



Contents lists available at ScienceDirect

American Heart Journal Plus: Cardiology Research and Practice

journal homepage: www.sciencedirect.com/journal/american-heart-journal-plus-cardiology-research-and-practice



Air pollution and cardiovascular disease: a systematic review of the effects of air pollution, including bushfire smoke, on cardiovascular disease[☆]

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ARTICLE INFO

Keywords:

Air pollution
Bushfires
Cardiovascular disease
Morbidity
Mortality

ABSTRACT

Objective: Particulate matter (PM) with diameter $\leq 2.5 \mu\text{m}$ (PM_{2.5}) and $\leq 10 \mu\text{m}$ (PM₁₀), including from bushfire smoke, is associated with cardiovascular disease (CVD) morbidity and mortality. This systematic review assesses how CVD morbidity and mortality is affected by type, duration, and level of air pollution exposure.

Data sources: A search was conducted on Ovid Medline, Embase and Scopus, spanning across 1 January 2012 to 30 July 2022. Primary quantitative studies exploring the effect of PM_{2.5}, PM₁₀ or bushfire smoke on CVD were included. Studies without adjustment for confounding factors were excluded. The Newcastle-Ottawa Scale was used to assess the risk of bias (ROB) in the studies, and meta-analysis was conducted on relevant outcomes.

Findings: A total 275 studies were obtained, and 80 studies were analysed with diseases ranging from ICD-10 I00-I99. For CVD morbidity, increased PM_{2.5} and PM₁₀ was associated with 1.92 (95 % CI: 0.58,3.26) years of life lost per $10 \mu\text{g}/\text{m}^3$ increase in exposure. Increased PM_{2.5} and PM₁₀ was associated with a 0.52 % (95 % CI: 0.37,0.68) increase in mortality per $10 \mu\text{g}/\text{m}^3$ increase in exposure. Bushfire smoke also presented similar trends. Two studies had high ROB, 42 had medium ROB, and 36 had low ROB. There was high heterogeneity between the studies, with I^2 values ranging between 88.09 % and 94.25 %.

Conclusion: Air pollution including bushfire smoke is associated with increased CVD morbidity and mortality. This effect ranges across different types, durations, and levels of air pollution exposure, making stringent climate change and air pollution mitigation strategies imperative.

1. Introduction and background

Currently, 90 % of the world's population is exposed to air pollution levels above World Health Organisation Air Quality Guidelines (WHO AQG) [1]. The 2022 guidelines recommend the daily maximum for particulate matter (PM) $\leq 2.5 \mu\text{m}$ (PM_{2.5}) to reach $15 \mu\text{g}/\text{m}^3$ daily, and $5 \mu\text{g}/\text{m}^3$ annually. For PM $\leq 10 \mu\text{m}$ (PM₁₀) these levels are $45 \mu\text{g}/\text{m}^3$ and $15 \mu\text{g}/\text{m}^3$ respectively [2]. Air pollution caused 6.7 million deaths in 2019, with over 50 % attributed to CVD [3]. Even low air pollution levels cause mortality, with individuals above 45 most at risk [4,5]. Bushfires smoke, a significant cause of air pollution, also increases CVD mortality, with the 2019–2020 Australian bushfires resulting in a 4.5 % increase in daily CVD mortality [6].

This systematic review explores how air pollution exposure affects CVD, with the relationship between air pollution and CVD becoming increasingly apparent. Air pollution is the 4th largest modifiable risk factor for CVD according to the global burden of disease study 2019 [7].

The Lancet Commission on pollution and health reported that air pollution led to 21 % of CVD deaths in 2015 [8]. Previous studies demonstrated that PM_{2.5} increases CVD morbidity and mortality, with heightened risk of ischaemic heart disease (IHD), arrhythmias and heart failure (HF) [9]. The latest umbrella review on the topic confirmed that short and long-term exposure to PM_{2.5} increases CVD mortality, along with higher rates of IHD, HF, stroke, hypertension, diabetes, arrhythmias and atherosclerosis [10]. The study examined systematic reviews until January 2021, leaving the COVID-19 pandemic unexplored. This research gap is of interest as global PM levels have reduced in highly polluted areas post-lockdown, with PM_{2.5} concentrations reducing by 200 % in Delhi, and by 31 % in Wuhan [11]. This review will also address the effects of bushfire smoke as a major source of air pollution impacting on CVD morbidity and mortality.

Thus, this systematic review aims to summarise the extent to which different types, durations and levels of air pollution continue to harm cardiovascular health. This will highlight the ongoing health threat

[☆] This article is part of a Special issue entitled: 'Climate Change and CV Health' published in American Heart Journal Plus: Cardiology Research and Practice.

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<https://doi.org/10.1016/j.ahjo.2025.100546>

Received 1 April 2024; Received in revised form 24 October 2024; Accepted 18 April 2025

Available online 19 April 2025

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posed by air pollution and can encourage further action to mitigate climate change and reduce air pollution levels.

2. Methods

This systematic review with meta-analysis was developed according to the PRISMA 2020 statement. A search was conducted by one reviewer (FI) on Ovid Medline, Embase and Scopus, limiting to articles published between 1 January 2012 and 30 July 2022, reporting human outcomes, with English text available. Broad terms were used to include all papers on PM or bushfire smoke exposure, and CVD morbidity or mortality. The search terms were: “air pollution” or “particulate matter” or “wildfire” or “bushfire” and “cardiovascular disease” or “heart disease” and “morbidity” or “mortality” (see Table S1 for search strategy). The final access date was 30 July 2022. Endnote20 was used to remove duplicates. The titles and abstracts of the remaining articles were screened by two reviewers (FI, SN), followed by a full-text screen. A third reviewer (PS) resolved discrepancies.

Included studies had English full-text available, explored the effect of PM_{2.5}, PM₁₀ or bushfire smoke on CVD, and reported quantitative measures of CVD morbidity or mortality. We excluded studies lacking adjustment for confounding factors such as ambient temperature or other pollutants (e.g. NO₂, ozone and noise). They were also excluded if the diseases explored were CVD risk factors (e.g. hypertension) or lacked ICD codes. Finally, non-primary studies were excluded.

Two reviewers (FI, SN) extracted the data, and another reviewer (PS) resolved discrepancies. We extracted authors; publication year; study

design and location; mean level, type, and duration of air pollution exposure; ICD code; and outcome. Morbidity outcomes included years of life lost (YLL), percentage increase in hospital, emergency department (ED) and general practice (GP) presentations, and incidence of disease per incremental increase in PM. Mortality was mostly reported as percentage increase in mortality per incremental increase in PM. Google sheets was used to tabulate the data. A narrative synthesis and meta-analysis was conducted to pool the results addressing morbidity and mortality associated with PM_{2.5}, PM₁₀ and bushfire smoke. Meta-analysis was performed using the random effects restricted maximum likelihood model in Stata/MP 17.0 when three or more studies reporting comparable outcomes could be pooled to obtain an overall effect size. Outcome measures of the same metrics were converted to a common scale where possible.

The Newcastle-Ottawa Scale was used to ascertain the risk of bias (ROB), assessing the selection and comparability of study groups and outcomes, thus judging whether the level of bias affects the validity of the publication's conclusions [12]. Studies with a score of 0–3 out of 9 were deemed to have a high ROB, whilst scores of 4–6 and 7–9 indicated medium and low ROB respectively. Publication bias was evaluated based on the funnel plots generated in Stata/MP 17.0 with each meta-analysis, and this assessment was based on the distribution of studies in comparison to the estimated effect size. I² values were used to evaluate study heterogeneity.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

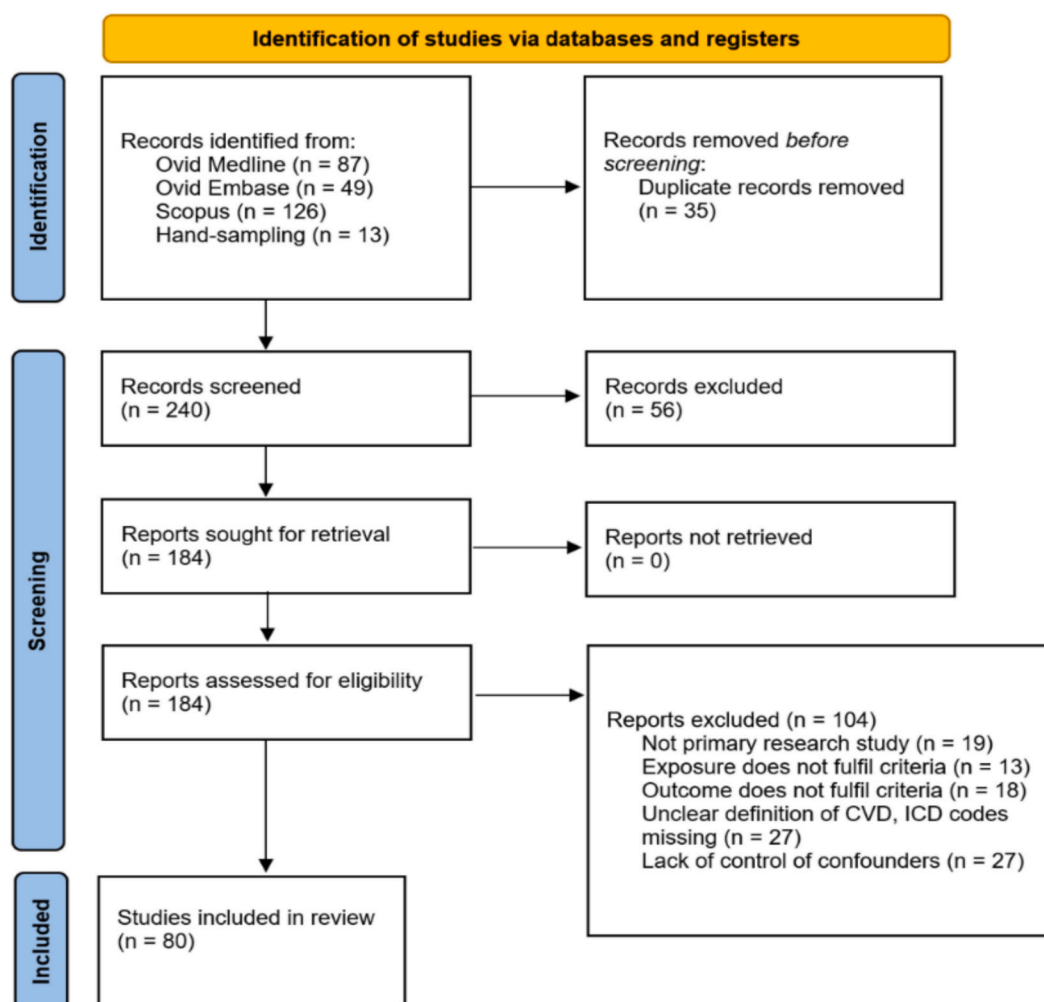


Fig. 1. PRISMA flowchart depicting the identification, screening and inclusion process for publications exploring the effects of air pollution on CVD.

3. Results

We found 275 studies through literature search and manual selection. Duplicates were eliminated, leaving 240 studies for title and abstract screening. We then excluded 56 irrelevant studies, leaving 184 for full-text screening. After applying our exclusion criteria, 80 articles were included in this systematic review (Fig. 1).

The studies included case-crossover, cohort, modelling, and time-series analysis designs. In terms of location, 38 studies were from Asia, 19 from Europe, 16 from the Americas, 4 from Oceania and 3 studies were global [13–92]. The effects of PM_{2.5} or PM₁₀ were explored in 73 studies [13–17,19–21,23–26,28–35,37–40,42–67,69–74,76–92], and 7 studies explored the effects of bushfire smoke [18,22,27,36,41,68,75]. Short-term effects were explored in 27 studies, whilst the remaining 53 explored the effect of long-term air pollution exposure on CVD morbidity and mortality. Mortality was reported in 60 studies, 10 reported morbidity and mortality, and 10 reported morbidity only. Mean air pollution levels higher than WHO AQG were reported in 68 studies, with only 12 studies reporting levels below the guidelines (Table 1).

The diseases explored included all-cause CVD (ICD-10 I00-I99), ischemic heart disease (IHD) including chronic IHD (ICD-10 I20-I25), acute myocardial infarction (AMI, ICD-10 I21-I22), atrioventricular conduction disorder (AVCD, ICD-10 I45), sudden cardiac death (ICD-10 I46), arrhythmias including atrial fibrillation (AF, ICD-10 I47-I49), heart failure (HF, ICD-10 I50), unspecified cardiac disease (ICD-10 I51) and peripheral vascular disease (PVD, ICD-10 I70-I79).

4. Effect of PM on CVD

4.1. Morbidity

Six studies explored the effect of short-term PM_{2.5} exposure on CVD morbidity, with 5 reporting PM_{2.5} levels higher than WHO AQG. Measures of morbidity included YLL (3 publications), percentage increase in ED presentations (1) and hospital admissions (2) per incremental increase in PM_{2.5}. The diseases explored were arrhythmias including AF, AMI, AVCD, unspecified cardiac disease, HF, IHD, sudden cardiac death and all-cause CVD. There was a negative association between all-cause CVD hospital admissions and PM_{2.5} exposure, with the percentage change in hospital admissions ranging from –1.06 % (95 % CI: –1.69,

0.5) to 0.65 % (95 % CI: 0.46,0.84). However, there was a positive association between PM_{2.5} and both ED presentations and YLL. Associations between short-term PM_{2.5} and other CVD morbidity outcomes are shown in Table 2. The study that reported PM_{2.5} levels below WHO AQG found a negative association between CVD hospital admissions and PM_{2.5} exposure, reporting a 1.09 % (95%CI: –1.52,-0.66) decrease in hospital admissions per 10 µg/m³ increase in PM_{2.5}. However, the study with PM_{2.5} levels above the guidelines reported a 0.81 % (95%CI: 0.53,1.10) increase for the same outcome. The pooled results for YLL reported 2.53 (95 % CI: 0.03,5.02) YLL per 10 µg/m³ increase in PM_{2.5}.

Eight studies explored the effect of long-term PM_{2.5} exposure on CVD morbidity, with PM_{2.5} levels above WHO AQG for all. Morbidity outcomes included an increase in hospital admissions (3), YLL (2), and incidence of disease (3) per incremental increase in PM_{2.5} exposure. Diseases explored included AF, AMI, IHD, HF and all-cause CVD. For all-cause CVD, there was a positive association between PM_{2.5} and both YLL and hospital admissions, with the YLL ranging between 0.88 (95 % CI: 0.19,1.57) and 8.02 (95 % CI: 4.94,11.1) per 10 µg/m³ increase in PM_{2.5}.

Overall CVD morbidity associated with PM_{2.5} exposure was measured by pooling the results from all studies assessing either short-term or long-term PM_{2.5}. Five out of 14 studies reported the YLL per 10 µg/m³ increase of PM_{2.5}, and the pooled results reported 1.00 (95 % CI: 0.47,1.53) YLL per 10 µg/m³ increase in PM_{2.5}.

Four studies explored the effect of short-term PM₁₀ exposure on CVD morbidity, with 2 reporting PM₁₀ levels below WHO AQG. Morbidity outcomes included percentage increase in hospital admissions (3) and YLL (1) per 10 µg/m³ increase in PM₁₀. The diseases explored were arrhythmias including AF, AMI, AVCD, unspecified cardiac disease, HF, IHD including chronic IHD and all-cause CVD. There was a positive association between PM₁₀ and both all-cause CVD hospital admissions and YLL, reporting 4.1 (95 % CI: 1.9,6.3) YLL per 10 µg/m³ increase in PM₁₀. Associations between short-term PM₁₀ and other CVD morbidity outcomes are shown in Table 2. The two studies that reported PM₁₀ levels below WHO AQG reported a 2.25 % (95 % CI: –3.24,7.73) increase in hospital admissions per 10 µg/m³ increase in PM₁₀. For the same outcome, the study that reported PM₁₀ levels above WHO AQG reported a 0.59 % (95 % CI: 0.39,0.79) increase. Pooled results showed a 1.68 % (95 % CI: –1.64,5.01) increase in CVD hospital admissions per 10 µg/m³ increase in PM₁₀ exposure.

Two studies explored the effect of long-term PM₁₀ exposure on CVD morbidity, with PM₁₀ levels above WHO AQG for both. Both studies reported the hazard ratio (HR) of disease incidence per 10 µg/m³ increase in PM₁₀. The diseases explored were AF, AMI, HF and IHD. HR ranged from 0.86 (95 % CI: 0.62,1.2) to 1.06 (95 % CI: 0.89,1.26) and 0.91 (95 % CI: 0.6,1.38) to 1.12 (95 % CI: 0.88,1.43) for IHD and HF respectively. The overall HR for the incidence of CVD was 1.20 (95 % CI: 0.71,1.68) per 10 µg/m³ increase in long-term PM₁₀ exposure.

A meta-analysis was conducted to ascertain the overall effect of PM on CVD morbidity (Fig. 2). There was a high level of heterogeneity amongst the pooled studies with an I² score of 88 %, attributable to variation in study designs and difference in outcomes reported. Six out of 20 data points reported the YLL per 10 µg/m³ increase in PM exposure. The pooled results found 1.92 (95 % CI: 0.58,3.26) YLL per 10 µg/m³ increase in PM exposure. The funnel plot did not demonstrate strong evidence of publication bias.

4.2. Mortality

Fourteen studies explored the effect of short-term PM_{2.5} exposure on CVD mortality, with 3 reporting PM_{2.5} levels below WHO AQG. Outcomes included percentage increase in mortality (10) per 10 µg/m³ increase in PM_{2.5}, and odds ratios (1) or risk ratios (3) for mortality. The diseases explored were arrhythmias including AF, AMI, HF, IHD including chronic IHD, sudden cardiac death, PVD and all-cause CVD. For all-cause CVD mortality, the percentage increase in mortality ranged between 0.25 % (95 % CI: –0.02,0.53) and 2.96 % (95 % CI:

Table 1
Summary of included studies, detailing type of exposure, outcome, length of exposure and level of exposure to air pollution.

Exposure explored	Outcome explored	Length of exposure	Air pollution levels (in comparison to WHO AQG)	Number of studies
PM2.5	Morbidity	Short term	Above	5
			Below	1
	Mortality	Long term	Above	8
			Below	0
		Short term	Above	11
			Below	3
PM10	Morbidity	Long term	Above	39
			Below	6
		Short term	Above	2
			Below	2
	Mortality	Long term	Above	2
			Below	0
		Short term	Above	6
			Below	2
Bushfire smoke	Morbidity	Long term	Above	15
			Below	1
	Mortality	Short term	Above	5
			Below	0
		Long term	Above	3
			Below	0

Table 2
Overall association between particulate matter exposure and measures of morbidity and mortality, by disease.

Outcome (↑/↓ Risk of mortality)				
	PM2.5		PM10	
Disease	Short term	Long term	Short term	Long term
Arrhythmias (Including AF)	Increased	Increased	Increased	Increased
AMI	Increased	Increased	Decreased	Increased
Unspecified cardiac disease		Increased		Increased
HF	Increased	Increased	Increased	Increased
IHD (Including chronic IHD)	Increased	Increased	Increased	Increased
Sudden cardiac death	Increased			
PVD	Increased			
All-cause CVD	Increased	Increased	Increased	Increased

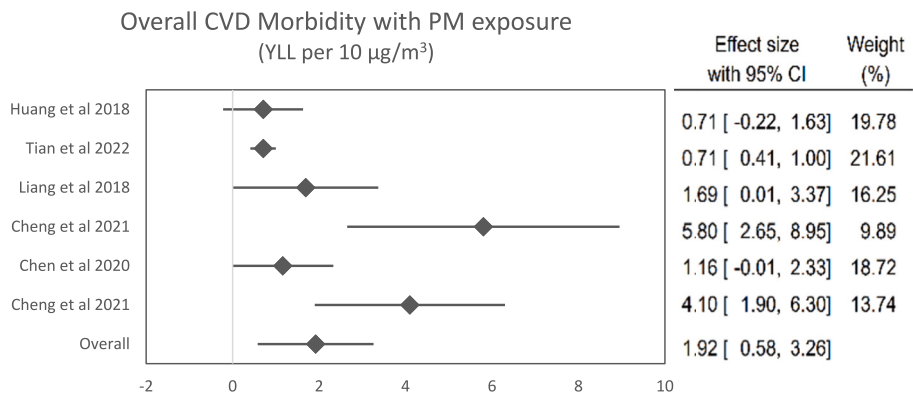


Fig. 2. Meta-analysis of CVD morbidity associated with overall PM_{2.5} exposure, measured as the YLL per 10 µg/m³ increase in PM_{2.5}.

–5.75,12.48) per 10 µg/m³ increase in PM_{2.5} (Table 2). The 3 studies that reported PM_{2.5} levels below WHO AQG found a 1.21 % (95 % CI: 0.44,1.98) increase in mortality per 10 µg/m³ increase in PM_{2.5}. The 11 studies with PM_{2.5} levels above WHO AQG reported a 1.23 % (95 % CI: 0.53,1.93) increase for the same outcome. The pooled results reported a 1.19 % (95 % CI: 0.64,1.74) increase in mortality per 10 µg/m³ increase in PM_{2.5}.

Forty-four studies explored the effect of long-term PM_{2.5} exposure on CVD mortality, with 6 reporting PM_{2.5} levels below WHO AQG. Outcomes included hazard (23), risk (4) or odds (1) ratios and percentage increase in mortality (15) per incremental increase in PM_{2.5}. Diseases explored included arrhythmias, AMI, IHD, HF, unspecified cardiac disease, and all-cause CVD. For all-cause CVD, there was a positive association with mortality (Table 2), with the percentage increase in mortality per 10 µg/m³ increase in PM_{2.5} ranging between –0.11 % (95 % CI: –0.92,0.71) and 2.58 % (95 % CI: –4.45,10.12). The 6 studies with PM_{2.5} levels below WHO AQG found a HR of 1.13 (95 % CI: 1.11,1.15) per 10 µg/m³. The 38 studies with PM_{2.5} levels above WHO AQG also found increased CVD mortality, with the HR ranging between 0.86 (95 % CI: 0.7,0.96) and 1.82 (95 % CI: 1.16,2.83). The pooled

results showed a 0.53 % (95 % CI: 0.35,0.71) increase in mortality per 10 µg/m³ increase in PM_{2.5} exposure.

Overall CVD mortality associated with PM_{2.5} exposure was measured by pooling the results from all studies assessing either short-term or long-term PM_{2.5}. Twenty-two out of 59 studies reported the percentage increase in mortality and reported an overall 0.71 % (95 % CI: 0.49,0.93) increase in CVD mortality per 10 µg/m³ increase in PM_{2.5}.

Eight studies explored the effect of short-term PM₁₀ exposure on CVD mortality, with 2 reporting PM₁₀ levels below the WHO AQG. Mortality was reported as percentage increase in mortality (7) and risk ratio (1) per 10 µg/m³ increase in PM₁₀. The diseases explored were arrhythmias including AF, AMI, HF, IHD including chronic IHD and all-cause CVD. Overall, there was an increase (Table 2) in all cause CVD mortality of 0.10 % (95 % CI: 0.02,0.18) per 10 µg/m³ increase in short-term PM₁₀ exposure. The 2 studies with PM₁₀ levels below WHO AQG found a 0.12 % (95 % CI: –1.34,1.57) increase in CVD mortality per 10 µg/m³ increase in PM₁₀. The 6 studies with PM₁₀ levels above the guidelines reported a 0.30 % (95 % CI: 0.10,0.50) increase for the same outcome. The pooled results revealed a 0.25 % (95 % CI: –0.07,0.58) increase in mortality per 10 µg/m³ increase in PM₁₀.

Sixteen studies explored the effect of long-term PM₁₀ exposure on CVD mortality, with 1 reporting PM₁₀ levels below WHO AQG. Outcomes were reported as percentage increase in mortality (7), hazard (7), risk (1) and odds (1) ratio per incremental increase in PM₁₀. The diseases explored were arrhythmias including AF, AMI, HF, IHD, unspecified cardiac disease and all-cause CVD. There was a positive association (Table 2) with all-cause CVD mortality and long-term PM₁₀ exposure, with the percentage increase in mortality per 10 µg/m³ increase in PM₁₀ ranging between 0.09 % (95 % CI: -0.06,0.24) and 0.18 % (95 % CI: 0.08,0.27). The overall percentage increase in mortality per 10 µg/m³ increase in long-term PM₁₀ exposure was 0.25 % (95 % CI: 0.14,0.37).

Overall CVD mortality associated with PM₁₀ exposure was measured by pooling the results from all studies assessing either short-term or long-term PM₁₀. Twelve out of 24 studies reported the percentage increase in mortality per 10 µg/m³ increase in PM₁₀, finding an overall 0.23 % (95 % CI: 0.10,0.35) increase in mortality per 10 µg/m³ increase.

A meta-analysis was conducted to ascertain the overall effect of PM

on CVD mortality (see Fig. 3). Again, there was high heterogeneity, with an I² value of 94.25 % depicting large variation in study design and subsequent variation in reported outcomes. Thirty-four out of 83 data points reported the percentage increase in mortality per 10 µg/m³ increase in PM exposure. The pooled results reported a 0.52 % (95 % CI: 0.37,0.68) increase in mortality per 10 µg/m³ increase in PM exposure. The funnel plot depicts asymmetrical distribution of effect size, suggesting a degree of publication bias favouring studies reporting positive findings.

5. Effect of bushfire smoke on CVD

5.1. Morbidity

Five studies assessed the effect of bushfire smoke on CVD morbidity. Outcomes explored included changes in GP, hospital, and ED admissions (5), disease incidence (1), and the risk of being prescribed medication

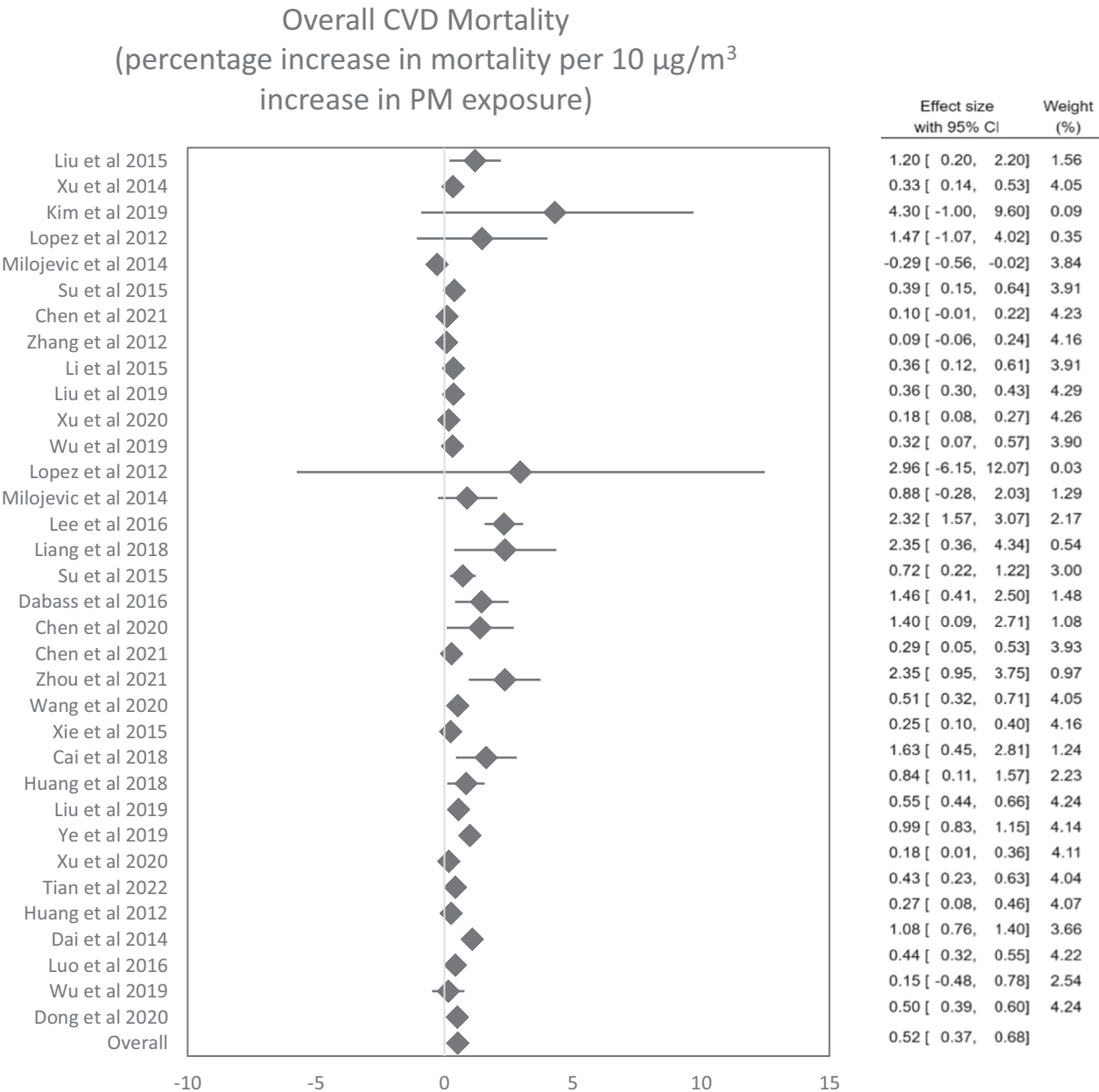


Fig. 3. Meta-analysis of overall CVD mortality associated with particulate matter, measured as the percentage increase in mortality per 10 µg/m³ increase in particulate matter.

for angina (1). Diseases explored included arrhythmias, AMI, HF, IHD, out-of-hospital-cardiac-arrests and all-cause CVD. For all cause CVD, bushfire smoke exposure was associated with increased GP and ED presentations and needing anti-anginal medications. However, there was a negative association between smoke exposure and CVD hospital admissions (Table 3).

5.2. Mortality

Three studies explored the effect of bushfire smoke on CVD mortality. Outcomes explored included odds ratio (1) and risk ratio (1) of mortality on smoke days, and percentage increase in mortality per 10 $\mu\text{g}/\text{m}^3$ increase in bushfire smoke (1). The diseases explored were IHD and all-cause CVD. An increase in mortality was seen with bushfire smoke exposure and IHD and all-cause CVD (Table 3), however the overall odds ratio of CVD mortality on smoke days was 1.00 (95 % CI:

Table 3

Overall association between bushfire smoke exposure and morbidity and mortality, by disease.

Outcome (↑↓ Risk of mortality)									
	PM2.5		PM10						
Disease	Short term	Long term	Short term	Long term					
Arrhythmias (Including AF)	Increased	Increased	Increased	Increased					
AMI	Increased	Increased	Decreased	Increased					
Unspecified cardiac disease		Increased		Increased					
HF	Increased	Increased	Increased	Increased					
IHD (Including chronic IHD)	Increased	Increased	Increased	Increased					
Sudden cardiac death	Increased								
PVD	Increased								
All-cause CVD	Increased	Increased	Increased	Increased					
Outcome (↑↓ Risk of morbidity)									
	PM2.5						PM10		
	Short term			Long term			Short term		Long term
Disease	ED presentations	Hospital admissions	YLL	Incidence of disease	Hospital admissions	YLL	Hospital admissions	YLL	Incidence of disease
Arrhythmias (including AF)		Decreased		Increased			Decreased		Increased
AMI		Increased	Increased			Increased	Increased		Increased
AVCD		Increased					Increased		
Unspecified cardiac disease		Increased					Increased		
HF		Increased		Increased	Increased		Increased		Increased
IHD (including chronic IHD)		Decreased	Increased	Increased	Increased	Increased	Decreased		Increased
Sudden cardiac death			Increased						
All-cause CVD	Increased	Decreased	Increased		Increased	Increased	Increased	Increased	

0.95,1.04), demonstrating no overall effect.

5.3. Risk of bias

Of 80 studies assessed for bias, 2 (2.5 %) studies were deemed to have a high ROB, 42 (52.5 %) had a medium ROB, and the remaining 36 (45 %) studies had a low ROB.

6. Discussion

This systematic review identified 80 primary studies that explored the effect of air pollution on CVD morbidity and mortality. Meta-analysis was used to examine the effect of PM_{2.5}, PM₁₀ and bushfire smoke on CVD morbidity and mortality, and their association with level or duration of air pollution exposure. Studies explored an array of CVDs, ranging from ICD-10 I00-I99, and data was stratified according to varying metrics of exposure. Outcomes were classified based on type, duration and level of exposure compared to WHO AQG. An additional comparison was made between studies conducted pre and post COVID.

6.1. Types of exposure

Overall, our study found an increase in CVD morbidity and mortality associated with air pollution exposure. Our meta-analysis reported exposure to PM was associated with 1.92 (95 % CI: 0.58,3.26) YLL per 10 µg/m³ increase in exposure. For mortality, PM was associated with 0.52 % (95 % CI: 0.37,0.68) increase in CVD mortality per 10 µg/m³ increase in exposure. The link with bushfire smoke was less robust, with some outcomes of morbidity such as CVD hospital admissions decreasing with smoke exposure. However, overall CVD morbidity increased with bushfire smoke exposure. Similarly, all-cause CVD mortality increased with bushfire smoke exposure, but pooled results from Doubleday et al.'s study revealed no overall effect of smoke exposure on mortality [68]. However, the small sample size of studies exploring bushfire smoke and CVD mortality affects the validity of these findings. Previous studies have similarly shown an overall increase in CVD morbidity and mortality associated with bushfire smoke, with the 2019–2020 Australian bushfires being responsible for a 4.5 % increase in CVD mortality and 1124 excess CVD hospitalisations [93].

6.2. Duration of exposure

Our study observed an increase in CVD morbidity with both short-term and long-term PM exposure. YLL and hospital admissions increased with each 10 µg/m³ increase in short-term PM_{2.5} and PM₁₀, and similarly long-term exposure to PM_{2.5} and PM₁₀ was associated with an increase in YLL and incidence in disease respectively. CVD mortality also increased with both short-term and long-term PM exposure, resulting in 1.19 % (95 % CI: 0.64,1.74) and 0.53 % (95 % CI: 0.35,0.71) increase in mortality per 10 µg/m³ increase in PM_{2.5} respectively. Previous studies have drawn similar conclusions, with large studies in the US reporting increases in cardiovascular hospital admissions with short-term air pollution exposure and rises in CVD mortality associated with long-term exposure [94].

6.3. Level of exposure

Level of exposure was categorised based on the WHO AQG. A considerable proportion of studies reported average air pollution levels above the WHO AQG, which reflects the ongoing need for reform of air quality control policies. For CVD morbidity, Milojevic et al.'s study reported that PM_{2.5} levels below WHO AQG were associated with a decrease in hospital admissions [23]. Due to a lack of other suitable studies exploring this outcome, it is difficult to draw a valid conclusion from this finding. However, PM₁₀ levels below the guidelines were associated with an increase in hospital admissions. For studies reporting

PM_{2.5} and PM₁₀ levels above WHO AQG, an increase in CVD morbidity was seen across all outcomes. Similarly, overall CVD mortality increased with all levels of PM_{2.5} and PM₁₀ exposure.

6.4. The effect of COVID 19

The COVID 19 pandemic brought about several strict periods of worldwide lockdown in an attempt to curb the widespread dissemination of disease. As a consequence decreased traffic, factory operations and various other reductions in emissions resulted in short term deduction of air pollution levels in highly polluted regions, with PM_{2.5} concentrations reducing by 200 % in Delhi, and by 31 % in Wuhan [11]. In terms of CVD mortality, the previous umbrella review by De Bont et al. revealed short term PM_{2.5} exposure was associated with 0.64 % (0.39; 0.97) to 1.00 % (1.00; 2.00) increase in all-cause CVD mortality per 10 µg/m³ increase in PM_{2.5} [10]. The pooled results for the same outcome in this publication revealed a 1.19 % (95 % CI: 0.64,1.74) increase in mortality per 10 µg/m³ increase in PM_{2.5}. This reveals that a slightly elevated risk was found when including studies during the COVID 19 pandemic, suggesting that other confounding factors could be at play, such as the effect of suffering from COVID. In terms of morbidity, De Bont et al.'s study revealed an increase in hospital admissions for all-cause CVD associated with incremental increase in PM_{2.5} exposure [10]. However, this publication including studies published during the pandemic revealed an overall decrease in hospital admissions per incremental increase in PM_{2.5} exposure, with the percentage change in hospital admissions ranging from −1.06 % (95 % CI: −1.69,−0.5) to 0.65 % (95 % CI: 0.46,0.84). Decreased levels of short-term air pollution levels may have played a role in this, however further research is required to ascertain this relationship.

6.5. Implications of findings

Ultimately, our findings further highlight the detrimental effects of air pollution on cardiovascular health. With the effects being present even with short-term and low levels of air pollution exposure, strategies to reduce air pollution globally are imperative.

6.6. Strengths and limitations

Strengths of this study include its wide temporal scope, including studies from 2012 to 2022. A broad search strategy was employed to minimise selection bias and ensure that all relevant studies would be included in our publication. We also explored a range of CVDs, to ascertain the overall effect of air pollution on CVD. Additionally, we categorised air pollution in terms of type, duration, and level of exposure which increases the real-life applicability of our findings. Limitations of this study include that conclusions were drawn based on meta-analysis that could only be obtained from studies reporting comparable outcomes, which is representative of a proportion of the studies. We also only included primary studies with quantitative data in our study, and thus future research may be required to synthesise any grey literature and qualitative data available.

7. Conclusion

Air pollution is associated with increased cardiovascular morbidity and mortality. This relationship is consistent across different types, durations, and levels of air pollution exposure. Increased global effort is imperative to mitigate this health threat by reducing air pollution exposure, as well as raise awareness of the deleterious effects of air pollution on cardiovascular health.

CRedit authorship contribution statement

Fariha Islam: Writing – review & editing, Writing – original draft,

Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Sai Keerthana Nukala:** Writing – review & editing, Investigation, Data curation. **Pallavi Shrestha:** Writing – review & editing, Data curation. **Tim Badgery-Parker:** Writing – review & editing, Validation, Supervision, Software. **Fiona Foo:** Writing – review & editing, Validation, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ahjo.2025.100546>.

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