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TH Scientific Statement

Air, Noise, and Light Pollution and Thromboembolic Cardiovascular Complications: A TH Scientific Statement

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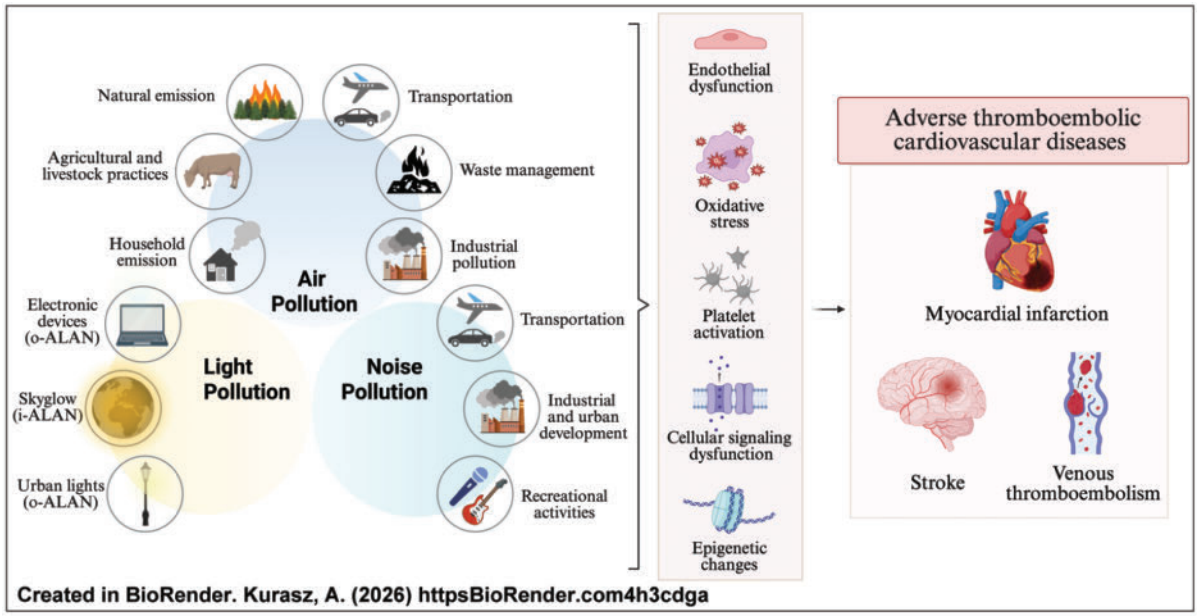
GRAPHICAL ABSTRACT



ABSTRACT

Thromboembolic cardiovascular diseases (CVD), including acute coronary syndromes, ischemic stroke, and venous thromboembolism, remain a leading cause of morbidity and mortality worldwide. Despite significant improvements in prevention and diagnosis, thromboembolic CVD remains a major global health challenge, reflecting the incomplete control of multifactorial vascular risk. Growing evidence indicates that air, noise, and light pollution are important yet under-recognized contributors to cardiovascular morbidity. Exposure to particulate matter ($PM_{2.5}$, PM_{10}), gaseous pollutants (NO_2, SO_2, CO, O_3), chronic noise, and artificial light at night promotes endothelial dysfunction, oxidative stress, inflammation, and platelet activation—key mechanisms fostering a prothrombotic setting. Although regulatory progress has been achieved, air pollution remains the most significant environmental determinant of cardiovascular health globally, and the combined effects of coexisting pollutants are not fully understood. The convergence of urbanization, industrialization, and increasing light exposure further amplifies environmental impacts on vascular health. This scientific statement aims to synthesize current epidemiological and mechanistic evidence, highlight the complex interactions among air, noise, and light pollution, identify critical research gaps, and provide a comprehensive conceptual framework for understanding how environmental stress contributes to thromboembolic cardiovascular complications. Strengthening multidisciplinary research, integrating exposome-based data, and implementing effective prevention policies are essential steps toward mitigating the cardiovascular burden of environmental pollution.

Keywords air pollution, noise, light, acute coronary syndrome, thromboembolic events, stroke

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Introduction

Thromboembolic cardiovascular diseases (CVD), encompassing acute coronary syndromes (ACS), ischemic stroke, and venous thromboembolism (VTE), represent a major global health burden, contributing to substantial morbidity, mortality, and healthcare costs.[1] Despite advances in prevention and treatment, their prevalence continues to rise, driven by aging populations, increasing cardiovascular risk factors, and persistent disparities in healthcare access.[2]

The pathophysiology of thromboembolic events involves a complex interplay between endothelial dysfunction, inflammation, oxidative stress, platelet activation, and coagulation abnormalities—processes that are increasingly recognized as being influenced by environmental factors. Among these, air pollution, noise, and light pollution have emerged as important, yet often underrecognized, contributors to CVD.[3]

Exposure to particulate matter (PM), gaseous pollutants, chronic noise stressors, and artificial light at night (ALAN)—the so-called environmental exposome—has been associated with vascular dysfunction, autonomic imbalance, and increased thrombogenicity.[4] With ongoing urbanization, industrialization, and climate change, the global exposure to environmental pollutants continues to intensify, necessitating a more precise evaluation of their prothrombotic mechanisms and clinical sequelae. Although the World Health Organization (WHO) has established environmental pollution guidelines, compliance remains variable across regions, and disparities in exposure are substantial. Indeed, epidemiological evidence increasingly supports associations between environmental pollution and thromboembolic outcomes, including ACS, stroke, and VTE—particularly in vulnerable populations.[5,6] However, the underlying mechanisms remain incompletely understood, and geographic heterogeneity in exposure complicates risk assessment.

To address these gaps, an exposome-based approach integrating high-resolution environmental and cardiovascular data is needed to clarify the causal pathways linking pollution and thrombosis. Developing real-time monitoring systems, precision exposure assessment tools, and targeted public health interventions will be essential to mitigate the cardiovascular burden of environmental pollution.

This scientific statement aims to synthesize current epidemiological and mechanistic evidence, highlight research gaps with future directions, and provide a comprehensive framework for understanding how air, noise, and light pollution contribute to thromboembolic cardiovascular complications.

The Global Burden of Thromboembolic Cardiovascular Complications and Environmental Pollution

Ischemic heart disease (IHD) remains the leading cause of death among CVDs, accounting for approximately 193 million disability-adjusted life years (DALYs) worldwide in 2023.[7] Within the IHD spectrum, ACS constitutes the predominant cause of mortality and represents a major global health burden, particularly in high-income countries where they account for a substantial proportion of cardiovascular deaths.[8] However, disparities in revascularization rates, secondary prevention, and long-term outcomes persist, with low- and middle-income countries bearing a disproportionate burden driven by increasing prevalence of cardiovascular risk factors and limited access to timely diagnosis and evidence-based management.[9]

Ischemic stroke similarly represents a major global health issue, with an 88% increase in incidence, a 55% increase in mortality, and a 52.4% rise in DALYs over the past three decades. Projections suggest a further increase by 2035, particularly among individuals over 45 years of age. Global rates are shaped by age, period, and cohort effects, showing higher incidence in older populations and declining trends in regions with a higher Socio-Demographic Index. Key modifiable risk factors include hypertension, elevated LDL cholesterol, and environmental influences (e.g., air pollution).[10]

Thrombosis contributes to approximately one in four deaths worldwide and remains a central pathophysiological process underlying myocardial infarction (MI), ischemic stroke, and VTE, including deep vein thrombosis (DVT) and pulmonary embolism (PE). While mortality from MI and stroke has declined in high-income countries over the past two decades due to advances in prevention and treatment, VTE-related mortality has remained largely unchanged. However, the COVID-19 pandemic temporarily reversed these favorable trends, with recent data indicating increased age-standardized mortality rates for MI, stroke, and VTE in the past 5 years.[2]

These observations underscore the urgent need for continued global efforts to reduce the impact of thromboembolic disease through equitable access to prevention, early diagnosis, and evidence-based treatment, with particular attention to low- and middle-income countries where the rising prevalence of cardiovascular risk factors and environmental exposures amplifies these effects.

Trends and Global Burden of Air, Noise, and Light Pollution

Environmental pollution is increasingly recognized as a significant contributor to the global burden of disease. Among nonclassical risk factors, air pollution remains the most extensively studied and well-established determinant of CVD.

In 2016, 90% of the global population lived in areas exceeding the WHO air quality guidelines for PM with aerodynamic diameter ≤ 2.5 µm ($PM_{2.5}$). Although this represents a 4.2 percentage point decline compared to 2010, the reduction was largely confined to North America and Europe, while exposure levels in other regions remained critically high.[11] However, looking at the newest data from the State of Global Air 2024 Report, 99% of the global population is exposed to $PM_{2.5}$ levels exceeding the WHO guideline limits.[12] Air pollution is responsible for over 8.3 million deaths annually, with 20% of CVD-related mortality attributed to its influence—surpassing established risk factors such as high LDL cholesterol, chronic kidney disease, and excessive BMI.[13,14]

Noise pollution also poses a significant public health challenge. According to the European Environment Agency, excessive noise from transport contributes to approximately 66,000 premature deaths annually and accounts for over one million healthy life years lost in Europe.[15] While exposure to road traffic noise is expected to remain stable, railway noise is projected to increase by 30%, whereas exposure from air traffic is anticipated to decline.[16] Notably, the European Environment Agency noise report indicates that over 20% of the European Union population resides in environments exceeding recommended noise levels, and this percentage is as high as 50% in urban populations.[15]

Light pollution, though less studied, is emerging as an important environmental health risk. For example, it has been reported that 80% of the global population and 99% of residents in the United States and Europe live under a light-polluted sky, with urban brightness increasing by 10% annually.[17,18] Growing evidence suggests detrimental health effects, including increased hospitalization rates and mortality linked to light exposure, including an association with coronary artery disease.[19] Given the vast number of individuals affected by these environmental factors, their impact on public health warrants urgent attention and further investigation.

Data on the proportion of the population exposed to the adverse effects of air, light, and noise pollution are presented in **Fig. 1**.

Current WHO Standards for Air, Noise, and Light Pollution

Environmental pollution standards are established by expert panels and compiled within the framework of the WHO and other United Nations health and environment guidelines, with updates across various domains between 2021 and 2024. Current recommendations address air quality and environmental noise exposure; however, there are still no established international standards for light pollution. The most recent WHO Air Quality Guidelines

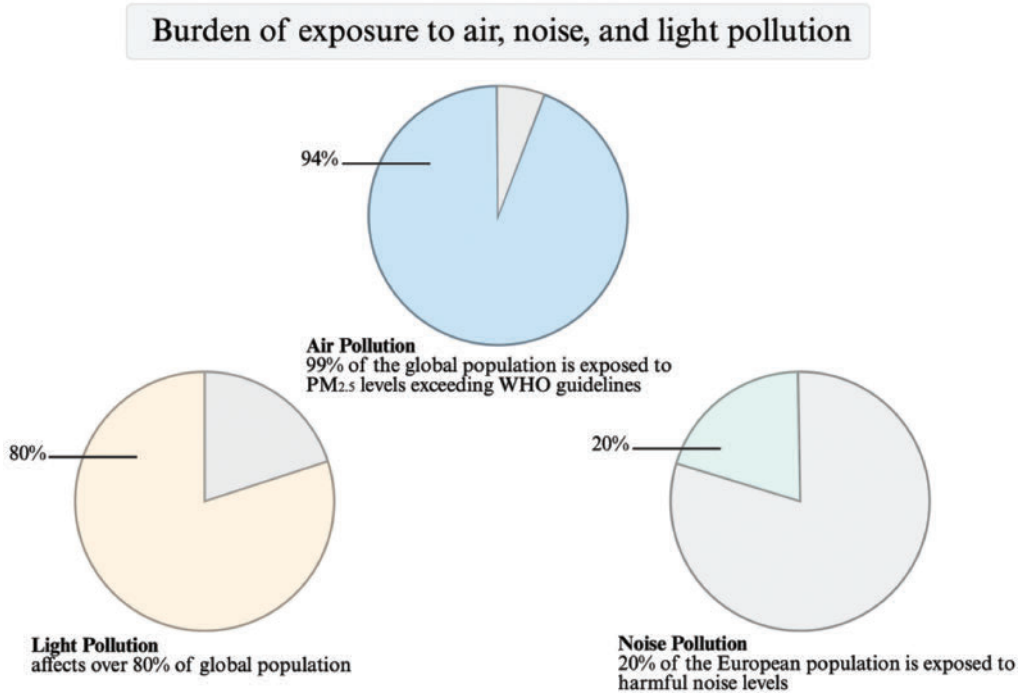

Fig. 1 Data on the proportion of the population exposed to the adverse effects of air, light, and noise pollution. Description: data for air pollution exposure from the "State of Global Air 2024" Report[12]; noise pollution from "Environmental noise in Europe 2025" report[15]; light pollution from "The new world atlas of artificial night sky brightness."[18] (Created in BioRender. Kurasz A. [2026] https://BioRender.com/4y4yqgb.)

Table 1 Recommended AQG levels

Pollutant	Averaging time	AQG level
$PM_{2.5}$, μg/m^3	Annual	5
	24-h[a]	15
PM_{10}, μg/m^3	Annual	15
	24-h[a]	45
O_3, μg/m^3	Peak season[b]	60
	8-h[a]	100
NO_2, μg/m^3	Annual	10
	24-h[a]	25
	1-h	200
SO_2, μg/m^3	24-h	40
	8-h	500
CO, mg/m^3	24-h[a]	4
	8-h	10
	1-h	35
	15-min	100

Abbreviations: CO, carbon monoxide; NO_2, nitrogen dioxide; O_3, ozone; $PM_{2.5}$, particulate matter with aerodynamic diameter $\leq$ 2.5 μm; PM_{10}, particulate matter with aerodynamic diameter $\leq$ 10 μm; SO_2, sulfur dioxide.
[a]Ninety-ninth percentile (i.e., 3–4 exceedance days per year).
[b]Average of daily maximum 8-hour mean O_3 concentration in the 6 consecutive months with the highest 6-month running average O_3 concentration.

(AQG), published in 2021, define target thresholds for six key pollutants: $PM_{2.5}$, PM with aerodynamic diameter $\leq$ 10 μm (PM_{10}), ozone (O_3), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), and carbon monoxide (CO). The proposed guideline values are summarized in **Table 1**.[20] Compared with the 2005 guidelines, the 2021 WHO AQG introduced more stringent standards for $PM_{2.5}$, PM_{10}, and NO_2, while the recommended limits for SO_2 were revised upward.

The environmental noise guidelines proposed in 2018, based on systematic reviews, are divided into average and night-time exposure.[21]

For average noise exposure, the following sound pressure levels are recommended:

- <53 dB for road traffic noise,
- <54 dB for railway noise,
- <45 dB for aircraft noise,
- <45 dB for wind turbine noise,
- Yearly average from all leisure source noises combined to $\leq$ 70 dB,
- Weekly average from leisure sources (such as personal listening devices) $\leq$ 80 dB or 1.6 Pa2h,
- Short-term average from occasional exposure to leisure source noise $\leq$ 100 dB.

For night noise exposure, the following sound pressure levels are recommended:

- <45 dB for road traffic noise,
- <44 dB for railway noise,
- <40 dB for aircraft noise.

Geographic Variability in Air, Light, and Noise Pollution

Exposure to environmental factors varies geographically and is determined by a complex interplay of urbanization, transportation patterns, industrial activity, socioeconomic conditions, and climatic factors. Air pollution represents a heterogeneous mixture of gaseous and particulate components, the proportions of which differ across regions. Distinct smog types have been identified, including the London, Los Angeles, and Polish smogs, each characterized by unique chemical compositions and formation mechanisms.[22–24]

London smog (classical smog) is composed primarily of SO_2, soot with PM, and carbon particulates. It typically develops under conditions of low atmospheric pressure, temperature inversion, and foggy, windless weather.[22] In contrast, Los Angeles smog (photochemical smog) arises mainly from NO_X, CO, CO_2, volatile hydrocarbons, and O_3, forming through photochemical reactions in strong sunlight and at air temperatures above 28 to 30°C.[23]

More recently, a distinct regional variant known as Polish smog has been described, which is characterized by elevated concentrations of PM and PAHs (benzo(a)pyrene), along with comparatively lower levels of SO_2 and NO_2. It originates primarily from low emissions associated with household heating using solid fuels such as coal, wood, and, in some cases, waste, and typically occurs under conditions of high atmospheric pressure and sub-zero temperatures.[24,25] In addition to anthropogenic sources, natural phenomena such as Saharan dust transport significantly influence air quality across Europe. Periodic intrusions of mineral dust from North Africa markedly deteriorate air quality by substantially increasing ambient concentrations of PM_{10} and $PM_{2.5}$.

Light and noise pollution intensities vary substantially between urban and rural environments, with both pollutants being more pronounced in urban areas. Light pollution, resulting from inappropriate or excessive artificial lighting, can be categorized according to its effects into light trespass, skyglow, glare, light clutter, and over-illumination. While light trespass may occur in both urban and rural settings, the remaining forms predominantly affect urbanized regions.[26] Noise pollution is likewise a growing concern in urban environments, particularly in areas affected by inadequate urban planning. Major sources include road traffic, railways, and aircraft operations, whereas in rural areas, wind turbines may represent a relevant source of noise exposure.[27] Ongoing urbanization and expanding transportation infrastructure will further increase population exposure to both light and noise pollution, amplifying their impact on public and cardiovascular health.

Potential Mechanisms of the Detrimental Impact of Air, Light, and Noise Pollution on Human Health in the Context of Thromboembolic Events

Among environmental factors, air pollution exerts the most direct and well-established influence on cardiovascular and thromboembolic risk. Air pollution inhalation triggers a cascade of local and systemic inflammatory responses.[28] The initial pulmonary

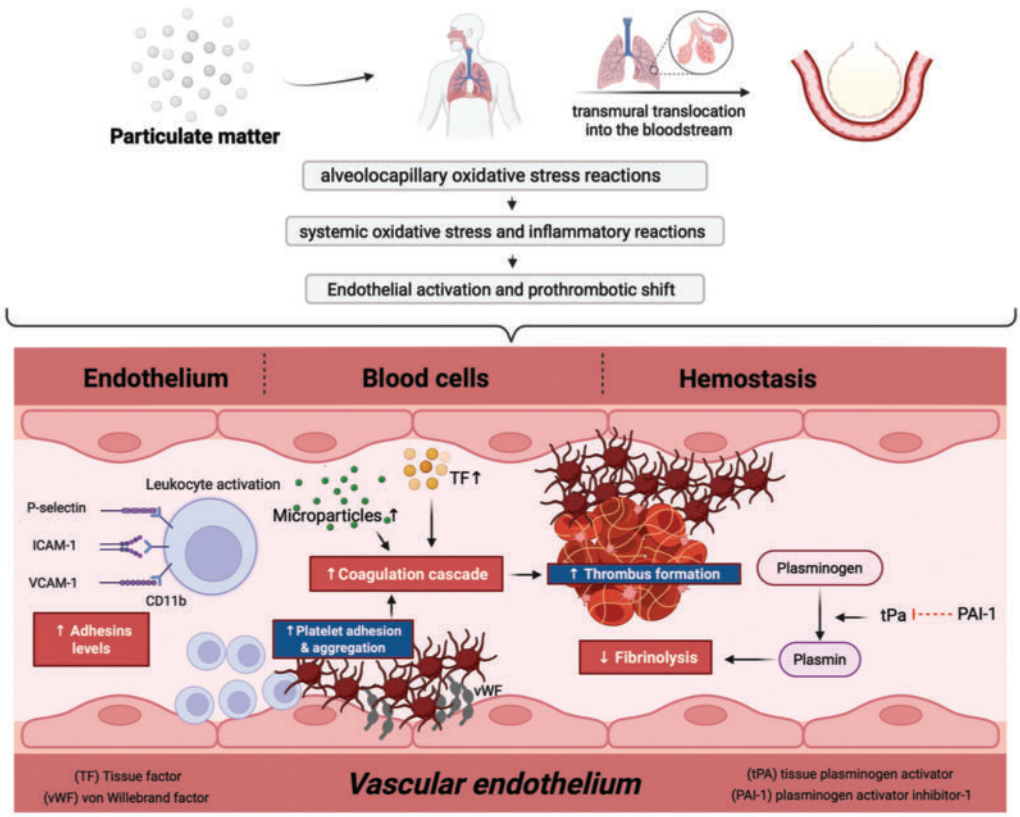

Fig. 2 Thrombotic pathways associated with environmental exposures. (Created in BioRender. Kurasz A. [2026] https://BioRender.com/55vjgwc.)

oxidative stress activates alveolar macrophages, leading to the release of pro-inflammatory mediators, including interleukin-6 and tumor necrosis factor-alpha. These cytokines promote endothelial activation, increase vascular permeability, and stimulate bone marrow activity, resulting in elevated circulating leukocytes and platelets.[29,30] This pro-inflammatory state promotes endothelial dysfunction and alters nitric oxide bioavailability, impairing vasodilation and predisposing to vasoconstriction. Concurrently, PM may penetrate the alveolar–capillary barrier and enter systemic circulation, directly interacting with endothelial cells and platelets to enhance adhesion molecule expression, coagulation factor activation, and thrombus formation (**Fig. 2**).[3,31] Chronic exposure further contributes to atherosclerotic plaque formation and vascular remodeling, while acute exposure episodes have been associated with plaque rupture and the onset of ACS and ischemic stroke.[32,33] Controlled human exposure studies by Newby et al demonstrated that short-term inhalation of dilute diesel exhaust acutely promotes myocardial ischemia and impairs endothelial fibrinolytic capacity, both in patients with coronary heart disease and in healthy individuals.[34,35] In the latter group, vascular dysfunction was also induced.[34] In addition, diesel exhaust exposure rapidly increases platelet activation and ex vivo thrombus formation within hours, providing direct human evidence for fast activation of atherothrombotic pathways linking traffic-related air pollution to acute MI.[36]

Noise exposure contributes to cardiovascular risk through stress-related and autonomic pathways. Chronic noise and related annoyance activate the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis, elevating catecholamine and cortisol levels.[37] These can promote atherosclerotic oxidative stress, impair endothelial nitric oxide production, and raise blood pressure.[38] Recurrent sleep disruption caused by nocturnal noise further amplifies these effects, leading to vascular inflammation, endothelial dysfunction, and a prothrombotic state.[39]

Light pollution, particularly exposure to ALAN, exerts its detrimental cardiovascular effects primarily through disruption of the circadian rhythm and suppression of melatonin secretion. The circadian system regulates numerous physiological processes, including heart rate, vascular tone, blood pressure, and the expression of genes involved in coagulation and inflammation. Disruption of this rhythm alters the timing of hormonal and autonomic activity, resulting in dysregulation of endothelial function.[40,41]

Although air, noise, and light pollution arise from different sources and act through distinct biological pathways, their combined and chronic exposure exerts additive or even synergistic effects on cardiovascular health (Graphical abstract). Individuals living in densely urbanized environments are often simultaneously exposed to multiple environmental stressors, amplifying vascular and autonomic dysregulation. Recent studies have shown that both air pollution and noise contribute substantially to cumulative risk indices for MI and stroke.[42,43] These overlapping exposures accelerate atherosclerosis, increase arterial stiffness, and augment blood coagulability—core processes underlying thromboembolic cardiovascular events.[44,45] Worth mentioning are also factors such as mental health, socioeconomic status, and level of

greenery, which can modify the effect of air pollution on human health.[46]

Current State-of-the-Art Research Regarding the Association Between Exposure to Air, Noise, and Light Pollution and Specific Thromboembolic Cardiovascular Events

Air, Noise, and Light Pollution and ACS

Air pollution, particularly $PM_{2.5}$, NO_2, and ozone, has the strongest evidence base linking it to ACS. Short-term spikes in $PM_{2.5}$ have been associated with increased hospitalizations for MI and out-of-hospital cardiac arrests.[47,48] Both case-crossover and large prospective cohorts show a stepwise rise in MI risk by 12 to 18%, with every 10 $\mu g/m^3$ increment in short-term $PM_{2.5}$, with effect estimates strongest within the first 24 h. Importantly, studies indicate that MI onset can occur within hours of exposure, with elevated fine-particle concentrations in the preceding 2-hour period and traffic exposure associated with MI onset within the subsequent hour, supporting the fast activation of atherothrombotic pathways.[49,50] Moreover, Liu et al observed that each 10-$\mu g/m^3$ increase in exposure to $PM_{2.5}$, PM_{10}, and NO_2 was significantly associated with a 4.14, 2.67, and 1.46% increase in odds of MI mortality, respectively.[51] Both short- and long-term air pollution exposure were associated with a higher incidence of ST-elevation MI.[52]

Noise pollution similarly been associated with cardiovascular morbidity. Chronic noise exposure contributes to annoyance and disrupts sleep, triggers sustained sympathetic nervous system activation, and elevates stress hormone levels.[38] These disturbances have been shown to provoke vascular inflammation, hypertension, and arrhythmias—conditions known to precipitate ACS.[39,53] Long-term road-traffic noise above 56dB independently raises MI incidence after adjustment for co-pollutants.[54] Experimental data link nocturnal noise exposure to surges in catecholamines, endothelial dysfunction, and blood-pressure spikes—creating a pro-ischemic milieu that persists into waking hours.[38]

Studies have shown associations between higher levels of nighttime light exposure and increased risk of ACS, particularly in urban settings.[41] Exposure to outdoor ALAN in the highest quartile is associated with 10 to 15% higher odds of first MI, even after accounting for greenness and air quality.[55] In a nationwide Chinese case-crossover study, authors observed that every 100 $nW/cm^2/sr$ increase in daily LAN was associated with increased mortality related to acute MI.[56] Laboratory studies demonstrate that indoor ALAN during sleep blunts vagal tone and elevates nocturnal heart-rate variability—a recognized harbinger of coronary events.[57,58]

The cardiovascular burden of environmental pollution is not uniformly shared; instead, it concentrates in populations where biological frailty and social inequity intersect. The aging vasculature accumulates oxidative damage and loses elastic reserve, so older adults mount larger blood pressure surges and redox imbalances when exposed to PM, translating into disproportionate spikes in MI admissions per 10 $\mu g/m^3$ rise in $PM_{2.5}$ compared with middle-aged cohorts.[59]

In people with cardio-metabolic comorbidity—hypertension, diabetes, or established atherosclerosis—the endothelium is already primed for thrombosis; transient, pollution-driven increments in inflammation or autonomic tone can therefore precipitate plaque rupture after modest additional insult.[60] The risk gradient steepens further in socio-economically disadvantaged or racially segregated communities, whose residents live closer to high-traffic corridors or industrial sources and face barriers to preventive care, creating a double jeopardy of higher dose and higher susceptibility.[61,62]

Sex differences also modify vulnerability: recent epidemiologic analyses reveal that the same $PM_{2.5}$ increment confers greater odds of obstructive coronary disease and MI in women, particularly around the menopausal transition when hormonal buffering wanes.[63] Finally, night-shift workers and outdoor laborers endure a syndicate of hazards—ALAN, circadian misalignment, and noise exposure—that up-regulate sympathetic tone, blunt insulin sensitivity, and destabilize plaques, cumulatively elevating ACS risk.[64] Mapping and mitigating these intersecting susceptibilities is pivotal for precision prevention, guiding multi-pollutant regulation, clinical risk stratification, and environmental-justice policy aimed at reducing pollution-triggered ACS worldwide.

A summary of particularly important studies regarding the association between exposure to air, noise, and light pollution and ACS can be found in **Table 2**.

Air, Noise, and Light Pollution and Ischemic Stroke

In 2022, stroke was the second leading cause of death worldwide, with projections suggesting that it will maintain this rank at least until 2050.[65] While the detrimental effect of air pollution on ischemic strokes is well established, the harmful impact of light and noise pollution remains debatable, with the WHO categorizing the quality of evidence as "low."[66]

Recent studies analyzing large cohorts provide increasing evidence of the harmful effects of air pollution exposure, both in the short- and long-term. One Chinese study linked hourly air pollution exposure to a higher total and ischemic stroke, with the strongest effects observed during the first 2 hours of exposure.[67] The EP-PARTICLES study demonstrated the harmful short-term effects of air pollution exposure on not only stroke incidence,[6,68,69] but also the incidence of atrial fibrillation (a well-known risk factor for stroke), even in areas with relatively low pollution levels.[70] Similar associations between air pollution exposure and atrial fibrillation have been reported across multiple populations, supporting the robustness of this relationship.[71–73] Moreover, in patients already diagnosed with atrial fibrillation, exposure to PM was associated with an increased risk of ischemic stroke.[74,75]

The long-term harmful effects of air pollution exposure have also been documented in recent literature, including studies on the population of Catalonia, where residents of areas with higher concentrations of PMs, black carbon, and NO_2 had an increased risk of ischemic stroke.[76] UK Biobank prospective analysis with nearly 14 years of follow-up provided further evidence of the detrimental impact of prolonged exposure to PMs and nitrogen oxides on ischemic stroke incidence.[77]

Table 2 A summary of important studies regarding the association between exposure to air, light, and noise pollution and acute coronary syndromes

Authors	Year of publication	Type of pollution	Country	Study population	Main conclusions
Zou et al[48]	2021	Air	Meta-analysis of 27 cohort studies. China	6,764,958	Long-term exposure to $PM_{2.5}$ and PM_{10} is a MI risk factor
Jiang et al[31]	2024	Air	China	2,110,000	Exposure to $PM_{2.5}$ is an ACS risk factor
Kuźma et al[5]	2024	Air	Poland	8,000,000	AP exposure increases the risk of NSTEMI and STEMI, especially in vulnerable groups.
Chen et al[121]	2022	Air	China	1,292,880	Exposure to $PM_{2.5}$, NO_2, SO_2, CO, increases risk of ACS
Dąbrowiecki et al[122]	2025	Air	Poland	88,467	Exposure to AP is associated with a higher risk of ACS
Cha et al[52]	2024	Air	South Korea	45,619	Higher PM_{10} levels are associated with higher STEMI risk
Liu et al[51]	2021	Air	China	151,608	Exposure to $PM_{2.5}$, PM_{10}, and NO_2 is associated with higher MI mortality
Ishii et al[123]	2020	Air	Japan	137,678	Exposure to $PM_{2.5}$ is associated with a higher risk of MI, and results showed seasonal differences in attributable risk.
Poulsen et al[124]	2023	Air	Denmark	1,964,702	AP exposure is associated with higher MI risk, especially in some vulnerable groups
Olanyian et al[125]	2022	Air	Canada	2,700,000	AP exposure is associated with higher MI risk
Héritier et al[126]	2019	Noise	Switzerland	Nationwide	Exposure to road, railway, and aircraft noise was associated with higher MI mortality
Lim et al[54]	2021	Noise	Denmark	22,378	Exposure to high road traffic noise is associated with higher MI risk
Vienneau et al[80]	2022	Noise	Switzerland	Nationwide	Exposure to road, railway, and aircraft noise was associated with higher MI mortality
Sun et al[19]	2021	Light	Hong Kong Special Administrative Region of the People's Republic of China	58,692	Light pollution exposure was associated with a higher risk of CAD hospitalizations and deaths

The Global Burden of Disease study analyses indicate that elderly people, as a group, are more vulnerable to environmental pollution, primarily air pollution.[78] However, available studies present conflicting results on that matter. A recent study originating from Asia presented that air pollution-related risk of stroke was more pronounced in men and nonelderly people.[67] A large European cohort study identified nonelderly females as the most sensitive phenotype for air pollution exposure and stroke risk, while also highlighting an increased risk among residents with harmful lifestyle habits.[6] Similar findings were demonstrated by researchers from Singapore, who also pointed to unfavorable lifestyle habits and highlighted patients with atrial fibrillation.[79] One UK Biobank study revealed an increased risk of air pollution-related ischemic stroke among individuals with elevated genetic susceptibility.[77]

As mentioned above, the quality of evidence regarding the associations between exposure to noise pollution is relatively low. One nationwide cohort study revealed a significant stroke mortality increase associated with an increment in exposure to road traffic and railway traffic noise, respectively.[80] Moreover, two meta-analyses showed an association between long-term exposure to road traffic noise and risk of stroke.[81,82] Although the aforementioned pathogenetic mechanisms of excessive light pollution exposure suggest its adverse impact on the cerebrovascular system, evidence remains scarce. A Chinese study was the first to demonstrate a positive correlation between O-ALAN exposure and cerebrovascular diseases, including ischemic stroke.[83] More recently, a large population-based analysis from eastern Poland reported that light pollution was associated with a 7% higher odds of hospitalization for acute ischemic stroke (OR: 1.07; 95% CI: 1.05–1.10), even after adjustment for air pollution, socioeconomic factors, and meteorological variables.[84] On the other hand, there remains a significant gap in evidence regarding the vulnerability of specific groups to light and noise pollution, as studies to date have not addressed this crucial issue.

A summary of particularly important studies regarding the association between exposure to air, noise, and light pollution and ischemic stroke can be found in **Table 3**.

Air, Noise, and Light Pollution and VTE

While there is a substantial body of evidence to support a link between ambient air pollution and CVD, less consistent is the clinical evidence of the association with VTE, including DVT and PE, potentially due in part to the fact that venous thrombotic

Table 3 A summary of particularly important studies regarding the association between exposure to air, light, and noise pollution and ischemic stroke

Authors	Year of publication	Type of pollution	Country	Study population	Main conclusions
Lv et al[67]	2023	Air	China	86,635	Exposure to specific air pollutants is associated with a greater incidence of stroke, including ischemic stroke
Święczkowski et al[6]	2024	Air	Poland	146,262	Exposure to air pollution, even at low levels, might increase the ischemic stroke incidence, particularly among nonelderly women, with harmful lifestyle habits potentially exacerbating its effects
Avellaneda-Gómez et al[76]	2022	Air	Spain	10,865	Long-term exposure to air pollution, especially NO_2, was linked to higher ischemic stroke incidence
Li et al[77]	2024	Air	United Kingdom	307,304	Prolonged exposure to air pollutants may increase ischemic stroke risk, especially in genetically susceptible individuals
Vienneau et al[80]	2022	Noise	Swiss Confederation	277,506	Railway and road traffic noise exposure is linked to increased ischemic stroke mortality, even below guideline limits
Fu et al[82]	2023	Noise	N/D	Meta-analysis	Long-term exposure to traffic noise, particularly from road traffic, is associated with an increased risk of CVDs, including ischemic stroke
Hao et al[81]	2022	Noise	N/D	Meta-analysis	This study reinforces the link between road traffic noise and higher risks of stroke, particularly in males
Wu et al[83]	2024	Light	China	1,278	Exposure to outdoor LAN was associated with an increased risk of cerebrovascular disease incidence, including ischemic stroke incidence
Ho et al[79]	2022	Air	Singapore	51,675	Short-term exposure to air pollution might increase the incidence of ischemic stroke, especially among AF patients and in those who are current or ex-smokers
Hasegawa et al[127]	2022	Air	Japan	335,248	Short-term air pollution exposure might increase ischemic stroke hospitalizations, with effects influenced by medication use and season
Yang et al[128]	2024	Air	China	67,150	Long-term exposure to PMs was associated with increased incidence of both types of strokes, with almost 2 times higher effect on hemorrhagic stroke
Stafoggia et al[129]	2022	Air	Italy	2,154,810	PM exposure was correlated with daily peaks in CVD admissions, including ischemic stroke, in both urban and rural areas
Jemielita et al[84]	2025	Light	Poland	483,688	An increase by one interquartile range (2,479.42 $nW/cm^2/sr$) in light pollution was linked to a 7% rise in odds of hospitalization for acute ischemic stroke
Święczkowski et al[68]	2025	Air	Poland	96,189	Short-term exposure to air pollution may be associated with intravenous thrombolysis-treated ischemic stroke, with a comparable effect across different sexes and age groups

events are less frequent than arterial ones, and thus are more difficult to study.[85,86]

Recent clinical studies evaluating the effects of short-term and long-term PM exposure have added important advances in our knowledge on this issue.[85] While a recent case-crossover study from China reviewed 18,616 cases of VTE and found a small but significant association with daily 10 mg/m^3 increments in both $PM_{2.5}$ and PM_{10} levels (OR: 1.039 [95% CI: 1.011–1.068] and OR: 1.011 [95% CI: 1.003–1.019], respectively).[87] Another large study of 219,952 PE and 275,506 DVT hospitalizations in Italy was unable to demonstrate an association between daily air pollutant exposure and VTE.[88] Likewise, in a long-term exposure study conducted in Korea recording 3,196 VTE events among 338,616 subjects followed for a 12-year period, the VTE risk was significantly associated with each 1 mg/m^3 increment in PM_{10}, with a HR of 1.064 (95% CI: 1.053–1.074).[89]

Conversely, no association was identified between VTE and proximity to a major road in the Atherosclerosis Risk in Communities Study (ARIC) study.[90] All in all, the literature data available, although limited by a wide inter-study heterogeneity, indicate a positive association between ambient air pollution and VTE risk, although it appears to be smaller than that observed for arterial thrombosis. This clinical observation is biologically plausible considering the predominant effect of air pollution on low-grade, long-term inflammation and the atherosclerotic process, which is the basis of arterial thrombotic events.[30,91,92] This smaller effect

Table 4 A summary of particularly important studies regarding the association between exposure to air, light, and noise pollution and venous thromboembolism

Authors	Year of publication	Type of pollution	Country	Study population	Main conclusion
Zhang et al[87]	2024	Air	China	18,616	Short-term exposure to dust PMs, especially $PM_{2.5}$, might increase the risk of pulmonary embolism, with a greater impact in the warm season
Di Blasi et al[88]	2022	Air	Italy	495,458	There was no association between exposure to air pollution and the incidence of pulmonary embolism and venous thrombosis
Gwon et al[89]	2022	Air	South Korea	3,196	Air pollution was identified as an independent risk factor for venous thromboembolism
Kan et al[90]	2011	Noise	USA	405	No association between exposure to noise pollution and venous thromboembolism
Hahad et al[93]	2024	Noise	Germany	14,639	Noise pollution was linked to the prevalence of CVDs, including venous thromboembolism, while its association with newly diagnosed cases was weak

on VTE is, however, globally relevant and deserves actions aimed at mitigating its burden on human health.

While the thrombotic risk associated with pollution from PM and other sources represents a major threat to the health of the world's population, this association is particularly evident for those individuals particularly fragile, such as, for instance, elderly people and cancer patients who are already at increased risk of VTE.[85] These populations may have an even greater risk in the presence of increased air pollution.

Scant data are available on the association of noise and light pollution with VTE. In a recent 10-year follow-up study, noise annoyance was independently associated with CVD risk, including VTE,[93] suggesting a possible relationship.

A summary of particularly important studies regarding the association between exposure to air, noise, and light pollution and VTE can be found in **Table 4**.

New Methods for Assessing the Impact of Air, Noise, and Light Pollution

The assessment of air, noise, and light pollution has traditionally relied on ground-based monitoring stations, which provide localized data and often fail to capture the broader spatial dynamics of these pollutants. The emergence of advanced satellite remote sensing technology has opened new avenues for comprehensive environmental pollution assessment. Satellite sensors can now provide high-resolution data (spatially, spectrally, and temporally) on various pollutants, enabling a more holistic understanding of their distribution and impact.[94]

One of the key advantages of using satellite data for pollution monitoring is the ability to observe and analyze pollutants over large geographic areas, often on a global scale.[95] Ground-based monitoring stations are inherently limited in their spatial coverage, whereas the analysis of in-orbit data using artificial intelligence (AI) can reveal the regional and even transboundary nature of air, noise, and light pollution.[96,97] This wide-ranging perspective is crucial for identifying hotspots, tracking the dispersion of pollutants, and evaluating the effectiveness of mitigation strategies. Furthermore, satellite-derived insights can be integrated with other geospatial information, such as population distributions, land use patterns, and transportation networks, to provide a comprehensive understanding of the drivers and impacts of environmental pollution.[98]

The integration of satellite data with advanced machine learning (ML) techniques can further enhance the capabilities of environmental pollution assessment.[94] ML algorithms help identify complex patterns, trends, and relationships—often difficult or impossible to determine by humans—within the satellite data, enabling more accurate modeling and prediction of pollution dynamics.[99] This can lead to more effective and scalable early warning systems, improved root-cause analysis, and targeted mitigation strategies. The application of AI to satellite pollution monitoring is rapidly evolving, with the potential to revolutionize our understanding and management of environmental pollution. Unsupervised methods can be highly valuable in the context of pollution monitoring. Unlike supervised techniques that require labeled training data, they can uncover hidden patterns and anomalies in the data without relying on costly ground-based observations.

In summary, utilizing satellite data for scalable and fast environmental pollution assessment enhances our ability to analyze the distribution, dynamics, and impacts of air, noise, and light pollution, enabling more effective mitigation strategies.

Leveraging Artificial Intelligence and Machine Learning to Model the Health Impacts of Environmental Risk Factors

AI, including ML techniques, has emerged as a powerful tool for modeling complex, multi-scale interactions, such as those between environmental exposures and health outcomes. While traditional statistical approaches remain foundational for causal inference and hypothesis testing, they often struggle to capture nonlinear relationships, high-dimensional interactions, and spatiotemporal variability. In contrast, AI methods are well-suited to

integrating diverse data streams and identifying latent structures that may underlie the health impacts of air, noise, and light pollution.

A growing body of research demonstrates the potential of AI in leveraging real-world data,[100] such as electronic health records, physiological monitoring, and mobile or satellite-based environmental sensing, for environmental health studies. For example, Pundi et al linked daily levels of $PM_{2.5}$ and Air Quality Index to cardiovascular function and behavior in over 28,000 patients with cardiac implantable electronic devices.[101] Using time-varying and case-crossover models, they observed significant associations between pollution exposure and premature ventricular contractions, thoracic impedance, and reduced physical activity. These findings highlight not only the acute effects of air pollution on cardiovascular health but also the feasibility of integrating high-resolution physiological and environmental data to characterize short-term exposure-response dynamics.

While advanced AI models offer powerful capabilities, traditional statistical techniques continue to play an important role in environmental epidemiology. A recent UK Biobank study assessed long-term exposure to multiple air pollutants and risk of IHD in nearly 400,000 participants.[102] Rather than relying on modern AI tools, the authors employed principal component analysis (PCA) to generate a composite air pollution score that captured the joint variance of five pollutant measures. This score was linearly associated with IHD incidence over a median follow-up of 12.5 years. Although PCA does not accommodate nonlinear effects or complex interactions, its continued use in large-scale cohort studies highlights the practical value of classical approaches for exposome-wide assessment.

The concept of the exposome (the totality of environmental exposures across the life course) has further broadened the scope of environmental cardiovascular research. In a recent review, Hahad et al summarized evidence linking air and noise pollution, reduced greenspace, low neighborhood walkability, and exposure to metals such as lead and cadmium with subclinical and clinical manifestations of atherosclerosis.[103] These environmental factors act through shared mechanisms, including oxidative stress, inflammation, and sympathetic nervous system activation. The authors emphasize the potential of remote sensing, AI, and integrative modeling by enabling multidimensional exposure profiling and mechanistic inference.

Altogether, these studies reflect the evolving landscape of environmental epidemiology, where AI augments traditional methods by enabling scalable, high-dimensional, and often real-time analyses of complex exposure-health relationships. From predictive modeling and unsupervised pattern recognition to network-based toxicology, AI offers a flexible and increasingly interpretable framework for elucidating the cardiovascular effects of environmental exposures. Future work should prioritize methodological validation, integration with mechanistic and longitudinal data, and the development of equitable, privacy-preserving models for public health and clinical translation.

The Use of in Silico Models

Biophysical models that mimic the process of thrombosis and hemostasis have been developed, ranging from the molecular level up to the blood vessel. These mathematical and in silico models, when personalized to an individual, can be termed digital twins or health virtual twins.[104] Developing these models for diagnosis and prevention of cardiovascular complications requires a multi-scale understanding of the mechanisms of thrombosis and its impact, ranging from the biochemical reactions up to the occlusion of flow in the vascular system.

Most models that aim to predict thromboembolism have grown out of well-established hemodynamic fluid-flow models. Due to the relative ease of imaging major vessels and the well-established biophysical principles of blood flow modeling, there are a number of models that use wall shear stress (WSS) as an identifier of individual vessels that are prone to thrombus formation (where low WSS correlates with thrombus formation).[105] These fluid-flow models are particularly useful when there are disturbances in flow in the pathology (e.g., atrial fibrillation). However, alone their use is limited as they do not consider the clotting cascade.

Extensions of the fluid-flow model include adding mechanisms of the clotting cascade and Virchow's triad. Due to the complexity of the clotting cascade, often these models will focus on the final stages of coagulation, where fibrin is generated.[106] Coupling these clotting models with fluid-flow models allows the assessment of blood stasis, hypercoagulability, and endothelial damage. This allows, for example, for the assessment of clotting risk in left atrial appendages in individuals with atrial fibrillation[106,107] or clotting risk for individuals with the placement of prosthetic valves or stents.[108] Simulations of treatments, such as heparin or warfarin, are also used to determine optimal treatment strategies for individuals.[109]

Beyond the biophysical models of thrombosis development and treatment, numerous models simulate the impact of thromboembolic complications, with stroke being the most critical one.[110] Researchers have developed simulations of stroke and its impact on brain perfusion and tissue death.[111] These models were also used within an in silico clinical trial framework to test thrombolysis and thrombectomy treatments in a purely computational clinical trial.[112] Complications that can arise poststroke include clot fragmentation, where clot fragments travel downstream and occlude the microvasculature. Models have been developed of this phenomenon, using these clot models as a test-bed for "what-if" scenarios, showing that clot fragmentation has only a minor impact on downstream perfusion.[113]

By understanding the properties of blood, the heart, and the vascular system, we can develop computational models (or digital twins) to help predict, prevent, and treat thromboembolic complications. Most current research focuses on thromboembolic events in the heart, atrial fibrillation, and the effects of stroke. However, there is still a large gap in personalizing these models to generate digital twins of thromboembolic events to optimize prevention and treatment strategies. Furthermore, as we improve our understanding of the clotting cascade mechanisms, and in particular, endothelial dysfunction, the models will also improve. Advanced multimodality imaging plays an increasing role in revealing the underlying pathophysiological mechanisms that potentially link environmental pollutants to CVD, by enabling the assessment of vascular inflammation and plaque burden.[92] To date, the authors are not aware of any models that account for

pollution in thromboembolic models, making it an area ripe for development and an opportunity to better understand the differential impact of various exposures in isolation or combination.

Future Directions and Areas of Future Research

Gaps in Evidence

Despite growing recognition of the external exposome as a major determinant of cardiovascular health, significant knowledge gaps remain in understanding its cumulative impact. One of the primary limitations is the scarcity of high-quality studies that comprehensively evaluate the combined effects of multiple environmental exposures on CVD. Most existing research focuses on isolated pollutants, such as air pollution or noise, without considering the potential synergistic interactions between different components of the exposome. Further, prospective studies of this type are lacking. Comparative impacts of the exposome in different populations are also needed, given the reported ethnic differences in clinical epidemiology[114] and cardiovascular-related outcomes.[115,116] Additionally, an improved understanding of why certain individuals are less likely to develop exposure-related CVD may help to characterize mechanisms of resilience.

Another critical issue is the lack of consistent evidence in identifying the most vulnerable populations. While epidemiological studies suggest that individuals with preexisting cardiovascular conditions, older adults, and those from socioeconomically disadvantaged backgrounds may be at higher risk, findings are often contradictory. This heterogeneity in results makes it difficult to develop targeted preventive measures. Indeed, more data are needed in under-represented and under-diagnosed high-risk patient subgroups such as those with dementia[117,118] and clinical complexity phenotypes.[119] Furthermore, there is no robust evidence demonstrating that personalized interventions to reduce exposome exposure can significantly lower the risk of CVD or improve life expectancy. While recommendations for reducing air pollution exposure exist, there are insufficient data to determine whether such interventions translate into meaningful health benefits at the individual level.

Finally, an important unresolved question is the cost-effectiveness of mitigation strategies. Given the substantial economic burden of CVD, it is crucial to determine which interventions provide the greatest health benefits relative to their cost.[120] Future research should focus on developing and validating scalable, cost-effective strategies to mitigate environmental risks while addressing existing health disparities. Addressing these evidence gaps is essential for advancing precision environmental medicine and improving cardiovascular health on a population level.

Areas for Future Research

The rapid advancement of technology and data science is reshaping cardiovascular epidemiology, including understanding the interplay between environmental factors and thromboembolic complications. Future research in this field will rely on novel data sources and advanced analytical approaches, enabling more precise risk assessment and the development of effective preventive strategies.

One of the most promising areas of progress is the integration of Big Data from Electronic Health Records into large-scale epidemiological studies. These datasets allow for real-time population-level analyses, identification of temporal trends, and assessment of long-term cardiovascular outcomes associated with environmental exposures. Additionally, Real-World Data from wearable devices offers continuous physiological monitoring, improving the detection of early warning signs of thromboembolic events and their triggers. Furthermore, satellite-based environmental monitoring is emerging as a crucial tool for quantifying exposure to air pollution, noise, and artificial light. Geostationary satellites provide high-resolution data at the national level, while Low Earth Orbit satellites enable precise exposure assessment on a citywide or regional scale. Smart city infrastructure sensors further complement these approaches, offering localized, real-time monitoring of environmental stressors that contribute to cardiovascular dysfunction. In this context, an extension of the classical exposome concept to a Sentinel Exposome was proposed by Łukasz Kuźma at the 2025 Congress of the Polish Cardiac Society: a continuously active system that monitors an individual's environmental exposures, acting as a "sentinel" that detects hazards and triggers protective actions. A key element is not only the integration of widely gathered external environmental data but also—via wearable devices—the coupling of exposures with the body's physiological response.

From a methodological perspective, population genomics will be pivotal in elucidating gene-environment interactions in the pathophysiology of thromboembolic CVDs. Epidemiological phenotyping will enhance the stratification of at-risk populations by integrating environmental, clinical, and genetic risk factors. Randomized Controlled Trials will remain the cornerstone of evaluating preventive and therapeutic interventions, including AI-driven predictive models and real-world evidence-based treatment strategies. The integration of advanced environmental data sources with cutting-edge cardiovascular epidemiology will enable a more comprehensive understanding of the mechanisms linking air, noise, and light pollution to thromboembolic CVD. This multidisciplinary approach is essential for informing public health policies and optimizing clinical interventions aimed at mitigating the cardiovascular burden of environmental exposure.

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Statements and Additional Information

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