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Air pollution and cardiovascular disease: a window of opportunity

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Abstract

The recent publication of *The Lancet* Commission on pollution and health is a watershed moment for one of the greatest challenges to cardiovascular health. In this Comment article, we discuss the global burden of air pollution on cardiovascular health.

Cardiovascular disease (CVD) is the leading cause of death worldwide, responsible for >17 million premature deaths in 2016 (REF. 1). Notably, >3 million of these deaths (19% of all cardiovascular deaths) are attributable to air pollution¹. *The Lancet* Commission on Pollution and Health aims to raise global awareness of the effects of pollution on overall health through six recommendations². In this Comment article, we evaluate how these recommendations can reduce the harmful effects of air pollution on cardiovascular health.

Air pollution exposures are often ascribed to two categories: household pollution and ambient pollution. Approximately 3 billion people are exposed to household air pollution emitted from cooking and heating with inefficient stoves that burn biomass and coal fuels^{1,2}. Nearly all individuals worldwide are exposed to ambient air pollution that originate from traffic, industrial facilities, and agricultural burning^{1–4}. In 2016, ambient air pollution and household air pollution were responsible for 2.3 million and 1.2 million cardiovascular deaths, respectively¹. The overwhelming majority of this burden is shouldered by low-income and middle-income countries. Mortality is also higher in certain susceptible subpopulations, including individuals of advanced age and lower socioeconomic status, and those with other traditional cardiovascular risk factors^{1, 3, 5}.

Fine particulate matter air pollution (particles >2.5 µm in diameter, or PM_{2.5}) is the pollutant with the most compelling observational and experimental evidence of association with increased risk of cardiovascular mortality^{1–6}. PM_{2.5} is composed of elemental carbon, transition metals, sulfates, nitrates, and complex organic molecules, and originates from household wood and coal burning, industrial facilities, vehicle emissions, and agricultural burning^{3–6}. These small particles can enter lung alveoli, triggering the release of proinflammatory mediators and vasoactive molecules, which result in oxidative stress, systemic inflammation, endothelial dysfunction, vasoconstriction, and autonomic

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Competing interests statement

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imbalance³⁻⁶. Consequently, acute exposures to PM_{2.5} can increase the risk of acute coronary syndrome, heart failure, venous thromboembolism, arrhythmia, and cardiac arrest³⁻⁶. Furthermore, chronic exposure to PM_{2.5} can increase the risk of developing hypertension, type 2 diabetes, and atherosclerosis, together increasing the risk of cardiovascular death. Conversely, reducing exposure to air pollution has been shown to decrease the immediate risk of acute coronary syndromes, arrhythmias, heart failure, and the long-term development and progression of atherosclerosis^{3,5,6}. The Clean Air Act increased regulation of industrial and vehicular particulate matter emissions, preventing an estimated 130,000 myocardial infarction events in the USA in 2010 (REF. 7).

The dose-response relationship between air pollution and CVD has been described using integrated exposure-response curves, which estimate the relative risk of different cardiovascular events at various levels of PM_{2.5} exposure⁸. The risk of ischaemic heart disease and stroke associated with PM_{2.5} exposure is supralinear; that is, the relationship is strongest at lower concentrations, and becomes weaker as air pollution increases. The shape of this curve implies that substantial health benefits will accrue only from interventions that reduce exposures to low levels. Consequently, improvements in cardiovascular health might be more difficult to achieve in communities suffering a greater burden of air pollution.

The Lancet Commission provided detailed recommendations for reducing air pollution exposures across populations, including the government-driven development of comprehensive, data-driven, and publicly available action plans with clear targets for combating pollution². A crucial part of this plan is the implementation and enforcement of regulations, subsidies, and penalties to shift economies towards affordable and renewable energy sources, cleaner vehicles, and reduced industrial emissions. Investments must be made to develop health-tracking systems with high-quality metrics to monitor air pollution exposure levels, the burden of pollution-attributable CVD, and the effect of interventions. Finally, a dedicated international funding pool is needed to finance research and interventions, particularly in low-resource settings.

Implementing the Commission's recommendations will do much to address pollution-attributable CVD at the level of populations and organizations. However, these recommendations must be paired with programmes that empower individuals to protect themselves from the harmful effects of air pollution within their own microenvironment (FIG. 1). Clinicians and health-care workers can be taught to screen individuals for pollution exposures based on household use of solid fuels and proximity to urban or industrial centres. Pollution exposures can be quantified in a variety of ways, including ground-based monitoring, satellite measurements, personal detection systems, and regression models that predict exposures based on home fuel use, proximity to traffic, and other factors.

To date, no randomized trials have investigated the efficacy of pollution-reducing interventions on cardiac outcomes. However, data from studies investigating intermediate end points (such as blood pressure and inflammatory markers) support the use of several interventions^{3,5}. Individuals identified as particularly vulnerable to pollution-attributable CVD can be provided with clean-burning stoves and fuel, improved household ventilation devices, and air-filtration devices, and can be educated on the use of automated air pollution

alert networks and strategies to reduce exposures to traffic and industrial emissions. Additionally, clinicians should emphasize treatment of traditional cardiovascular risk factors to these at-risk individuals. Such interventions often benefit from partnership with governments, nongovernmental organizations, or insurance providers.

Extensive research is still needed to quantify exposures and validate interventions to reduce the burden of pollution-attributable CVD². Important future tasks include: the development of a model to estimate individual absolute risk of cardiac events based on pollution exposure levels and co-morbidities; calculation of how much pollution exposure levels must be reduced in order to achieve cardiovascular benefits; and determination of the relationship between cardiovascular health and other air pollutants, such as nitrogen dioxide, carbon monoxide, sulfur dioxide, and other volatile organic compounds. Furthermore, the efficacy, feasibility, cost-effectiveness, and co-benefits (such as increased productivity or reduced greenhouse gas emissions) of interventions to reduce ambient and household air pollution exposures should be calculated, and both health and environmental benefits of reducing pollution should be characterized. To guide these investigations, a consortium should provide standard research methods and facilitate data aggregation and meta-analysis⁹.

The burden of pollution-attributable disease is shouldered disproportionately by low-income and middle-income countries, which account for >99% of deaths due to household air pollution and 89% of deaths due to ambient air pollution². Moreover, within countries, the burden of pollution-associated disease is most concentrated among children, women, the elderly, and the poor. This disease burden is an infringement on the human right to a healthy environment, and a threat to equal protection of environmental laws and regulations². The global health community has a critical window of opportunity to invest in research, regulation, and intervention, particularly among low-resource communities, to reduce the global burden of pollution-attributable CVD.

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References

1. GBD 2016 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 390, 1345–1422 (2017).28919119
2. Landrigan PJ The Lancet Commission on pollution and health. *Lancet* 10.1016/S0140-6736(17)32345-0 (2017).
3. Brook RD Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 121,2331–2378 (2010).20458016
4. Cosselman KE , Navas-Acien A & Kaufman JD Environmental factors in cardiovascular disease. *Nat. Rev. Cardiol* 12, 627–642 (2015).26461967
5. Newby DE Expert position paper on air pollution and cardiovascular disease. *Eur. Heart J* 36, 83–93 (2015).25492627
6. Ruckerl R , Schneider A , Breitner S , Cyrys J & Peters A Health effects of particulate air pollution: a review of epidemiological evidence. *Inhal. Toxicol* 23, 555–592 (2011).21864219

7. Environmental Protection Agency Office of Air and Radiation. The Benefits and Costs of the Clean Air Act from 1990 to 2020, Final Report — Rev. A. ERA <https://www.epa.gov/sites/production/files/2015-07/documents/summaryreport.pdf> (2011).
8. Shin HH Meta-analysis methods to estimate the shape and uncertainty in the association between long-term exposure to ambient fine particulate matter and cause-specific mortality over the global concentration range. *Risk Analysis* 36, 1813–1825 (2016).26040916
9. Anderson HR Implications for the science of air pollution and health. *Lancet Respir. Med* 5, 916–918 (2017).29066089

Figure 1]. Conceptual diagram illustrating a combined population-level and individual-level approach to mitigating air pollution exposures and protecting cardiovascular health.

In addition to *The Lancet* Commission's recommendations² for population-level interventions led by governments and global alliances, we advocate individual-level interventions, including screening, reductions in emissions and exposure, and relevant funding mechanisms. RCT, randomized, controlled trial.