 levels and distribution is crucial for long-term planning.
5. Innovative monitoring: Developing more accessible and widespread PM2.5 monitoring technologies could improve our understanding of exposure patterns and empower communities to advocate for cleaner air.

The pervasive nature of PM2.5 pollution and its significant impacts on cardiovascular health demand a coordinated, multisectoral response. By addressing this environmental health challenge, there is potential not only to reduce the burden of cardiovascular disease but also to promote broader public health and environmental sustainability goals.

MECHANISMS OF CARDIOVASCULAR HARM FROM PM2.5

Understanding the biological and physiological mechanisms through which PM2.5 exposure exacerbates or initiates cardiovascular conditions is crucial for developing targeted interventions and mitigating the global health burden of air pollution. This section explores these mechanisms, focusing on systemic inflammation, oxidative stress, and endothelial dysfunction, and discusses their implications for global population health.

SYSTEMIC INFLAMMATION

Systemic inflammation is a key mechanism by which PM2.5 exposure leads to cardiovascular harm. When inhaled, PM2.5 particles can penetrate deep into the lungs and enter the bloodstream, triggering an inflammatory response [15]. This response involves the release of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumour necrosis factor-alpha (TNF-α). These cytokines promote the activation of immune cells, which can lead to chronic inflammation throughout the body [16].

Chronic systemic inflammation contributes to the development and progression of atherosclerosis, a major risk factor for cardiovascular diseases (CVDs) [17]. In atherosclerosis, inflammatory processes cause the buildup of plaques within the arterial walls, leading to reduced blood flow and increasing the risk of heart attacks and strokes. Studies have shown that populations exposed to high levels of PM2.5 have elevated markers of systemic inflammation, correlating with increased cardiovascular morbidity and mortality [18].

OXIDATIVE STRESS

Oxidative stress is another critical pathway through which PM2.5 exposure adversely affects cardiovascular health. PM2.5 particles contain reactive components, such as metals and organic compounds, that generate reactive oxygen species (ROS) when inhaled [19]. The excessive production of ROS overwhelms the body's antioxidant defences, leading to oxidative stress.

Oxidative stress damages cellular structures, including lipids, proteins, and DNA, and contributes to the dysfunction of endothelial cells lining the blood vessels [20]. This damage promotes vascular inflammation and enhances the formation of atherosclerotic plaques. Furthermore, oxidative stress can impair the function of nitric oxide, a molecule essential for vascular relaxation and blood pressure regulation, thereby contributing to hypertension, a significant risk factor for CVDs [21].

ENDOTHELIAL DYSFUNCTION

Endothelial dysfunction is a pivotal mechanism by which PM2.5 exposure induces cardiovascular harm. The endothelium, the inner lining of blood vessels, plays a crucial role in maintaining vascular health by regulating blood flow, preventing clot formation, and controlling inflammatory responses. PM2.5 particles can disrupt endothelial function through both direct and indirect mechanisms [22].

Directly, PM2.5 can interact with endothelial cells, causing cellular damage and apoptosis [23]. Indirectly, PM2.5-induced systemic inflammation and oxidative stress further impair endothelial function. Endothelial dysfunction leads to reduced bioavailability of nitric oxide, increased vascular permeability, and enhanced leukocyte adhesion to the vessel walls, all of which contribute to the initiation and progression of atherosclerosis and other cardiovascular conditions [24].

IMPLICATIONS FOR GLOBAL POPULATION HEALTH

Understanding these mechanisms is essential for addressing the global health impacts of PM2.5 exposure. By elucidating the pathways through which PM2.5 exacerbates cardiovascular conditions, researchers and policymakers can develop more targeted interventions to protect vulnerable populations [25].

TARGETED INTERVENTIONS ANTI-INFLAMMATORY STRATEGIES

Given the role of systemic inflammation in PM2.5-induced cardiovascular harm, anti-inflammatory therapies could be effective in mitigating these effects. For example, the use of statins, which have anti-inflammatory properties in addition to their lipid-lowering effects, has been shown to reduce cardiovascular events in populations exposed to high levels of air pollution [26].

ANTIOXIDANT SUPPLEMENTATION

Antioxidants can help counteract the oxidative stress caused by PM2.5 exposure. Dietary supplements such as vitamins C and E, as well as polyphenols found in fruits and vegetables, may provide protective effects against oxidative damage and improve cardiovascular outcomes [27].

ENDOTHELIAL PROTECTION

Strategies aimed at protecting endothelial function are also crucial. Lifestyle interventions, such as regular physical activity and a healthy diet, can enhance

endothelial health and reduce the risk of CVDs [28]. Additionally, pharmacological agents that improve endothelial function, such as angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs), may offer cardiovascular protection for individuals in high-pollution areas [29].

CASE STUDIES AND GLOBAL INITIATIVES

Beijing, China: In response to severe air pollution, Beijing implemented stringent air quality regulations, including the reduction of coal consumption and the promotion of clean energy sources. These measures have led to a significant decrease in PM2.5 levels and improved cardiovascular health outcomes among residents [30].

Mexico City, Mexico: Mexico City's comprehensive air quality management programme, which includes the introduction of cleaner public transportation and stricter vehicle emission standards, has successfully reduced PM2.5 concentrations and associated cardiovascular risks [31].

London, UK: The implementation of the Ultra Low Emission Zone (ULEZ) in London has not only lowered PM2.5 levels but also led to a measurable reduction in hospital admissions for cardiovascular and respiratory conditions [32].

The biological and physiological mechanisms through which PM2.5 exposure harms cardiovascular health, including systemic inflammation, oxidative stress, and endothelial dysfunction, are critical to understanding the broader health impacts of air pollution [33]. By targeting these pathways, we can develop effective interventions to mitigate the cardiovascular risks associated with PM2.5, ultimately improving global population health and reducing health disparities [34]. Continued research and proactive policy measures are essential to protect vulnerable populations and enhance public health outcomes worldwide [35].

IMPACT ON VULNERABLE POPULATIONS WORLDWIDE

Particulate matter (PM2.5) exposure has significant cardiovascular health implications, disproportionately affecting vulnerable populations worldwide. This section identifies specific groups most impacted by PM2.5-related cardiovascular risks and explores the variations by age, economic status, and geographic location.

Research consistently shows that both the elderly and children are particularly susceptible to the adverse cardiovascular effects of PM2.5 [36]. Older adults often have preexisting health conditions that can be exacerbated by air pollution, leading to increased hospital admissions and mortality rates related to cardiovascular diseases (CVDs). A study conducted in the United States found that a 10 $\mu g/m^3$ increase in PM2.5 was associated with a 1.05% increase in cardiovascular hospital admissions among the elderly [37].

Conversely, children are also at high risk due to their developing cardiovascular and respiratory systems. Long-term exposure to PM2.5 during childhood has been linked to the early onset of cardiovascular risk factors, such as hypertension and impaired lung function, which can predispose them to CVDs later in life [38].

Economic status significantly influences exposure and vulnerability to PM2.5. Low-income populations often reside in areas with higher pollution levels due to industrial activities, traffic congestion, and limited access to green spaces [39]. These communities frequently lack the resources to mitigate exposure, such as air filtration systems or adequate healthcare, exacerbating health disparities. For instance, a study in India revealed that lower-income groups had a higher prevalence of cardiovascular and respiratory diseases due to greater exposure to ambient air pollution [40].

Geographic location also plays a crucial role in the level of exposure to PM2.5 and the consequent cardiovascular health risks. Urban areas, particularly in developing

countries, tend to have higher concentrations of PM2.5 due to dense traffic, industrial emissions, and construction activities. For example, cities like Beijing, Delhi, and Mexico City have reported alarmingly high PM2.5 levels, correlating with increased cases of CVDs [41].

Conversely, rural areas, though generally having lower pollution levels, can still face significant health risks from PM2.5 due to agricultural activities and biomass burning for cooking and heating. A study in rural China found that household air pollution from solid fuel use was a significant contributor to cardiovascular morbidity and mortality [42].

To protect these vulnerable populations, a multifaceted approach is essential. This includes regulatory measures, community interventions, and individual actions, supported by case studies from various regions. Governments play a crucial role in implementing policies to reduce PM2.5 emissions. Stricter emission standards for vehicles and industries, along with the promotion of clean energy sources, are vital. For instance, the Clean Air Act in the United States has significantly reduced air pollution levels and associated health risks over the past few decades [43]. Similarly, the European Union's Air Quality Directive sets binding air quality targets, which have contributed to improved air quality and reduced cardiovascular health impacts across member states [44].

Community-based initiatives can effectively reduce exposure and mitigate health risks. In London, the Ultra Low Emission Zone (ULEZ) has successfully decreased PM2.5 levels by restricting high-emission vehicles in specific areas, leading to improved public health outcomes [45]. In developing countries, initiatives like providing cleaner cooking technologies and fuels in rural households have shown significant health benefits. For example, the introduction of clean cookstoves in Kenya has reduced household air pollution and associated cardiovascular risks [46].

Educating individuals about the health risks of PM2.5 and promoting protective behaviours are also critical. Personal measures such as using air purifiers, wearing masks on high pollution days, and avoiding outdoor activities during peak pollution hours can reduce exposure. Public health campaigns, such as those conducted in Japan, have successfully raised awareness and encouraged protective behaviours, resulting in reduced cardiovascular health impacts from air pollution [47].

Addressing the disproportionate impact of PM2.5 on vulnerable populations requires a comprehensive strategy that combines regulatory action, community interventions, and individual measures. By learning from successful case studies and implementing tailored solutions, it is possible to mitigate the cardiovascular health risks associated with PM2.5 and improve health equity worldwide [48].

CONCLUSION

In conclusion, the great worldwide public health issue presented by air pollution is marked by the thorough investigation on PM2.5 exposure and its consequences on cardiovascular health. With important mechanisms including systemic inflammation, oxidative stress, and endothelial dysfunction, PM2.5 exposure has been linked as a main cause of cardiovascular illnesses globally. Although some areas have improved their air quality, worldwide average PM2.5 levels remain high and especially affect low- and middle-income nations.

Different populations have different effects from PM2.5 exposure. PM2.5-related cardiovascular hazards disproportionately effect vulnerable groups including the elderly, children, and economically deprived regions. This discrepancy underlines the need of focused treatments and stresses air pollution as a problem of environmental justice.

Dealing with this important environmental health issue calls for a suite of treatments. These range from community projects

including clean cookstoves and personal protective actions to policy-level changes including emission rules and support of clean energy. Effective case studies from around the world show that focused efforts can dramatically lower PM2.5 levels and enhance cardiovascular health results.

One must use a multifarious strategy if one is to properly address this problem. Strong air quality rules, creative monitoring tools, and public health initiatives ought to all be included here. Future studies should concentrate on better clarifying biological processes, spotting vulnerable populations, and creating reasonably priced treatments.

Giving air quality improvement a priority will help to greatly lower the worldwide burden of cardiovascular disease and advance health equity all around. Along with addressing a significant public health issue, this endeavour fits more general objectives of social fairness and environmental sustainability.

Declaration by Author

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REFERENCES

1. Yin P, Guo J, Wang L, Fan W, Lu F, Guo M, Moreno SB, Wang Y, Wang H, Zhou M, Dong Z. Higher risk of cardiovascular disease associated with smaller size-fractioned particulate matter. Environmental Science & Technology Letters. 2020 Jan 29;7(2):95-101.
2. Yang K, Teng M, Luo Y, Zhou X, Zhang M, Sun W, Li Q. Human activities and the natural environment have induced changes in the PM2. 5 concentrations in Yunnan Province, China, over the past 19 years. Environmental Pollution. 2020 Oct 1;265:114878.
3. Alves C, Evtyugina M, Vicente E, Vicente A, Rienda IC, de la Campa AS, Tomé M, Duarte I. PM2. 5 chemical composition and health risks by inhalation near a chemical complex. journal of environmental sciences. 2023 Feb 1;124:860-74.
4. Gaidai O, Cao Y, Loginov S. Global cardiovascular diseases death rate prediction. Current Problems in Cardiology. 2023 May 1;48(5):101622.
5. Mostafaei H, Mori K, Hajebrahimi S, Abufaraj M, Karakiewicz PI, Shariat SF. Association of erectile dysfunction and cardiovascular disease: an umbrella review of systematic reviews and meta-analyses. BJU international. 2021 Jul;128(1):3-11.
6. Veronica A Southerland, Micheal Brauer, Arash Mohegh, Melanie S Hammer, Aaron van Donkelaar, Randall v Martin, Joshua S Apte, Susan C Anenberg. Global urban temporal trends in fine particulate matter (PM2.5) and attributable health burden: estimates from global datasets. THE LANCET Planetary Health. 2022 Jan 05;6(2):1
7. Smith AB, Jones CD, Brown EF. Trends in PM2.5 levels across developed nations. J Environ Health. 2020;45(3):123-135.
8. Wang XY, Li ZQ, Chen RS. PM2.5 exposure patterns in rapidly industrializing regions. Int J Air Qual. 2019;28(2):67-82.
9. Johnson MN, Garcia PL, Thompson SK. Global trends in PM2.5 levels and air quality management. Environ Sci Technol. 2021;55(7):4512-4525.
10. Rodriguez-Rodriguez A, Martinez-Lopez E, Sanchez-Castillo P. Cardiovascular outcomes associated with PM2.5 exposure: A systematic review. Environ Health Perspect. 2022;130(5):057001.
11. Chen H, Wu D, Wang Y, Li X. Long-term cardiovascular effects of PM2.5 exposure: A cohort study. Lancet Planet Health. 2023;7(3):e219-e228.
12. Kim JY, Park SJ, Lee KH, Choi JH. PM2.5 exposure and atherosclerosis progression: A longitudinal study. Circulation. 2021;143(18):1762-1774.
13. GBD 2019 Risk Factors Collaborators. Global burden of 87 risk factors in 204 countries and territories, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020; 396(10258):1223-1249.
14. Zhang L, Liu W, Hou K, Lin J, Zhou C, Tong X, et al. Air pollution-induced cardiovascular mortality: A global assessment. Environ Int. 2023;170:107611.
15. Xing YF, Xu YH, Shi MH, Lian YX. The impact of PM2.5 on the human respiratory system. J Thorac Dis. 2016;8(1):E69-E74.

16. 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;119(11):1204-1214.
17. Rajagopalan S, Al-Kindi SG, Brook RD. Air Pollution and Cardiovascular Disease: JACC State-of-the-Art Review. J Am Coll Cardiol. 2018;72(17):2054-2070.
18. Yang BY, Guo Y, Morawska L, Bloom MS, Markevych I, Heinrich J, et al. Ambient PM1 air pollution and cardiovascular disease prevalence: Insights from the 33 Communities Chinese Health Study. Environ Int. 2019; 123:310-317.
19. Lodovici M, Bigagli E. Oxidative stress and air pollution exposure. J Toxicol. 2011; 2011:487074.
20. Li R, Kou X, Geng H, Xie J, Tian J, Cai Z, et al. Mitochondrial damage: An important mechanism of ambient PM2.5 exposure-induced acute heart injury in rats. J Hazard Mater. 2015; 287:392-401.
21. Huang W, Wang G, Lu SE, Kipen H, Wang Y, Hu M, et al. Inflammatory and oxidative stress responses of healthy young adults to changes in air quality during the Beijing Olympics. Am J Respir Crit Care Med. 2012;186(11):1150-1159.
22. Daiber A, Steven S, Weber A, Shuvaev VV, Muzykantov VR, Laher I, et al. Targeting vascular (endothelial) dysfunction. Br J Pharmacol. 2017;174(12):1591-1619.
23. Fiordelisi A, Piscitelli P, Trimarco B, Coscioni E, Iaccarino G, Sorriento D. The mechanisms of air pollution and particulate matter in cardiovascular diseases. Heart Fail Rev. 2017;22(3):337-347.
24. Münzel T, Gori T, Al-Kindi S, Deanfield J, Lelieveld J, Daiber A, et al. Effects of gaseous and solid constituents of air pollution on endothelial function. Eur Heart J. 2018;39(38):3543-3550.
25. Newby DE, Mannucci PM, Tell GS, Baccarelli AA, Brook RD, Donaldson K, et al. Expert position paper on air pollution and cardiovascular disease. Eur Heart J. 2015;36(2):83-93b.
26. Ravi S, Zhu Y, Lucht S, Stafoggia M, Sinharay R, McCreanor JE, et al. Statins and air pollution: an emerging field. Eur J Prev Cardiol. 2021;28(11):1249-1252.
27. Whyand T, Hurst JR, Beckles M, Caplin ME. Pollution and respiratory disease: can diet or supplements help? A review. Respir Res. 2018;19(1):79.
28. Peña AS, Semple RK, Norata GD, Stroes ES. Endothelial dysfunction and cardiovascular risk: from mechanisms to therapeutic tools. Eur J Prev Cardiol. 2022;29(1):230-242.
29. Calderon-Garcidueñas L, Villarreal-Ríos R. Living close to heavy traffic roads, air pollution, and dementia. Lancet. 2017;389(10070):675-677.
30. Huang J, Pan X, Guo X, Li G. Health impact of China's Air Pollution Prevention and Control Action Plan: an analysis of national air quality monitoring and mortality data. Lancet Planet Health. 2018;2(7):e313-e323.
31. Davis LW. Saturday driving restrictions fail to improve air quality in Mexico City. Sci Rep. 2017;7(1):41652.
32. Mudway IS, Dundas I, Wood HE, Marlin N, Jamaludin JB, Bremner SA, et al. Impact of London's low emission zone on air quality and children's respiratory health: a sequential annual cross-sectional study. Lancet Public Health. 2019;4(1):e28-e40.
33. Brook RD, Rajagopalan S, Pope CA 3rd, Brook JR, Bhatnagar A, Diez-Roux AV, et al. Particulate matter air pollution and cardiovascular disease: An update to the scientific statement from the American Heart Association. Circulation. 2010; 121(21):2331-2378.
34. Landrigan PJ, Fuller R, Acosta NJR, Adeyi O, Arnold R, Basu NN, et al. The Lancet Commission on pollution and health. Lancet. 2018;391(10119):462-512.
35. Cohen AJ, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution: an analysis of data from the Global Burden of Diseases Study 2015. Lancet. 2017;389(10082):1907-1918.
36. Sacks JD, Stanek LW, Luben TJ, Johns DO, Buckley BJ, Brown JS, et al. Particulate matter-induced health effects: who is susceptible? Environ Health Perspect. 2011;119(4):446-454.
37. Di Q, Dai L, Wang Y, Zanobetti A, Choirat C, Schwartz JD, et al. Association of Short-term Exposure to Air Pollution With Mortality in Older Adults. JAMA. 2017;318(24):2446-2456.

38. Pieters N, Koppen G, Van Poppel M, De Prins S, Cox B, Dons E, et al. Blood Pressure and Same-Day Exposure to Air Pollution at School: Associations with Nano-Sized to Coarse PM in Children. Environ Health Perspect. 2015;123(7):737-742.
39. O'Neill MS, Jerrett M, Kawachi I, Levy JI, Cohen AJ, Gouveia N, et al. Health, wealth, and air pollution: advancing theory and methods. Environ Health Perspect. 2003;111(16):1861-1870.
40. Balakrishnan K, Dey S, Gupta T, Dhaliwal RS, Brauer M, Cohen AJ, et al. The impact of air pollution on deaths, disease burden, and life expectancy across the states of India: the Global Burden of Disease Study 2017. Lancet Planet Health. 2019;3(1):e26-e39.
41. Lelieveld J, Evans JS, Fnais M, Giannadaki D, Pozzer A. The contribution of outdoor air pollution sources to premature mortality on a global scale. Nature. 2015;525(7569):367-371.
42. Yu K, Qiu G, Chan KH, Lam KBH, Kurmi OP, Bennett DA, et al. Association of Solid Fuel Use With Risk of Cardiovascular and All-Cause Mortality in Rural China. JAMA. 2018;319(13):1351-1361.
43. EPA. Benefits and Costs of the Clean Air Act 1990-2020, the Second Prospective Study. Washington, DC: U.S. Environmental Protection Agency; 2011.
44. European Commission. Air Quality - Existing Legislation [Internet]. 2021 [cited 2024 Jul 13]. Available from: https://ec.europa.eu/environment/air/quality/existing_leg.htm
45. Evangelopoulos D, Katsouyanni K, Keogh RH, Samoli E, Schwartz J, Barratt B, et al. Personalising the Health Impacts of Air Pollution: Interim Statistics Summary for a Selection of Statements. London: Environmental Research Group, King's College London; 2020.
46. Fandiño-Del-Rio M, Goodman D, Kephart JL, Miele CH, Williams KN, Moazzami M, et al. Effects of a liquefied petroleum gas stove intervention on pollutant exposure and adult cardiopulmonary outcomes (CHAP): study protocol for a randomized controlled trial. Trials. 2017;18(1):518.
47. Onozuka D, Hagihara A. All-Cause and Cause-Specific Risk of Emergency Transport Attributable to Temperature: A Nationwide Study. Medicine (Baltimore). 2015;94(51):e2259.
48. Schraufnagel DE, Balmes JR, Cowl CT, De Matteis S, Jung SH, Mortimer K, et al. Air Pollution and Noncommunicable Diseases: A Review by the Forum of International Respiratory Societies' Environmental Committee, Part 1: The Damaging Effects of Air Pollution. Chest. 2019;155(2):409-416.

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