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Review article

Impact of air pollution and noise exposure on cardiovascular disease incidence and mortality: A systematic review

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ABSTRACT

Background: The relationship between environmental pollutants, specifically air pollution and noise, and cardiovascular disease is well-recognized. However, their combined effects on cardiovascular health are not fully explored.
Objectives: To review evidence on the correlation between air pollution and noise exposure and cardiovascular disease incidence and mortality.
Methods: Following the PRISMA 2020 guidelines, we identified relevant studies through multiple databases and snowballing. We focused on studies published between 2003 and 2024. Studies were selected based on a PEOS framework, with a focus on exposure to air pollution or noise and clinical cardiovascular outcomes and evaluated for bias using the ROBINS-E tool.
Results: A total of 140 studies met our inclusion criteria. Most studies suggested a consistent association between long-term exposure to air pollutants and an increased risk of cardiovascular diseases, notably ischemic heart disease and stroke. While air pollution was often studied in isolation, the interaction effects between air pollution and noise exposure were less commonly investigated, showing mixed results. The majority of these studies were conducted in Western countries, which may limit the generalizability of the findings to global populations. No studies were found to use time-updated confounders, despite the long durations over which participants

Abbreviations: AF, Atrial Fibrillation; AMI, Acute Myocardial Infarction; BC, Black Carbon; BMI, Body Mass Index; CINAHL, Cumulative Index to Nursing and Allied Health Literature; CO, Carbon Monoxide; CV, Cardiovascular; CVD, Cardiovascular Disease; EC, Elemental Carbon; ELAPSE, Effects of Low-Level Air Pollution: A Study in Europe; HF, Heart Failure; IHD, Ischemic Heart Disease; IQR, Interquartile Range; LAeq, Equivalent noise level over a 24-h period with a penalty of 10 dB(A) for noise during the hours of 23:00–07:00; Lden, Day-evening-night noise level, the A-weighted, Leq over a whole day, with a penalty of 10 dB(A) for night-time noise and 5 dB(A) for evening noise; LMIC, Low- and Middle-Income Countries; MSM, Marginal Structural Model; NGO, Non-Governmental Organization; NO2, Nitrogen Dioxide; O3, Ozone; PEOS, Population, Exposure, Outcome, Study Design; PM10, Particulate Matter less than 10 μm; PM2.5, Particulate Matter less than 2.5 μm; PRISMA, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; PROSPERO, Prospective Register of Systematic Reviews; ROBINS-E, Risk of Bias in Non-randomized Studies – of Exposures; SO2, Sulfur Dioxide; US EPA, United States Environmental Protection Agency; WHO, World Health Organization.

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were followed, which could influence the accuracy of the results. Moreover, none of the studies incorporated both residential and occupational addresses in exposure assessments, suggesting a need for future studies to include these multiple exposure points to improve measurement precision and accuracy.
Conclusion: Air pollution exposure is increasingly linked to cardiovascular disease risks. Although individual air pollution and noise exposures are recognized as significant risk factors, the combined interaction between these exposures needs further exploration.
Registration: PROSPERO (CRD42023460443).

1. Introduction

The link between air pollution and cardiovascular disease (CVD) is increasingly acknowledged [1]. Air pollution, primarily arising from combustion processes, industrial emissions, and traffic, infiltrate the human respiratory system, with finer particles, depending on their size and other characteristics, may penetrate deeper into the respiratory system, including the alveolar regions, although the exact deposition varies based on particle size, airflow, and other factors [2,3]. The inflammatory response triggered by these particles can lead to systemic effects [4], including alterations in blood pressure, endothelial function, and development of atherosclerotic lesions. This can lead to manifest CVD [5].

Noise pollution, particularly from transportation sources, is an established non-traditional cardiovascular risk factor [6]. Chronic exposure to high levels of environmental noise activates the stress axis, leading to the release of stress hormones such as cortisol, adrenaline, and noradrenaline, which are known to promote hypertension, atherosclerosis, and other cardiovascular pathologies. While air pollution has been widely studied, the body of research on noise exposure is comparatively smaller but growing. Transportation noise, particularly from road traffic, can independently and synergistically affect cardiovascular health. The effects of noise, including sleep disruption and increased sympathetic activation, warrant detailed consideration alongside air pollution, given that both stressors can have overlapping yet distinct pathways affecting cardiovascular health [7,8]. Moreover, noise-induced stress aggravates sleep disturbances, further exacerbating cardiovascular (CV) risk [9].

Leading journals, NGOs, and governmental bodies have emphasized the urgency of further research into the association between air pollution and CVD [1]. In 2018, The Lancet characterized air pollution as a *public health emergency* [10], mirroring the stance of the American Heart Association [11] and Nature Reviews Cardiology [12], which identify air pollution as a CV risk. The World Health Organization (WHO) attributes approximately 7 million deaths annually to air pollution [13], although some estimates put the number of deaths well above 9 million [14], which emphasize its role as a significant risk factor. Similarly, agencies such as the U.S. EPA [15], European Commission [16], American Lung Association [17], and the non-profit Clean Air Task Force have platforms underscoring the CV repercussions of air pollution and advocating for more research [18–20]. Preliminary studies hint at potential CV risks linked to noise [7], with some suggesting a correlation with heart diseases and strokes in high noise environments [8].

The effects of air pollution continue to gain recognition, but it is imperative to also acknowledge the potential role of noise in the development of CVD. While the individual contributions of air pollution to CVD is increasingly recognized, air pollution and noise and their synergistic effects remain underexplored [7]. Drawing public attention to this aspect is important and prompts institutions to further research into the interplay between noise and air pollution and CVD.

The aim of this review was to methodically examine the literature concerning correlation between exposure to air pollution or noise and clinical CV outcomes. A particular emphasis was placed on identifying whether the studies considered air pollution and noise as potential effect modifiers or confounders. Included studies were quantitatively evaluated to appraise the robustness of the evidence. In line with our objectives, this paper intended to determine the reliability of the body of evidence and evaluating the confidence level in asserting an association between the exposures and the outcomes.

2. Methods

For this systematic review, a search strategy and reporting in accordance with the PRISMA 2020 guidelines was employed [21]. The protocol was registered at the International Prospective Register of Systematic Reviews (PROSPERO, register ID: CRD42023460443). Detailed information on the research question, PEOS framework, and inclusion and exclusion criteria is provided in the Supplemental Material (A.1., A.2., and A.3. Table S1).

2.1. Eligibility criteria and search strategy

A PEOS (Population, Exposure, Outcome, and Study) framework was established to structure our research analysis. Each domain of the PEOS framework was accompanied by specific inclusion and exclusion criteria tailored to the outcomes of interest. The emphasis of this review was on clinical cardiovascular effects (rather than pre- or subclinical) discerned within the broader population. We included cohort, case-control, cross-sectional, and intervention studies that used individual-level data.

2.2. Population

This review targeted studies examining the general human population aged 18 and older, exposed to air pollution or noise. It included individuals from both high-income and low-income areas, covering both urban, suburban, and rural settings. Studies qualified if they documented exposure to air pollution or noise among these broad groups. The selection did not limit based on the geographical locations or jobs of the participants. We wanted to capture a broad range of environmental exposures and their effects on cardiovascular health outcomes.

2.3. Exposure

Studies that documented short-term (days to months) or long-term (months to years) exposure to air pollution or noise were included. Studies eligible for inclusion reported exposure in clear quantitative terms, enabling us to assess the intensity and duration of pollution exposure and its correlation with cardiovascular outcomes. We excluded studies that solely assessed exposure to pollution in occupational settings or indoor environments, aiming to concentrate on the broader environmental factors affecting the general population.

2.4. Comparator

In this systematic review, the traditional comparator component of the PEOS framework was intentionally omitted. This decision was informed by the nature of epidemiological research, where the association between continuous exposure and the risk of adverse health outcomes is evaluated without a classic comparator. Such studies inherently compare the risk of outcomes between subjects exposed to relatively high versus low concentrations of pollutants. Including a specific comparator would not align with the objective of understanding the dose-response associated with varying levels of air pollution or noise exposure.

2.5. Outcome

The review focused on the incidence and prevalence of clinical cardiovascular diseases and cardiovascular disease-specific mortality. We aimed to evaluate the impact of exposure to air pollution or noise on the development and outcomes of CVD, specifically targeting measurable, clinically diagnosed diseases. This focus ensured our review directly addressed the significant health impacts of pollution on observable and severe cardiovascular events. We deliberately excluded studies that concentrated on pre-clinical or subclinical manifestations of cardiovascular disease, such as early markers of vascular injury or functional changes without diagnosed cardiovascular conditions.

2.6. Study

Cohort, case-control, cross-sectional, and intervention studies that used individual-level data was included. This inclusion covered both prospective and retrospective approaches. Only studies published in English were considered.

Studies where no original data were analyzed, methodological studies, genome-wide association studies (GWAS), and other -omics research, as well as non-human studies was excluded. Grey literature, including conference abstracts and unpublished data, to maintain the highest level of evidence quality was also omitted. Although reviews and systematic reviews were not directly included in our analysis, they were assessed to identify potential primary studies. Our focus on individual-level data from well-defined epidemiological studies was chosen to provide clear, applicable insights into the relationship between pollution exposure and CVD.

2.7. Study selection and data collection

We screened PubMed, Embase, Web of Science, Global Health Database, and CINAHL. Initial study screening was performed by SPM and KER, who independently reviewed titles and abstracts for relevance to our research question and PEOS framework. This preliminary screening was facilitated by the use of Covidence (2023 Covidence.org), an online tool designed for systematic reviews, which supported data accuracy, comprehensive review, risk of bias assessment, and streamlined data extraction.

Following the abstract screening, potentially relevant studies were subjected to a full-text assessment to confirm their eligibility based on our predefined criteria. Throughout this stage, any disagreements between SPM and KER were resolved through discussion and consensus.

To systematically extract data from the included studies, we developed and piloted a data extraction template with ten articles. This template was specifically designed to capture essential information from each study: study ID, title, country of conduct, study aim, design, participant inclusion criteria, sample size, study duration, setting, age, gender distribution, baseline health status, and relevant demographics. Additionally, detailed data on the type of pollution, pollution measurement methods and timings, pollutants investigated, exposure duration, exposure settings, clinical cardiovascular disease outcomes, methods used for disease incidence or prevalence determination, statistical analyses, main results relating to cardiovascular diseases, factors controlled for in the analysis, and study limitations were extracted for each study. The full data extraction table is available in the Supplemental Materials (A.4.).

2.8. Risk of bias (RoB) assessment

In evaluating the risk of bias within the included studies, a modified version of the Risk of Bias in Non-randomized Studies – of Exposures (ROBINS-E) tool to assess the methodological quality and potential biases in the included studies was created. It was applied through the Covidence platform for a structured and systematic approach. The risk of bias assessment was conducted by SPM and KER, with any disagreements resolved through discussion and consensus.

The assessment of *bias due to confounding* identified key confounders. Each study's approach to adjusting for these confounders was carefully reviewed, categorizing the risk levels from low to critical or noting the absence of information, with justifications provided for each decision.

The examination of *bias in the selection of participants into the study* reviewed participant selection methods and identified potential sources of selection bias. The risk of bias was assessed across the same categories, with detailed justifications for the assigned risk levels.

For *bias in classification of exposures*, the method of pollutant measurement and the potential for misclassification were evaluated. This included whether measurements were personal or ambient and their frequency, assigning risk levels with corresponding justifications.

The assessment of *bias due to missing data* looked at the volume and reasons behind missing data and how studies addressed this issue, assigning a risk level and providing justifications for the assessment.

Bias in measurement of outcomes focused on how cardiovascular outcomes were measured and the specificity of those outcomes, considering the potential for outcome misclassification and assigning risk levels with justifications.

Bias in selection of the reported result involved assessing the potential for selective reporting by the studies, particularly the omission of negative or non-significant results, with risk levels assigned and supported by detailed justifications.

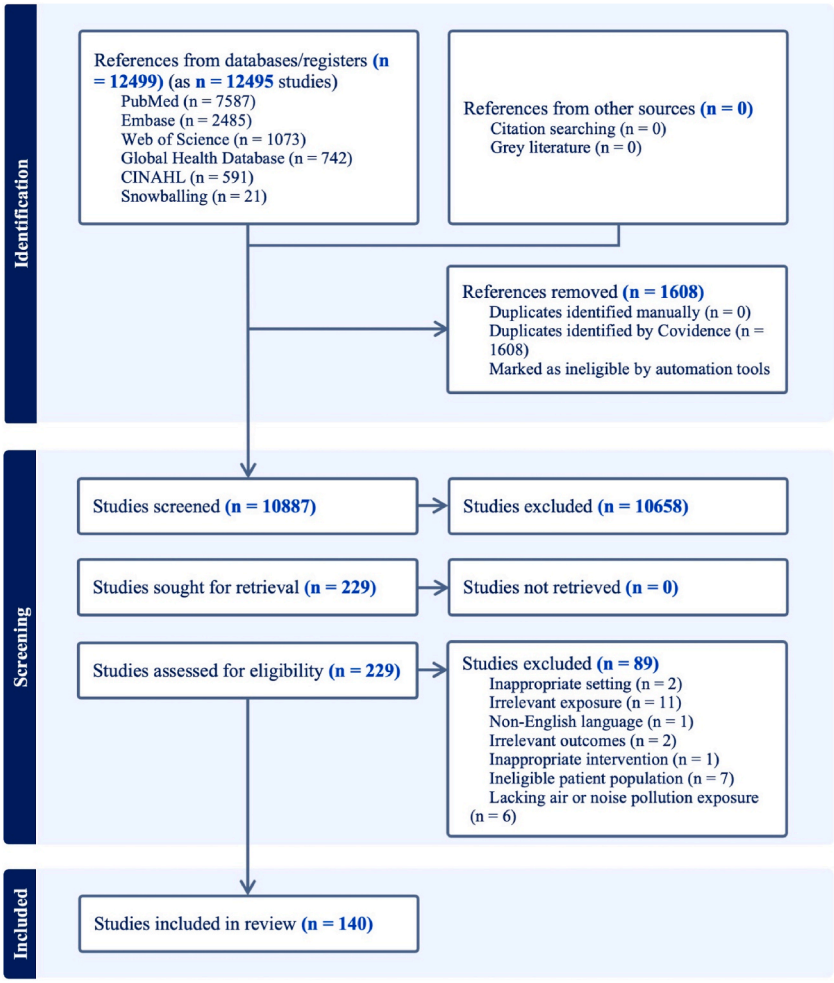

Fig. 1. PRISMA 2020 flowchart of assessment of eligible studies.

Fig. 1 shows the PRISMA 2020 Flowchart, illustrating the study selection process. It provides the number of studies identified, screened, eligible, and included, showing the exclusion of studies at each step. Specific reasons for exclusions are noted as well.

For the *overall risk of bias*, the highest risk of bias identified across the domains was initially considered the overall risk level for each study. However, adjustments were made if compounded issues across several domains suggested a higher overall risk. This process included predicting the overall direction of bias based on the domain assessments, though acknowledging the complexity of such predictions. The full RoB quality assessment table is available in the Supplemental Materials (A.5.).

3. Results

3.1. Article selection and description

Initially, we identified 12,499 articles across six major databases and through snowballing (Fig. 1). The databases included PubMed (7,587 studies), Embase (2,485 studies), Web of Science (1,073 studies), Global Health Database (742 studies), and CINAHL (591 studies). Furthermore, an additional 21 studies were identified through a snowballing approach, ensuring that key references cited in preliminary articles were captured. All searches were independently conducted by SPM and KER to ensure accuracy and comprehensiveness.

Key terms used in our search strings involved “air pollution”, “noise pollution”, “particulate matter”, “exposure”, “cardiovascular disease”, “ischemic heart disease”, “coronary artery disease”, “atrial fibrillation”, “heart failure”, “myocardial infarction”, “stroke”, and “coronary disease”. Searches were restricted to studies published between 2003 and 2024, adult populations, and articles exclusively published in English. We further refined our search by excluding reviews, meta-analyses, and scoping reviews. However, we did snowball reference lists from the excluded reviews. We collaborated with a librarian from the University of Southern Denmark (SDU) and consulted with two Senior Teaching Fellows in Public Health from the Department of Primary Care & Public Health at Imperial College London to develop our search strategy. Full search terms are included in the Supplemental Materials (A.6.).

Following the initial retrieval, 1,608 duplicates were removed, leaving 10,887 studies for title and abstract screening. Of these, 10,658 were excluded based on abstract content. This exclusion was due to the abstract content not meeting our specific inclusion criteria. Abstracts were assessed for relevance, and those providing information about content outside of our scope or not within the study period were removed from further consideration. This resulted in 229 studies assessed for full-text eligibility. After an in-depth evaluation, 89 studies were subsequently excluded for reasons mentioned in Fig. 1. Consequently, 140 studies met our predefined criteria and were included in the review.

3.2. Study characteristics

Table 1 provides characteristics for the included studies [6,22-100,101-160].

3.2.1. Study summary and description

We identified a predominant number of studies from Denmark, with 23 studies, followed by China with 19 studies, the United States also with 19 studies, Sweden with 16 studies, and the United Kingdom with 12 studies.

Prospective cohort studies were the most common study design with 70 studies. We also reviewed 31 retrospective cohort studies, and we identified 10 case-crossover studies, 10 case-control studies, and 8 cross-sectional studies.

A large variability in sample size was observed, ranging from a minimum of 32 to a maximum of 8,600,000 participants, with a mean sample size of 644,641. The median sample size was 50,154.

A wide range in study durations was noted, from 1 year to 44 years, with an average follow-up of 11.55 years and a median of 10.5 years.

The mean age of participants at study entry across the 100 studies, that provided participants’ age, was 57 years, with a median age of 56 years. The ages ranged from a minimum of 24 years to a maximum of 82 years. The interquartile range of 12 years indicates variability in the participant demographics.

Ninety-one studies solely investigated air pollution, demonstrating a clear focus on this type of exposure. Conversely, 36 studies examined both air pollution and noise.

The majority of the studies investigated long-term effects, with 121 studies, which are crucial for understanding chronic outcomes associated with pollution exposure. Seventeen studies focused on short-term effects. Additionally, 2 studies examined both short and long-term effects.

One hundred thirty-two studies based their pollution exposure assessments on participants’ home addresses, reflecting the common practice of evaluating residential exposure as a significant factor in long-term health studies. Eight studies focused on regional exposure assessments. While regional data can provide insights into broader environmental impacts and public health trends, this method typically results in less precise measurements of individual exposure to pollutants. The regional approach might dilute specific correlations between pollution exposure and cardiovascular outcomes due to the inability to accurately capture the variability in individual exposure levels across a larger area. None of the studies combined home and work addresses, which indicate a potential area for further research to understand occupational or dual-location exposures.

Table 2 presents a summary of the included studies. Detailed descriptive and summary statistics of data extraction variables are available in the Supplemental Materials (A.7.).

3.2.2. Confounders

We identified the most common confounders controlled for in the studies. Age was the most frequently adjusted variable [114].

Table 1
Study characteristics for included studies.

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Alexeeff 2023	United States	Retrospective Cohort study	3,798,078	7	Both	41	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD
Andersen 2012	Denmark	Prospective Cohort study	57,053	13	Urban	56 (median)	Air Pollution	NO_2	Long-term	Home address	Stroke
Andersen 2021	Denmark	Retrospective Cohort study	23,528	44	Both	53	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	AF
Andersson 2020	Sweden	Prospective Cohort study	6,304	36	Urban	58	Air and Noise Pollution	NO_x, Noise	Long-term	Home address	CV Mortality, IHD, Stroke
Atkinson 2013	UK	Retrospective Cohort study	836,557	4	Both	Not stated	Air Pollution	NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Long-term	Home address	IHD
Bai 2019	Canada	Retrospective Cohort study	1,135,817	17	Urban	51	Air Pollution	NO_2, $PM_{2.5}$, UFP	Long-term	Regional	AMI, HF
Bai 2019	Canada	Retrospective Cohort study	5,100,000	14	Both	53	Air Pollution	NO_2, O_3, $PM_{2.5}$	Long-term	Home address	AMI, HF
Bai 2020	Canada	Retrospective Cohort study	4,900,000	11	Both	51	Noise Pollution	Noise	Long-term	Home address	AMI, HF
Banerjee 2014	India	Cross sectional study	909	1	Urban	Not stated	Noise Pollution	Noise	Long-term	Home address	IHD
Beckerman 2012	Canada	Cross sectional study	2,360	7	Urban	60	Air Pollution	EC, NO_2, O_3, $PM_{2.5}$	Long-term	Home address	IHD
Beelen 2009	Netherlands	Prospective Cohort study	120,852	9	Both	64 (median)	Air and Noise Pollution	EC, Noise	Long-term	Home address	All-cause Mortality, CV Mortality, IHD
Bodin 2016	Sweden	Prospective Cohort study	13,512	10	Both	Not stated	Air and Noise Pollution	NO_x, Noise	Long-term	Home address	AMI
Cai 2018	UK	Retrospective Cohort study	355,732	15	Both	53	Air and Noise Pollution	EC, NO_2, Noise, PM_{10}, $PM_{2.5}$	Long-term	Home address	IHD, Stroke
CaiJunFang 2018	China	Retrospective Cohort study	3,112,366	5	Urban	Not stated	Air Pollution	CO, NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Long-term	Home address	AMI, CV Mortality, HF, IHD, Stroke
Canterbury 2020	United States	Prospective Cohort study	2,000	Not stated	Urban	59	Air Pollution	BC, $PM_{2.5}$	Long-term	Home address	All-cause Mortality
Carey 2016	UK	Retrospective Cohort study	211,016	6	Urban	Not stated	Noise Pollution	NO_x, O_3, $PM_{2.5}$	Long-term	Home address	AMI, HF
Carlsen 2022	Sweden	Prospective Cohort study	6,103	20	Urban	58	Air Pollution	BC, NO_x, PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI, HF, IHD, Stroke
Cesaroni 2013	Italy	Prospective Cohort study	1,265,058	15	Urban	Not stated	Air Pollution	NO_2, $PM_{2.5}$	Long-term	Home address	All-cause Mortality, CV Mortality, IHD
Chen 2005	United States	Prospective Cohort study	3,239	22	Both	Not stated	Air Pollution	NO_2, O_3, PM, SO_2	Long-term	Regional	CV Mortality

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Chen 2013	Canada	Retrospective Cohort study	205,440	Not stated	Urban	52	Air Pollution	NO_2	Long-term	Home address	CV Mortality, IHD, Stroke
Chen 2022	Taiwan	Retrospective Cohort study	1,362,284	4	Both	44	Air Pollution	$PM_{2.5}$	Long-term	Home address	Stroke
Cole-Hunter 2021	Denmark	Prospective Cohort study	25,660	14	Both	53	Air and Noise Pollution	NO_2, NO_x, Noise, PM_{10}, $PM_{2.5}$	Long-term	Home address	Stroke
Cole-Hunter 2022	Denmark	Prospective Cohort study	24,994	17	Both	53	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	AMI, IHD, Stroke
Cramer 2020	Denmark	Prospective Cohort study	22,882	19	Both	53	Air and Noise Pollution	NO_2, NO_x, Noise, PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI
Crouse 2012	Canada	Retrospective Cohort study	2,124,986	11	Both	Not stated	Air Pollution	$PM_{2.5}$	Long-term	Regional	All-cause Mortality, CV Mortality
Dabass 2016	United States	Time-stratified case-crossover	62,135	12	Urban	Not stated	Air Pollution	O_3, $PM_{2.5}$	Short-term	Home address	CV Mortality
Dehbi 2017	UK	Prospective Cohort study	7,071	25	Both	49	Air Pollution	NO_2, NO_x, PM_{10}, $PM_{2.5}$, SO_2	Long-term	Home address	CV Mortality
deKluizenaar 2013	Netherlands	Prospective Cohort study	18,213	13	Urban	46	Air and Noise Pollution	EC, NO_2, Noise, PM_{10}	Long-term	Home address	IHD
Dirgawati 2019	Australia	Prospective Cohort study	11,627	16	Urban	72	Air Pollution	NO_2, NO_x, $PM_{2.5}$	Long-term	Home address	All-cause Mortality, Stroke
Dzhambov 2016	Bulgaria	Cross sectional study	513	NA	Urban	Not stated	Noise Pollution	Noise	Long-term	Regional	IHD
Feng 2019	China	Prospective Cohort study	45	1	Urban	24	Air Pollution	$PM_{2.5}$	Long-term	Home address	AF
Floud 2013	UK, Germany, Netherlands, Sweden, Greece, Italy	Cross sectional study	4,712	NA	Both	58	Air and Noise Pollution	NO_x, Noise	Long-term	Home address	IHD, Stroke
Gaines 2023	Israel	Retrospective Cohort study	74,052	5	Both	72	Air Pollution	$PM_{2.5}$	Long-term	Home address	Stroke
Gan 2012	Canada	Prospective Cohort study	445,868	5	Urban	60	Air and Noise Pollution	BC, NO_2, $PM_{2.5}$	Long-term	Home address	CV Mortality, IHD
Halonen 2015	UK	Ecological study	8,600,000	7	Urban	Not stated	Air and Noise Pollution	NO_x, Noise, O_3, $PM_{2.5}$	Long-term	Regional	All-cause Mortality, IHD, Stroke
HaoGuang 2022	UK	Prospective Cohort study	342,566	9	Both	56	Air and Noise Pollution	Noise, $PM_{2.5}$	Long-term	Home address	All-cause Mortality, CV Mortality, IHD, Stroke

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Hart 2015	United States	Prospective Cohort study	114,537	2	Both	64	Air Pollution	PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI, IHD, Stroke
Hayes 2020	United States	Prospective Cohort study	566,398	Not stated	Urban	63 (median)	Air Pollution	$PM_{2.5}$	Long-term	Home address	CV Mortality, IHD, Stroke
Heinonen-Guzejev 2007	Finland	Case control study	1,988	15	Both	56	Noise Pollution	Noise	Long-term	Regional	CV Mortality, IHD
Heritier 2017	Switzerland	Prospective Cohort study	4,410,000	8	Both	52	Noise Pollution	Noise	Long-term	Home address	AMI, CV Mortality, HF, IHD, Stroke
Heritier 2018	Switzerland	Prospective Cohort study	4,410,000	8	Both	52	Noise Pollution	Noise	Long-term	Home address	AMI, CV Mortality, HF, Stroke
Hoffmann 2006	Germany	Cross sectional study	3,399	5	Urban	60	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, IHD
Hoffmann 2009	Germany	Prospective Cohort study	4,814	Not stated	Urban	Not stated	Air and Noise Pollution	Noise, $PM_{2.5}$	Long-term	Home address	AMI, IHD
Hoffmann 2015	Germany	Prospective Cohort study	4,814	8	Urban	59	Air and Noise Pollution	CO, Noise, PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD, SCD, Stroke
Huang 2018	China	Time-series study	10,322	5	Both	Not stated	Air Pollution	NO_2, $PM_{2.5}$, SO_2	Short-term	Home address	IHD
Huss 2010	Switzerland	Retrospective Cohort study	4,600,000	5	Both	51 (median)	Air and Noise Pollution	Noise, PM_{10}	Long-term	Home address	AMI, CV Mortality
Hvidtfeldt 2019	Denmark	Prospective Cohort study	49,564	22	Urban	56	Air and Noise Pollution	BC, NO_2, O_3, PM_{10}, $PM_{2.5}$	Long-term	Home address	CV Mortality
Hystad 2020	Canada, Saudi Arabia, Sweden, UAE, Argentina, Brazil, Chile, Malaysia, Poland, South Africa, Turkey, Chine, Colombia, Iran, Palestine, the Phillipines, Bangladesh, India, Pakistan, Tanzania, Zimbabwe	Prospective Cohort study	157,436	3	Both	50	Air Pollution	$PM_{2.5}$	Long-term	Regional	AMI, CV Mortality, IHD, Stroke
Jalali 2021	Iran	Prospective Cohort study	3,081	15	Both	50	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, SCD, Stroke
Jin 2022	United States	Retrospective Cohort study	7,280,359	17	Both	73	Air Pollution	NO_2, O_3, $PM_{2.5}$	Long-term	Home address	HF, Stroke
Jones 2023	Australia	Retrospective Cohort study	11,249	21	Both	72	Air Pollution	$PM_{2.5}$	Long-term	Home address	HF, IHD
Katsoulis 2014	Greece	Prospective Cohort study	2,752	14	Urban	47	Air Pollution	NO_2, PM_{10}	Long-term	Home address	IHD, Stroke

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Kaufman 2012	United States	Prospective Cohort study	7,562	10	Both	Not stated	Air Pollution	BC, $PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, HF, IHD, Stroke
Kim 2017	South Korea	Retrospective Cohort study	136,094	7	Both	42	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, HF, Stroke
Kim 2020	South Korea	Retrospective Cohort study	436,933	Not stated	Both	48	Air Pollution	O_3, $PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, HF, Stroke
Kim 2020	South Korea	Retrospective Cohort study	196,167	9	Both	47	Air Pollution	PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI, HF, IHD, Stroke
Kim 2021	South Korea	Retrospective case-control study	10,775	2	Both	Not stated	Air Pollution	CO, NO_2, NO_x, O_3, PM_{10}, SO_2	Short- and Long-term	Home address	IHD
Korek 2015	Sweden	Prospective Cohort study	20,070	19	Urban	60	Air Pollution	NO_x, PM_{10}	Long-term	Home address	Stroke
Kuzma 2022	Poland	Retrospective Cohort study	4,838	9	Both	74	Air Pollution	CO, NO_2, NO_x, PM_{10}, $PM_{2.5}$, SO_2	Short-term	Home address	Stroke
Kwon 2019	South Korea	Retrospective Cohort study	124,010	11	Urban	49	Air Pollution	CO, NO_2, NO_x, O_3, PM_{10}, $PM_{2.5}$, SO_2	Short- and Long-term	Home address	AF
Lanki 2006	Germany, Spain, Finland, Italy, Sweden	Observational study	27,000	Not stated	Urban	Not stated	Air Pollution	CO, NO_2, O_3, PM_{10}	Long-term	Home address	AMI
Li 2020	China	Retrospective Cohort study	118,229	23	Both	51	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD
Li 2022	China	Prospective Cohort study	29,141	11	Urban	62	Air Pollution	NO_2, PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI, HF, IHD
Liang 2020	China	Prospective Cohort study	116,972	15	Both	51	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, IHD, Stroke
Liang 2022	China	Prospective Cohort study	90,672	7	Both	43	Air Pollution	BC, CO, O_3, $PM_{2.5}$	Long-term	Home address	CV Mortality, IHD, Stroke
Lim 2021	Denmark	Prospective Cohort study	22,189	21	Both	60	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	HF
Lisabeth 2008	United States	Ecological	3,508	4	Urban	72 (median)	Air Pollution	O_3, $PM_{2.5}$	Short-term	Home address	Stroke
Liu 2015	China	Case-crossover design	8,955	3	Urban	Not stated	Air Pollution	NO_2, PM_{10}, SO_2	Short-term	Home address	CV Mortality
Liu 2020	Canada	Time-stratified case-crossover design	6,142	8	Urban	65	Air Pollution	CO, NO_2, NO_x, O_3, PM_{10}, $PM_{2.5}$, SO_2	Short-term	Home address	AMI
Liu 2021	China	Retrospective Cohort study	14,331	6	Both	45	Air Pollution	BC, CO, EC, O_3, $PM_{2.5}$	Long-term	Home address	AMI, HF, IHD, Stroke

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Liu 2021	China	Time-stratified case-crossover study	151,608	6	Both	75	Air Pollution	CO, NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Short-term	Home address	AMI
Liu 2022	China	Prospective Cohort study	512,689	13	Both	52	Air Pollution	CO, O_3, $PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD, Stroke
LjungmanPLS 2019	Sweden	Prospective Cohort study	114,758	21	Urban	Not stated	Air Pollution	BC, PM_{10}, $PM_{2.5}$	Long-term	Home address	IHD, Stroke
Loop 2018	United States	Retrospective Cohort study	17,126	6	Both	63 (median)	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD
Lu 2019	Pakistan	Time-series study	31,749	1	Urban	Not stated	Air Pollution	BC, CO, EC, $PM_{2.5}$	Short-term	Home address	HF, IHD
Madrigano 2013	United States	Case control study	4,467	8	Urban	25	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI
Maheswaran 2005	UK	Ecological study	199,682	5	Both	Not stated	Air Pollution	CO, NO_x, PM_{10}	Long-term	Home address	CV Mortality, IHD
Maheswaran 2012	UK	Ecological study	2,610	17	Urban	Not stated	Air Pollution	NO_2, PM_{10}	Long-term	Home address	Stroke
Maheswaran 2016	UK	Time-stratified case-crossover	2,590	11	Urban	72	Air Pollution	NO_2, O_3, PM_{10}	Short-term	Home address	Stroke
Mai 2022	China	Cross sectional study	13,484	2	Both	61	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, HF, IHD, Stroke
Matsuo 2016	Japan	Case-crossover study	6,885	NA	Both	82	Air Pollution	$PM_{2.5}$	Short-term	Home address	Stroke
Miller 2007	United States	Prospective Cohort study	65,893	6	Both	63 (median)	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, CV Mortality, IHD, Stroke
Mo 2023	China	Space-time stratified case-crossover	12,927	2	Both	74	Air Pollution	EC, O_3, $PM_{2.5}$	Short-term	Home address	AMI
Monrad 2016	Denmark	Prospective Cohort study	57,053	15	Urban	56 (median)	Air and Noise Pollution	NO_2, NO_x, Noise	Long-term	Home address	AF
NautiyalJ3rd 2007	India	Cross sectional study	200	NA	Both	Not stated	Air Pollution	CO, NO_x, PM_{10}, $PM_{2.5}$	Long-term	Home address	IHD
Nishiwaki 2013	Japan	Prospective Cohort study	62,142	Not stated	Both	Not stated	Air Pollution	NO_2, PM, SO_2	Long-term	Home address	AMI, IHD, Stroke
Nuvolone 2011	Italy	Case-crossover	11,450	1	Urban	73	Air Pollution	CO, NO_2, NO_x, PM_{10}	Short-term	Home address	AMI
Occelli 2020	France	Ecological study	3,268	3	Urban	Not stated	Air Pollution	CO, EC, NO_2, PM_{10}	Long-term	Home address	IHD
Olaniyan 2022	Canada	Prospective Cohort study	2,700,000	10	Both	Not stated	Air Pollution	NO_2, O_3, $PM_{2.5}$	Long-term	Home address	AMI, Stroke
Ostro 2010	United States	Prospective Cohort study	45,000	5	Urban	54	Air Pollution	$PM_{2.5}$	Long-term	Home address	All-cause Mortality, CV Mortality, IHD

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Oudin 2011	Sweden	Case control study	7,244	5	Both	Not stated	Air Pollution	NO_x	Long-term	Home address	Stroke
PopeCA3rd 2006	United States	Case-crossover study	12,865	10	Urban	63	Air Pollution	EC, $PM_{2.5}$	Short-term	Home address	AMI, IHD
PopeCA3rd 2015	United States	Prospective Cohort study	669,046	22	Both	Not stated	Air Pollution	$PM_{2.5}$	Long-term	Home address	CV Mortality
Poulsen 2023	Denmark	Prospective Cohort study	1,964,702	12	Both	58 (median)	Air Pollution	EC, NO_2, $PM_{2.5}$, UFP	Long-term	Home address	AMI
Poulsen 2024	Denmark	Prospective Cohort study	1,964,702	13	Both	58	Air and Noise Pollution	CO, EC, NO_2, Noise, $PM_{2.5}$, UFP	Long-term	Home address	AMI
Puett 2009	United States	Prospective Cohort study	66,250	10	Urban	62	Air Pollution	$PM_{2.5}$	Long-term	Home address	AMI, IHD
Pyko 2019	Sweden	Prospective Cohort study	20,012	Not stated	Urban	60	Noise Pollution	Noise	Long-term	Home address	IHD, Stroke
Pyko 2023	Scandinavia (DK, NO, SE)	Pooled cohort study	132,801	NA	Urban	Not stated	Air and Noise Pollution	Noise	Long-term	Home address	IHD
Raaschou-Nielsen 2012	Denmark	Prospective Cohort study	52,061	14	Urban	56	Air Pollution	NO_2	Long-term	Home address	All-cause Mortality, CV Mortality
Recio 2016	Spain	Time-stratified case-crossover	342,566	7	Urban	Not stated	Air and Noise Pollution	CO, NO_2, Noise, $PM_{2.5}$	Short-term	Home address	CV Mortality
Rodins 2020	Germany	Prospective Cohort study	4,105	14	Urban	59	Air Pollution	CO, EC, NO_2, O_3, PM10, $PM_{2.5}$	Long-term	Home address	AMI, IHD
Roscoe 2023	United States	Prospective Cohort study	114,116	30	Both	68	Noise Pollution	Noise	Long-term	Home address	IHD, Stroke
Rosenlund 2006	Sweden	Case control study	1,397	2	Urban	61	Air Pollution	CO, NO_2, NO_x, PM_{10}, SO_2	Long-term	Home address	AMI
Rosenlund 2008	Italy	Prospective Cohort study	6,513	7	Urban	Not stated	Air Pollution	NO_2	Long-term	Home address	AMI, IHD
Rosenlund 2009	Sweden	Case control study	301,273	11	Urban	79	Air Pollution	CO, EC, NO_2, NO_x, PM_{10}	Long-term	Home address	AMI
Roswall 2017	Denmark	Prospective Cohort study	50,744	14	Both	57	Air and Noise Pollution	NO_2, Noise	Long-term	Home address	AMI
Roswall 2021	Denmark and Sweden	Prospective Cohort study	135,951	20	Both	54	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	CV Mortality, Stroke
Sarnat 2006	United States	Panel study	32	NA	Both	71	Air Pollution	CO, EC, NO_2, NO_x,	Short-term	Home address	AF/VT/VF

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
								O_3, $PM_{2.5}$, SO_2			
Seidler 2018	Germany	Case control study	1,026,670	5	Urban	76 (median)	Noise Pollution	EC, Noise	Long-term	Home address	Stroke
Selander 2009	Sweden	Case control study	3,666	24	Urban	Not stated	Air and Noise Pollution	NO_2, Noise	Long-term	Home address	AMI
Shi 2021	China	Prospective Cohort study	4,866	1	Both	65	Air Pollution	CO, NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Long-term	Home address	HF
So 2020	Denmark	Prospective Cohort study	24,541	17	Both	53	Air and Noise Pollution	NO_2, Noise, PM_{10}, $PM_{2.5}$	Long-term	Home address	CV Mortality
Sørensen 2011	Denmark	Prospective Cohort study	57,053	13	Urban	56 (median)	Air and Noise Pollution	NO_x, Noise	Long-term	Home address	Stroke
Sørensen 2012	Denmark	Prospective Cohort study	57,053	10	Urban	59	Air and Noise Pollution	NO_x, Noise	Long-term	Home address	AMI
Sørensen 2014	Denmark	Prospective Cohort study	57,053	10	Both	Not stated	Air and Noise Pollution	NO_2, NO_x, Noise	Long-term	Home address	Stroke
Sørensen 2017	Denmark	Prospective Cohort study	57,053	13	Urban	Not stated	Air and Noise Pollution	NO_2, Noise	Long-term	Home address	HF
Sorensen 2021	Denmark	Retrospective Cohort study	3,620,000	20	Both	53	Noise Pollution	Noise	Long-term	Home address	Stroke
Stockfelt 2015	Sweden	Prospective Cohort study	7,494	34	Urban	53	Air Pollution	NO_x	Long-term	Home address	AMI, CV Mortality, IHD
Stockfelt 2017	Sweden	Prospective Cohort study	10,350	21	Urban	Not stated	Air Pollution	NO_x, PM_{10}, $PM_{2.5}$	Long-term	Home address	HF, IHD, Stroke
Thacher 2022	Denmark	Retrospective Cohort study	3,600,000	17	Both	50	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	AF
Thacher 2022	Denmark	Retrospective Cohort study	2,538,395	9	Both	59	Air and Noise Pollution	NO_2, Noise, $PM_{2.5}$	Long-term	Home address	AMI, HF, IHD
Thurston 2016	United States	Prospective Cohort study	445,860	22	Urban	56.6	Air Pollution	CO, EC, $PM_{2.5}$	Long-term	Home address	CV Mortality, IHD
Tonne 2007	United States	Case control study	5,049	8	Urban	71	Air Pollution	NO_2, O_3, $PM_{2.5}$	Long-term	Home address	AMI
Tonne 2013	UK	Prospective Cohort study	154,204	4	Both	68	Air Pollution	NO_2, NO_x, PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI
Tseng 2015	Taiwan	Retrospective Cohort study	43,227	16	Urban	41	Air Pollution	CO, EC, NO_2, NO_x, O_3, $PM_{2.5}$, SO_2	Long-term	Home address	CV Mortality

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Table 1 (*continued*)

Study ID	Country in which the study conducted	Study design	Sample size (n)	Duration of study (years)	Setting (Urban, Rural, Both)	Age in years (mean, if not otherwise stated)	Type of Pollution	Type(s) of pollutant(s)	Duration of Exposure	Place of exposure	Type of CVD Studied
Ueda 2012	Japan	Prospective Cohort study	7,250	24	Both	Not stated	Air Pollution	PM	Long-term	Home address	CV Mortality, IHD, Stroke
Vienneau 2022	Switzerland	Prospective Cohort study	4,190,000	15	Both	Not stated	Noise Pollution	CO, EC, Noise, $PM_{2.5}$	Long-term	Home address	CV Mortality
Vienneau 2023	Switzerland	Prospective Cohort study	4,190,000	14	Both	70	Air and Noise Pollution	BC, CO, NO_2, Noise, O_3, $PM_{2.5}$	Long-term	Home address	All-cause Mortality, CV Mortality
Wen 2023	China	Prospective Cohort study	26,851	2	Both	50	Air Pollution	BC, CO, NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Long-term	Home address	AMI, HF, IHD, Stroke
Wichmann 2013	Denmark	Other: Case-crossover	4,657	11	Urban	Not stated	Air Pollution	CO, NO_2, NO_x, O_3, PM_{10}, $PM_{2.5}$	Short-term	Regional	Cardiac arrest
Wolf 2015	Finland, Sweden, Denmark, Germany, Italy	Prospective Cohort study	100,166	15	Both	55	Air Pollution	PM_{10}, $PM_{2.5}$	Long-term	Home address	AMI
Wolf 2021	Sweden, Denmark, the Netherlands, and Germany	Prospective Cohort study	137,148	17	Both	54	Air Pollution	BC, NO_2, O_3, $PM_{2.5}$	Long-term	Home address	AMI, IHD, Stroke
Yang 2018	Taiwan	Cross sectional study	663	1	Urban	45	Air and Noise Pollution	NO_2, Noise, PM_{10}	Long-term	Home address	AMI, HF, IHD, Stroke
Yang 2021	China	Prospective Cohort study	38,140	10	Urban	44	Air Pollution	$PM_{2.5}$	Long-term	Home address	Stroke
Yang 2023	UK	Prospective Cohort study	424,767	12	Both	62	Air and Noise Pollution	EC, NO2, Noise, $PM_{2.5}$	Long-term	Home address	AMI, HF
Yankoty 2021	Canada	Retrospective Cohort study	1,065,414	14	Urban	Not stated	Noise Pollution	NO_2, Noise	Long-term	Home address	AMI
Yankoty 2022	Canada	Retrospective Cohort study	1,065,414	8	Urban	Not stated	Air and Noise Pollution	NO_2, Noise	Long-term	Home address	Stroke
Yu 2018	China	Time-series study	5,545	2	Both	Not stated	Air Pollution	NO_2, O_3, PM_{10}, $PM_{2.5}$, SO_2	Short-term	Home address	AMI
Zhang 2014	China	Retrospective Cohort study	39,054	12	Urban	44	Air Pollution	PM_{10}	Long-term	Home address	CV Mortality, HF, IHD, Stroke
Zhang 2020	South Korea	Prospective Cohort study	182,488	4	Urban	37	Air Pollution	PM_{10}, $PM_{2.5}$	Long-term	Home address	AF

Table 2
Summary of included studies.

Countries	Denmark [23], China [19], USA [19], Sweden [16], UK [12], Canada [11], Germany [8], Italy [6], South Korea [6], Switzerland [5], Finland [3], India [3], Taiwan [3], Japan [3], Netherlands [3], Australia [2], Poland [2], Greece [2], Pakistan [2], Spain [2], Iran [2], Tanzania [1], Zimbabwe [1], Bangladesh [1]. The Philippines [1], Palestine [1], Colombia [1]. Myanmar [1] Turkey [1]. South Africa [1], Saudi Arabia [1]. Malaysia [1]. Chile [1], Brazil [1]. Argentina [1], UAE [1], Scandinavia (DK/NO/SE) [1], France [1], Bulgaria [1], and Israel [1]
Study design overview	Prospective [70], retrospective [31], case-cross over [10], case-control [10], cross sectional [8], and others [11]
Sample size statistics	Min: 32, max: 8,600,000, mean: 644,641, median: 50,154
Duration of studies	1–44 years, mean: 11.55 years, median: 10.5 years
Age of participants	24–82 years, mean: 57, median: 56, IQR: 12 years
Pollution focus	Air [91], air & noise [36], noise [13]
Exposure duration	Long-term [121], short-term [17], both [2]
Exposure location	Home [132], regional [8]

This was followed by sex/gender, controlled for in 102 studies, and smoking, which was accounted for in 98 studies. Education was considered in 67 studies, and alcohol consumption was included in 50 studies.

Additionally, physical activity was adjusted for in 40 studies, while BMI was considered in 39 studies. Both income and socio-economic status were included as confounders in 36 studies each. Occupation was controlled for in 22 studies, and race/ethnicity appeared in 19 studies. Diet was the least frequently controlled confounder, adjusted for in only 7 studies.

3.2.3. Limitations

We identified recurring limitations across the studies. A concern was exposure misclassification. The majority of studies relied on residential addresses and modeled estimates, which is the nature of such environmental studies. This, approach may not capture the true exposure levels, potentially skewing the understanding of the relationship. Although, it is crucial to note that these models are capable of capturing the difference in concentration levels between urban and rural areas, known as the urban increment. This distinction can be more critical than capturing absolute exposure values, as the differences that follow population densities drive the observed health impacts.

Another significant limitation we noted was residual confounding. The studies frequently lacked comprehensive data on important

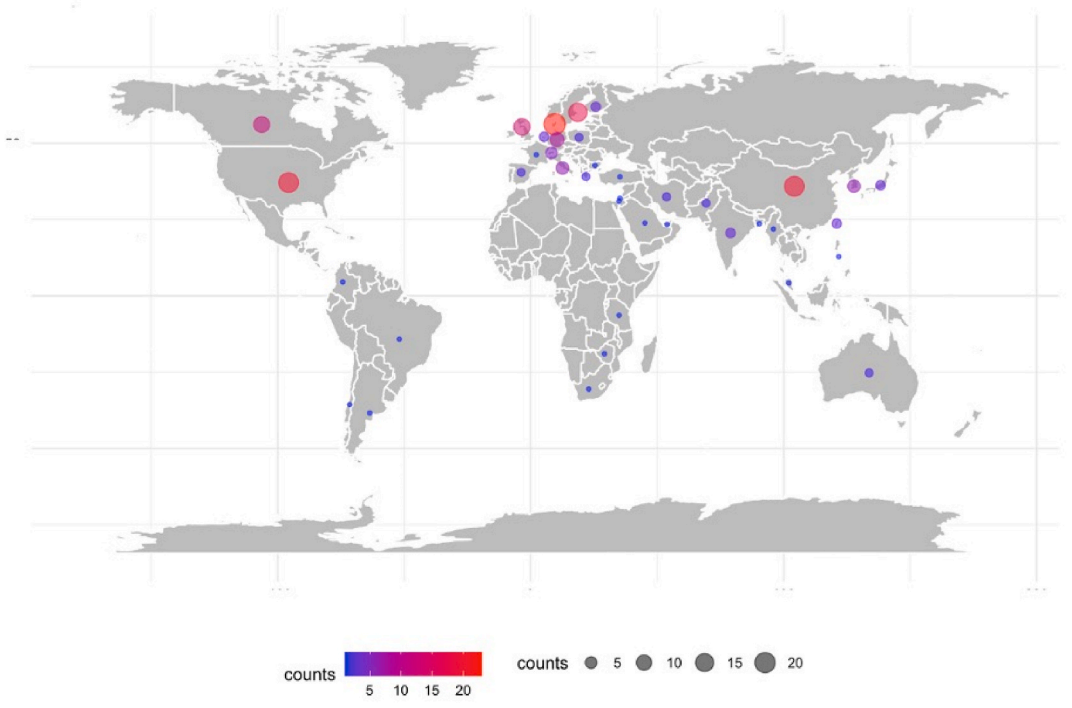

Fig. 2. Geographical distribution of studies.
Fig. 2. The map shows the number of studies from different countries. Each circle represents a country, with the circle's size corresponding to the number of studies from that location. The color gradient represents the count of studies, with cooler colors indicating fewer studies and warmer colors showing more. Countries with a larger volume are highlighted with bigger, warmer colored circles. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

variables such as socioeconomic factors and personal behaviors, such as smoking, diet, and physical activity. This absence raises the possibility that unmeasured factors could influence the observed associations and diminish the reliability of the findings.

We also observed concerns regarding the generalizability of results. The findings from many studies were based on specific populations or geographic settings (Fig. 2), which does not necessarily represent broader populations. Furthermore, recent studies suggest that the exposure-response function may become supra-linear at very low concentrations, such as those found in the Nordic countries, potentially affecting the applicability of these findings to areas with different pollution levels [66,114,115,161].

The cross-sectional design of some studies was another limitation we identified. This design restricts the ability to establish causal relationships, as it does not confirm that exposure precedes the cardiovascular events, complicating the interpretation of causal effects.

3.2.3.1. Risk of bias (RoB) assessment. Our results on the risk of bias revealed varying degrees of risk across the included studies. The most common risk was due to confounding, with 80 studies at a moderate risk level, indicating that while many studies attempted to account for important confounders, there remained some uncertainty regarding their influence on the observed outcomes. Low risk was attributed to 36 studies, suggesting a more thorough adjustment for potential confounders.

Fig. 3 shows a summary of the RoB assessment for all included articles. For detailed information of each study's RoB, see Table S2 (A.8.).

In the overall risk of bias, we identified a trend towards a low to moderate risk. This trend is shown by the methodological strengths, mostly the comprehensive outcome measurement and solid reporting. Despite these strengths, a moderate risk of bias persists, mostly due to challenges in accurately classifying exposures and the possibility of unmeasured confounding factors. Concerns highlight the limitations of ambient exposure assessments as a surrogate for personal exposure levels. Moreover, while registry-based data and observational study designs provide a foundation for outcomes, they also carry potential limitations that could introduce classification and information biases. Our assessment acknowledges the rigorous approaches employed in the study designs, suggesting that the body of evidence in general is somewhat robust. The systematic and comprehensive nature of national cohort data, along with extensive confounder adjustments in select large studies [8,10,12,22,54,59,60,67,75,86,102,109,114,140,141,144,147,148,151,160,162], are recognized as significant strengths that reduce certain risks and establish a strong basis for trusting the identified correlation.

Our RoB assessment confirmed what is already mostly known; the included retrospective cohort studies had the advantage of including large national participant pools. However, these studies were limited in their ability to adjust for a wide range of specific confounding factors, primarily individual-level data, because they relied on national registers or previously collected data that may not have accounted for all potential influencers.

On the other hand, the included prospective cohort studies allowed for the collection of detailed data on targeted confounders. These studies were designed with future analyses in mind, enabling researchers to identify and measure a wide array of relevant variables. While this allowed for a more nuanced and precise understanding of the relationships between exposures and outcomes, the prospective studies had smaller participant numbers compared to their retrospective counterparts.

Participant selection presented less of a bias risk, with the majority of studies [76] falling into the moderate risk category. This suggests a general awareness and control of selection bias among the reviewed studies. However, 62 studies were assessed at low risk, indicating strong confidence in their selection methods.

The classification of exposures was an area where bias was more prevalent, with a notable 123 studies categorized at moderate risk, possibly due to the challenges inherent in modelling pollutants accurately. Serious risk was noted in 11 studies, and only a small fraction [5] was considered at low risk, reflecting precise exposure assessment methods.

Missing data significantly increased the risk of bias in our review, as 83 studies failed to provide information on missing data. This absence of data prevents a clear understanding of whether missing values could have affected the study outcomes. The lack of transparency in reporting missing data raises concerns about the accuracy and reliability of the findings. For the studies that did report on missing data, their approaches varied widely, which suggests a lack of consistency in handling potentially bias-inducing missing

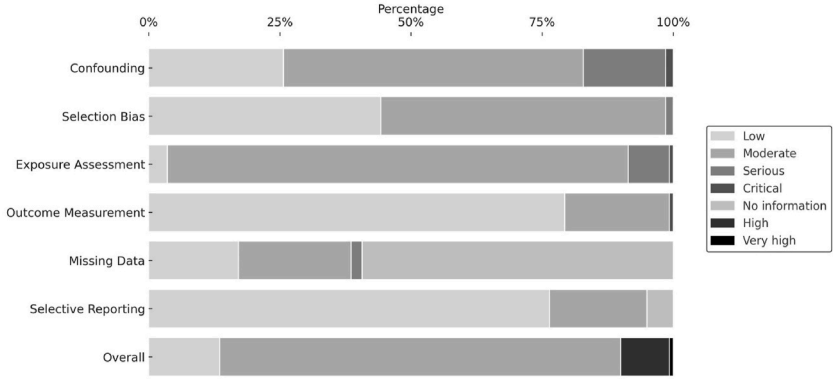

Fig. 3. RoB assessment summary.
Fig. 3 shows a summary of the risk of bias assessment for the studies included in the review, categorized by risk domains. The assessment used the ROBINS-E tool to highlight the methodological strengths and potential biases across the included studies.

data across studies.

Outcome measurement was a strength among the included studies, with a low risk of bias identified in 111 studies. This reflects a high degree of confidence in how cardiovascular outcomes were ascertained across most studies. This confidence is largely due to the fact that the majority of studies utilized national medical registries, which are trusted sources that ensure accuracy through the use of ICD-10 coding.

Selective reporting of results was well-controlled, as reflected by the 107 studies rated at low risk. The risk of reporting bias appears to be minimal. The majority of studies reported outcomes transparently and comprehensively.

Overall, the risk of bias was predominantly moderate across the studies, with 107 studies falling into this category. Low risk was less common, with 19 studies, and high risk was identified in 13 studies, indicating some concerns that warrant careful consideration. Only one study was rated at very high risk of bias, suggesting significant issues across multiple domains.

4. Discussion

4.1. Summary of evidence

Our systematic review evaluated the body of evidence investigating the effects of air pollution and noise on cardiovascular outcomes. Our findings demonstrate a consistent association between prolonged exposure to $PM_{2.5}$ and NO_2, and elevated risks of coronary heart disease and stroke incidents. This effect persists across geographics and varying levels of pollution. Short-term studies presented mixed results, with some identifying positive associations between pollution and cardiovascular outcomes, while others found no significant associations.

The consistency of our results with prior studies indicates a robust association between pollution and cardiovascular health risks. Similar systematic reviews have highlighted the cardiovascular risks associated with long-term exposure to air pollution [7,163–165]. Notably, our findings show a lack of the combined effects of air pollution and noise, which could provide a deeper understanding of their synergistic impacts, and which have been less explored in previous studies [7]. This could contribute new insights to the literature, suggesting that the joint evaluation of these pollutants is important for assessing full cardiovascular risk profiles.

4.2. Heterogeneity of studies and risk of bias

The majority of the studies included in our review were conducted in developed countries, with Denmark [23], China [19], the United States [19], and other European nations making significant contributions. The concentration of studies in developed countries benefits from well-established research infrastructures, comprehensive health registries, and relatively homogeneous healthcare access which enhances the quality and consistency of data collection. Such environments facilitate longitudinal studies with large cohorts that are essential for tracking long-term health outcomes related to pollution exposure. These regions often have robust environmental monitoring systems, allowing for precise quantification and characterization of pollution, which is crucial for assessing exposure-response relationships.

However, the majority of data from high-income countries raises concerns about the generalizability of the findings. While pollutants like $PM_{2.5}$ and NO_2 are present globally, the distribution of components that contribute to $PM_{2.5}$ can differ significantly across regions. More critically, the levels of air pollution differ drastically, with parts of India experiencing annual mean $PM_{2.5}$ concentrations as high as 150 $\mu g/m^3$ [166], far exceeding levels typically observed in Europe and North America. These substantial differences in pollution levels and the composition of pollutants highlight the potential limitations in applying findings from high-income countries to low- and middle-income countries (LMICs).

The underrepresentation of LMICs is particularly concerning given that these regions are often the most impacted by pollution due to rapid urbanization, less strict environmental regulations, and greater use of biomass fuels. The health outcomes in these populations may be influenced by different baseline health statuses, nutritional deficiencies, and other local factors that could modify the effects of pollution. Moreover, healthcare access and public health infrastructure in many LMICs may not support extensive health monitoring, leading to underdiagnosis of cardiovascular conditions and other pollution-related health issues.

We found that the duration of the long-term studies varied significantly. They ranged from 1 to 44 years, with a median duration of 10.5 years. Long-term studies are crucial for understanding chronic exposure effects, but the wide range in study durations can lead to variability in outcome assessments. Similarly, the sample sizes ranged from 32 to over 8.6 million participants, with a median of 50,154. We found that the larger studies provided more robust data and improved the power of statistical analyses. The variety in study durations and sample sizes across the reviewed studies highlights the need for a balanced research approach that incorporates both extensive, long-term studies for comprehensive risk assessment and shorter, targeted studies. These short-term studies are essential for capturing changes during quick exposure peaks that might not be evident in long-term analyses. Such transient peaks may exacerbate or provoke acute cardiovascular events. This can provide insights into the immediate effects of pollution exposure on CVD events.

Our review identified that while a large number of studies focused solely on air pollution, there were some studies that examined both air pollution and noise, and few that focused solely on noise. A significant portion of the studies (91 out of 140) exclusively investigated air pollution. This focus is likely due to the well-documented pathways through which air pollutants, particularly $PM_{2.5}$ and NO_2, impact cardiovascular health—by promoting inflammation, oxidative stress, and endothelial dysfunction [167]. These mechanisms are critical in the progression of atherosclerosis and other cardiovascular diseases.

While our systematic review found evidence linking long-term exposure to air pollutants like $PM_{2.5}$ and NO_2 with increased CV risk, the data on noise exposure was comparatively limited. Nevertheless, emerging studies underscore that noise exposure is more than just

a secondary risk factor. Noise-induced CV effects are primarily mediated through stress mechanisms, including sleep disturbances, chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis, and heightened autonomic responses. These mechanisms differ from those of air pollution, which largely exerts its effects through inflammatory and oxidative stress pathways.

Noise exposure is typically characterized by short-term peaks, such as those caused by traffic, with immediate physiological responses like elevated blood pressure and heart rate variability, compared to the often continuous, chronic nature of air pollution exposure. This difference in exposure patterns and physiological responses suggests that while air pollution and noise may interact, they also have independent pathways that must be evaluated separately.

In light of these insights, we acknowledge the importance of giving noise exposure more weight in future research and public health considerations, particularly when addressing the cumulative impacts of multiple environmental stressors on cardiovascular disease.

Thirty-six studies explored the combined effects of air pollution and noise. These studies are key as they begin to investigate the interactions between the two stressors and their synergistic effects on CVD. The combined impact of these pollutants may not just be additive but could potentially amplify each other's effects. This complicates the risk assessments and intervention strategies. For instance, noise exposure can lead to stress and disturbed sleep, which in turn exacerbates the cardiovascular strain caused by air pollution.

We identified varying degrees of risk of bias across the included studies (Fig. 3). This affects the validity and reliability of the conclusions drawn. For example, few studies used broad regional pollution data as a proxy for individual exposure. This might not capture the true exposure dynamics experienced by individuals. Such approximations can introduce exposure misclassification bias.

The biases observed in relation to confounding and exposure classification necessitate a cautious interpretation of the current evidence. Of the 140 included studies, 114 adjusted for age, 98 adjusted for smoking, 50 and 40 studies adjusted for alcohol and physical activity, respectively. For instance, many studies lacked adequate control for socioeconomic factors (36 studies adjusted for socioeconomics), which can be a significant confounder given its association with both increased pollution exposure and higher cardiovascular risk. This oversight can lead to an overestimation or underestimation of the true effect of pollution. The inconsistent control for lifestyle factors such as smoking, and diet further complicates the ability to isolate the specific contributions of air pollution and noise.

All the reviewed studies utilized residential addresses to estimate pollution exposure, with none exploring occupational addresses or a combination of both. This oversight is significant as individuals often move between different environments throughout the day for work and other activities, which could alter their actual exposure compared to what is estimated based solely on their home location. Currently, it is unclear whether this results in an overestimation or underestimation of exposure levels, but it is crucial that future studies address this gap. By incorporating both residential and occupational exposure data, research can reduce exposure assessment errors and provide a more accurate assessment of true exposure levels. Although this limitation is common in environmental studies, it is important to bear in mind as we interpret the findings and seek to improve study designs in the future.

To address these challenges effectively, future studies could use registry data to track residential addresses retrospectively and, when feasible, gather information on occupational history and types during participant inclusion. This approach will enhance the classification of individual exposure levels by considering both home and workplace environments over time. Furthermore, comprehensive adjustment for a wide range of potential confounders is essential. For example, future studies should consider detailed modeling of socioeconomic variables, dietary intake, medications, comorbidities, and other individual lifestyle factors to refine the estimation of pollution's impact on cardiovascular disease.

Prospective cohort studies that include well designed, real-time exposure assessment methods offer a more accurate approach to understanding the effects of pollution. These studies should be designed with the capability to capture longitudinal data on both residential and occupational exposures. This will provide a more nuanced view of how different environments contribute to the overall exposure. Moreover, using a detailed, pre-defined list of confounders for adjustment in the analysis phase can help minimize the risk of residual confounding. While our review found that most studies were prospective cohorts, followed by retrospective cohorts, it is notable that the retrospective cohorts included a significantly larger number of participants. This is often characteristic of such study designs. However, to enhance the validity of future research outcomes, there should be a concerted effort to focus on larger prospective cohorts that incorporate detailed adjustments for all relevant confounders and evaluate both air pollution and noise concurrently.

4.3. Methodological challenges in the reviewed studies

The use of prospective cohort designs in many studies included in our review stands out as a strength. These designs allow for the establishment of temporal relationships between exposure and the development of cardiovascular outcomes. For example, we found studies conducted in Sweden, Denmark, and the US that utilized this to track the impacts of pollution over more than 30 years [23,25,122,139]. They provide evidence of its chronic effects on cardiovascular disease. The prospective nature of these studies minimizes biases associated with retrospective data collection, which in turn enhance the validity of their findings. Also, the capability to track changes over time using longitudinal data is important in epidemiological research. This strengthens the evidence of association between pollution exposure and cardiovascular outcomes and also allows for the examination of potential delayed effects that shorter-term studies might miss. Longitudinal data provide researchers with insights into the progression and escalation of disease states in response to varying levels of pollution exposure over time. Moreover, the majority of the included reviewed studies are longitudinal studies, which enable the analysis of individual trajectories, which can vary significantly based on personal, environmental, and socioeconomic factors. This individual-level data is essential for identifying high-risk groups and understanding differential responses to pollution.

The different settings and large sample sizes in our reviewed studies enhance the applicability and generalizability of the findings.

One notable study, the ELAPSE study [151], is a multi-national cohort across Europe, which includes both urban and rural populations. This allows for a broader analysis of the impacts across different geographical and socioeconomic contexts. Such variety is important for understanding the universal and localized effects of pollution and can provide a clearer picture of its impact across different societal strata.

Precision in determining exposure levels is important when linking pollution to specific health outcomes. Many studies employed high-resolution spatial data to estimate personal exposure levels rather than relying on broader regional data. For instance, a study from the UK utilized GPS data combined with local air monitoring stations to track individual exposure more accurately [48]. Additionally, studies from Denmark have developed resolutions at address levels. This captures rather precise individual-level exposure and, hence, significantly enhances the accuracy of exposure assessments [23,43,45,66,114,136,137,141,151]. This methodological precision is important for reducing misclassification of exposure. However, as previously described, it is important to note that all included studies model exposure based on residential addresses, which does not account for the significant amount of time individuals may spend away from home. This will potentially lead to exposure misestimations.

Exposure misclassification remains the most significant challenge in environmental health studies in general. Many of the studies included in this review relied on generalized models using residential addresses to estimate exposure to air pollution and noise. We noted that recent advancements have led to the use of better assessment. This is a significant improvement over earlier methods, such as Land Use Regression (LUR) or satellite models [168]. Future studies should strive to use the most advanced, state-of-the-art models available, or innovate new technologies for accurate exposure assessments. Although there is an increasing call for more sophisticated technological and methodological approaches like personal monitoring devices or comprehensive environmental tracking systems, these methods often face practical challenges in large-scale applications due to their logistical complexities and high costs.

The lack of detailed individual-level data, particularly concerning lifestyle factors like diet, physical activity, and smoking habits, were frequently cited as limitations in our review. For example, most studies only classified smoking status as current, former, or never, without considering the amount smoked, duration of smoking, or changes in smoking habits over time. This can lead to significant residual confounding in prospective cohort studies. Another significant issue arises with the long-term follow-up of participants, which in some of the identified studies extended over 30 years. The studies capture data on confounders and covariates – such as medication usage, diet, smoking habits, and comorbidities – only at the baseline inclusion visit. This approach fails to account for changes in these variables over time. This will significantly influence the study's outcomes. For example, a participant's smoking status or medication regimen might change multiple times, which impacts their health trajectory. Yet, if these changes are not recorded and adjusted for throughout the follow-up period, the resulting data may reflect outdated or inaccurate relationships between exposure and health outcomes. To address this, there is a strong case for using time-updated methods to measure these confounders [169–171]. By integrating data that captures these changes, researchers can better understand the dynamics between pollution exposure and CVD and the confounding effects of relevant covariates. This approach enhances the accuracy of the findings and strengthens the understanding of how long-term exposures interact with personal health behaviors and treatments over time. Such advancements in methodological approaches could prove difficult but would provide more reliable evidence.

Generalizability remains a challenge across our identified studies. For instance, a significant proportion of the studies in our systematic review originate from high-income, urban settings in Scandinavia, Europe, and North America. While these studies offer valuable insights, they do not reflect the conditions in rural or low-income areas where pollution dynamics and health outcomes might differ significantly. For example, urban centers typically have better pollution control technologies and health care infrastructures, which can influence the severity and reporting of cardiovascular incidents compared to rural areas. Rural regions might face different types of pollution sources, such as agricultural activities or less regulated industries, which are not typically the focus in urban-centric studies. Moreover, lifestyle factors and baseline health conditions can vary between urban and rural populations, potentially modifying the effects of pollution. This geographic and demographic skew in data sources limits the ability to apply findings universally. It underlines the necessity for studies that are deliberately designed to include diverse settings. Expanding research to include underrepresented regions, such as rural areas in low and middle-income countries, would enhance the external validity of the findings and provide a more nuanced understanding of the global impacts of pollution on cardiovascular health.

4.4. Implications

Our systematic review demonstrates the association between air pollution, noise, and increased risks of CVD. This suggests that current air quality standards may need re-evaluation, as even low particulate matter levels in urban settings significantly affect CVD outcomes. Public health bodies may consider stricter thresholds, as highlighted by differences between WHO guidelines ($PM_{2.5}$ annual mean $\leq$ 5 $\mu g/m^3$) and EU directives ($\leq$25 $\mu g/m^3$) [172,173].

Our findings also emphasize the need for future research with longitudinal designs that incorporate time-updated confounders like smoking and BMI, given their potential to influence long-term exposure outcomes. Furthermore, the generalizability of our results is limited by the predominance of studies from high-income, urban settings. Future studies should focus on diverse populations, including those from rural and low-income regions, to provide a more global perspective.

This review highlights the established relationships between air pollution and cardiovascular diseases, while also identifying a key gap in the literature: the interaction between air pollution and noise pollution. The combined effects of these environmental stressors are not well understood and could have potentially synergistic impacts on cardiovascular health. Future studies should aim to address this gap by exploring how these pollutants interact, particularly in real-world settings where co-exposure is common.

5. Conclusion

Air pollution and noise are important risk factors for CVD, with evidence linking long-term exposure to increased CV risks. However, the combined effects of air pollution and noise are less well-understood, and current findings on their interaction remain variable and inconsistent.

Further research is needed to explore the joint impact of these exposures, particularly through studies that include both residential and occupational addresses. Well-powered, prospective studies with comprehensive confounder adjustments are crucial for understanding these interactions and informing effective public health strategies.

CRediT authorship contribution statement

Stephan Peronard Mayntz: Conceptualization, Data curation, Formal analysis, Funding acquisition, Investigation, Methodology, Resources, Software, Validation, Visualization, Writing – original draft, Writing – review & editing. **Kasper Emil Rosenbech:** Data curation, Formal analysis, Investigation, Methodology, Validation, Writing – review & editing. **Roda Abdulkadir Mohamed:** Writing – review & editing. **Jes Sanddal Lindholt:** Supervision, Validation, Writing – review & editing. **Axel Cosmus Pyndt Diederichsen:** Supervision, Validation, Writing – review & editing. **Lise Marie Frohn:** Supervision, Validation, Writing – review & editing. **Jess Lambrechtsen:** Conceptualization, Funding acquisition, Methodology, Project administration, Resources, Supervision, Validation, Writing – review & editing.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Stephan Peronard Mayntz reports financial support was provided by Region of Southern Denmark. If there are other authors, they 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

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