



Air Pollution as a Cardiovascular Risk Factor: What a Cardiologist Can Legitimately Advise a Patient to Do

Alessio Tortorella^{1*}, Ida Vestergaard-Krog² and Rui Manuel Bragança³

¹Department of Environmental Epidemiology, Campanian Heart Centre, Naples, Italy

²Department of Preventive Cardiology, Vejle Heart Centre, Vejle, Denmark

³Centre for Public Health, Vouga Heart Centre, Aveiro, Portugal

*Correspondence to: Alessio Tortorella, Department of Environmental Epidemiology, Campanian Heart Centre, Naples, Italy

E-mail: a.tortorella@chc-naples.it

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Abstract

Ambient air pollution is now estimated to cause about 4.2 million deaths worldwide each year, of which roughly 2.94 million -- nearly 70% -- are cardiovascular: 1.9 million from ischemic heart disease and 0.9 million from stroke. A 2012 meta-analysis of short-term exposure found significant, same-week increases in myocardial infarction risk for particulate matter, nitrogen dioxide, sulfur dioxide, and carbon monoxide (relative risk 1.006-1.048 per pollutant increment), and a 2013 meta-analysis found comparable increases in heart failure hospitalization and mortality (0.46-3.52% per increment). Long-term cohort data from the European ESCAPE Project (100,166 participants) found the coronary-event hazard associated with fine particulate matter (PM_{2.5}) did not disappear below the European Union's former annual exposure limit (HR 1.18, 95% CI 1.01-1.39, restricted to exposures under 25 µg/m³), and the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) linked higher long-term exposure to faster coronary artery calcium progression, a marker of subclinical atherosclerosis. Set against this evidence, what an individual cardiologist can responsibly tell a patient is narrower than the epidemiology might suggest: a single 40-person randomized trial found indoor HEPA filtration lowered systolic blood pressure by 3.4 mmHg, but no trial has tested whether personal protective measures -- filtration, masks, or activity timing -- reduce hard cardiovascular events, and the American Heart Association's own 2024 policy statement frames these as reasonable adjuncts for higher-risk individuals rather than the primary solution, reserving that role for population-level air-quality policy. This review sets out that evidence, and that honest gap between population-level causation and individual-level actionability, without prescribing a diet, exercise, or supplement regimen no trial has shown to counteract ambient exposure.

Keywords: Air pollution; Particulate matter; Cardiovascular disease; Myocardial infarction; Heart failure; Preventive cardiology

Introduction

Cardiologists are accustomed to risk factors a patient can act on directly -- blood pressure, LDL cholesterol, glycemic control, tobacco use. Ambient air pollution belongs to a different category. The exposure that drives its cardiovascular

harm is set largely by where a patient lives, works, and commutes, by regional industry and traffic density, and by national regulation -- not by an individual choice made in a clinic visit. Yet the size of the association is too large to set aside: air pollution is now estimated to be responsible for millions of cardiovascular deaths every year, more than most

single risk factors a cardiology clinic screens for routinely.

This creates an uncomfortable asymmetry. The evidence that ambient air pollution causes cardiovascular disease, at a population level, is now about as strong as evidence in cardiovascular epidemiology gets: multiple large cohorts, a plausible and partly elucidated mechanism, and a dose-response relationship documented across very different exposure ranges and study designs. The evidence that any specific action available to an individual patient meaningfully reduces their own cardiovascular risk from air pollution is, by contrast, thin -- confined mostly to small trials of surrogate markers rather than outcome trials of myocardial infarction, stroke, or death.

This review sets out what is actually known about air pollution and cardiovascular disease, then asks the question a treating cardiologist actually faces: given that exposure is largely structural, what, if anything, can be legitimately recommended to an individual patient sitting in front of them.

The evidence base establishing air pollution as a cardiovascular, rather than purely respiratory, concern is now two decades old. A widely cited 2004 reanalysis of the American Cancer Society's Cancer Prevention Study II cohort -- roughly 500,000 adults followed from 1982 to 1998 -- found each 10 $\mu\text{g}/\text{m}^3$ increase in long-term PM_{2.5} exposure associated with a 12% increase in cardiovascular mortality (95% CI 8-15%) and an 18% increase specifically in ischemic heart disease mortality (95% CI 14-23%), alongside a 13% increase in deaths from arrhythmia, heart failure, or cardiac arrest (RR 1.13, 95% CI 1.05-1.21) [10]. That single cohort reanalysis, alongside the accumulating evidence summarized below, was substantial enough that the American Heart Association issued a dedicated scientific statement in 2004 and updated it in 2010, concluding that the overall body of evidence was consistent with a causal relationship between PM_{2.5} exposure and cardiovascular morbidity and mortality [3]. Two decades on, the cardiovascular case is no longer contested; what remains genuinely unsettled is set out in the final Results section below.

That 2010 update also set out the mechanistic pathways proposed to link inhaled particulate matter to cardiovascular events: systemic inflammation and oxidative stress triggered by deposited particles, autonomic nervous system imbalance favoring sympathetic activation, endothelial dysfunction impairing normal vascular tone, and a shift toward a prothrombotic state -- pathways that, in combination, are consistent with both the acute triggering of events described below and the slower atherosclerotic progression this review returns to later [3]. None of these mechanistic pathways implies an action available to an individual patient beyond reducing their own exposure, which is the question this review returns to after setting out the epidemiological evidence in full.

Material and Methods

This is a narrative review, not a systematic review with a pre-registered protocol. Sources were identified through the epidemiological and clinical literature on ambient air pollution and cardiovascular disease, prioritizing large meta-analyses and cohort studies with published, peer-reviewed effect estimates, alongside the current World Health Organization air-quality guidelines and the most recent American Heart Association scientific and policy statements on the subject. All percentages, hazard ratios, relative risks, and confidence intervals reported here are taken directly from the cited sources; none has been invented, estimated, or extrapolated. Where a source reported a finding as not statistically significant, that is stated explicitly rather than omitted. A second, separate question this review asks -- what follows for an individual patient -- was assessed against a deliberately stricter standard: a measure is described here as evidence-supported for an individual only where a randomized or otherwise controlled human study was identified testing that specific measure, and the type of endpoint tested (a surrogate marker such as blood pressure, versus a hard cardiovascular outcome) is stated explicitly rather than left to be inferred from the strength of the underlying population-level epidemiology.

Results

The scale of the problem

Ambient air pollution is estimated to have caused about 4.2 million deaths worldwide in 2019, of which roughly 2.94 million -- close to 70% -- were cardiovascular: 1.9 million attributed to ischemic heart disease and 0.9 million to stroke [1]. Figure 1 sets this breakdown out directly. Cardiovascular disease is therefore not a secondary or incidental consequence of air pollution exposure; on these estimates it is the single largest category of harm attributed to it, larger than the combined respiratory and cancer mortality burden usually associated with the exposure in public discussion.

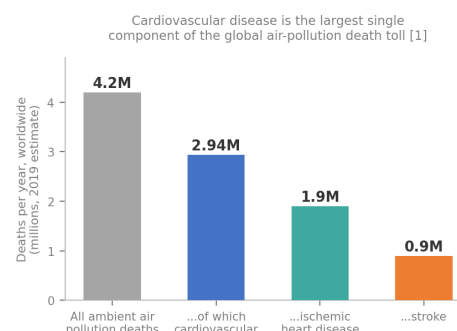


Figure 1: Global deaths attributable to ambient air pollution, and the cardiovascular share, 2019 estimate [1].

Short-term exposure can trigger acute events

A 2012 systematic review and meta-analysis by Mustafic et al. pooled short-term (up to seven-day) exposure studies and found statistically significant increases in myocardial infarction risk for particulate matter, nitrogen dioxide, sulfur dioxide, and carbon monoxide, though the size of the effect varied considerably by pollutant: relative risk 1.025 (95% CI 1.015-1.036) per 10 $\mu\text{g}/\text{m}^3$ increase in PM_{2.5}, 1.006 (1.002-1.009) per 10 $\mu\text{g}/\text{m}^3$ increase in PM₁₀, 1.011 (1.006-1.016) per 10 $\mu\text{g}/\text{m}^3$ increase in NO₂, 1.010 (1.003-1.017) per 10 $\mu\text{g}/\text{m}^3$ increase in SO₂, and 1.048 (1.026-1.070) per 1 ppm increase in CO; ozone showed no significant association (RR 1.003, 95% CI 0.997-1.010) [4]. Figure 2 sets these six estimates out together. A comparable pattern holds for heart failure: a 2013 meta-analysis by Shah et al. found same-day increases in heart failure hospitalization or mortality of 0.46-3.52% per pollutant increment, again significant for every pollutant studied except ozone [7]; the full heart-failure estimates are reported in detail, and set out graphically, later in this section.

Long-term exposure carries chronic risk, without an evident safe floor

The European ESCAPE Project pooled 11 cohorts totalling 100,166 participants followed for a mean of about 11.5 years, during which 5,157 coronary events occurred. Long-term PM_{2.5} exposure was associated with a hazard ratio of 1.13 (95% CI 0.98-1.30) per 5 $\mu\text{g}/\text{m}^3$ across the whole cohort; restricted to participants whose exposure never exceeded the European Union's former annual limit of 25 $\mu\text{g}/\text{m}^3$, the association strengthened to 1.18 (95% CI 1.01-1.39) and became statistically significant [5]. Figure 3 sets these two estimates side by side. PM₁₀ showed a comparable pattern, HR 1.12 (95% CI 1.01-1.25) per 10 $\mu\text{g}/\text{m}^3$ [5]. The finding that risk did not attenuate, and if anything strengthened, at exposures below a regulatory limit is one of the more consequential results in this literature: it implies no exposure threshold below which long-term PM_{2.5} is cardiovascularly inert, a conclusion consistent with the World Health Organization's decision to halve its own annual PM_{2.5} guideline from 10 to 5 $\mu\text{g}/\text{m}^3$ in 2021 [2]. Table 1 sets out that full revision alongside the 2005 guideline it replaced.

Pollutant	2005 annual guideline	2021 annual guideline
PM _{2.5}	10 $\mu\text{g}/\text{m}^3$	5 $\mu\text{g}/\text{m}^3$
PM ₁₀	20 $\mu\text{g}/\text{m}^3$	15 $\mu\text{g}/\text{m}^3$
NO ₂	40 $\mu\text{g}/\text{m}^3$	10 $\mu\text{g}/\text{m}^3$

Table 1: WHO global air quality guideline annual mean values, 2005 versus 2021 revision [2].

A plausible mechanism: accelerated subclinical atherosclerosis

The Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) followed 6,795 participants across six US metropolitan areas for roughly ten years, repeating coronary artery calcium (CAC) scoring to track subclinical atherosclerosis progression directly. Each 5 $\mu\text{g}/\text{m}^3$ higher long-term PM_{2.5} exposure was associated with 4.1 additional Agatston units of CAC progression per year (95% CI 4.0-6.8), and each 40 ppb higher NO_x exposure with 4.8 additional Agatston units per year (95% CI 0.9-8.7) [6]. Figure 4 sets both estimates out together. This gives the epidemiological associations in Figures 2 and 3 a concrete, imageable substrate: air pollution exposure tracks with faster buildup of coronary calcium, not only with a statistical excess of downstream events.

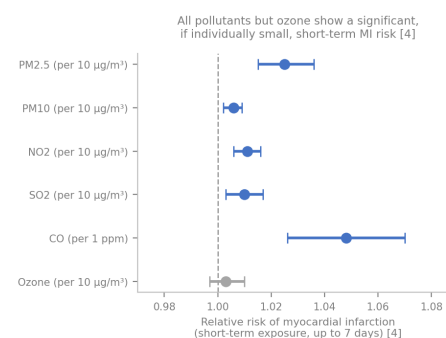


Figure 2: Relative risk of myocardial infarction per short-term pollutant increment (up to 7 days), by pollutant [4].

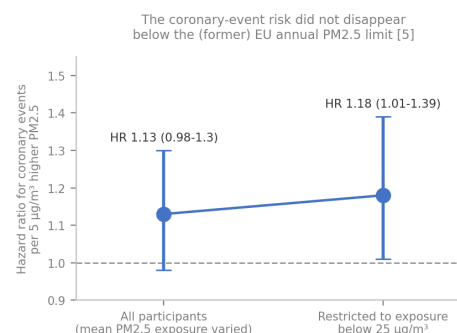


Figure 3: Hazard ratio for coronary events per 5 $\mu\text{g}/\text{m}^3$ higher long-term PM_{2.5}, all participants versus those exposed below the former EU limit, ESCAPE Project [5].

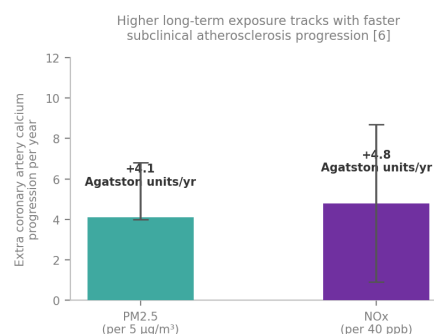


Figure 4: Additional coronary artery calcium progression per year, per long-term PM_{2.5} and NO_x increment, MESA Air [6].

Heart failure specifically

Restating the Shah et al. findings alongside the long-term and subclinical-mechanism data above places heart failure squarely inside this literature rather than at its margin: carbon monoxide carried the largest same-day association with heart failure hospitalization or mortality (3.52% per 1 ppm, 95% CI 2.52-4.54), followed by SO₂ (2.36% per 10 ppb), PM_{2.5} (2.12% per 10 µg/m³), NO₂ (1.70% per 10 ppb) and PM₁₀ (1.63% per 10 µg/m³); ozone again showed no significant association [7]. Figure 5 orders all six estimates from largest to smallest. That carbon monoxide -- a combustion byproduct closely tied to traffic -- carries the largest same-day heart-failure signal is consistent with a decompensation-triggering mechanism acting on an already-vulnerable myocardium, distinct from the slower atherosclerotic mechanism Figure 4 illustrates. Table 2 summarizes the design, exposure metric, and headline result of the five principal sources reviewed so far, for reference before turning to what any of this means for an individual patient.

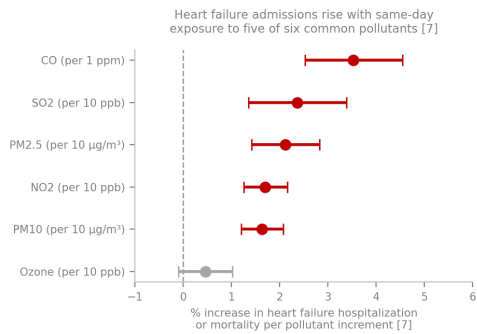


Figure 5: Percent increase in heart failure hospitalization or mortality per same-day pollutant increment, ordered by magnitude [7].

Study / source	Design	Exposure metric	Outcome	Key result
World Heart Federation [1]	GBD 2019 estimate	All ambient air pollution	Global mortality	2.94M cardiovascular deaths/yr
Mustafic et al. [4]	Meta-analysis, 34 studies	PM2.5, per 10 µg/m³	Myocardial infarction	RR 1.025 (1.015-1.036)
Cesaroni et al. (ESCAPE) [5]	11-cohort pooled analysis, n=100,166	PM2.5, per 5 µg/m³	Coronary events	HR 1.13-1.18
Kaufman et al. (MESA Air) [6]	Prospective cohort, n=6,795	PM2.5, per 5 µg/m³	Coronary calcium progression	+4.1 Agatston units/yr
Shah et al. [7]	Meta-analysis	PM2.5, per 10 µg/m³	Heart failure hospitalization/death	+2.12% (1.42-2.82)

Table 2: Design, exposure metric, and headline result of the principal epidemiological sources reviewed here [1,4-7].

What can an individual cardiologist legitimately advise?

Given the scale and consistency of the population-level evidence above, it is tempting to translate it directly into individual patient advice. The evidence base for doing so is, however, much narrower than the epidemiology, and the difference matters clinically. The best available trial of a personal protective measure is a 40-participant randomized crossover study by Morishita et al., which found that portable HEPA filtration reduced personal PM_{2.5} exposure by roughly 40% and lowered systolic blood pressure by a mean 3.4 mmHg compared with sham filtration, with a larger 6-10 mmHg reduction in participants with obesity [8]. This is a genuine, randomized, mechanistically coherent result -- and it is also a surrogate-endpoint finding from a single small trial, not evidence that filtration reduces myocardial infarction, stroke, or cardiovascular death. No comparable outcome trial exists for masks, cabin air filters, or timing outdoor activity around published air-quality indices, despite these being commonly suggested measures.

The American Heart Association's own 2024 policy statement on reducing the cardiovascular burden of ambient air pollutants is explicit about where it places the weight of its recommendation: personal protective measures -- portable air cleaners, high-efficiency HVAC and vehicle cabin filters, N95 respirators, avoiding heavily polluted areas -- are framed as reasonable for higher-risk individuals and higher-exposure settings, while population-level policy (air-quality standards, vehicle and industrial emissions limits, urban planning that reduces traffic-adjacent exposure) is presented as the primary lever for reducing the burden documented in Figure 1 [9]. The statement offers no quantified efficacy figures for the individual-level measures it names, and does not claim that any of them has been shown, in a controlled outcome trial, to lower an individual's cardiovascular risk. Figure 6 sets this asymmetry out directly, alongside what this review deliberately does not do: it does not convert this literature into a prescriptive diet, exercise, or supplement plan, because no trial cited here tested one against ambient air-pollution exposure.

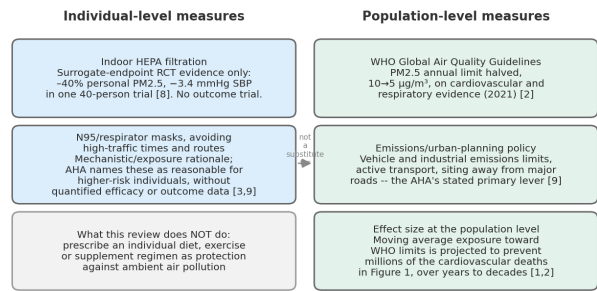


Figure 6: Individual-level and population-level responses to ambient air pollution, set against their evidence base [1,2,3,8,9].

Discussion

Taken together, these findings support a causal, dose-related relationship between ambient air pollution and cardiovascular disease that operates across short-term triggering (Figures 2 and 5), long-term chronic risk without an apparent safe floor (Figure 3), and a documented subclinical mechanism (Figure 4), on a scale (Figure 1) that places air pollution among the largest cardiovascular risk factors identified in the epidemiological literature. That conclusion is not seriously in dispute among the sources reviewed here.

What is genuinely open is the second question this review set out to answer: what follows for an individual patient. The honest complication is that the strength of the population-level causal evidence does not transfer automatically to strength of evidence for any specific individual-level countermeasure. Indoor filtration has one small randomized trial behind it, on a surrogate blood-pressure endpoint, not a cardiovascular-outcome endpoint. Masks and activity-timing advice, commonly given in clinical practice and public messaging alike, rest on mechanistic plausibility and exposure-reduction logic rather than any outcome trial identified here. The American Heart Association's own policy statement, the body most likely to overstate individual-level efficacy if any professional body were inclined to, instead places its emphasis on population-level policy [9] -- which is itself a form of evidence about where this field currently believes the actionable lever actually sits.

The pollutant-specific pattern running through Figures 2, 4, and 5 reinforces the same point from a different angle. Carbon monoxide, a combustion byproduct concentrated near traffic, carried the largest same-day risk signal for both myocardial infarction and heart failure decompensation, while the coronary artery calcium data in Figure 4 point to a slower process operating over years rather than hours. Neither pathway is one a patient controls by changing an individual behavior at the point of a clinic visit: the CO exposure is set by traffic density and vehicle emissions standards along a patient's usual routes, and the calcium-progression exposure by years of ambient background concentration wherever that patient has lived. This is precisely the structural character of the exposure that Figure 6 tries to make explicit, and it is why the same policy statement that names personal protective measures for higher-risk individuals nonetheless devotes most of its own recommendations to vehicle emissions, urban planning, and air-quality standards rather than to patient-facing advice [9].

A cardiologist can therefore honestly tell a patient that ambient air pollution is a real, sizeable, and well-documented contributor to their cardiovascular risk, and that measures such as indoor filtration have plausible, trial-supported effects on intermediate markers like blood pressure. What a cardiologist cannot honestly tell a patient is that any of these measures has

been shown to reduce their risk of myocardial infarction, stroke, or death from air pollution exposure -- because, on the evidence assembled here, that trial has not been done.

Study limitations

This is a narrative review, not a systematic review with a pre-registered protocol and search strategy, and it draws on a purposively selected set of large, prominently cited sources rather than an exhaustive search of the primary literature. The GBD-derived global burden estimate reported in Figure 1 depends on modelled exposure-response functions applied across highly heterogeneous settings, and carries wider uncertainty than a single cohort's hazard ratio; it should be read as an order-of-magnitude estimate, not a precise count. The ESCAPE, MESA Air, and Shah et al. analyses are predominantly European and North American; the pollutant mixtures and background exposure levels in those cohorts may not generalize to settings with substantially higher ambient concentrations, where both absolute risk and the shape of the dose-response curve could differ. The Morishita filtration trial enrolled only 40 participants in a single low-income senior facility, over a three-day exposure window per condition -- too small and too short to support claims about sustained blood-pressure control, let alone cardiovascular outcomes, and its findings should not be extrapolated to masks, cabin filters, or other measures it did not test. Finally, this review reports what current evidence does and does not show; it takes no position on air-quality policy beyond describing the guidance documents cited.

Conclusion

Ambient air pollution is a documented, dose-related cardiovascular risk factor operating through both acute triggering and chronic, mechanistically plausible pathways, responsible on current estimates for millions of cardiovascular deaths worldwide each year. The evidence base for any specific action available to an individual patient is far thinner than the evidence for the underlying risk itself, resting on one small surrogate-endpoint trial for indoor filtration and on mechanistic reasoning, not outcome trials, for masks and activity timing. A cardiologist can accurately convey the scale of the risk and the modest, trial-supported benefit of indoor filtration on blood pressure; recommending any of these measures as a proven means of preventing myocardial infarction, stroke, or cardiovascular death would overstate what the literature reviewed here actually shows.

One further figure is worth restating alongside Figure 1's ambient-exposure estimates, because it changes what "structural" means in practice for some patients rather than others. The same GBD-based analysis attributes a comparable cardiovascular toll to household air pollution -- chiefly indoor combustion of solid fuels for cooking and heating -- estimating

roughly 1.696 million cardiovascular deaths per year worldwide from that exposure route (1 million ischemic heart disease, 0.7 million stroke), concentrated in low- and middle-income countries [1]. Unlike ambient outdoor pollution, household exposure is a route where a cardiologist's own counsel about a patient's home cooking and heating arrangement can occasionally intersect with an exposure a patient or family can influence directly -- a genuine exception to this review's broader conclusion that the exposure itself sits mostly outside individual control, though even here no cardiovascular-outcome trial of a household-level intervention was identified for this review.

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