Combustion Aerosols as an Underrecognized Contributor to the Twentieth-Century Coronary Heart Disease Epidemic

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Abstract

Coronary heart disease (CHD) emerged as one of the leading causes of mortality in the United States and many industrialized nations during the twentieth century. Traditional explanations for the rise and subsequent decline of CHD have emphasized cigarette smoking, dietary changes, serum cholesterol levels, hypertension, and advances in medical treatment. However, a growing body of epidemiological, toxicological, and clinical evidence indicates that fine particulate air pollution (PM2.5) is an important risk factor for cardiovascular disease and acute myocardial infarction. This paper proposes that the dramatic increase in combustion-derived aerosols during the industrial expansion of the twentieth century—including emissions from coal combustion, municipal waste incineration, industrial processes, and motor vehicle exhaust—may have contributed substantially to the observed epidemic of coronary heart disease. Historical trends in particulate pollution broadly coincide with the period of increasing CHD mortality from the early twentieth century through the mid-1960s, while subsequent reductions in particulate pollution following environmental regulation coincide with declining cardiovascular mortality. Although PM2.5 was unlikely to be the sole driver of the CHD epidemic, evidence suggests it may represent a significant and underappreciated contributor. This hypothesis provides a framework for integrating environmental exposure history into the broader understanding of cardiovascular disease epidemiology.

Introduction

Coronary heart disease became a dominant public health concern during the twentieth century. Mortality rates from ischemic heart disease rose steadily through the first half of the century and peaked in many industrialized nations during the 1960s before entering a prolonged decline [1,2].

The conventional explanation for this pattern emphasizes several major risk factors, including cigarette smoking, elevated serum cholesterol, dietary changes, hypertension, and sedentary lifestyles. Improvements in medical treatment, smoking cessation, and risk-factor management are widely credited for the decline observed after the 1960s and 1970s [1,2].

However, another major environmental change occurred during the same period. Industrialization, urbanization, coal combustion, municipal waste incineration, and rapid growth in automobile use

dramatically increased atmospheric aerosol concentrations. Fine particulate matter (PM2.5) reached historically elevated levels in many urban and industrial regions. Concurrently, widespread atmospheric aerosol loading produced the phenomenon now known as global dimming, characterized by reductions in surface solar radiation from approximately the 1950s through the 1980s [3].

This paper examines the hypothesis that combustion-derived aerosols were a significant but underrecognized contributor to the twentieth-century coronary heart disease epidemic.

Historical Trends in Combustion Aerosols

Coal Combustion

Coal consumption increased dramatically throughout the late nineteenth and early twentieth centuries. Coal-fired power plants, industrial facilities, rail transportation, and residential heating emitted large quantities of soot, sulfates, fly ash, and other particulate pollutants.

Historical photographs and air-quality records indicate that many American and European cities experienced severe smoke pollution. Urban visibility degradation became sufficiently severe that smoke-abatement programs emerged decades before modern environmental regulation.

Municipal Waste Incineration

Municipal solid waste incineration became increasingly common throughout the twentieth century. Incinerators emitted substantial quantities of fine particulate matter and metal-containing aerosols. Prior to modern emission controls, these facilities represented significant urban pollution sources.

Motor Vehicle Emissions

Automobile ownership expanded rapidly after World War II. Internal combustion engines emitted particulate matter directly and generated secondary particulate pollution through atmospheric chemical reactions involving sulfur dioxide, nitrogen oxides, and hydrocarbons.

Everyday Evidence of Particulate Deposition

A largely overlooked indicator of historical particulate loading is found in ordinary domestic practice. Through the 1950s and 1960s, indoor surfaces accumulated soot and particulate residue so rapidly that routine seasonal “spring cleaning” commonly included the washing of interior walls and ceilings to remove deposited grime—a chore driven largely by coal-fired heating, industrial soot, and uncontrolled combustion emissions. As particulate air pollution standards reduced ambient PM10 and PM2.5 through the 1970s, such deposition diminished markedly, and the once-routine washing of walls and ceilings largely disappeared from household maintenance. Although

anecdotal and observational rather than quantitative, this shift offers a tangible illustration of how dramatically ambient particulate concentrations fell following the introduction of emission controls.

Global Dimming

Between approximately 1950 and 1985, widespread increases in atmospheric aerosols reduced the amount of solar radiation reaching Earth's surface. Numerous studies have documented declines in surface solar radiation across North America, Europe, and Asia during this period, followed by a reversal toward "global brightening" after roughly 1985 as developed nations curtailed aerosol emissions [3].

The existence of global dimming provides independent corroboration that atmospheric aerosol concentrations increased substantially during the same period that coronary heart disease mortality rose to historic levels. Because global dimming is a measure of surface radiation rather than of breathing-zone exposure, it is best interpreted as supporting evidence of regional aerosol loading rather than as a direct proxy for inhaled PM2.5.

Acute Pollution Disasters and the Acute–Subacute Distinction

A series of well-documented air pollution disasters during the mid-twentieth century demonstrated both the extreme particulate and sulfurous pollution levels of the era and their direct lethality. In the Meuse Valley, Belgium (1930), a temperature inversion trapped industrial emissions and produced roughly sixty excess deaths over a few days [5]. In Donora, Pennsylvania (1948), emissions of sulfur dioxide, sulfuric acid, fluorine, and metal-bearing particulates from local steel and zinc works were trapped by an inversion, killing approximately twenty people within days and an estimated additional fifty within the following month, while sickening nearly half of the town's population [6]. The London "Great Smog" of December 1952, in which sulfur dioxide from coal combustion was oxidized to sulfuric acid aerosol, was associated with an estimated 4,000 immediate excess deaths and, by later analyses, as many as 12,000 deaths over the following months [4].

These episodes are universally recognized for their acute mortality, and the excess deaths were unambiguously attributed to the pollution. Acute mortality, however, represents only the most visible endpoint of extreme exposure. The disasters are equally important as evidence of the magnitude of routine ambient pollution during this era: conditions severe enough to produce mass-casualty events during temperature inversions imply a baseline of chronic exposure that persisted, at lower but still substantial levels, for decades.

The central concern of the present hypothesis is therefore not the acute death spikes themselves but the subacute and chronic cardiovascular consequences of sustained particulate exposure—including the promotion of atherosclerosis and the triggering of acute myocardial infarction. Such effects would not appear in disaster death tolls, yet, distributed across entire exposed populations over decades, they could contribute substantially to population-level coronary mortality.

Cardiovascular Effects of PM2.5

A substantial modern literature demonstrates that PM2.5 exposure is associated with cardiovascular disease [7]. Numerous cohort studies have reported increased risks of:

- Coronary heart disease
- Acute myocardial infarction
- Stroke
- Heart failure
- Cardiac arrhythmias
- Cardiovascular mortality

Foundational cohort investigations, including the Harvard Six Cities Study [8] and analyses from the Women's Health Initiative [9], reported increased cardiovascular morbidity and mortality with higher long-term PM2.5 exposure. Meta-analyses consistently demonstrate elevated cardiovascular risk with increasing PM2.5 exposure, and recent studies suggest measurable effects even at concentrations below current regulatory standards [7].

The American Heart Association has concluded that particulate matter exposure contributes directly to cardiovascular disease and that PM2.5 should be regarded as a modifiable risk factor [7].

Biological Plausibility

Several mechanisms support a causal relationship between PM2.5 exposure and coronary heart disease [7].

Systemic Inflammation

Fine particles trigger inflammatory responses in the respiratory system that propagate throughout the body. Elevated inflammatory markers are known contributors to atherosclerosis progression and plaque instability.

Oxidative Stress

PM2.5 exposure increases production of reactive oxygen species, resulting in endothelial injury and vascular dysfunction.

Endothelial Dysfunction

Particulate matter impairs vascular endothelial function, reducing nitric oxide availability and promoting vasoconstriction.

Hypercoagulability

Exposure to particulate pollution increases blood coagulability and platelet activation, increasing the likelihood of thrombosis.

Plaque Destabilization

Inflammatory responses associated with PM2.5 exposure may increase the probability of plaque rupture, a primary trigger of acute myocardial infarction.

Together, these mechanisms provide a biologically plausible pathway connecting long-term particulate exposure with increased rates of coronary heart disease and acute cardiac events.

Historical Comparison of Aerosol and Coronary Disease Trends

Several broad observations support the hypothesis that particulate pollution contributed to twentieth-century cardiovascular disease trends.

First, aerosol emissions increased substantially during the same decades that coronary heart disease mortality increased.

Second, the period of maximum aerosol loading approximately coincided with peak coronary mortality during the 1960s [1,2].

Third, environmental regulations introduced during the 1960s and 1970s led to significant reductions in particulate pollution.

Fourth, epidemiological studies have demonstrated that reductions in PM2.5 are associated with measurable increases in life expectancy and reductions in cardiovascular mortality; one analysis estimated that a 10 μg/m³ decrease in PM2.5 corresponded to an increase in mean life expectancy of approximately 0.77 years [10].

Although correlation alone cannot establish causation—and the trajectories of cigarette consumption and dietary change followed broadly similar time courses, complicating attribution—the temporal correspondence is notable and warrants further investigation. A spatial test, comparing the timing and magnitude of coronary mortality peaks across regions with differing historical combustion-aerosol burdens after adjustment for smoking and diet, would provide a stronger test of the hypothesis than temporal correlation alone.

Relationship to Established Risk Factors

This hypothesis does not challenge the importance of smoking, cholesterol, hypertension, obesity, or medical treatment.

Instead, it proposes that particulate pollution operated alongside these factors as an environmental amplifier of cardiovascular risk.

The twentieth-century coronary heart disease epidemic was likely multifactorial. Smoking may have served as a dominant individual-level risk factor, while combustion aerosols may have increased baseline cardiovascular risk across entire populations.

Under this framework, the observed epidemic reflects the combined effects of multiple simultaneous environmental and behavioral exposures.

Lead: A Competing and Complementary Combustion-Related Exposure

Lead exposure warrants particular attention as a competing and potentially complementary combustion-related cardiovascular exposure. Like fine particulate matter, lead was released in large quantities by twentieth-century combustion—chiefly through leaded gasoline, but also coal burning and metal smelting—and a substantial literature now identifies it as an independent cardiovascular risk factor. A systematic review found consistent, dose-dependent associations between lead exposure and hypertension, coronary heart disease, stroke, and peripheral arterial disease [11].

The magnitude of this contribution may be considerable. In a population-based cohort drawn from the Third National Health and Nutrition Examination Survey (NHANES-III), low-level lead exposure was associated with 28.7% of cardiovascular and 37.4% of ischemic-heart-disease mortality, corresponding to an estimated 256,000 cardiovascular deaths annually in the United States—implicating a combustion-derived metal in much of the same cardiovascular mortality examined here [12].

This convergence raises an important caveat. Leaded-gasoline consumption rose after World War II, peaked in the early 1970s, and was phased out between 1975 and 1995—a trajectory closely paralleling both the rise and regulatory decline of combustion aerosols and the global dimming-and-brightening sequence. Because these exposures are strongly collinear in time, temporal correlation alone cannot apportion twentieth-century cardiovascular trends among PM2.5, lead, and co-occurring behavioral factors such as smoking and diet. Rather than weakening the case for an environmental contribution, this convergence arguably strengthens it: multiple combustion-derived exposures plausibly acted in concert. Disentangling their relative contributions will require

spatial and individual-level analyses that exploit regional differences in exposure history, together with explicit recognition that pre-1999 PM2.5 concentrations must be reconstructed from proxies rather than measured directly.

Implications for Public Health

Recognizing particulate pollution as a contributor to historical cardiovascular disease has important implications.

First, it suggests that the health benefits of air pollution regulation may be larger than traditionally estimated.

Second, it highlights the importance of continued reductions in combustion-related emissions.

Third, it may help explain portions of cardiovascular mortality trends not fully accounted for by traditional risk factors.

Finally, this perspective may be particularly relevant for rapidly industrializing regions that continue to experience elevated particulate pollution.

Conclusions

Modern epidemiological and toxicological evidence firmly establishes PM2.5 as a cardiovascular risk factor capable of increasing the incidence of myocardial infarction and coronary heart disease [7].

Historical evidence indicates that combustion-derived aerosols increased dramatically during the same period that coronary heart disease became a leading cause of mortality in industrialized nations. Subsequent reductions in particulate pollution coincided with improved cardiovascular outcomes and increased life expectancy [10].

While smoking, cholesterol, hypertension, and medical treatment remain central components of the conventional explanation for twentieth-century coronary disease trends, particulate pollution may represent a substantial and underrecognized environmental contributor. Future research should seek to quantify the contribution of historical aerosol exposures to long-term cardiovascular mortality trends and to evaluate the role of particulate pollution in shaping the epidemiological transition of cardiovascular disease during the industrial era.

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Competing Interests

The authors have declared that no competing interests exist.

Data Availability

This is a hypothesis/review article; no new data were generated. All data discussed are available within the cited published studies and their respective sources.

Keywords

PM2.5; particulate matter; coronary heart disease; myocardial infarction; cardiovascular disease; air pollution; global dimming; global brightening; coal combustion; aerosols; lead; leaded gasoline; environmental epidemiology.

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