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INVITED EDITORIAL

From heavy smog to healthy hearts: China's clean air act as a blueprint for cardiovascular disease prevention

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This editorial refers to 'Cardiovascular disease averted by China's clean air act and the potential benefit of further air pollution intervention: evidence based on the parametric G-computation', by J. Xie *et al.*, https://doi.org/10.1093/eurjpc/zwag039.

Air pollution has evolved from being perceived primarily as a respiratory hazard into one of the most important cardiovascular health risks of our time. The epidemiological signal is overwhelming. Ambient air pollution contributes to an estimated 8.3 million deaths annually worldwide,[1] with more than half of these deaths attributable to cardiovascular causes, predominantly ischaemic heart disease and stroke. In recent global evaluations of mortality risk factors, ambient air pollution now ranks second, surpassed only by arterial hypertension.[2]

When disability-adjusted life years (DALYs) are considered, air pollution has been ranked as the leading contributor to global disease burden. These numbers are not abstract statistics; they reflect a profound and preventable cardiovascular health burden affecting billions of individuals.[3]

Despite this, cardiovascular prevention frameworks still focus on traditional risk factors such as hypertension, dyslipidaemia, smoking, diabetes, obesity, and sedentary lifestyle. While these remain critically important, the failure to fully incorporate environmental exposures into prevention strategies represents a substantial gap. Unlike lifestyle choices, exposure to polluted air is largely involuntary, shaped by energy systems, transport policies, and urban design. Addressing air pollution, therefore, represents not only an environmental objective but one of the most powerful population-level cardiovascular intervention options available today.

Mechanistically, air pollution fulfils all criteria of a causal cardiovascular risk factor. Fine particulate matter ($PM_{2.5}$) and ultrafine particles trigger oxidative stress, promote systemic and vascular inflammation, and impair endothelial function by reducing nitric oxide bioavailability.[4] Activation of NADPH (nicotinamide adenine dinucleotide phosphate) oxidases, lipid oxidation, dysfunctional HDL, and immune dysregulation accelerate atherosclerosis progression and contribute to plaque instability and thrombosis.[5] Autonomic imbalance and neuroinflammation further compound cardiovascular vulnerability. These pathways do not operate in isolation but often interact with co-exposures such as noise and heat, amplifying vascular injury.[6,7] The biological coherence of these mechanisms reinforces the epidemiological evidence: air pollution is a cardiovascular toxin because it directly disrupts vascular homeostasis.

Against this background, the study by Xie and colleagues provides timely and policy-relevant evidence on this issue.[8] The authors evaluate the cardiovascular impact of China's Clean Air Act (CCAA) using data from the Sichuan sub-cohort of the China Multi-Ethnic Cohort, comprising 21 592 adults aged 30–79 years recruited between 2018 and 2019 and followed through the end of 2022. Incident cardiovascular events were identified through provincial hospital surveillance systems using

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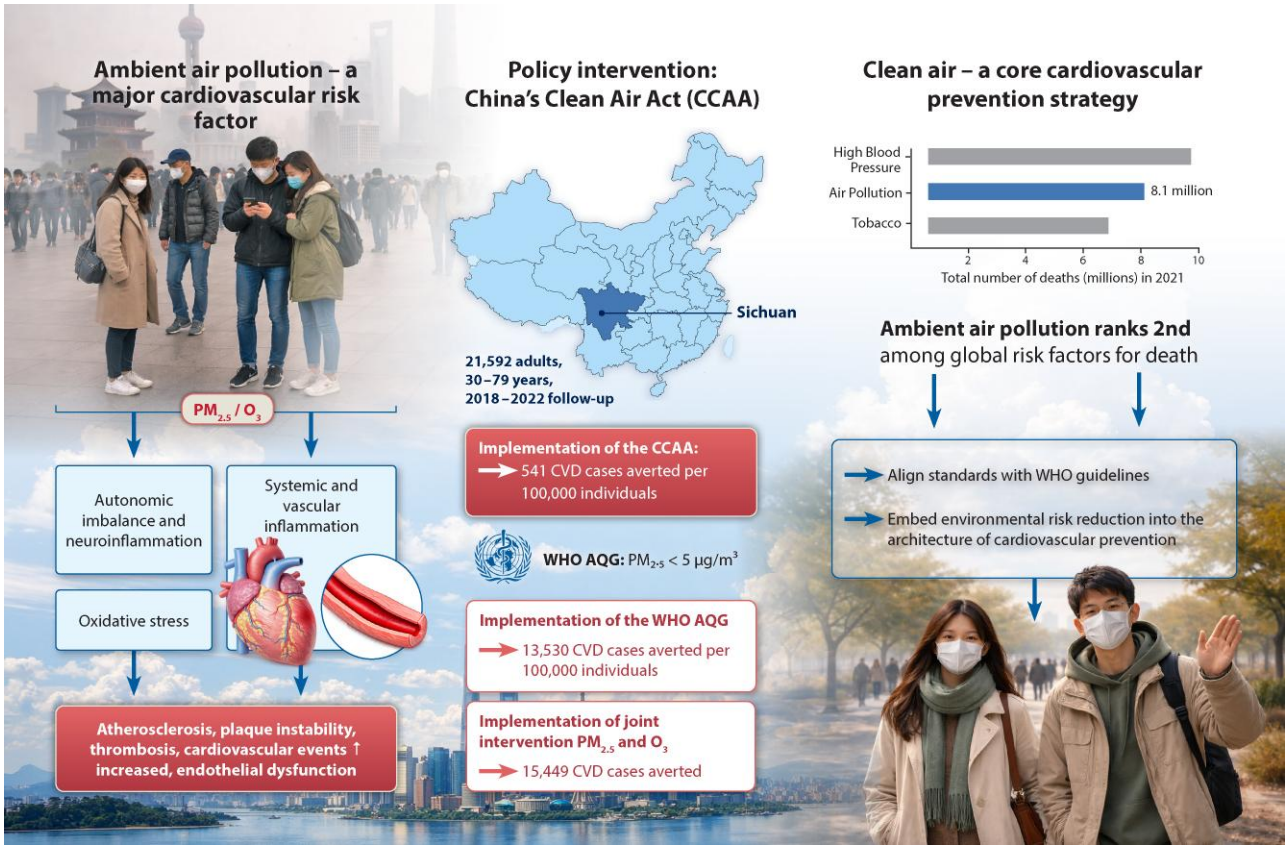

Figure 1 Clean air policy as cardiovascular prevention. Ambient air pollution, particularly $PM_{2.5}$ µm and ozone (O_3), is a major environmental driver of cardiovascular disease. Exposure induces oxidative stress, systemic inflammation, autonomic imbalance, and endothelial dysfunction, promoting atherosclerosis, plaque instability, thrombosis, and cardiovascular events. China's Clean Air Action Plan (CCAA) demonstrates that regulatory air-quality interventions can substantially reduce cardiovascular burden. In a cohort of 21 592 adults aged 30–79 years (2018–2022), improved air quality was associated with approximately 541 fewer cardiovascular disease cases per 100 000 individuals. Modelling further indicates that aligning standards with the WHO $PM_{2.5}$ guideline (<5 µg/m^3) could prevent substantially more events, highlighting clean air policy as a powerful strategy for population-level cardiovascular prevention.[8]

ICD-10 codes. Importantly, the investigators applied parametric g-computation to simulate counterfactual scenarios and estimate the number of cardiovascular events prevented under the CCAA, as well as the number of additional cases that could be averted under stricter pollution-control strategies targeting $PM_{2.5}$ and ozone.

This modelling framework moves beyond conventional association studies. By estimating cases averted per 100 000 individuals under defined intervention scenarios, the authors translate epidemiological findings into policy-relevant metrics. Such an approach provides decision-makers with a tangible estimate of the health dividend associated with environmental regulation.

The results are striking.[8] The authors estimate that implementation of the CCAA was associated with 541 cardiovascular cases averted per 100 000 individuals (95% CI 184–914) attributable to reductions in $PM_{2.5}$. When more ambitious scenarios were simulated, particularly threshold interventions aligning pollutant levels with WHO Air Quality Guidelines, the potential gains were substantially larger. Limiting $PM_{2.5}$ and ozone to WHO guideline levels was associated with 13 530 cases averted per 100 000 for $PM_{2.5}$, 6498 for ozone, and 15 449 for combined $PM_{2.5}$ and ozone interventions (*Figure 1*). These findings underscore a crucial principle: policy matters, and more ambitious targets yield disproportionately greater cardiovascular benefits.

Several aspects of this study are noteworthy. First, the focus on preventing cases provides a language that resonates with health ministries and finance departments. Second, the comparison of multiple intervention strategies, threshold, ratio, and phased reductions, reflects real-world regulatory pathways. Third, the results reinforce the concept of a near-linear exposure–response relationship at contemporary pollution levels, consistent with broader burden analyses suggesting that health benefits accrue even at relatively low concentrations.[1]

As with all modelling studies based on observational data, certain limitations must be acknowledged. The validity of parametric g-computation depends on correct model specification and adequate adjustment for confounders. Residual confounding and unmeasured time-varying factors cannot be excluded. Exposure assessment relied on ambient concentrations at residential locations, which may not fully capture individual-level

exposures influenced by occupational settings, commuting patterns, and indoor environments. Furthermore, the analysis was based on a provincial sub-cohort; replication across diverse regions of China and extended follow-up would strengthen generalisability and allow better assessment of long-term cumulative benefits.

Nevertheless, these limitations do not diminish the central message. Rather, they highlight the need for continued refinement. Future research should extend these analyses nationally, integrate multi-pollutant and multi-exposure frameworks consistent with the exposome concept, and incorporate intermediate vascular phenotypes and biomarkers to further strengthen causal inference. Attention should be given to vulnerable populations, including older adults, individuals with established cardiovascular disease, diabetes, and socioeconomically disadvantaged groups, who may derive disproportionate benefit from pollution reduction.

In our recent comprehensive expert review on environmental risk factors and cardiovascular disease ('Environmental risk and CVD'), we emphasised that environmental exposures must be embedded within mainstream cardiovascular prevention.[9] The findings by Xie and colleagues provide compelling empirical support for this perspective: clean air is not a peripheral environmental goal but a core strategy for cardiovascular disease prevention.

Recent modelling further highlights the magnitude of potential health gains. Lelieveld and colleagues estimated that approximately 5.1 million premature deaths annually are attributable to air pollution from fossil fuel combustion, suggesting that a global fossil fuel phase-out and transition to clean energy could prevent a substantial share of this burden. Importantly, more than half of these deaths arise from cardiometabolic diseases, particularly ischaemic heart disease and stroke.[1] Fossil fuel emissions account for roughly 82% of avoidable mortality from anthropogenic air pollution, underscoring the enormous public health potential of reducing their use.

Real-world policy evidence supports this view. Satellite observation-based analyses indicate that China's air-quality policies have already prevented ~655 000 deaths annually compared with moderate mitigation scenarios and up to 1.28 million deaths per year compared with a scenario without emission controls.[10]

Integrating environmental health into cardiovascular prevention is overdue. Clean air policy saves hearts, and must become an integral pillar of modern preventive cardiology.

Author contributions

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