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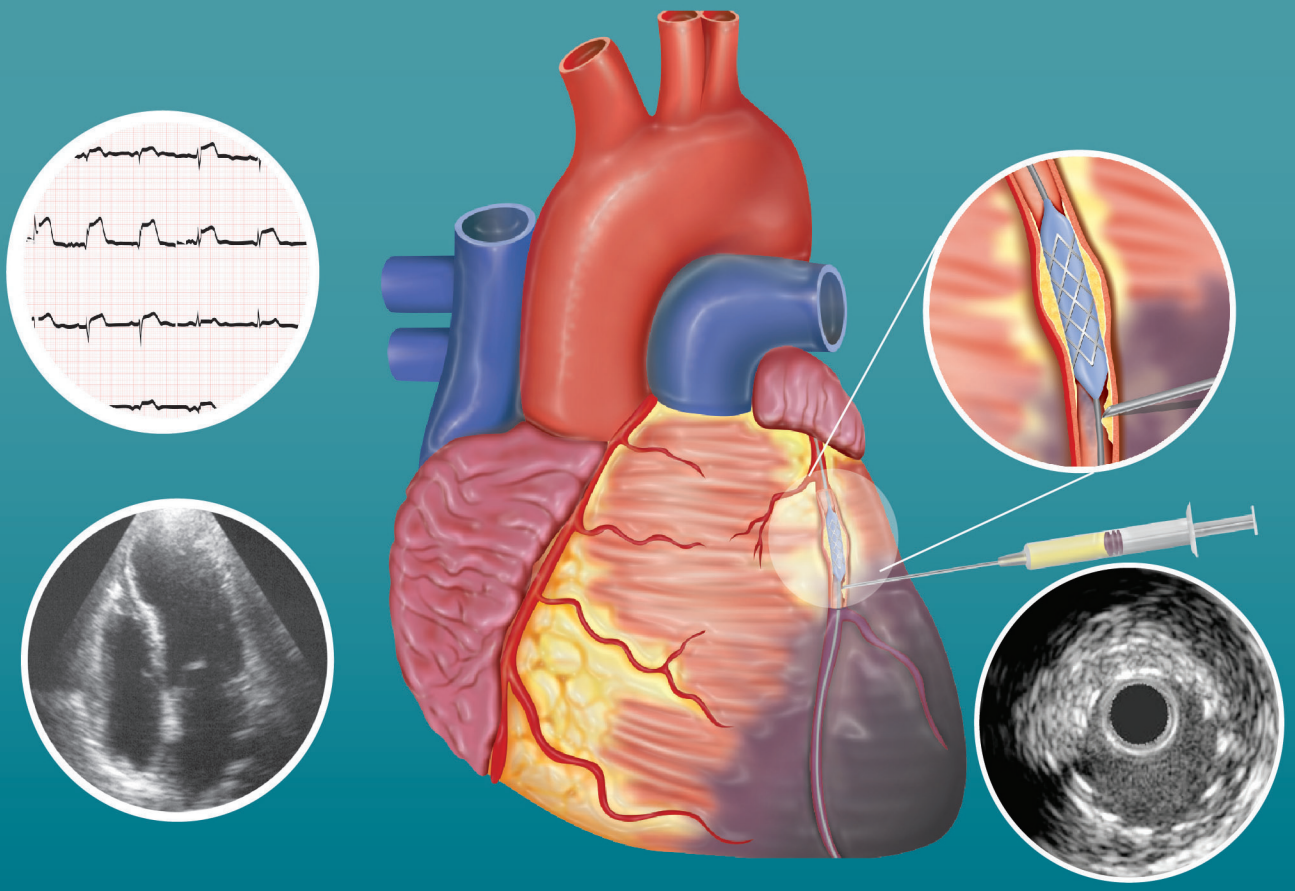
STEMI

A Cardiology Update



HK CHOPRA
SAMEER MEHTA

Foreword
Ashok Seth

JAYPEE

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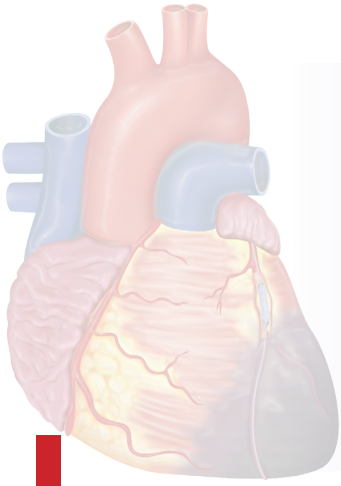
CHAPTER



Air Pollution and Heart Disease

Bhargav Krishna, Dorairaj Prabhakaran

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INTRODUCTION

Cardiovascular disease (CVD) is considered as the leading cause of death and disability globally, with close to 80% of the burden now occurring in low and middle income countries (LMIC).[1] Not only is the onset of CVD in LMIC populations like in India is sooner than in western nations, there is also a significant economic burden associated with it.[2] While the risk factor profile appears to be relatively similar between developing and developed nations, there is a need to examine the causal mechanisms for the earlier onset of CVD. Air pollution exposure could be one such factor that putatively heightens this risk. The purpose of this chapter, therefore, is to examine the association between air pollution and CVD, the pathophysiological mechanisms of this association, and the gaps in our knowledge that need to be addressed.

Air pollution exposure was historically not considered as a risk factor for CVDs. However, research conducted in the last 10–15 years has shown that it is indeed a serious risk factor (include citations to said research). This culminated with the Global Burden of Disease comparative risk assessment exercise carried out in 2010, which showed that the combined exposure to smoke from solid cooking fuels and the ambient environment was the leading environmental risk factor for mortality and morbidity worldwide among the more than 60 risk factors examined (Fig. 1).

Estimates released by the World Health Organization (WHO) in 2014 revealed that globally, around 7 million people (one in eight) died in the year 2012 as a result of exposure to air pollution, with the South-East Asian and Western Pacific regions contributing the largest share of the burden. Further, over 80% of the deaths attributable to ambient air pollution (AAP) exposure and 60% of the deaths attributable to household air pollution (HAP) exposure, were due to CVDs such as stroke and ischemic heart disease.[3] For example, HAP and AAP together contributed to 9% of the national disease burden in India, which translated to 1.6 million premature deaths and 49 million disability-adjusted life years (DALYs) in 2010, a large majority of which were attributable to CVD. This effectively means that taken together, HAP and AAP constitute the single largest risk factor for ill health in India.

Exposure to air pollution occurs through two primary mechanisms—(1) the burning of solid fuels such as coal, wood, dung, charcoal, etc. indoors for either cooking or heating; (2) exposure in the ambient environment where pollutants are released from a variety of sources including vehicular transport, trash burning, construction dust, industrial emissions, etc. The scale of exposure can only truly be perceived when one understands that over 700 million persons in India use biomass or solid fuels as their primary energy source for cooking or heating. This number has not declined in the last 20 years although proportionally fewer people use solid fuels now.[4] The scale of exposure to ambient air pollution in India received wide attention in mid-2014 when the WHO released an Ambient Air Pollution database, a dataset that characterized pollution levels across hundreds of cities in dozens of countries around the world. Thirteen of the top 20 cities with the highest annual average air pollution levels were Indian, and many of them recorded levels 10 times higher than those considered safe by the WHO (Fig. 2).[5]

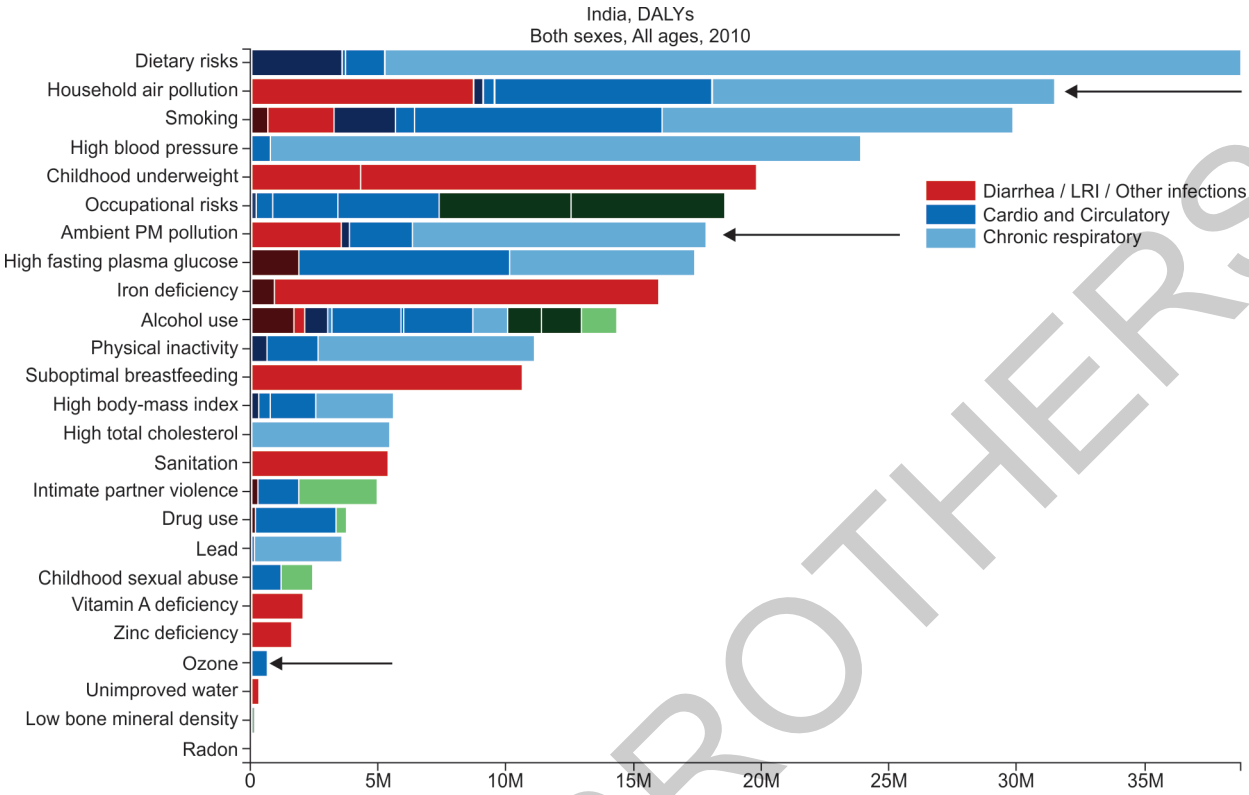

Fig. 1: Ranking of leading risk factors contributing to the national burden of disease in India (IHME, 2013)[1]
Abbreviations: DALYs, disability-adjusted life years; LRI, lower respiratory tract infection

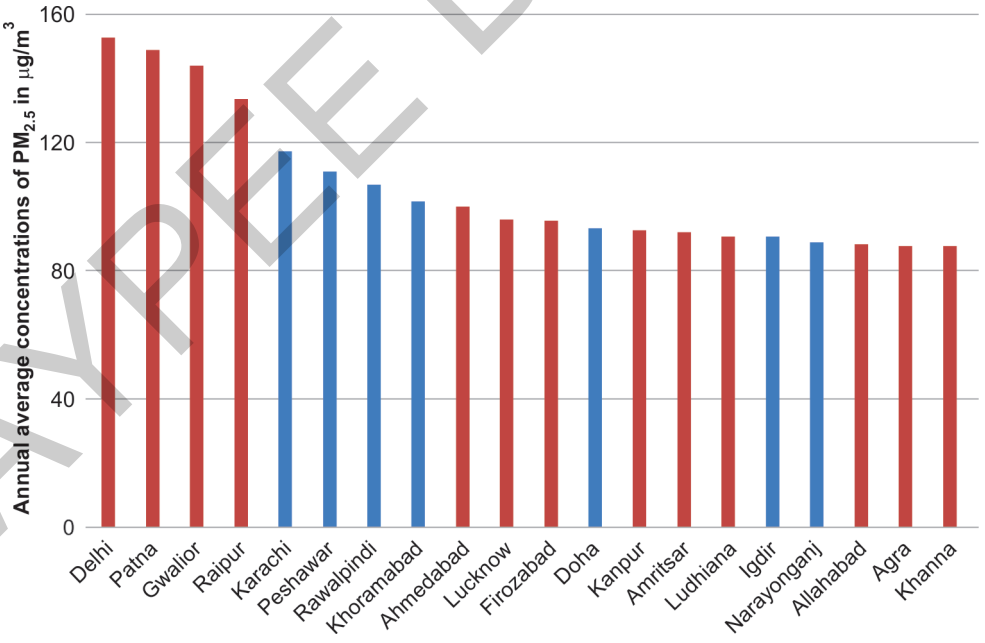

Fig. 2: Top 20 cities with highest annual average $PM_{2.5}$ (particulate matters less than 2.5 microns in size) levels[5]

Pollutants present in household and ambient air pollution include sulfur dioxide (SO_2), nitrogen oxides (NO_x), carbon monoxide (CO), ozone, PM_{10} and $PM_{2.5}$ (particles less than 10 and 2.5 microns in size, respectively). Other toxic and carcinogenic byproducts from biomass burning include formaldehyde, benzene, and polyaromatic hydrocarbons.

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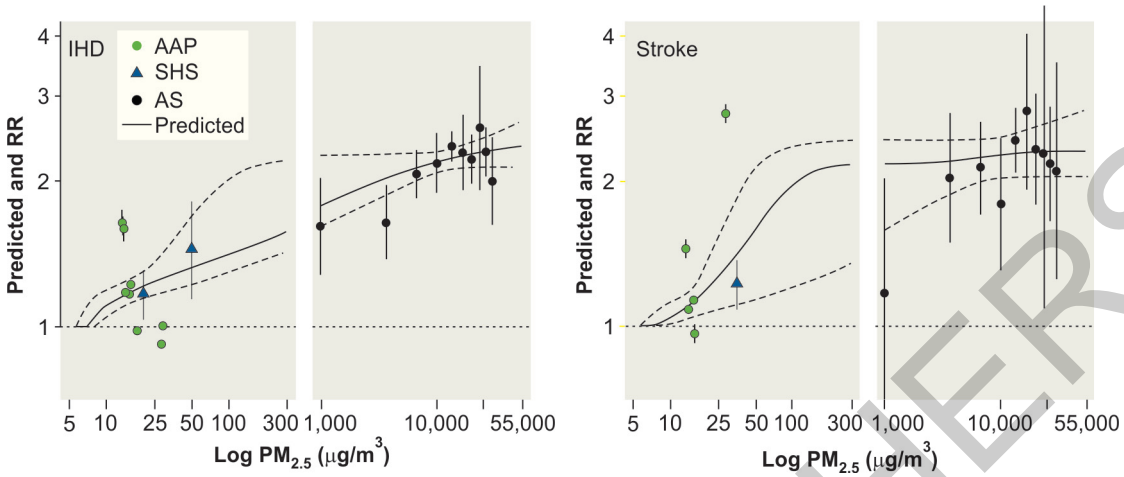

Fig. 3: Integrated exposure-response curve for exposure to ambient air pollution (AAP), household air pollution (HAP), second-hand smoking (SHS) and active tobacco smoking (AS), as developed for the 2010 Global Burden of Disease study[19]
(*Source:* Reproduced with permission from Environmental Health Perspectives.)
Abbreviations: IHD, ischemic heart disease; RR, relative risk

Exposure to the earlier listed pollutants can result in several chronic and acute respiratory and cardiovascular health outcomes. While exposure to SO_2, NO_x, CO and ozone can be detrimental to health, exposure to PM_{10} or $PM_{2.5}$ is considered to be the most harmful to human health. $PM_{2.5}$ primarily consists of organic/elemental carbon, soot and sulfates, but may also contain ammonia, nitrates and non-crustal material depending on the source of the emission.[6] Short-term exposure to $PM_{2.5}$ contributes to acute cardiovascular mortality and morbidity, and longer term exposures reduce life expectancy. While the mechanistic pathways for some of these health outcomes remain understudied, the epidemiological evidence till date suggests a sufficiently strong link to necessitate actions that reduce exposure drastically, especially in developing countries where exposure levels are often much higher than safe limits.[7]

The WHO ambient air quality guidelines state that 24-hour exposures to $PM_{2.5}$ should be no greater than 25 µg/m^3, and annual average exposures no greater than 10 µg/m^3. However, the WHO's ambient air quality database of 2014 reveals that the vast majority of cities, especially those in developing countries, far exceed those limits. Measurements of 24-hour exposure in kitchens where solid fuels are used for cooking, have shown that women and children are subjected to average $PM_{2.5}$ levels around 450 µg/m^3, and peaks of around 800 µg/m^3 in some cases, which is the equivalent of smoking around 400 cigarettes an hour.[8,9]

Acute and chronic exposures to air pollution, and $PM_{2.5}$ in particular, have been associated at varying degrees of certainty with a range of cardiovascular effects including ischemic heart disease, heart failure, stroke, thrombosis, arrhythmias, and hypertension.[10-15] Time-series studies have shown that an increase in $PM_{2.5}$ of 10 µg/m^3 can increase the relative risk of cardiovascular mortality in a population.[16] The ubiquitous and iniquitous nature of exposure also ensures that theoretical statistical risks ascribed to populations are not equally distributed.[17] At the levels of exposure observed in most countries, $PM_{2.5}$ poses the most serious threat to susceptible populations such as the elderly and those with preexisting conditions.[18] Given the nature and levels of exposure, the epidemiological evidence available, and the concomitant prominence of $PM_{2.5}$ as a risk factor over all other criteria air pollutants, we will briefly examine the mechanistic pathways by which exposure to $PM_{2.5}$ manifests on the cardiovascular system in the following section (Fig. 3).[19]

EXPOSURE AND RISK

While public discourse around the deleterious effects of air pollution exposure tends to focus on respiratory illnesses, the reality is that the vast majority of mortality attributable to air pollution exposure is from CVD.[1] Exposure to PM either in the ambient air or in households, for short or long terms, is known to result in a range of CVD end points including myocardial infarction, ischemic heart disease, stroke, and conditions including thrombosis, hypertension, heart rate variability and arrhythmias.

To date, the Global Burden of Disease comparative risk assessment of 2010 is the most comprehensive work carried out to estimate the risk associated with exposure and to calculate the proportional disease burden attributable to

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air pollution exposure (household and ambient). Given the plethora of evidence published on the exposure-response relationship for many disease endpoints in relation to $PM_{2.5}$ exposure, either through smoking or other combustion particle exposures, the Global Burden of Disease 2010 exercise developed integrated exposure response (IER) estimates. The development of the IERs allowed the investigators to develop a consistent response across many similar categories of combustion particle exposure (namely active tobacco smoking, second-hand smoke, household air pollution and ambient air pollution) and thereby produce accurate estimates where detailed exposure studies are not as well developed. Using daily average $PM_{2.5}$ exposure as the metric also allowed the IERs to straddle across three orders of magnitude of exposure, thereby allowing future work to estimate end-point specific disease burdens for higher exposure levels.[19]

The IERs as explained in Figure 3 reveal an exponential link between $PM_{2.5}$ exposure and cardiovascular health outcomes, with exposure even at low levels carrying a level of risk beyond acceptability.[19] Exposure, for instance, of 25 $\mu g/m^3$ which is within WHO guidelines for safe exposure levels, carry a relative risk of mortality between 1.2 and 1.3. The curves also reveal that the most discernible improvements in CVD end-points are achievable at very low levels of exposure, contrary to the reality in many developing countries such as India and China, where exposure levels are often 10 times the advised limits. Given the similarities in exposure-response, the oft-used statement that people need to completely stop smoking to protect their health holds true in this case as well.

While the Global Burden of Disease exercise characterized the most commonly studied health end-points, several others have been studied in fair detail epidemiologically. Some of these are highlighted below in Table 1.

TABLE 1: Cardiovascular effects associated with exposure to air pollution (PM) in human subjects

Cardiovascular effect	*Increased risk (CI)*	*Source (Ref.)*
Myocardial infarction	HR = 1.13 (0.98–1.30) RR = 1.16 (1.04–1.29)	20 21
Ischemic heart disease	OR = 1.17; (0.98–1.40)	10
Heart failure	RR = 1.21 (1.14–1.28)	11
Stroke	RR = 1.19; (0.88–1.62)	12
Thrombosis	OR = 1.70; (1.30–2.23)	13
Hypertension	B = 2.97 mm Hg, P = 0.002 for systolic BP; and β = 1.87 mm Hg, P = 0.005 for diastolic BP	15
	1.393 mm Hg, 95% CI (0.874–1.912) and 0.895 mm Hg, 95% CI (0.49–1.299) per 10 $\mu g/m^3$ increase for SBP and DBP, respectively	22
Arrhythmias	OR = 1.24 (0.87–1.76)	14

Abbreviations: CI, confidence interval; OR, odds ratio; SBP, systolic blood pressure; DBP, diastolic blood pressure; RR, relative risk; HR, hazard ratio

PATHOPHYSIOLOGY

At our current level of understanding, there are three hypotheses that outline the association between exposure to PM and cardiovascular disease with varying levels of certainty attached to each, as shown in Figure 2.[7] The first asserts that the entry of PM into the lungs provokes inflammatory responses that promote systemic oxidative stress, thereby leading to increased risk of thrombosis, endothelial dysfunction, atherosclerosis progression, and dyslipidemia. This hypothesis is best supported by the evidence.

The second hypothesis asserts that pulmonary exposure leads to pathological alterations in vasoconstriction, increased heart rate variability, hypertension, and platelet aggregation. The pathway for these pathological alterations was through the activation of lung autonomic nervous system (ANS) arcs mediated by transient receptor potential (TRP) channels.[23]

Supported by only a handful of studies, the third hypothesis emphasizes that inhalation of PM into the lungs, and their subsequent absorption into the blood stream results in tissue-level interactions resulting in platelet aggregation, vasoconstriction, endothelial dysfunction, and sometimes even chemical modification of the DNA, resulting in epigenetic changes in gene expression.[23]

Systemic Inflammation and Oxidative Stress

$PM_{2.5}$ and its constituents, including diesel exhaust, have been shown to release cytokines into the blood stream, resulting in oxidative stress. Exposure to $PM_{2.5}$ also triggers the release of oxidized phospholipids in the lungs, thereby mediating a systemic inflammatory response through Toll-like receptor 4 (TLR 4)/ reduced form of nicotinamide adenine dinucleotide phosphate (NADPH).[24] Short-term exposures to $PM_{2.5}$ have also shown increased levels of circulating cytokines such as interleukin-1 beta (IL-1β), IL-6 and tumor necrosis factor alpha (TNF-α), indicating systemic inflammation.[25] Positive associations have also been found between exposure to $PM_{2.5}$ and changes in the expression of genes in both mice and human subjects.[26]

Thrombosis, Coagulation and Hypertension

A study conducted by Lucking et al. revealed increased thrombus formation and increases in platelet neutrophils and platelet monocytes on exposure to particulates from

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diesel exhaust.[27] Studies have also shown that in-vehicle exposure to $PM_{2.5}$ results in an attendant increase in von Willebrand factor.[28] Another study shows that a larger exposure to PM resulted in a 3.9% higher von Willebrand factor level in diabetics.[29] Further credence is lent to the theory that oxidative stress and vascular inflammation are the key mechanisms through which PM-mediated atherogenesis occurs, by studies in mice.[30]

Vascular Dysfunction and Atherosclerosis

A study conducted in 2010 in Los Angeles found that persons living in closer proximity to highways and thus exposed to higher $PM_{2.5}$ levels from traffic pollution had accelerated CIMT (carotid intima-medial thickness) progression [5.5 µm/year (95% CI: 0.13–10.79; p = 0.04)] more than twice the population mean progression.[31] Recent results from the Multi-Ethnic Study of Atherosclerosis and Air Pollution (MESA Air) have supported this hypothesis that long-term exposure to $PM_{2.5}$ is associated with increased progression of IMT of the common carotid artery, an indicator of atherosclerosis. The study found that in the same metropolitan area, persons exposed to slightly higher levels of $PM_{2.5}$ exhibited greater IMT progressions (around 5 µm/year) than others living in the same region.[32]

Myocardial Ischemia/STEMI

A study conducted in Finland in 2007 noted that in a small cohort of nonsmoking elderly patients with coronary heart disease, short-term exposures to $PM_{2.5}$ were associated with ST segment depressions, increasing the risk of myocardial ischemia.[33] A similar study conducted in 2010 with a more comprehensive outcome assessment [24-hour beat to beat electrocardiogram (ECG) with 12-lead Holter], corroborated the earlier findings, with the most immediate effects felt within 2 hours of the exposure.[34] A further study from 2011 linked ST-Segment depression to quasi-ultrafine particulate exposure (PM < 0.25 micron).

Others

A systematic review of the epidemiological studies conducted on air pollution and its links to heart failure until 2012 noted that an increase in atmospheric PM concentration of 10 µg/m^3 was associated with increased risk of hospitalization or death from heart failure (2.12% per 10 µg/m^3 of $PM_{2.5}$; 95% CI 1.42–2.82). The same study also revealed that a mean reduction in $PM_{2.5}$ of 3.9 µg/m^3 would prevent 7,978 heart failure hospitalizations and save one-third of a billion US dollars a year.[11] Although limited research on human subjects to identify the causal pathways behind this link exists, studies on mice seem to confirm the epidemiologic links established in human subjects (Table 2).[35]

TABLE 2: Pathophysiological mechanisms observed on exposure to air pollution (PM) in human subjects

Pathophysiological mechanism	*Source (Ref.)*	*Findings*
Systemic inflammation	25	Increased levels of IL-1β, IL-6 and TNF-α
Systemic oxidative stress	24	Increased levels of oxidized phospholipids in lungs that then mediate systemic cellular inflammatory response
Thrombosis and coagulation	27 29 32	Increasing IMT of the carotid artery on exposure to $PM_{2.5}$, increased thrombogenicity on exposure to diesel exhaust and increased von Willebrand factor levels
Hypertension	22 15,36	Increased systolic and diastolic blood pressure
Vascular dysfunction and atherosclerosis	31 32 37	Faster progression of CIMT on exposure to $PM_{2.5}$, and "abnormal accumulations of oxidized lipids, such as 7-KCh, in air pollution-induced atherosclerosis progression"
Heart rate variability/ arrhythmias	38 14 39 40	Small increases in heart rate for short-term exposure, and observations of ventricular tachycardia in older populations
Myocardial fibrosis and heart failure	11 35	Epidemiologic studies show a strong link, but no clinical studies have yet been conducted. Studies in mice echo epidemiologic links
Myocardial ischemia	33 34 41	Exposure to combustion related pollutants (acute and chronic) associated with ST-segment depression

Abbreviations: IL, interleukin; TNF-α, tumor necrosis factor alpha; CIMT, carotid intima-medial thickness

LEGISLATIVE MECHANISMS AND MULTISECTORAL ACTION

A growing global recognition of the health impacts of air pollution exposure was crystallized in the outcome document of the Open Working Group on the Sustainable Development Goals. Mandated by the Member States of the United Nations at the Rio+20 Conference on Sustainable Development, the Open Working Group has for the first time included a target to "substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water, and soil pollution and contamination" by 2030.[42] This

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recognition of an otherwise ignored but key risk factor has spurred the dialogue around multisectoral action to address air pollution as a risk factor for ill health, particularly through mechanisms of the WHO. One such mechanism is the "Global Action Plan for the prevention and control of noncommunicable diseases (NCDs) 2013–2020", which sets out 9 global targets to reduce NCD (including CVD) related mortality and morbidity.[43] The WHO South-East Asia regional office (SEARO) region, in an expression of policy-informed decision making, has included a 10th target to reduce household air pollution exposure, thereby recognizing a key risk factor for CVD.

This global movement towards this within the health sector reached a crescendo in 2015, when the World Health Assembly, under Indian leadership, passed a resolution to address the health impacts of air pollution,[44] highlighting the key role that national authorities have to play in awareness building and exposure mitigation. The resolution highlights the need for strong action across sectors, the integration of health in all policies, and developing and maintaining monitoring and surveillance systems. This declaration will be followed by the development of a roadmap for an enhanced global response to the issue to be presented at the 69th Assembly.

The case made for strong action at the national level, has also been ensconced in national policies, with some countries in particular leading the way. In the United States for instance, the Environmental Protection Agency (EPA) plays a strong agenda-setting and regulatory role, with a regular appraisal of the evidence to examine the need for re-evaluating standards. It also participates in awareness building through its AirNow (http://www.airnow.gov/) forecasting program, which issues regular health warnings through an air quality index (AQI). The European Union has also set health-based targets for air quality through its *thematic strategy on air pollution*, which aims to cut the annual number of premature deaths by 2020 by 40%.[45]

CONCLUSION

While the evidence described earlier makes a sufficiently strong case for considering air pollution exposure a key risk factor for adverse cardiovascular health outcomes including MI, the majority of the evidence base consists of cross-sectional, case-control or time-series studies, which while good for establishing causation, are not ideal at developing the evidence for pathophysiological mechanisms. Therefore, there is a need to conduct a series of cohort studies like the Nurse's Health Study or Six Cities Study in the USA, or the MESA Air study in Europe where long-term cardiovascular effects can be studied in greater detail. This is especially relevant in the context of developing countries that are experiencing high levels of exposure such as those in South or South-East Asia.

The evidence base for CVD effects of air pollution exposure comprises mainly studies conducted in developed countries, with exposure levels far below that experienced in countries like India. This lack of a local evidence base is often also cited as a reason for policy inaction in these countries, even though the health effects of air pollution exposure are likely the same, if not worse. Given the ubiquity of sources and combined contributions of ambient and household air pollution to population level exposures, and the common threads in terms of health outcomes, there is also a need to conduct epidemiological studies that analyze exposure and health responses across rural and urban settings such as in the case of a recently established rural-urban cohort in South India.[46] The interconnectedness of air pollution with other environmental effects such as climate change, also necessitates studying the effects of increased temperatures and of extreme weather events on cardiac health in greater detail.[47]

Moreover, knowledge amongst health professionals of the adverse effects of air pollution exposure is limited at best (citation if possible). Physicians and public health professionals are at the front line of addressing adverse health outcomes, and are the best source of engagement with the public. Physicians are also trusted interlocutors with the general public. Thus, utilizing them as mediums to disseminate information and educate the public about the ill effects of air pollution exposure can help in mitigating exposure, and in fostering the kind of public pressure needed to effect multisectoral action on this key developmental issue.

Localized evidence in developed countries has also shown that the effect of episodic events of high air pollution adversely affect vulnerable populations and exacerbate all-cause mortality specifically through CVD pathways.[14,40,41] Thus, there is a need to establish health systems protocols and action plans to address the excess mortality and morbidity associated with episodic events of high pollution. In conclusion, as the recent inclusion of HAP as a target for NCD reduction by countries of the WHO SEARO region has shown, there is a growing understanding of the need to embed a response to this growing public health burden into health systems at all levels.

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- *STEMI: A Cardiology Update* is the first publication in the form of a textbook from India, under the editorship of HK Chopra, New Delhi, India; and Sameer Mehta, Miami, USA.
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