

ปัจจัยสิ่งแวดล้อมสำคัญบางประการที่ขับเคลื่อนการเกิดโรคหัวใจและหลอดเลือด: การทบทวนเชิงบรรยายด้านกลไกและนัยสำคัญต่อสุขภาพประชากรและสาธารณสุข Selected Environmental Drivers of Cardiovascular Disease: A Narrative Review of Mechanisms and Implications for Population and Public Health

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Received: December 8, 2025 Revised: August 24, 2026 Accepted: August 24, 2026

Abstract

Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality worldwide. Environmental exposures may contribute to cardiovascular risk beyond established clinical and behavioural risk factors. This review aimed to synthesise evidence on environmental exposures associated with cardiovascular risk, including shared biological pathways, interactions between exposures, population vulnerability, and implications for prevention, public-health policy, and environmental justice. A narrative review informed by a structured literature search was conducted using PubMed, Scopus, and Web of Science for publications from January 2000 to December 2024. Reference lists and selected institutional and technical sources were also examined. Of 642 records identified, 231 duplicates were removed, leaving 411 records for screening; 92 full-text publications were assessed for eligibility. 22 sources met the eligibility criteria and were included in the principal narrative synthesis. Evidence was interpreted according to methodological limitations, consistency across studies, exposure–response relationships, biological plausibility, and convergence between epidemiological and mechanistic evidence. PM_{2.5} had the strongest and most consistent evidence of cardiovascular harm. Extreme heat and transportation noise showed substantial but context-dependent associations, whereas evidence concerning artificial light at night, persistent chemical pollutants, and microplastics and nano plastics remained heterogeneous or emerging. Shared biological pathways included oxidative stress, systemic inflammation, endothelial dysfunction, autonomic and circadian dysregulation, metabolic dysregulation, and thrombosis. Co-exposures may have additive or interactive effects, although evidence for synergistic effects remains exposure-specific. In Thailand, a modelling study using 2009 exposure data

estimated a population-attributable fraction of approximately 14.6% for cardiovascular mortality associated with ambient PM_{2.5} exposure. Supplementary contextual evidence indicated increasing attention to PM_{2.5}; however, evidence concerning cardiovascular risk awareness, policy effectiveness, health outcomes, and equity remains limited. Cardiovascular prevention in Thailand should consider environmental exposure assessment, protection of vulnerable populations, source-specific exposure reduction, and evaluation of policy effects using cardiovascular and equity outcomes.

Keywords: Cardiovascular disease, Environmental exposure, Air pollution, Climate-related stressors, Environmental justice

บทคัดย่อ

โรคหัวใจและหลอดเลือดเป็นสาเหตุสำคัญของการเจ็บป่วยและการเสียชีวิตทั่วโลก โดยปัจจัยด้านสิ่งแวดล้อมอาจเพิ่มความเสี่ยงนอกเหนือจากปัจจัยทางคลินิกและพฤติกรรม การทบทวนนี้มีวัตถุประสงค์เพื่อสังเคราะห์หลักฐานเกี่ยวกับปัจจัยด้านสิ่งแวดล้อมที่สัมพันธ์กับความเสี่ยงต่อโรคหัวใจและหลอดเลือด รวมทั้งกลไกทางชีวภาพ ปฏิสัมพันธ์ระหว่างการสัมผัส ความเปราะบางของประชากร และนัยต่อการป้องกันและนโยบายสาธารณสุข การศึกษานี้เป็นการทบทวนวรรณกรรมเชิงพรรณนาโดยสืบค้นอย่างมีโครงสร้างจาก PubMed, Scopus และ Web of Science ครอบคลุมบทความที่เผยแพร่ระหว่าง ค.ศ. 2000–2024 รวมทั้งแหล่งข้อมูลที่เกี่ยวข้อง จากเอกสาร 642 รายการ ตัดรายการซ้ำ 231 รายการ คัดกรอง 411 รายการ และประเมินฉบับเต็ม 92 รายการ โดยมี 22 แหล่งข้อมูลผ่านเกณฑ์และนำเข้าสู่การสังเคราะห์หลักฐาน ผลการทบทวนพบว่า PM_{2.5} มีหลักฐานผลกระทบต่อระบบหัวใจและหลอดเลือดที่ชัดเจนและสอดคล้องมากที่สุด ความร้อนจัดและเสียงรบกวนจากการคมนาคมมีหลักฐานสนับสนุนแต่ขึ้นอยู่กับบริบท ขณะที่หลักฐานเกี่ยวกับแสงประดิษฐ์ในเวลากลางคืน สารเคมีที่คงอยู่ยาวนานในสิ่งแวดล้อม และไมโครพลาสติกและนาโนพลาสติกยังมีความหลากหลายหรืออยู่ในระยะเริ่มต้น กลไกสำคัญร่วมกัน ได้แก่ ภาวะเครียดออกซิเดชัน การอักเสบ ความผิดปกติของเยื่อหลอดเลือด การรบกวนระบบประสาทอัตโนมัติ และจังหวะชีวภาพ ความผิดปกติของการเผาผลาญ และการเกิดลิ้มเลือด การสัมผัสหลายปัจจัยอาจมีผลแบบเพิ่มร่วมหรือปฏิสัมพันธ์กันสำหรับประเทศไทย การศึกษาแบบจำลองจากข้อมูลการสัมผัส ค.ศ. 2009 ประมาณการว่าสัดส่วนการเสียชีวิตจากโรคหัวใจและหลอดเลือดที่สัมพันธ์กับ PM_{2.5} อยู่ที่ประมาณร้อยละ 14.6 ดังนั้น การป้องกันโรคหัวใจและหลอดเลือดควรคำนึงถึงการสัมผัสด้านสิ่งแวดล้อม การคุ้มครองกลุ่มเปราะบาง การลดการสัมผัสที่แหล่งกำเนิด และการประเมินนโยบายด้านสุขภาพและความเป็นธรรม

คำสำคัญ : โรคหัวใจและหลอดเลือด, การสัมผัสสิ่งแวดล้อม, มลพิษทางอากาศ, ปัจจัยกดดันที่เกี่ยวข้องกับสภาพภูมิอากาศ, ความเป็นธรรมด้านสิ่งแวดล้อม

Introduction

Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality worldwide. Although hypertension, dyslipidemia, diabetes, smoking, and physical inactivity are central to cardiovascular prevention, these established risk factors do not fully explain geographical and population-level differences in CVD burden. Environmental determinants are increasingly recognized as important contributors to cardiovascular risk across the life course. Environmental exposures of particular concern include ambient air pollution, climate-related stressors, transportation noise, artificial light at night, and persistent chemical contaminants. These issues are particularly relevant to Thailand, where seasonal biomass burning, urban traffic, industrial activity,

and extreme heat represent important environmental health challenges.

Air pollution and climate-related stressors represent closely related but distinct environmental threats. Long-term exposure to fine particulate matter (PM_{2.5}) has been associated with ischaemic heart disease, stroke, heart failure, arrhythmias, and premature mortality^(1,2). Climate-related stressors may also increase cardiovascular risk through heatwaves, temperature variability, wildfires, and other extreme events. Heat exposure can increase thermoregulatory and circulatory demands, promote dehydration and haemoconcentration, and precipitate acute cardiovascular events, particularly among older adults and people with pre-existing cardiovascular disease. Climate-related

hazards may also interact with air pollutants. For example, a large case-crossover study in China reported interactive effects of heatwaves and ozone exposure on cardiovascular mortality⁽³⁾. This finding highlights the importance of considering concurrent climatic and air-pollution stressors rather than evaluating individual exposures in isolation. In Thailand, a modelling study using 2009 exposure data estimated that the population-attributable fraction of cardiovascular mortality associated with ambient PM_{2.5} exposure was approximately 14.6%⁽⁴⁾. However, evidence concerning the combined cardiovascular effects of heat, air pollution, and other environmental stressors remains limited.

Despite their diversity, environmental exposures may influence cardiovascular health through overlapping biological pathways, including oxidative stress, systemic inflammation, endothelial dysfunction, autonomic dysregulation, thrombogenic responses, circadian disruption, and metabolic dysregulation. Transportation noise has been associated with cardiovascular risk through pathways involving neuroendocrine stress and sleep disturbance⁽⁵⁾, whereas artificial light at night may disrupt circadian rhythms, melatonin regulation, and metabolic homeostasis⁽⁶⁾. Persistent chemical contaminants have also been associated with vascular and metabolic disturbances, although the strength and directness of the evidence vary across compounds. Emerging contaminants such as microplastics and nano plastics have raised additional cardiovascular concerns, with experimental and observational evidence suggesting potential inflammatory, vascular, and pro-thrombotic effects^(7,8). However, their long-term cardiovascular implications and causal relevance remain uncertain.

Environmental stressors rarely occur in isolation. Individuals living in urban, industrial, or high-traffic settings may experience simultaneous exposure to air pollution, heat, transportation noise, artificial light at night, and chemical contaminants. These co-exposures may act through shared or intersecting biological pathways and may have additive or interactive effects in some settings; however, evidence remains limited, and most studies continue to evaluate individual exposures separately.

Environmental cardiovascular risks are also socially patterned. Socioeconomically disadvantaged populations and communities located near major roads or industrial facilities may experience greater environmental exposures while having fewer resources to avoid or mitigate them⁽⁹⁾. Environmental determinants of CVD are therefore closely linked to population vulnerability, health inequalities, and environmental justice.

Important gaps remain in understanding exposure-response relationships, the combined effects of multiple environmental stressors, variation in population susceptibility, and the applicability of international evidence to Thailand. Evidence is particularly limited for several emerging exposures and for multipollutant and exposome-based approaches in middle-income settings. These limitations constrain the development and evaluation of evidence-informed approaches to environmental cardiovascular prevention.

Accordingly, this narrative review aimed (1) to synthesise current evidence on selected environmental exposures associated with cardiovascular disease risk; and (2) to examine shared biological pathways, potential additive and interactive effects of co-exposures, population vulnerability, and implications for clinical prevention, public-health policy, and environmental justice, with particular attention to the Thai context.

Objectives

1. To synthesise current evidence on selected environmental exposures associated with cardiovascular disease risk; and
2. To examine shared biological pathways, potential additive and interactive effects of co-exposures, population vulnerability, and implications for clinical prevention, public-health policy, and environmental justice.

Methods

This narrative review employed a structured literature search to synthesize evidence on environmental determinants of cardiovascular disease (CVD), their biological mechanisms, and their implications for population

health, clinical practice, and public policy. The review encompassed physical environmental factors, including climate-related stressors, environmental noise, artificial light at night, and built-environment characteristics, as well as chemical and toxicant exposures, including air pollutants, persistent organic pollutants, endocrine-disrupting chemicals, microplastics and nano plastics, and contaminants of soil and water. Particular attention was given to shared biological pathways, exposure-response relationships, co-exposures, population vulnerability, environmental justice, and relevance to Thailand.

The identification and screening of relevant publications were guided by principles adapted from the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 framework.⁽¹⁰⁾ However, this review was narrative in design and was not conducted as a systematic review or meta-analysis. Given the breadth and heterogeneity of the evidence, findings were synthesised narratively rather than through statistical pooling or meta-analysis.

Population and Sample

The population of interest comprised peer-reviewed epidemiological, clinical, experimental, mechanistic, modelling, and review publications examining environmental exposures relevant to cardiovascular health or providing evidence on plausible cardiovascular mechanisms. Publications were eligible if they reported a cardiovascular outcome, an intermediate vascular or cardiometabolic outcome, a quantitative disease-burden estimate, or a directly relevant biological mechanism. No geographical restrictions were applied to the principal literature search; however, evidence from Thailand and comparable middle-income settings was prioritized when available.

Conference abstracts, editorials without substantive evidence, duplicate publications, studies unrelated to environmental exposure or cardiovascular health, and publications with insufficient methodological information were excluded. Institutional and technical reports were included only as contextual sources when they provided authoritative information on disease burden or policy

and were distinguished from empirical studies when causal evidence was interpreted.

The search identified 642 records: 312 from PubMed, 198 from Scopus, 101 from Web of Science, and 31 from additional sources. After removal of 231 duplicates, 411 records underwent title and abstract screening, of which 319 were excluded. 92 full-text publications were assessed for eligibility, and 69 were excluded because they did not adequately examine an environmental determinant of CVD, provided insufficient methodological information, or were retained solely as contextual sources. 22 sources were included in the final narrative synthesis, comprising primary epidemiological, experimental, and modelling studies, together with systematic, narrative, and scientific reviews or statements. The study-selection process is presented in Figure 1.

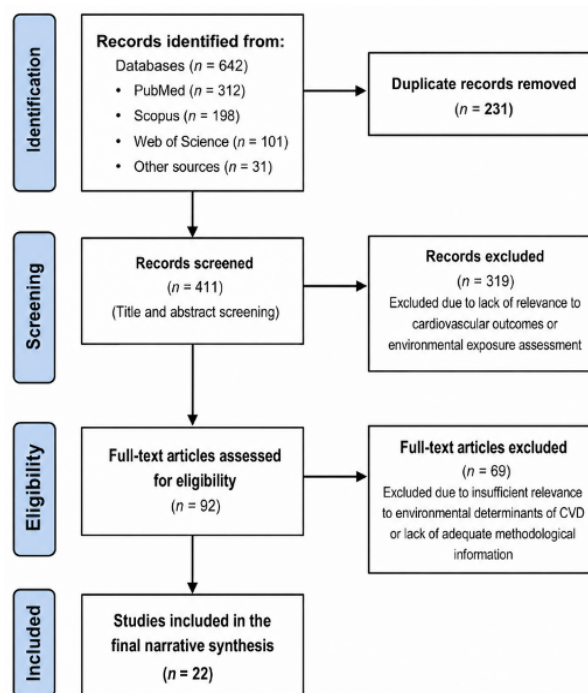


Figure 1 PRISMA Flow Diagram of the Study-Selection Process

Research Instruments

A standardized data-extraction form was used to systematically record publication details, study location, study design, population or experimental model, sample size, environmental exposure, exposure-assessment

method, exposure level or comparison group, cardiovascular outcome, principal effect estimate and uncertainty measure, adjustment for potential confounders, proposed biological mechanisms, and methodological limitations.

Methodological appraisal tools were selected according to study design. Observational cohort, case-control, analytical cross-sectional, and systematic-review evidence was appraised using the relevant Joanna Briggs Institute (JBI) critical appraisal checklists.⁽¹¹⁾ Case-crossover studies were assessed using the JBI Case-Control Checklist adapted to the methodological characteristics of the case-crossover design. Experimental animal studies were assessed using the SYRCLE risk-of-bias tool,⁽¹²⁾ whereas in-vitro toxicological studies were evaluated using ToxRTool.⁽¹³⁾ Narrative reviews were appraised using the Scale for the Assessment of Narrative Review Articles (SANRA).⁽¹⁴⁾ Health-burden and exposure-modelling studies were assessed using the Guidelines for Accurate and Transparent Health Estimates Reporting (GATHER),⁽¹⁵⁾ supplemented by consideration of input-data quality, exposure modelling, counterfactual assumptions, uncertainty, and external validity. Institutional and technical reports were assessed using the Authority, Accuracy, Coverage, Objectivity, Date, and Significance (AACODS) checklist⁽¹⁶⁾ and were treated as contextual rather than causal evidence.

Appraisal findings were recorded according to the structure and domains of the instrument appropriate to each study design. Because the appraisal instruments assessed different methodological domains and did not provide directly comparable numerical metrics across study types, the results were not converted into a common percentage score or a universal high-, moderate-, or low-quality classification. Instead, the principal methodological limitations, directness of evidence, and evidential role of each source were considered in the interpretation of findings. Study-level methodological appraisal findings are presented in Supplementary Table S1.

Data Collection

PubMed, Scopus, and Web of Science were searched for literature published between January 2000

and December 2024. Search terms describing environmental exposures were combined with terms related to cardiovascular outcomes. Exposure terms included “air pollution,” PM_{2.5}, ozone, nitrogen dioxide, climate change, heat exposure, heatwave, environmental noise, transportation noise, artificial light at night, persistent organic pollutants, PFAS, endocrine-disrupting chemicals, microplastics, Nano plastics, soil contamination, water contamination, and built environment. Cardiovascular terms included “cardiovascular disease,” coronary heart disease, myocardial infarction, stroke, hypertension, heart failure, arrhythmia, atherosclerosis, cardiovascular mortality, endothelial dysfunction, and thrombosis.

Boolean operators, truncation, and database-specific subject headings were applied where appropriate. Reference lists of relevant publications were manually screened to identify additional eligible sources. Database-specific search strategies, search dates, filters, and retrieval counts are presented in Supplementary Table S2.

Authoritative institutional and technical reports were selectively examined to provide context regarding global disease burden and policy. Additional Thai governmental and institutional sources published after the principal search period were consulted to update information on public awareness, air-quality monitoring, cross-sector initiatives, and clean-air policy developments. The PFAS socioeconomic report⁽¹⁷⁾ and supplementary Thai policy sources were identified separately and were not included among the 22 sources in the principal evidence synthesis or used independently to establish causal exposure–outcome relationships.

One reviewer performed the initial data extraction using the standardized data-extraction form, and a second reviewer independently verified the extracted information against the full-text publications. Methodological appraisal was conducted using the design-specific appraisal approaches described above. Disagreements arising during data extraction or appraisal were resolved through discussion, consensus, and re-examination of the original publication.

Data Analysis

The included sources were classified into two overarching environmental-health domains: physical environmental factors and chemical and toxicant exposures. Physical environmental factors comprised climate-related stressors, environmental noise, artificial light at night, and built-environment characteristics. Chemical and toxicant exposures comprised air pollution, persistent organic pollutants, endocrine-disrupting chemicals, microplastics and nano plastics, and contaminants of soil and water. Evidence relating to shared biological pathways, co-exposures, population vulnerability, environmental justice, and clinical and public-health implications was subsequently synthesized across both domains.

Statistical pooling and meta-analysis were not performed because of substantial heterogeneity in environmental exposures, study designs, exposure metrics, populations, cardiovascular outcomes, and effect measures. Findings were therefore synthesized narratively by considering the direction and magnitude of associations, methodological limitations, consistency across independent studies, evidence of exposure–response relationships, biological plausibility, and convergence between epidemiological and mechanistic evidence.

The synthesis distinguished direct human cardiovascular evidence from indirect mechanistic, experimental, review, and contextual evidence. Greater interpretive weight was given to evidence derived from methodologically robust and directly relevant studies, findings that were consistent across populations, quantitative exposure–response relationships, and convergence between epidemiological and mechanistic evidence. Methodological appraisal findings informed the interpretation of individual sources but were not used as automatic exclusion criteria. Claims concerning additive, interactive, or synergistic effects were made only when supported by studies that directly evaluated joint exposures. Where evidence was heterogeneous, indirect, or insufficient to support causal inference, the uncertainty was explicitly acknowledged.

Research Ethics

This study was a narrative review based exclusively on previously published literature and publicly available institutional and technical sources. No primary data were collected from human participants, no participants were recruited, and no identifiable individual-level information was accessed or analysed. Accordingly, the study did not involve direct human-participant research. All included sources were reviewed, synthesised, and cited in accordance with established principles of research integrity and academic scholarship.

Results

Study Selection and Evidence Profile

The search identified 642 records: 312 from PubMed, 198 from Scopus, 101 from Web of Science, and 31 from additional sources. After removal of 231 duplicates, 411 records underwent title and abstract screening, of which 319 were excluded. 92 full-text publications were assessed for eligibility, and 69 were excluded because they did not adequately examine an environmental determinant of CVD, lacked sufficient methodological information, or were retained solely as contextual sources. 22 sources were included in the final narrative synthesis.

The evidence base comprised primary epidemiological, experimental, and modelling studies, together with systematic, narrative, and scientific reviews or statements. Direct human cardiovascular evidence was available for PM_{2.5}, transportation noise, heat-related exposure, selected endocrine-disrupting chemicals, and microplastics detected in carotid plaques. Evidence concerning other persistent pollutants, artificial light at night, and built-environment characteristics was derived from a combination of observational, experimental, and mechanistic studies. Institutional and technical reports were used separately to provide disease-burden and policy context and were not included in the principal evidence synthesis or used to infer causal exposure–outcome relationships. Study characteristics and methodological appraisal findings are presented in Supplementary Table S1.

Physical Environmental Factors

Climate-Related Stressors

Climate-related cardiovascular risks arise through exposure to extreme heat, temperature variability, wildfires, and associated disruptions to health and social systems. Heat exposure increases the demand for cutaneous blood flow and cardiac output while promoting dehydration, haemoconcentration, electrolyte disturbances, and systemic inflammation. These physiological responses may increase susceptibility to myocardial ischaemia, arrhythmia, stroke, and heart-failure exacerbation, particularly among older adults and individuals with pre-existing cardiovascular or renal disease.

Climate-related stressors may also interact with air pollution. In a population-based case-crossover study, Xu et al.⁽³⁾ reported positive exposure response associations between ozone, heatwaves, and cardiovascular mortality. The reported odds ratios ranged from 1.03 to 1.11 across ozone-exposure levels and from 1.14 to 1.65 across heatwave definitions or intensities. Joint exposure was associated with a greater risk of cardiovascular mortality than exposure to either stressor considered separately, although the study did not establish a universal threshold applicable across populations. These findings suggest that cardiovascular risk may increase across a range of exposure levels and underscore the importance of considering con-current heat and air-pollution events rather than treating these exposures as independent hazards.

Wildfires represent an additional pathway linking climate change to cardiovascular risk because they increase ambient concentrations of PM_{2.5} and reactive gases. These pollutants may activate oxidative and inflammatory pathways similar to those associated with urban air pollution^(1,18). Climate change may also influence cardiovascular outcomes indirectly through displacement, disruptions to healthcare and medication access, and psychological stress. Across these pathways, vulnerability is particularly relevant among older adults, people with chronic diseases, outdoor workers, and populations with limited access to cooling, healthcare, or safe housing.

Transportation Noise

Transportation noise was associated with hypertension, ischaemic heart disease, myocardial infarction, stroke, heart failure, and atrial fibrillation^(5,19). The evidence suggests that transportation noise may act as a biological stressor rather than solely as an environmental nuisance. Repeated exposure may activate the sympathetic nervous system and the hypothalamic-pituitaryadrenal axis, increasing catecholamine and cortisol concentrations, vascular tone, and blood pressure. These responses may contribute to oxidative stress, endothelial dysfunction, vascular inflammation, and remodeling.

Cardiovascular effects may be particularly relevant during chronic night-time exposure because noise-related microarousals and sleep fragmentation can occur without conscious awakening. These disturbances may reduce restorative sleep, impair nocturnal bloodpressure dipping, alter glucose regulation, and sustain autonomic activation. Reviews included in this synthesis reported adverse cardiovascular associations at chronic environmental noise levels above approximately 50–55 dB and an exposure-response relationship, with the risk of hypertension increasing by approximately 14% per 10-dB increase in night-time road-traffic noise^(5,19). These estimates should be interpreted as populationlevel associations rather than absolute clinical thresholds because they may vary according to noise source, exposure-assessment method, outcome definition, and adjustment for co-exposure to air pollution.

Transportation noise and traffic-related air pollution commonly originate from the same sources and frequently occur together. Their independent cardiovascular effects may therefore be difficult to disentangle completely, and residual confounding remains possible. Nevertheless, the convergence of epidemiological and mechanistic evidence supports an association between transportation-noise exposure and cardiovascular risk, particularly with chronic night-time exposure and concurrent exposure to high levels of air pollution.

Artificial Light at Night

Artificial light at night was associated with circadian disruption, altered melatonin secretion, sleep disturbance, and impaired metabolic regulation⁽⁶⁾. Experimental evidence suggests that short-wavelength light may suppress melatonin secretion and alter autonomic and hormonal rhythms. These changes may contribute to oxidative stress, insulin resistance, elevated blood pressure, and endothelial dysfunction. Observational studies have also reported associations between nighttime light exposure or shift work and cardiometabolic outcomes, including obesity, diabetes, and hypertension.

However, the cardiovascular evidence for artificial light at night was less consistent than that for PM_{2.5} or transportation noise. Exposure definitions varied substantially across studies and included satellite-derived outdoor illumination, bedroom light exposure, screen exposure, and shift-work patterns. These exposure metrics are not directly interchangeable and do not support the identification of a single cardiovascular exposure threshold. Accordingly, the available evidence suggests that artificial light at night is an emerging and potentially modifiable environmental exposure, while the magnitude and independence of its association with cardiovascular risk remain uncertain.

Built and Social Environments

Built-environment characteristics may influence cardiovascular health by shaping physical activity, exposure to environmental pollutants, heat, noise, food access, and psychosocial stress. A systematic review found that greater greenspace exposure was associated with several favourable health outcomes, although substantial heterogeneity existed across exposure definitions and study designs⁽²⁰⁾. **A multicountry cross-sectional study further reported that walkable, mixed-use urban environments were associated with higher levels of physical activity⁽²¹⁾**, providing a potential indirect pathway through which urban form may influence cardiovascular risk.

In contrast, residence near major roads may increase simultaneous exposure to PM_{2.5}, nitrogen dioxide, ultrafine particles, and transportation noise⁽⁵⁾. Lower-income and structurally disadvantaged neighborhoods may experience greater environmental burdens while having more limited

access to greenspace, safe housing, cooling, healthcare, and active-transport infrastructure. Taken together, the evidence suggests that built and social environments may shape both the intensity of environmental exposures and the capacity of individuals and communities to avoid or mitigate those exposures.

Chemical and Toxicant Exposures

Air Pollution

Air pollution had the strongest and most consistent evidence of cardiovascular harm among the environmental determinants reviewed. Long-term PM_{2.5} exposure was associated with ischemic heart disease, stroke, heart failure, arrhythmia, and premature mortality⁽¹⁾. Burnett et al.⁽²⁾ reported a non-linear relationship between long-term outdoor PM_{2.5} exposure and non-accidental mortality across a broad concentration range. Their global model attributed approximately 8.9 million deaths annually to outdoor PM_{2.5} exposure, indicating that substantial health effects may occur at concentrations below those traditionally examined in heavily polluted settings.

The cardiovascular effects associated with PM_{2.5} exposure are supported by several converging biological mechanisms. Inhaled particles may initiate pulmonary oxidative stress and inflammation, followed by systemic inflammatory signaling, endothelial dysfunction, altered autonomic balance, platelet activation, and thrombosis⁽¹⁾. Ultrafine particles or particle constituents may also enter the circulation and interact directly with vascular tissues. Chronic exposure may contribute to atherosclerotic progression and vascular remodelling, whereas short-term increases in exposure may precipitate acute cardiovascular events among susceptible individuals.

Traffic-related air pollution comprises PM_{2.5}, ultrafine particles, black carbon, nitrogen dioxide, and carbon monoxide. Exposure to these pollutants has been associated with vascular dysfunction and increased cardiovascular risk; however, their high correlation and frequent co-occurrence make pollutant specific effects difficult to disentangle. In Thailand, ambient air pollution was estimated to

account for approximately 14.6% of the population-attributable fraction of cardiovascular mortality⁽⁴⁾. This national estimate supports the relevance of the broader international evidence to the Thai context, although the modelling assumptions and exposure estimates should be considered when interpreting the magnitude and regional distribution of the burden.

Persistent Organic Pollutants

Persistent organic pollutants included polychlorinated biphenyls (PCBs), dioxins, dichlorodiphenyltrichloroethane (DDT), polybrominated diphenyl ethers (PBDEs), and per-polyfluoroalkyl substances (PFAS). These compounds persist in the environment, accumulate in biological tissues, and may disrupt lipid metabolism, endocrine signaling, mitochondrial function, and inflammatory regulation^(22,23). Observational studies have reported associations between persistent pollutant exposure and dyslipidemia, hypertension, metabolic dysfunction, and atherosclerotic outcomes, although the strength and consistency of these associations varied across compounds and populations.

Experimental studies provided additional support for the biological plausibility of these associations. Perinatal DDT exposure induced hypertension and cardiac hypertrophy in adult mice⁽²⁴⁾, **whereas chronic exposure to dioxins and PCBs was associated with cardiovascular pathology in rats⁽²⁵⁾**. Cellular evidence further suggested that PCB exposure may activate vascular inflammatory pathways and that modulation of peroxisome proliferator-activated receptor alpha (PPAR- α) signaling may reduce PCB-induced endothelial activation⁽²⁶⁾. Collectively, these findings support oxidative stress, mitochondrial injury, altered gene expression, and endothelial inflammation as plausible biological mechanisms. Nevertheless, direct human cardiovascular evidence was less consistent than that for PM_{2.5}, and heterogeneity in compounds, exposure assessment, and exposure mixtures precluded identification of a single dose-response threshold.

PFAS were addressed using supplementary contextual and socioeconomic evidence and were not included in the principal 22 source evidence synthesis. Goldenman et al.⁽¹⁷⁾ described the long-term societal, environmental, and regulatory costs associated with PFAS persistence and exposure. Because this report did not provide direct primary evidence on cardiovascular outcomes, it was not used to establish a

causal association between PFAS exposure and CVD or to define a cardiovascular exposure threshold.

Endocrine-Disrupting Chemicals

Endocrine-disrupting chemicals examined in the included evidence comprised bisphenols, phthalates, chlorpyrifos, and dioxin-related compounds. Available evidence suggested that these substances may affect metabolic, inflammatory, endothelial, and myocardial pathways; however, the directness and strength of the evidence varied by compound and study design^(25,27,28)

Human evidence was most direct for phthalates. Su et al.⁽²⁹⁾ reported an association between phthalate exposure and subclinical atherosclerosis in a young population, supporting a potential vascular effect before the development of overt clinical disease. However, the cross-sectional design limited causal inference and did not establish temporality. Evidence for bisphenol A and bisphenol S was derived largely from mechanistic and review literature, which suggested potential links between these chemicals and oxidative stress, pancreatic β -cell dysfunction, endocrine disruption, and vascular injury^(27,28).

Experimental animal evidence provided additional support for biological plausibility. Intermittent chlorpyrifos exposure was associated with cardiac hypertrophy and oxidative stress in rats⁽³⁰⁾. **Dioxin- and PCB-related experimental studies also identified cardiovascular pathology and endothelial inflammatory activation^(25,26)**. Collectively, these findings support the biological plausibility of cardiovascular effects; however, extrapolation from experimental doses to typical human exposure levels remains uncertain. The available evidence therefore raises concern regarding potential cardiovascular toxicity but remains insufficient to define uniform exposure thresholds for cardiovascular effects in humans.

Quantitative exposure response evidence was available for only a subset of the environmental determinants examined. The most clearly characterized relationships involved PM_{2.5}, transportation noise, ozone, and heatwaves, whereas evidence for emerging contaminants was generally based on comparisons between exposure groups or internal measures of exposure rather than externally defined dose thresholds. The principal quantitative findings are summarized in Table 1.

Table 1 Quantitative Evidence Linking Environmental Exposures to Cardiovascular Outcomes

Exposure	Exposure measure or comparison	Study design/ population	Cardiovascular outcome	Effect estimate	Interpretation	Reference
PM _{2.5}	Long-term exposure across a broad concentration range	Global exposure-response modelling	Non-accidental mortality	Non-linear exposure-response relationship; approximately 8.9 million attributable deaths annually	Strong and consistent evidence of an exposure-response association across a broad concentration range; no universal safe threshold was established	(2)
Transportation noise	Per 10-dB increase in night-time road-traffic noise	Cohort and case-control evidence summarized in reviews	Hypertension	Approximately 14% higher risk	Supports an exposure-response association; estimates vary by noise metric and population	(5,19)
Ozone	Study-specific exposure categories	Population-based case-crossover study	Cardiovascular mortality	OR 1.03–1.11	Association varied according to exposure level and heatwave conditions	(3)
Heatwaves	Study-specific duration and intensity definitions	Population-based case-crossover study	Cardiovascular mortality	OR 1.14–1.65	Stronger associations were observed under more severe heatwave definitions	(3)
microplastics and nano plastics in carotid plaques	Detected versus not detected	Prospective observational study	Composite of myocardial infarction, stroke, or all-cause death	HR 4.53; 95% CI: 2.00–10.27	Prognostic association; causality and an external exposure threshold were not established	(8)
Ambient air pollution in Thailand	Modelled population exposure	GIS-based burden study	Cardiovascular mortality	Approximately 14.6% population-attributable fraction	Supports the national relevance of ambient air pollution; the estimate depends on modelling assumptions and exposure estimates	(4)

Microplastics and Nano plastics

Evidence concerning microplastics and nano plastics was emerging and substantially less mature than that for PM_{2.5}. A narrative review identified ingestion and inhalation as major exposure pathways and described oxidative stress, mitochondrial dysfunction, endothelial injury, platelet activation, and immune signaling as plausible cardiovascular mechanisms⁽⁷⁾. Experimental findings suggested that smaller particles may cross biological barriers and interact with vascular and cardiac tissues; however, the relevance of many experimental concentrations to typical human exposure levels remains uncertain.

The most direct human evidence came from Marfella et al.⁽⁸⁾, who detected microplastics or nano plastics in carotid atherosclerotic plaques. Patients in whom these particles were detected had a higher subsequent risk of the composite outcome of myocardial infarction, stroke, or death from any cause than those without detected particles. The adjusted hazard ratio was 4.53 (95% CI: 2.00–10.27) over approximately 34 months of follow-up. This finding demonstrated a prognostic association but did not establish that the detected particles caused the cardiovascular events. Residual confounding, exposure misclassification, and the selected surgical population limit the generalizability of the findings. Accordingly, microplastics and nano plastics should be regarded as an emerging cardiovascular concern rather than an established causal risk factor.

Soil and Water Contaminants

The included evidence provided limited direct evidence on cardiovascular outcomes associated with metals and other soil or water contaminants. Although broader toxicological literature has reported associations of lead, cadmium, arsenic, nitrate, and disinfection by-products with vascular or cardiometabolic effects, these studies were not sufficiently represented in the included evidence base to support detailed exposure–response conclusions. Accordingly, soil and water contamination was retained as an important contextual domain; however, specific cardiovascular associations could not be characterised reliably within the scope of

the present review. Further conclusions would require the inclusion and methodological appraisal of directly relevant studies.

Shared Biological Pathways and Potential Interactive Effects

Across the physical and chemical domains, five recurring biological pathways were implicated in associations between environmental exposures and cardiovascular injury: oxidative stress, systemic inflammation, endothelial dysfunction, autonomic or neuroendocrine imbalance, and metabolic or circadian dysregulation. PM_{2.5}, persistent pollutants, endocrine-disrupting chemicals, and microplastics were associated with convergent oxidative, inflammatory, mitochondrial, and endothelial pathways^(1,7,18,23,28). **Transportation noise and artificial light at night were linked predominantly to sympathetic activation, sleep disturbance, and circadian disruption**^(5,6,19). Heat exposure may impose additional thermoregulatory and hemodynamic stress, potentially increasing cardiovascular vulnerability among individuals with underlying vascular disease.

The available evidence also suggested that some co-exposures may have additive or interactive effects. Concurrent exposure to heatwaves and ozone was associated with greater cardiovascular mortality than exposure to either stressor considered separately⁽³⁾, **while road traffic represents a shared source of transportation noise and a complex mixture of air pollutants**^(5,19). However, most studies examined individual exposures, and the available evidence was insufficient to conclude that co-exposures generally act synergistically. The magnitude and direction of potential interactions may depend on exposure intensity, timing, duration, population susceptibility, and the cardiovascular outcome evaluated.

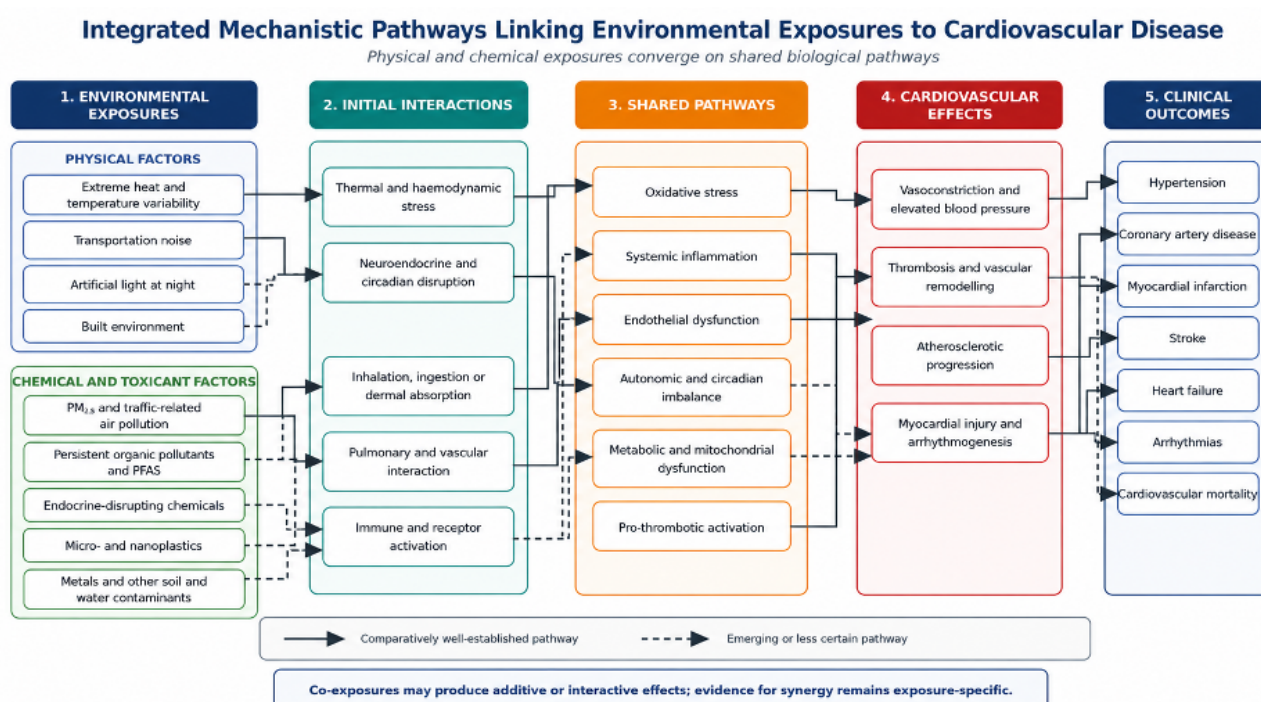


Figure 2 Integrated mechanistic pathways linking physical and chemical environmental exposures to cardiovascular disease.

Environmental exposures may converge on shared biological pathways, including oxidative stress, systemic inflammation, endothelial dysfunction, autonomic and circadian dysregulation, metabolic dysregulation, and mitochondrial injury. These processes may contribute to vascular remodeling, thrombosis, atherosclerotic progression, elevated blood pressure, myocardial injury and arrhythmogenesis, and ultimately to clinical outcomes including hypertension, coronary artery disease, myocardial infarction, stroke, heart failure, arrhythmias, and cardiovascular mortality. Solid arrows represent comparatively well-established pathways, whereas dashed arrows represent emerging or less certain relationships. Evidence supporting pathways involving PM_{2.5} and transportation noise is comparatively more established, whereas pathways involving artificial light at night, persistent organic pollutants, endocrine-disrupting chemicals, and microplastics and nano plastics remain supported to varying degrees by mechanistic, experimental, and observational evidence^(1,5-8,18,19,23,28).

Population Vulnerability and Environmental Justice

Environmental cardiovascular risks were not

distributed equally across populations. Urban and low-income communities, people living near major roads or industrial facilities, outdoor workers, shift workers, older adults, and individuals with pre-existing cardiometabolic disease may experience greater environmental exposures, increased physiological susceptibility, or both. Socially and economically disadvantaged populations may also face multiple concurrent stressors, including air pollution, noise, heat, limited access to greenspace, and restricted access to preventive resources⁽⁹⁾.

Evidence concerning environmental justice suggests that social and structural disadvantage may shape both exposure and the capacity to avoid, mitigate, or recover from environmental hazards. Factors such as housing quality, land use, transport systems, occupation, healthcare access, and access to cooling or clean indoor air may influence vulnerability to the cardiovascular effects of environmental exposures. Collectively, these findings suggest that environmental exposures may contribute to existing cardiovascular health inequalities; however, direct evidence from Thailand and other middle-income settings remains limited.

Clinical and Public-Health Implications Identified from the Evidence

The synthesis identified PM_{2.5}, transportation noise, and extreme heat as environmental factors with comparatively well-established evidence and immediate relevance to cardiovascular prevention. Evidence concerning persistent chemicals, artificial light at night, and microplastics and nano plastics was less sufficient to define clinical exposure thresholds but highlighted the need for continued surveillance and further research. Assessment of environmental exposure history, including residential proximity to major roads or industrial sites, occupational conditions, household fuel use, shift work, and exposure during episodes of extreme heat or air pollution, may help identify individuals with increased environmental vulnerability.

At the population level, the available evidence suggests potential cardiovascular benefits from measures aimed at reducing air-pollutant emissions, transportation noise, heat exposure, and unequal environmental burdens. Evidence concerning the built environment also suggests that access to greenspace and active-transport infrastructure may contribute indirectly to cardiovascular health through pathways such as increased physical activity and reduced environmental stressors^(20,21). However, the effectiveness of specific clinical, public-health, or policy interventions was not systematically evaluated in this review. These implications should therefore be interpreted as evidence-informed considerations rather than formal clinical or policy recommendations. Detailed recommendations and research priorities are presented separately in the Potential Implications and Research Priorities section.

Discussion

This review indicates that environmental cardiovascular risk is best understood as arising from the interplay of exposure intensity, biological susceptibility, and social context rather than from isolated environmental hazards. Across the evidence base, diverse physical and chemical exposures were associated with

convergent biological pathways involving oxidative stress, systemic inflammation, endothelial dysfunction, autonomic and circadian dysregulation, metabolic dysregulation, and thrombosis. This convergence supports biological plausibility; however, the maturity and directness of the evidence varied across exposures. Evidence concerning PM_{2.5} was comparatively well established, heat and transportation noise showed substantial but context-dependent associations, and evidence concerning artificial light at night, persistent chemicals, and microplastics and nano plastics remained heterogeneous, emerging, or compound-specific.

PM_{2.5} provided a particularly clear example of convergence between epidemiological and mechanistic evidence. Long-term exposure has been associated with ischemic heart

disease, stroke, heart failure, arrhythmia, and premature mortality, while mechanistic evidence supports inflammatory, endothelial, autonomic, and pro-thrombotic pathways⁽¹⁾. **The non-linear exposure-response relationship observed across a broad concentration range further suggests that adverse health effects are not confined to episodes of extremely high pollution⁽²⁾.** Nevertheless, burden estimates depend on modelled exposures, counterfactual assumptions, and other methodological choices and should therefore be interpreted as population-level estimates rather than precise measures of individual risk.

Climate-related stressors and transportation noise may contribute to cardiovascular risk through partly distinct but intersecting pathways. Heat imposes thermoregulatory and hemodynamic strain, whereas transportation noise has been associated with sleep disruption and neuroendocrine activation^(5,19). **The cardiovascular consequences of these exposures may also be influenced by concurrent air pollution. The joint association of heatwaves and ozone with cardiovascular mortality reported by Xu et al.⁽³⁾** is consistent with the importance of considering co-exposures. However, evidence from one exposure combination should not be generalized to all

multipollutant settings. Overall, the available literature suggests the potential for additive or interactive effects in some settings but does not establish universal synergistic effects.

Evidence concerning artificial light at night and chemical contaminants was less direct. Artificial light at night may affect cardiovascular health through circadian misalignment, melatonin suppression, sleep disturbance, and metabolic dysfunction⁽⁶⁾; **however, heterogeneous exposure measures and potential confounding by occupation, urbanization, and behaviour limit estimation of its independent cardiovascular association. Persistent pollutants and endocrine-disrupting chemicals similarly showed plausible associations with vascular and metabolic injury**⁽²²⁻³⁰⁾. Animal and cellular studies provided evidence of endothelial activation, oxidative stress, mitochondrial injury, and cardiac remodeling; however, experimental doses cannot be directly translated into human exposure thresholds. These findings support concern regarding potential cardiovascular effects while emphasizing the need to interpret evidence separately according to compound, exposure characteristics, and study design.

Microplastics and nano plastics illustrate the distinction between biological plausibility and causal certainty. Detection of microplastics or nano plastics in carotid atherosclerotic plaques was associated with a higher subsequent risk of myocardial infarction, stroke, or death from any cause⁽⁸⁾, **consistent with proposed inflammatory and pro-thrombotic mechanisms**⁽⁷⁾. However, this association does not establish causality, define external exposure levels, or demonstrate generalizability beyond the selected surgical population. micro-plastics and nano plastics should therefore be regarded as an emerging cardiovascular concern requiring longitudinal confirmation, standardized exposure assessment, and replication across diverse populations.

A central finding across the exposure domains is that environmental cardiovascular risks are socially patterned. Communities with fewer socioeconomic resources and populations living near major roads or industrial facilities may experience greater exposure to

pollution, heat, noise, and chemical contaminants while having less capacity to avoid or mitigate these exposures⁽⁹⁾. The resulting cardiovascular burden may reflect both multiple concurrent exposures and reduced resilience associated with inadequate housing, limited access to greenspace, occupational constraints, and restricted access to healthcare. Environmental justice should therefore be considered an integral dimension of environmental cardiovascular risk because social and structural conditions may shape both the distribution of exposures and the capacity to respond to them.

These findings are particularly relevant to Thailand. Ambient air pollution has been estimated to account for approximately 14.6% of the population-attributable fraction of cardiovascular mortality⁽⁴⁾. The Thai context includes urban traffic, industrial activity, seasonal biomass burning, transboundary haze, and increasing exposure to extreme heat. Although the distribution of these hazards varies geographically, multiple exposures may converge among vulnerable populations, including older adults, people with established CVD, outdoor workers, and residents of densely populated or industrial-adjacent communities. However, the Thai evidence included in this review was concentrated primarily on air pollution and was insufficient to quantify the cardiovascular burden attributable to transportation noise, heat, artificial light at night, or complex chemical mixtures.

Despite the relevance of environmental cardiovascular risks to Thailand, evidence evaluating policy implementation, achieved exposure reductions, cardiovascular outcomes, and distributional equity was insufficiently represented in the included evidence base. Evidence concerning public awareness and private-sector contributions was also insufficient to support specific conclusions within the scope of this review. These areas should therefore be regarded as important knowledge gaps requiring further investigation rather than as established findings.

This review has several limitations. Its narrative design, heterogeneous evidence base, and inclusion of epidemiological, experimental, review, and contextual

sources precluded quantitative pooling and limited direct comparisons across exposures. Differences in exposure assessment, cardiovascular outcomes, confounder adjustment, and followup also reduced comparability. Most studies examined individual exposures despite the prevalence of real-world co-exposures, and much of the human evidence originated from high-income countries. These limitations are particularly important for emerging contaminants, for which inconsistent sampling and analytical methods and limited longitudinal evidence restrict causal inference. In addition, because the review was narrative rather than systematic in design, the findings should not be interpreted as providing exhaustive coverage of all environmental cardiovascular evidence.

Future research should therefore move beyond isolated exposure–outcome associations. Longitudinal studies in Thailand and comparable middle-income settings should combine harmonized exposure assessment with validated cardiovascular outcomes and explicit consideration of social vulnerability. Multipollutant and exposome-based approaches are needed to determine whether environmental stressors act independently, additively, or interactively. Intervention and natural-experiment studies should also evaluate whether environmental policies are associated with measurable reductions in exposure and cardiovascular burden and whether these benefits are equitably distributed among highly exposed and vulnerable populations.

Overall, the evidence supports a differentiated interpretation across environmental exposures. $PM_{2.5}$ has comparatively well-established cardiovascular evidence; heat and transportation noise have substantial but context-dependent evidence; and artificial light at night, persistent chemicals, and microplastics and nano plastics remain emerging, heterogeneous, or compound-specific concerns. The principal implication is not that all environmental exposures carry equal causal weight, but rather that cardiovascular prevention should account for interacting environmental, biological, and social determinants.

Thailand: Public Understanding, Policy Implementation, and Cross-Sector Action

Supplementary contextual evidence from Thailand provides additional insight into public understanding, policy implementation, and cross-sector responses to environmental health risks. A 2025 Department of Health poll of 623 respondents found that 70.11% regularly monitored $PM_{2.5}$ levels and 77.21% reported knowing and applying protective measures. However, only 48.64% reported knowing how to create a clean-air room, while other respondents considered such measures difficult or unsuitable for their homes or reported not knowing how to implement them⁽³¹⁾. These findings suggest that awareness may not necessarily translate into feasible protective action. Interpretation should remain cautious because the poll was not nationally representative and women and government-sector workers were overrepresented among respondents. Moreover, the poll assessed general health awareness and protective practices rather than knowledge of cardiovascular risk specifically. Evidence concerning public understanding of the cardiovascular consequences of environmental exposures in Thailand therefore remains limited.

Contextual policy evidence indicates increasing attention to air-pollution management in Thailand. Air-quality surveillance, pollution alerts, industrial emission-control measures, actions addressing agricultural burning, and mechanisms for managing seasonal and transboundary haze have been implemented. In 2025, the Pollution Control Department reported 100 air-quality monitoring stations across 69 provinces and plans to expand monitoring coverage, supported by the integration of ground-based monitoring, satellite observations, and forecasting systems⁽³²⁾. However, these contextual sources were not designed to evaluate cardiovascular outcomes, and the available evidence was insufficient to determine whether these measures were associated with sustained reductions in $PM_{2.5}$ exposure, cardiovascular morbidity or mortality, or geographical inequalities in exposure and health outcomes. Future policy evaluations should therefore extend beyond implementation activities to assess exposure trends, enforcement, population reach, cardiovascular outcomes, and equity.

Cross-sector initiatives have also been used to support technological and monitoring activities. Collaborative activities involving local authorities, academic institutions, state enterprises, industry organizations, and technology providers have included sensor deployment, source identification, forecasting, and public communication^(32,33). These initiatives illustrate potential mechanisms for strengthening air-quality management; however, the available evidence concerning their effectiveness remains primarily operational and contextual. The contextual sources considered here were not designed to determine whether these initiatives independently reduced population exposure or improved cardiovascular outcomes. Cross-sector and public-private initiatives should therefore be interpreted as potential implementation mechanisms rather than established cardiovascular interventions.

Overall, the available contextual evidence indicates increasing multisectoral attention to PM_{2.5} and environmental health in Thailand. Nevertheless, important evidence gaps remain. Public understanding of and communication about the cardiovascular consequences of PM_{2.5} remain insufficiently characterized; the effects of policy implementation on cardiovascular outcomes have not been adequately evaluated; and the distribution of potential policy benefits across haze-affected, urban, low-income, and industrial-adjacent communities remains unclear. Addressing these gaps will require nationally representative studies of environmental health literacy, transparent indicators of policy implementation and effectiveness, and stronger integration of environmental monitoring with cardiovascular surveillance.

Recommendation

Clinical and Individual-Level Actions

Thai healthcare providers could consider piloting the inclusion of a brief environmental exposure history as part of cardiovascular risk assessment, particularly for older adults, patients with established cardiovascular disease (CVD), outdoor workers, shift workers, and residents of areas with high levels of air pollution or substantial industrial activity. Such an assessment could

include residential proximity to major roads or industrial sites, occupational exposures, household fuel use, shift work, and recent exposure to episodes of elevated PM_{2.5} or extreme heat. Air pollution and extreme heat have been associated with increased cardiovascular risk⁽¹⁻³⁾. During periods of elevated environmental risk, clinicians could provide advice consistent with established public-health guidance, including reducing strenuous outdoor activity, maintaining adequate hydration, ensuring continuity of prescribed medications, and seeking prompt medical attention for worsening cardiovascular symptoms. Advice concerning transportation noise and artificial light at night could focus on reducing night-time exposure and protecting sleep, while acknowledging that evidence for direct cardiovascular benefits from these interventions remains limited^(5,6,19). The feasibility, acceptability, and effectiveness of incorporating these approaches into routine cardiovascular care should be evaluated before widespread implementation.

Health-System Actions

Thai health authorities and healthcare facilities could consider piloting mechanisms that link air-quality and heat alerts with targeted risk communication for people with cardiovascular conditions, including heart failure, coronary artery disease, hypertension, and arrhythmias. Before routine implementation, the feasibility, acceptability, privacy, and data-governance implications of recording relevant residential, occupational, and environmental exposure information in electronic health records should be evaluated. Pilot programmers could assess process indicators, such as the proportion of eligible high-risk patients receiving environmental alerts, undergoing environmental exposure screening, and receiving documented counselling, alongside relevant health outcomes, including cardiovascular emergency visits and hospitalizations during periods of elevated air pollution or extreme heat. Such evaluations could help determine whether these approaches are feasible and acceptable and whether their implementation is associated with measurable clinical and public-health benefits.

Environmental and Public-Policy Actions

Given the documented cardiovascular burden associated with ambient air pollution in Thailand⁽⁴⁾, environmental and public-health authorities could consider population-level measures aimed at reducing environmental exposure at source, alongside timely public warnings and individual-level protective guidance. Potential measures include reducing traffic and industry-related emissions, preventing open burning, supporting alternatives to agricultural burning, reducing emissions from household combustion, and strengthening approaches to managing seasonal and transboundary haze. Environmental monitoring could also be expanded or strengthened to better characterize exposure patterns in haze-affected areas, densely populated urban settings, industrial zones, and underserved communities. Future evaluations could assess trends in PM_{2.5} concentrations, the frequency and duration of exceedances of national air-quality standards, source-specific emission trends, population reach, and geographical inequalities in exposure.

Local and regional authorities could also consider incorporating cardiovascular health considerations into heat-adaptation and urban planning strategies. Potential approaches include improving access to shaded pedestrian environments and cooling areas, expanding greenspace and active-transport infrastructure, reducing traffic-related emissions and transportation noise, and limiting unnecessary night-time light exposure. Particular attention could be given to populations experiencing multiple environmental and social disadvantages, as cumulative exposures and limited access to protective resources may increase vulnerability to adverse cardiovascular outcomes⁽⁹⁾. Future implementation and evaluation efforts should assess both the effectiveness and equity of these approaches using environmental exposure measures, relevant cardiovascular outcomes, and indicators of population reach.

Research Priorities

Future research in Thailand should priorities longitudinal studies that integrate harmonized environmental exposure assessment with validated cardiovascular

outcomes. Studies should examine geographical and contextual variation across urban, haze-affected, industrial, and rural settings and adequately represent populations that may experience greater exposure or susceptibility, including older adults, outdoor workers, shift workers, and people with established CVD. Multipollutant and exposome-based approaches are needed to determine whether environmental stressors, including heat, air pollution, transportation noise, artificial light at night, and chemical exposures, act independently, additively, or interactively.

Research on microplastics and nano plastics should priorities the development and application of standardized sampling, analytical, and quality-control methods, together with longitudinal assessment of both external and internal exposures and cardiovascular outcomes. Given the current limitations of the evidence, future studies should focus on characterizing exposure response relationships and clarifying causal pathways rather than attempting to define cardiovascular exposure thresholds prematurely. Environmental policy and intervention studies should also assess changes in environmental exposures and relevant cardiovascular outcomes, including cardiovascular morbidity, hospitalizations, and mortality, while examining whether potential benefits are equitably distributed across highly exposed and socially vulnerable populations

Conclusion

Environmental exposures are associated with cardiovascular risk through overlapping inflammatory, vascular, autonomic, circadian, and metabolic pathways, although the strength and directness of the evidence vary across exposures. PM_{2.5} has the most consistent epidemiological and mechanistic support, whereas evidence for extreme heat and transportation noise is substantial but context dependent. Evidence concerning artificial light at night, persistent chemicals, and microplastics and nano plastics remains emerging, heterogeneous, or compound-specific. In Thailand, the documented cardiovascular burden associated with

ambient air pollution highlights the importance of considering environmental exposures in cardiovascular prevention and strengthening coordinated approaches across environmental, health, and urban sectors. However, locally specific evidence for several environmental exposures remains limited. Future research should prioritise longitudinal, multipollutant, and intervention studies that generate locally relevant evidence and evaluate whether reductions in environmental exposures and associated health benefits are equitably distributed across highly exposed and socially vulnerable populations.

Acknowledgement

The authors would like to express their sincere gratitude to their advisors and academic mentors for their valuable guidance, constructive feedback, and support throughout the development of this review. Their expertise and thoughtful suggestions contributed to refining the conceptual framework, critical analysis, and overall quality of the manuscript.

Declaration of AI Use in Manuscript Preparation

The authors used ChatGPT (OpenAI, San Francisco, CA, USA) as an AI-assisted tool during manuscript preparation to support language editing, grammar checking, text refinement, organisation, and improvements in clarity and academic presentation. The AI tool was used solely as an editorial aid and did not replace the authors' scholarly judgment. All literature searches, source selection, reference verification, critical appraisal, interpretation of the evidence, and scientific conclusions were independently reviewed and verified by the authors. The authors critically reviewed and revised all AI-assisted content and take full responsibility for the accuracy, validity, originality, and integrity of the final manuscript.

Declarations

Conflict of Interest: The authors declare no conflict of interest.

Funding: This review received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Ethics Approval: Not applicable, as this narrative review was based exclusively on previously published literature and did not involve human participants, identifiable personal data, or animal subjects.

Author Contributions: All authors contributed to the conception of the review, literature search and evidence synthesis, drafting, and critical revision of the manuscript. All authors reviewed and approved the final version of the manuscript.

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SUPPLEMENTARY MATERIAL

Supplementary Table S1

Design-specific methodological appraisal and interpretive role of sources included in the principal narrative synthesis

Ref.	Source	Design/source type	Appraisal approach	Appraisal result*	Principal methodological limitation(s)*	Interpretive role
1	Brook et al., 2010	Scientific statement and evidence review	Contextual assessment of transparency, evidence selection, and conflicts of interest	Contextual assessment completed; no numerical score presented	Not a systematic review; study-selection and study-level appraisal procedures were not fully reproducible; some conclusions relied on indirect evidence	Authoritative cardiovascular and mechanistic context
2	Burnett et al., 2018	Global exposure-response modelling study	GATHER reporting criteria, supplemented by assessment of model assumptions, input-data quality, exposure estimates, uncertainty, and external validity	Assessment completed; no numerical score presented	Dependent on model specification and exposure estimates; uncertainty in regions with sparse data; limited individual-level information; possible exposure misclassification	Direct quantitative population-level evidence
3	Xu et al., 2024	Population-based case-crossover study	JBIChecklist for Case-Control Studies, adapted to the case-crossover design	Appraisal completed; no numerical score presented	Area-level exposure estimates; possible exposure misclassification; residual time-varying confounding; limited generalisability beyond the study setting	Direct quantitative evidence of heatwave-ozone interaction
4	Pinichka et al., 2017	GIS-based comparative risk-assessment and burden-modelling study	GATHER reporting criteria, supplemented by assessment of exposure modelling, input-data quality, uncertainty, and external validity	Assessment completed; no numerical score presented	Modelled rather than individually measured exposure; externally derived exposure-response functions; reliance on 2009 exposure data; uncertainty in baseline mortality and spatial estimates	Direct Thai population-burden evidence
5	Münzel et al., 2024	Narrative/state-of-the-art review	SANRA	Appraisal completed; numerical score not presented	No fully reproducible systematic search or formal study-level risk-of-bias assessment; heterogeneity in noise-exposure metrics and cardiovascular outcomes	Mechanistic and clinical context for transportation noise
6	Cho et al., 2015	Literature review	SANRA	Appraisal completed; numerical score not presented	Narrative search and selection procedures; heterogeneous definitions and measurements of artificial light at night; limited longitudinal evidence	Contextual evidence for artificial light at night

Ref.	Source	Design/source type	Appraisal approach	Appraisal result*	Principal methodological limitation(s)*	Interpretive role
7	Zheng et al., 2024	Narrative review	SANRA	Appraisal completed; numerical score not presented	Evidence derived mainly from experimental and cross-sectional studies; heterogeneous methods for detecting micro- and nano-plastics; limited prospective human evidence	Emerging microplastic and nanoplastic cardiovascular context
8	Marfella et al., 2024	Prospective observational cohort study	JBIChecklist for Cohort Studies	Appraisal completed; no numerical score presented	Selected surgical population; residual confounding; external exposure source and timing were not quantified; limited generalisability to the general population	Direct human cardiovascular evidence
9	Khadke et al., 2024	Ecological cross-sectional study using census-tract data	JBIChecklist for Cross-Sectional Studies Checklist, adapted for ecological data	Appraisal completed; no numerical score presented	Ecological design and potential ecological fallacy; inability to establish individual exposure or temporality; residual spatial and socioeconomic confounding; reliance on aggregated data	Population-level evidence concerning environmental injustice and cardiovascular health
18	Rajagopalan and Brook, 2012	Mechanistic narrative review	SANRA	Appraisal completed; numerical score not presented	Narrative evidence selection; heterogeneous experimental and observational evidence; unable to provide a pooled quantitative estimate	Indirect mechanistic evidence concerning air-pollution-related metabolic effects
19	Münzel et al., 2020	Narrative review	SANRA	Appraisal completed; numerical score not presented	No fully reproducible systematic search or formal study-level risk-of-bias assessment; heterogeneity in noise metrics, exposure windows, and cardiovascular outcomes	Mechanistic and public-health context for night-time transportation noise
20	Twohig-Bennett and Jones, 2018	Systematic review and meta-analysis	JBIChecklist for Systematic Reviews and Research Syntheses	Appraisal completed; no numerical score presented	Heterogeneous definitions and measures of greenspace; predominance of observational studies; residual confounding; possible publication bias	Synthesised evidence concerning greenspace and health
21	Sallis et al., 2016	Multicountry cross-sectional study	JBIChecklist for Cross-Sectional Studies Checklist	Appraisal completed; no numerical score presented	Cross-sectional design; residual confounding; differences in environmental measures across countries; physical activity rather than CVD was the principal outcome	Indirect population evidence on urban form and physical activity

Ref.	Source	Design/source type	Appraisal approach	Appraisal result*	Principal methodological limitation(s)*	Interpretive role
22	Guo et al., 2019	Narrative/comprehensive review	SANRA	Appraisal completed; numerical score not presented	Broad but non-systematic evidence selection; heterogeneous exposure definitions, populations, and outcomes; variable quality of included evidence	Exposure and toxicological context for persistent organic pollutants
23	Lind and Lind, 2020	Narrative review	SANRA	Appraisal completed; numerical score not presented	Predominantly observational evidence; heterogeneous chemical mixtures and exposure measurements; possible residual confounding	Cardiovascular context for persistent organic pollutants
24	La Merrill et al., 2016	Experimental animal study	SYRCLE Risk-of-Bias Tool	Appraisal completed; several domains unclear because of incomplete reporting	Randomisation, allocation concealment, and blinded outcome assessment were not fully reported; single animal model; limited direct generalisability to humans	Direct experimental evidence concerning DDT-related cardiovascular effects
25	Jokinen et al., 2003	Experimental animal study	SYRCLE Risk-of-Bias Tool	Appraisal completed; several domains unclear because of incomplete reporting	Incomplete reporting of randomisation, allocation concealment, and blinding; species-specific responses; uncertain comparability with chronic human exposure	Direct experimental cardiovascular evidence concerning dioxin/PCB exposure
26	Arzuaga et al., 2007	In-vitro mechanistic study	ToxRTool for in-vitro studies	Appraisal completed; numerical score not presented	Cell-based model does not represent whole-body physiology; selected concentrations may not reflect population exposure; short-term cellular outcomes; limited clinical generalisability	Direct cellular mechanistic evidence concerning PCB-induced endothelial activation
27	Banerjee et al., 2022	Narrative/comprehensive review	SANRA	Appraisal completed; numerical score not presented	No fully reproducible systematic search; heterogeneous experimental and epidemiological evidence; limited prospective human evidence	Indirect mechanistic evidence concerning BPA
28	Zhang et al., 2020	Narrative toxicological review	SANRA	Appraisal completed; numerical score not presented	Evidence derived mainly from experimental and cross-sectional studies; heterogeneous doses and outcome measures; limited long-term human evidence, particularly for BPS	Mechanistic and toxicological context for BPA and BPS

Ref.	Source	Design/source type	Appraisal approach	Appraisal result*	Principal methodological limitation(s)*	Interpretive role
29	Su et al., 2019	Analytical cross-sectional study	JBIC Analytical Cross-Sectional Studies Checklist	Appraisal completed; no numerical score presented	Cross-sectional design; possible exposure misclassification; residual confounding; reverse causation cannot be excluded; a single measurement may not represent long-term exposure	Direct human vascular evidence concerning phthalate exposure
30	Minassa et al., 2022	Experimental animal study	SYRCLE Risk-of-Bias Tool	Appraisal completed; several domains unclear because of incomplete reporting	Randomisation and blinding were not fully reported; experimental dose and duration may not represent usual human exposure; interspecies limitations	Direct experimental evidence concerning chlorpyrifos-related cardiovascular effects

***Note.** Appraisal findings informed the interpretation and evidential role of each source and were not used as automatic exclusion criteria. Numerical scores are not presented because the appraisal approaches assess different methodological domains and do not provide directly comparable metrics across study designs. For health-burden and exposure-modelling studies, GATHER reporting criteria were supplemented by consideration of input-data quality, exposure modelling, model assumptions, uncertainty, and external validity. Domains judged as unclear primarily reflected insufficient methodological reporting rather than confirmed methodological failure.

Abbreviations: BPA, bisphenol A; BPS, bisphenol S; CVD, cardiovascular disease; DDT, dichlorodiphenyltrichloroethane; GATHER, Guidelines for Accurate and Transparent Health Estimates Reporting; JBI, Joanna Briggs Institute; PCB, polychlorinated biphenyl; SANRA, Scale for the Assessment of Narrative Review Articles; SYRCLE, Systematic Review Centre for Laboratory Animal Experimentation.