REVIEW

Impact of Indoor Air Pollutants on the Cardiovascular Health Outcomes of Older Adults: Systematic Review

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Abstract: Indoor air pollution accounts for approximately 3.8 million inopportune deaths annually at global level. Due to spending more time indoors, children and older adults are especially susceptible to the health risks of indoor air pollution. This review seeks to summarise existing knowledge on the cardiovascular health effects of three common indoor air pollutants, namely carbon monoxide (CO), particulate matter ($PM_{2.5}$ and PM_{10}), and Nitrogen dioxide (NO_2), focusing on older adults. We systematically reviewed the literature (PROSPERO CRD42024479220) on PubMed, Google Scholar, Scopus, Web of Science and Embase. The search yielded 20,914 records. Two independent reviewers screened the articles using titles, abstracts, and full-length articles written in English. Upon a detailed assessment of all the records, the review considered 38 full-length articles. Several studies reported mortality, myocardial infarction, stroke, increased hospitalisation and increased emergency room visits due to exposure to indoor air pollutants. The increased mortality, morbidity, hospitalization, and emergency rooms visits resulting from indoor air pollution associated CVDs makes indoor air pollution a health risk for older adults. There is, therefore, a need to synthesize information on studies relate d to how the selected indoor air pollutants affected the cardiovascular health of older adults.

Keywords: particulate matter, PM_{2.5}, PM₁₀, nitrogen dioxide, myocardial infarction, stroke, hospitalisation, mortality, older adults

Introduction

Indoor air pollution is an emerging problem which negatively affects health of people worldwide. While air pollutants are a natural occurrence, their types and concentrations have skyrocketed due to industrialization and urbanization, driven by human activities.¹ Air pollution is a major global health threat, contributing to millions of deaths annually.² Outdoor pollutants alone cause over 4 million deaths, while indoor air pollution is responsible for an additional 2.3 million.³ This exposure raises the risk of various cardiovascular, respiratory, and neurological diseases.³ While the specific sources of indoor air pollution can differ depending on location, some common culprits are inefficient fuel burning for cooking and heating. The most used biomass fuels include wood, crop residues, and animal dung, while the solid mass fuels include charcoal and coal.⁴ Cooking and heating with these usually take place indoors using inefficient stoves, leading to incomplete combustion due to the limited oxygen supply. The result of incomplete combustion of these fuels is the production of carbon monoxide, black carbon, and complex organic carbon compounds.⁵ The situation is further aggravated by the inadequate ventilation prevalent in many kitchens in low income settings of developing countries. Indoor cooking with solid fuels often generates smoke containing particulate matter (PM) levels as high as 1000 mg/m³, with even higher concentrations having been documented.⁴ These levels far exceed the national ambient air quality standards for PM set by the US Environmental Protection Agency (EPA) and the guidelines established by the World Health Organization (WHO). In addition, use of household products such as cleaning detergents, pesticides, and solvents

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Cardiovascular diseases (CVDs) are among the top causes of mortality and disability globally with over 17.9 million deaths recorded in 2019^{7,8} and this is predicted to escalate to 32.3 million in 2050. The risk of death from cardiovascular disease (CVD) increases dramatically with age.⁹ After 50, the number of fatalities from CVD roughly doubles with each passing decade.⁹ A disproportionate burden, three-quarters (75%) of these CVD deaths, falls on low- and middle-income countries (LMICs).⁷ Limited access to primary care in these regions hinders early detection and treatment, leading to delayed diagnoses and tragically, earlier deaths.⁷ The growing burden of cardiovascular diseases (CVDs) and their associated risk factors stems from a complex interplay of factors.¹⁰ This includes the ongoing shift in population demographics known as the epidemiological transition.¹⁰ Factors like increasing urbanization, changing lifestyles and diets, socioeconomic development, and modernization all play a role.¹⁰ Furthermore, growing evidence suggests that environmental factors significantly contribute to CVD risk. As much as a quarter of all ischemic heart disease (IHD) cases may be linked to an unhealthy environment – factors like air pollution and even traffic noise can have a significant impact.¹

People from middle- and low-income countries particularly experience the highest rates of health challenges related to poor air quality¹¹ because they lack the resources to obtain cleaner fuels and devices.¹² The key pollutants identified are particulate matter, sulphur dioxide, nitrogen dioxide, volatile organic compounds carbon monoxide, and hydrocarbons.¹³ The most common forms of indoor air pollution in low-income settings come from the biomass fuels which produce particulate matter (PM_{2.5} and PM₁₀) and carbon monoxide (CO).¹⁴ African cities are believed to have much higher levels of fine particulate matter (PM_{2.5}) compared to most European and North American cities.¹¹ PM_{2.5} concentrations in African cities are estimated to be around five times higher, reaching 100 micrograms per cubic meter (mg/m3), whereas most European and North American cities have levels below 20mg/m3.¹¹ In low-income countries, a significant portion of the population, an estimated 57%, is directly exposed to unhealthy concentrations of PM2.5, a dangerous air pollutant.² Long-term exposure to particulate matter has been linked to oxidative stress and inflammation in the respiratory system, contributing to respiratory and cardiovascular diseases (CVDs).^{15,16} This oxidative stress occurs either through direct inhalation of reactive oxygen species (ROS) or indirectly via toxic aerosol compounds from sources like combustion. Inhaled particles can reach the alveoli, triggering inflammatory responses, while some ultrafine particles may enter the bloodstream, affecting cardiovascular function and the autonomic nervous system.¹⁵ Numerous studies have found that air pollution is associated with increased inflammatory biomarkers and subclinical effects such as oxidative stress and autonomic imbalance, emphasizing its impact on public health.¹⁷

Age is a well-established risk factor for a range of cardiovascular diseases (CVDs). This includes conditions like coronary artery disease, high blood pressure (hypertension), heart failure, and stroke.¹⁸ The global burden of cardiovascular disease (CVD) is increasing with age, and this trend is particularly concerning in low-income countries. Adults in these settings experience disproportionately higher rates of CVD compared to those in wealthier nations.¹⁹ Overtime, the body undergoes aging, a process marked by gradual buildup of cellular damage, a decline in how well our organs and systems function, and a greater risk of developing various diseases.¹⁸ With age comes a heightened risk of chronic illnesses like heart disease, diabetes, and cancer.²⁰ These pre-existing conditions in older adults can significantly worsen the impact of air pollutants on their health.²¹

Methods

The protocol for this review was registered with the International prospective register of systematic reviews (PROSPERO) in accordance with PRISMA guidelines (PROSPERO CRD42024479220).

Search Strategy

Online databases, particularly PubMed, Google Scholar, Scopus, Web of Science and Embase were used to conduct the systematic review between November 2023 and March 2024. Full Length prospective cohort studies, retrospective cohort studies, case-control studies, cross-sectional studies, randomized controlled trials (RCTs) and longitudinal studies with follow-up periods sufficient to assess cardiovascular health outcomes were used in this systematic review. Only those

Table I Search Strategy

Category	Keywords used
Population	"Elderly" OR "Seniors" OR "Geriatric population" OR "Aging population" OR "Older individuals".
Exposure	"Particulate matter (PM _{2.5} & PM ₁₀)" OR "Nitrogen dioxide" OR "Carbon monoxide".
Comparator	"Low pollution areas" OR "Clean air environments" OR "Reduced exposure to pollutants" OR "Control groups".
Outcome	"Cardiovascular disease" OR "Stroke" OR "Myocardial infarction" OR "Hypertension" OR "Arrhythmia" OR "Heart failure" OR
	"Mortality"

articles reported in English between 2013 and 2024 were considered for the review. The Population Exposure Comparison Outcome (PECO) tool was used to develop the search criteria as shown in Table 1. Briefly, the population of interest was older adults aged 65 years and above, the exposure was indoor air pollutants (NO₂; SO₂; PM_{2.5}; PM₁₀), comparator population was adults above 65 years living in less polluted/cleaner environments and the outcome was cardiovascular health. The retrieved documents were manually screened by two reviewers independently. Instances of discordance were addressed through deliberation to facilitate merging of extracted data. Ancillary sources, such as editorials, reviews, and case reports, were excluded from consideration, and materials classified as grey literature were also omitted from the analysis.

Data Extraction

The study titles were screened and records meeting the predetermined criteria were subsequently transferred to Microsoft Excel spreadsheet. Data such as the study title, abstract, and year of publication was extracted from these chosen studies. We removed duplicate entries and irrelevant studies. This process ensured only full-text articles relevant to our systematic review were included for further analysis.

Results

The details of the study selection process are shown in Figure 1. Results for the systematic review of literature are shown in Table 2.

All thirty-four studies included in this review were from only 3 continents (Asia, Europe and North America). More than half of the studies (22) included in this systematic review were from Asia, 8 from North America and the remaining 8 were from Europe. The most reported cardiovascular health outcomes from all the studies in descending order included mortality, 26,27,29,34,35,40,51,55 myocardial infarction, 23-25,34,38,39,46,55 stroke, 24,26,34,41,44,49,54 increased CVD hospital admissions,^{22,28,41,42,47,49} heart failure,^{24,32,34,41,47} Ischaemic heart disease,^{26,32,41,53} increased emergency room visits (4 studies),^{37,45,48,50} hypertension^{15–17,30,56} and arrhythmia.^{24,39,48} The study conducted in the United States by Haves et al revealed that a 10µg/m3 increase in PM2.5 was associated with a 16% increase in Ischaemic Heart disease-related mortality and a 14% increase in stroke-related mortality.²⁶ A study done in the Hubei province of China by Liu et al reported that short-term exposure to PM_{2.5}, PM₁₀, and NO₂ was associated with an increased risk of myocardial infarction mortality, and the association was stronger with NO2.55 Interestingly, 5 out of 8 studies reporting on mortality attributed it to NO2.27,29,51,53,55 All studies reporting myocardial infarction as a resultant cardiovascular health outcome of indoor air pollution, except for one study by Atkinson et al, attributed the increased risk of myocardial infarction to exposure to $PM_{2.5}$.^{23,25,34,38,39,46,55} More specifically, the study done in Belgium on adults \geq 75 years by Argacha et al reported that exposure to PM2.5 and NO2 increased the risk of ST-elevation myocardial infarction (STEMI). Furthermore, they reported that the risk related to PM_{2.5} appears to be greater in the elderly compared to younger individuals.²³ A study conducted in Ontario by Bai et al reported a linear relationship between exposure to PM2.5 and acute myocardial infarction (AMI).²⁵ Contrary to these reports, Atkinson et al's study on older adults in the UK reported a weak association between indoor air pollution exposure and myocardial infarction.¹⁷



Figure I Study selection process.

Discussion

This systematic review investigated the impact of indoor air pollution on the cardiovascular health outcomes of older adults. Most studies came from Asia, possibly because of the high population density² and rapid industrialisation in countries like China, India, and South Korea⁵⁷ which has prompted extensive studies to understand its impacts. Majority (nearly 99%) of people are exposed to air pollution exceeding the safety limits set by the World Health Organization (WHO),³ with low- and middle-income countries being the most exposed.¹² Low-income countries face a double burden of air pollution due to weaker regulations, older machinery, subsidized fossil fuels, congested transportation, rapid industrial growth, and agricultural practices that all contribute to significantly higher pollution levels.² Grey literature has shown that airborne pollutants in Sub-Saharan African cities are primarily composed of gases and vapours (over 90%), namely particulate matter, NO₂, volatile organic compounds (eg, benzene), SO₂ and CO.¹³ Our systematic review did not find any studies reporting on the impact of indoor air pollution on the cardiovascular health outcomes of older adults in Africa despite the continent being flagged as potentially exposed to high pollution, as highlighted in grey literature.^{12,13} However, some studies have been done on indoor air quality and CVD risk in children in South Africa.^{58,59} The present findings indicate a notable deficiency in comprehension and awareness on the adverse impacts on cardiovascular well-being attributable to

Country	Study Design	Sample Size	Population	Exposure	Outcome	Reference
China	Longitudinal	460 938	≥65 years	Short term PM _{2.5}	Increased admission due to CHDs	[22]
Belgium	Case crossover	2598	≥75 years	10µg/m3 of PM _{2.5} , NO ₂	 Increased the risk of MI 	[23]
United Kingdom	Retrospective cohort	8336 557	40-89 years	Long term, PM, SO ₂ & NO ₂	• Weak association between exposure MI, stroke, or	[24]
					arrhythmia	
					 Increased development of HF 	
Ontario, Canada	Retrospective Cohort	5100 000	35–85 years	PM _{2.5}	 Increased risk for acute MI 	[25]
US	Longitudinal	565 477 men and	50–71 years	10 μg/m3 PM _{2.5}	 Increased mortality due to IHD and stroke 	[26]
		women				
China	Longitudinal	21,816	≥75 years	PM ₁₀ , SO ₂ , and NO ₂	 Increased CVD mortality 	[27]
Iran	Longitudinal	84,114	65–74 years	PM ₁₀ , NO ₂ , SO ₂ , CO	 Increased hospital admissions for CVD 	[28]
Britain	Longitudinal	7569	40–69 years	Long term PM _{2.5} , PM ₁₀ , PMcoarse,	 Increased CVD Mortality 	[29]
				SO ₂ , NO ₂		
China	Prospective cohort	3754	≥65years	Biomass fuel	 Increased risk of hypertension 	[30]
Shiraz, Iran	Case crossover		≥65 years	Short term exposure, PM_{10} , SO_2 ,	 Increased CVD morbidity 	[31]
				and NO ₂		
New York City	Case control	837523	67±15 years	NO ₂ , PM _{2.5} , SO ₂	 Increased risk of CVD, IHD, and HF 	[32]
US	Prospective cohort study	6795	45–84 years	PM _{2.5}	• Increased coronary calcification, increased progression of	[33]
					atherosclerosis	
Korea	Retrospective cohort study	900845	≥65 years	PM _{2.5}	 Increased cardiovascular mortality, 	[34]
					Acute MI	
					Congestive heart failure	
					• Stroke.	
Northern	Retrospective cohort	169714	60.8±15.6	PM _{2.5}	 Increased risk of cardiovascular mortality 	[35]
California						
US		502	29–89 years	PM _{2.5}	 Increased risk of AF 	[36]
Beijing	Longitudinal	82,430	≥65 years	NO ₂ , SO ₂ , PM ₁₀	 Increased cardiovascular emergency room admissions 	[37]
North Carolina	Retrospective cohort	5679	60.8±12.1	Daily, long term PM _{2.5}	 Increased CAD and incidence of MI 	[38]
Wales	Case cross over		60–81 years	PM _{2.5}	Arrhythmias and AF	[39]
				NO ₂	 Increased risk for MI 	
North-Eastern Iran	Retrospective Cohort	50045	40–75 years	Biomass fuels	 Increased risk for CVD mortality 	[40]
Vietnam	Case crossover	135 101	≥65 years	PM _{2.5} ; PM ₁ ; SO ₂	 Increased daily hospital admissions due to CVD conditions 	[41]
					(IHD, CF, stroke)	
Thailand	Retrospective cohort	503 105	≥65 years	NO_2 , SO_2 , PM_{10}	 Increase in CVD hospital admission 	[42]
Vietnam	Retrospective cohort	N/A	≥65 years	PM ₁₀ ; NO ₂ & SO ₂	Increased cardiovascular admissions	[43]
Western	Prospective Cohort	4105	45–76 years	PM _{2.5} and PM ₁₀	 Increased risk of stroke 	[44]
Germany						
New Mexico	Case crossover	4739	≥65 years	PM _{2.5} and PM ₁₀	 Increased emergency room visits for CVDs 	[45]

Table 2 Cardiovascular Health Outcomes of Older Adults Exposed to Indoor Air Pollution

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(Continued)

Table 2 (Continued).

Country	Study Design	Sample Size	Population	Exposure	Outcome	Reference
Finland	Case crossover	N/A	65.5±12.5	PM _{2.5}	• Increased risk of acute MI	[46]
			years			
China	Prospective cohort	4866	65.2±13.5	PM _{2.5} , PM ₁₀ , CO, SO ₂	 Increased risk of HF readmission 	[47]
Beijing China	Time series	N/A	45–75 years	SO3, NO3, PM10	 Increased CVD emergency room visits 	[48]
		-	,	PM _{2.5}	 Increased arrhythmia 	
Beijing, China	Retrospective cohort	63,956	≥65 years	Short term PM _{2.5}	Increased hospitalisation for Ischaemic stroke	[49]
Shanghai, China	Cross sectional	N/A	≥65 years	PM _{2.5} , PM ₁₀	• Increased risk of CHD outpatients and emergency depart-	[50]
					ment visit	
Hong Kong, China	Prospective cohort	66820	≥65 years	Long term PM _{2.5} , NO ₂	Elevated risk of cardiovascular mortality.	[51]
China	Cross sectional	24,845	18–74 years	PM 1, PM _{2.5}	 Increased prevalence of CVDs 	[52]
Wuhan, China	Case cross over	8955	≥65 years	PM10, NO2, SO2	 Increased risk of IHD 	[53]
				NO ₂	Cardiovascular mortality	
Okayama, Japan	Case crossover	10,949	≥65 years	Particulate matter	 Increased risk of haemorrhagic and ischemic stroke 	[54]
Hubei Province,	Case cross over	15 1608	70–85 years	PM _{2.5} , PM ₁₀ , and NO ₂	 Increased MI mortality. 	[55]
China					 The association was stronger with NO₂ 	
Germany	Randomised controlled	54	65–79 years	PM_1 , $PM_{2.5}$ and PM_{10}	• $PM_{2.5}$ and PM_{10} increased systolic blood pressure	[15]
	exposure	25				FF (1
Beijing, China	Randomised crossover	35	Average 66.26	PM2.5 and black carbon	• PM _{2.5} increased BP	[56]
Change China	Intervention	24	years			F 1 77
Chongqing, China	Randomised double-blind	24	61-97 years	PIM_{1} , $\operatorname{PIM}_{2.5}$ and PIM_{10}	 Increased markers of blood inflammation and heart rate 	[17]
C	crossover trial		(5.70)			F 1 77
Germany	exposure	55	65–19 years	$PI_{1,}$ $PI_{2.5}$ and PI_{10}	 Piti, and Piti2.5 increased arterial stiffness indices 	[17]

Abbreviations: NO₂, Nitrogen dioxide; SO₂, Sulphur dioxide; CO, Carbon monoxide; PM, Particulate matter; PM_{2.5}, Particulate matter 2.5; PM₁₀, Particulate matter 10; MI, Myocardial infarction; CF, Cardiac failure; CVD, cardiovascular diseases; CHD, coronary heart diseases; CAD, coronary artery diseases; AF, Atrial fibrillation; IHD, Ischaemic heart diseases; HF, Heart failure; BP, Blood pressure.

air pollution across all life stages in the African continent. It is known that cardiovascular aging is a major trigger for cardiovascular diseases in elderly people. As cardiovascular function declines with age, the risk of atherosclerosis, stroke, heart attack (myocardial infarction), and other diseases with poor outcomes all rise significantly.⁹ Stroke has previously been reported as the major contributor to the total CVD deaths in people older than 85 years.⁶⁰ While ischemic stroke is more common overall, intracerebral hemorrhage causes the most disability-adjusted life years (DALYs) and deaths in people under 70.⁶¹ Our study confirms the reports by previous studies; mortality, myocardial infarction, and stroke were the top 3 reported cardiovascular health outcomes of older adults in this systematic review, and in some studies, mortality was attributed to myocardial infarction or stroke. This illustrates the extent of deleterious effects of exposure to atmospheric pollutants on cardiovascular health outcomes among the elderly demography.

Particulate matter (PM), particularly PM_{2.5} was the most reported air pollutant with most studies reporting myocardial infarction. This could be because PM2.5, the major component of air pollution, easily enters the bloodstream due to its tiny size, potentially affecting organs more readily than larger PM10 particles. Although there are finer particles than PM2.5, known as the ultra-fine particles with an aerodynamic diameter of less than 0.1µm, also known as UFPs, the latter are less abundant in the atmosphere than PM25.⁶² However, both PM25 and even finer particles called UFPs can travel deep into the lungs and enter the bloodstream, potentially causing harm throughout the body.⁶³ Inhaling air pollution, particularly PM2.5, can trigger harmful consequences within the lungs. These tiny particles reach the alveoli, the air sacs where oxygen exchange occurs. PM can directly generate reactive oxygen species (ROS), molecules that damage cells or indirectly cause their production.⁶⁴ This process, known as oxidative stress, can also be initiated by inhaling other toxic compounds in air pollution.⁵ The lungs respond to PM exposure by activating immune cells called alveolar macrophages. These macrophages release inflammatory molecules (cytokines) that can travel throughout the body, potentially affecting heart function and blood vessel control. PM enters the body through the respiratory tract, with larger particles (>50µm) often trapped by the nose. PM10 may lodge in the upper airways, while the tiny PM2.5 particles can penetrate deep into the lungs and even enter the bloodstream. It's important to note that carbon monoxide (CO), another air pollutant, has a different mechanism of harm. Unlike PM, CO toxicity does not involve oxidative stress but rather its ability to disrupt oxygen transport in the blood at very high concentrations (not typically found in ambient air).⁶⁴ When inhaled, CO travels to the lungs and reaches the alveoli.⁶⁵ Once in the alveoli, CO diffuses across the thin alveolar-capillary membrane into the bloodstream, displacing O₂ in haemoglobin and binds irreversibly to haemoglobin thus depriving organs of oxygen leading to hypoxia.⁶⁴ This systematic review reported CO in only 2 studies,^{28,47} possibly because it usually does not occur in ambient air. A study by Shah et al found a link between rising carbon monoxide (CO) levels and an increased risk of hospitalization or death from heart failure (HF).⁶⁴ This risk increased by 3.52% for every one part per million increase in CO levels.⁶⁴ The confidence interval (CI) indicates a 2.52% to 4.54% increase in risk.⁶⁴ One of the studies included in this review also reported that CO increased CVD hospitalisations,²⁸ while that by Shi et al reported an increased risk in HF readmissions.⁴⁷

Nitrogen Dioxide (NO2) is a gas formed from both natural processes and human activities like burning fossil fuels in power plants and cars.^{66,67} It can also contribute to the formation of other harmful air pollutants.⁶⁶ Studies have shown a link between NO2 exposure and negative health effects, particularly on the heart. In people with weakened heart muscles (dilated cardiomyopathy), NO2 has been associated with structural changes in the heart itself.⁶⁸ Long-term exposure to NO2 may also increase the risk of death from heart disease and other causes, with one study finding a 13% rise in cardiovascular deaths for every 10 micrograms per cubic meter increase in annual NO2 levels.⁶⁹ Research by Huang et al further suggests that long-term NO2 exposure can increase the risk of death from various causes, including respiratory and cardiovascular problems⁶⁷.

In the current study, NO₂ was the most dominant air pollutant in mortality reports, 27,29,51,53,55 confirming the previously reported association of this pollutant with increased mortality. A study by Zhang et al in China investigating the link between air pollution and cardiovascular deaths found no statistically significant effect of NO₂ on CVD mortality.²⁷ This suggests that the impact of NO₂ on heart health may vary depending on other factors not explored in any of the studies included in this systematic review. Later in 2021, Liu et al carried out a study in China on short term exposure to air pollution and mortality from MI. Their study found that the risk from PM_{2.5} and PM₁₀ exposures rose sharply until certain levels (PM_{2.5} at 33.3 mg/m³ and PM₁₀ at 57.3 mg/m³), which levelled off. In contrast, the risk from NO₂ exposure increased steadily nearly linearly.⁵⁵ This implies minimal risk associated with PM2.5 and PM10 exposure levels beyond the threshold points. Meanwhile, with the increase in NO₂ levels, the risk of adverse health outcomes

increases consistently, without a noticeable breakpoint where the risk changes dramatically. Interestingly, the study also found that the link between NO₂ exposure and death from heart attack (MI) was stronger in older adults compared to younger people.⁵⁵ This contradicts previous findings suggesting that NO₂ has a greater impact on younger patients.²³ While NO₂ is often used to indicate overall air pollution, research is ongoing to determine its exact independent contributions to health problems. However, increasing evidence suggests that NO₂ can have harmful effects on its own beyond simply reflecting the presence of other pollutants.^{55,67,69} The connection between exposure to these pollutants and adverse cardiovascular outcomes in older adults is shown in Figure 2 and the two possible mechanisms linking air pollutants to CVDs are summarized in Figure 3.



Figure 2 Connection between exposure to indoor air pollutants and adverse cardiovascular outcomes in older adults. An illustration of the cardiovascular health impacts of indoor air pollution from the use of biomass and solid fuels for cooking. Pollutants such as particulate matter, sulphur dioxide, nitrogen dioxide, and carbon monoxide are released during combustion and inhaled into the body. These pollutants contribute to cardiovascular conditions, including stroke, ischemic heart disease (IHD), arrhythmia, heart failure, myocardial infarction, and ultimately increased mortality.



Figure 3 Two possible mechanisms by which air pollutants affects the cardiovascular system. I: PM_{10} and $PM_{2.5}$ can penetrate deep into the respiratory system and even enter the bloodstream. Once in the body, they can trigger inflammation. PM-induced inflammation can lead to endothelial dysfunction, oxidative stress, and activation of immune cells. Inflammation plays a crucial role in the development and progression of cardiovascular diseases such as atherosclerosis, hypertension, and heart failure. 2: NO_2 can impact the autonomic nervous system, which regulates involuntary bodily functions such as heart rate, blood pressure, and respiratory rate. This imbalance in autonomic tone can contribute to the development of cardiovascular diseases by promoting hypertension, arrhythmias, and myocardial ischemia.

Conclusion

Studies have linked exposure to indoor air pollutants with a higher risk of cardiovascular problems in older adults. This includes an increased chance of death from cardiovascular disease (CVD), heart attack (MI), and stroke. Additionally, older adults exposed to indoor air pollution may experience more hospital admissions and emergency room visits for cardiovascular issues. $PM_{2.5}$ was associated with most reports of myocardial infarction, while NO₂ was associated with most mortalities reported by studies in this review. These findings imply that exposure to PM_{10} , $PM_{2.5}$ and NO₂ can potentially reduce the quality of life of older adults. The paucity of data on the impact of indoor air pollutants on cardiovascular health in some continents poses a public health challenge as individuals may be exposed unknowingly leading to potential cardiovascular problems such as MI, stroke and even death. Additionally, in the absence of comprehensive data, public health policies and regulations may not adequately address indoor air quality issues, resulting in failure to mitigate exposure to harmful pollutants, leaving populations vulnerable to associated with exposure to indoor air pollutants.

Disclosure

The authors report no conflicts of interest in this work.

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