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Transportation Noise and Cardiovascular Health: Evidence, Mechanisms, and Policy Imperatives

ABSTRACT

Environmental noise, particularly from road, rail, and aircraft traffic, is now firmly recognized as a widespread risk factor for cardiovascular disease. About 1 in 3 Europeans is exposed to chronic noise exposure above the guideline thresholds recommended by the World Health Organization (WHO), thus contributing substantially to cardiovascular morbidity and mortality. Robust evidence from recent meta-analyses links transportation noise to ischemic heart disease, heart failure, stroke, hypertension, and type 2 diabetes mellitus. Findings from experimental studies in both humans and animal models reveal biologically plausible mechanisms involving sympathetic nervous system activation, oxidative stress, inflammation, endothelial dysfunction, and disruption of circadian rhythms. The adverse health effects are particularly pronounced for nighttime noise, which disrupts restorative sleep and alters neurohormonal balance. Emerging research using the exposome framework highlights the cumulative toll of environmental stressors, including noise, on vulnerable populations. This review combines the latest evidence from epidemiology, mechanistic research, and intervention studies and outlines a roadmap for incorporating noise into cardiovascular prevention strategies. Figures illustrate key concepts such as the noise reaction model, oxidative stress pathways, and practical mitigation measures. The conclusion calls for noise to be treated as a fully recognized cardiovascular risk factor—in parallel with traditional risks—and to address it through coordinated efforts in urban planning, public health policy, and clinical practice.

Keywords: Cardiovascular disease, endothelium, nitric oxide, noise pollution, oxidative stress, preventive cardiology

INVITED REVIEW

INTRODUCTION

Environmental noise is ubiquitous in urban life and has long been regarded as a mere nuisance or quality-of-life issue. However, a growing body of scientific evidence indicates that transportation noise emanating from cars, trucks, trains, and aircraft contributes significantly to the global burden of non-communicable diseases, especially cardiovascular conditions. According to the European Environment Agency (EEA) 2025 report, more than 110 million Europeans—over 20% of the population—are exposed to transportation noise levels exceeding the European Union (EU) reporting thresholds for health effects. When assessed against the stricter WHO guideline values, this figure rises to approximately 150 million people, corresponding to more than 30% of the European population. Each year, chronic exposure to transport noise is estimated to cause 66 000 premature deaths, 50 000 new cases of cardiovascular disease, and 22 000 new cases of type 2 diabetes. Overall, around 1.3 million disability-adjusted life years (DALYs) are lost annually due to transport noise in Europe, with an estimated economic cost of at least €95.6 billion per year, equivalent to roughly 0.6% of the EU's gross domestic product.[1,2]

Unlike auditory damage, the cardiovascular effects of noise occur via indirect pathways. The central "noise reaction model" (Figure 1) outlines how the perception of sound, particularly during sleep, activates limbic structures, leading to stimulation of the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system (SNS).[3]

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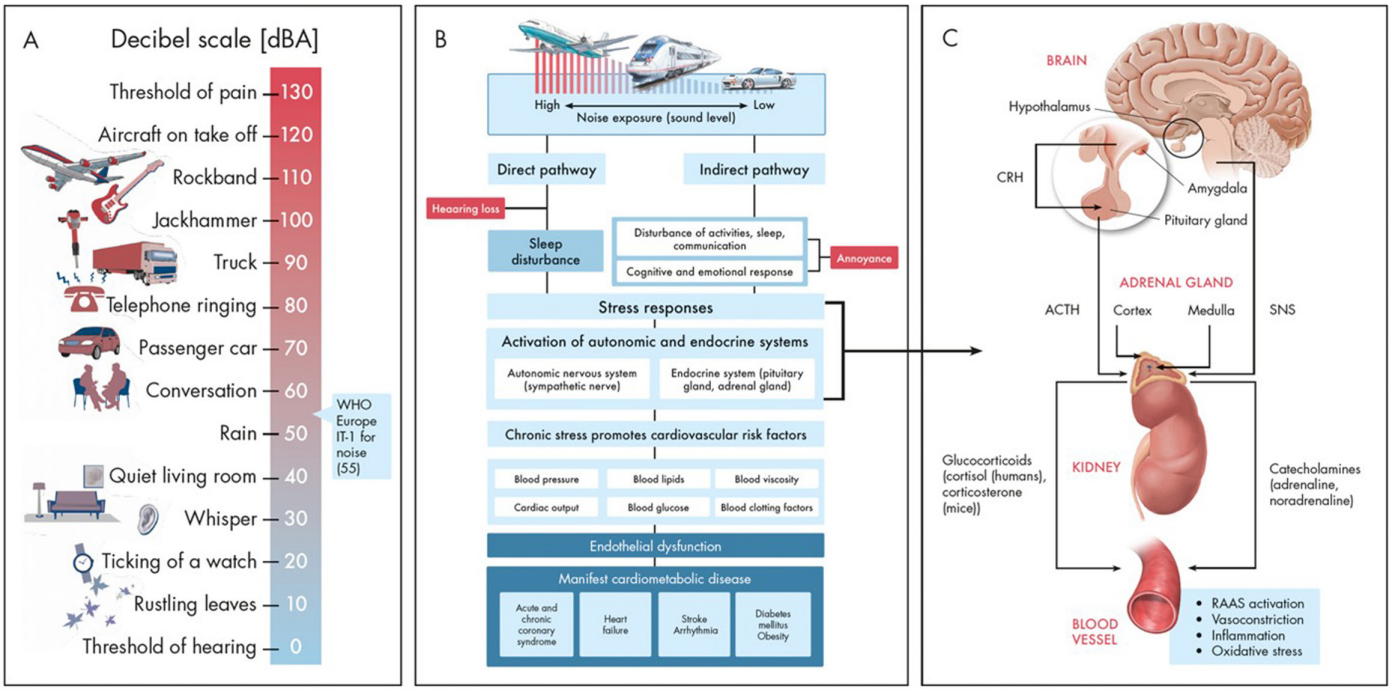

Figure 1. A: Sound pressure levels (SPLs) of different noise sources.[4] B: Noise reaction model for noise exposure's direct (auditory) and indirect (non-auditory) effects.[5] C: Neuronal activation (arousals) induced by noise triggers signaling via the hypothalamic–pituitary–adrenal axis and sympathetic nervous system (SNS). This leads to release of corticotropin-releasing hormone (CRH) from the hypothalamus into the pituitary gland, which stimulates the release of adrenocorticotropic hormone (ACTH) into the blood. ACTH induces the production of glucocorticoids by the adrenal cortex, and the activation of the SNS stimulates the production of catecholamines by the adrenal medulla. The release of glucocorticoids and catecholamines, in turn, leads to the activation of other neurohormones and pathways, such as the renin–angiotensin–aldosterone (RAAS) system, and increased inflammation and oxidative stress. Panel A reprinted from[4] with permission. Copyright © 2018 The Authors. Panel B and C adapted from[6] with permission. Copyright © 2021, Springer Nature Limited.

This results in elevated levels of stress hormones, cortisol, adrenaline, and norepinephrine, which contribute to inflammation, oxidative stress, and ultimately vascular dysfunction.[7] These pathophysiological processes overlap significantly with those triggered by air pollution and other environmental stressors.

This brief review provides an integrated perspective on transportation noise as a cardiovascular risk factor. The historical development of the field, epidemiological associations with cardiovascular and metabolic diseases, insights from human and animal experiments, interactions with air pollution, and mitigation approaches are summarized.

HIGHLIGHTS

- About one third of all European citizens are exposed to environmental noise exceding WHO treasholds.
- Chronic noise exposure induces oxidative stress, inflammation, endothelial dysfunction, and circadian disruption, which are all hallmarks of cardiovascular pathophysiology.
- Environmental noise, as a risk factor, should be integrated into public health guidelines, exposome research, and preventive cardiology frameworks.

Recent reviews, including the 2023 Umbrella+ analysis[8] and the 2025 EEA report,[1] advocate for recognition of noise as a priority in cardiovascular prevention strategies.

HISTORICAL MILESTONES IN UNDERSTANDING NOISE AS A CARDIOVASCULAR RISK FACTOR

Although the detrimental effects of noise were first recognized in the context of occupational hearing loss, non-auditory impacts have been described for over half a century. In the 1960s and 1970s, pioneering studies by Levi and colleagues demonstrated that industrial noise exposure led to increased blood pressure and catecholamine release in workers.[9] Kryter and colleagues proposed that noise acts as a generalized stressor capable of inducing physiological changes even at subauditory thresholds.[10]

Animal studies by Yamamura and others later revealed that intermittent and low-frequency noise provokes stronger autonomic responses than continuous or high-frequency sound.[11] These findings anticipated modern insights into the role of noise-induced arousals and disrupted sleep architecture. In the 1990s, Babisch formalized the "noise reaction model," laying the groundwork for mechanistic studies of cardiovascular outcomes.[3]

The past 2 decades have seen a surge in interest in environmental cardiology, with transportation noise emerging as

a leading non-traditional risk factor. In 2014, the European Heart Journal published a comprehensive review outlining the clinical, epidemiological, and mechanistic evidence for noise-induced vascular damage.[5] Subsequent WHO guidelines and EEA reports have validated and expanded these findings, emphasizing the need for preventive action.

EPIDEMIOLOGY OF TRANSPORTATION NOISE AND CARDIOVASCULAR DISEASE: EVIDENCE, THRESHOLDS, AND POLICY IMPLICATIONS

Meta-analyses confirm that road traffic noise is associated with higher risks of ischemic heart disease (IHD), stroke, and heart failure. A 2023 Umbrella+ review that meta-analyzed data from over 100 studies estimated that a 10 dB(A) increase in road noise is associated with a 4.1% higher risk of IHD, a 4.6% higher risk of stroke, and a 4.4% higher risk of heart failure.[8] Aircraft and railway noise showed weaker but still positive associations, particularly for nighttime exposure.

Noise is likely to also be associated with acute cardiovascular events. A Swiss case-crossover study demonstrated that exposure to nighttime aircraft noise (>50 dB L_{max}) is associated with a 50% increased risk of cardiovascular death in the subsequent 2 hours.[12] These findings suggest that noise not only contributes to chronic disease development but may also trigger acute cardiovascular events.

Numerous cohort and cross-sectional studies have investigated the impact of noise on cardiometabolic risk factors. A recent meta-analysis reported that each 10 dB(A) increment in road noise exposure is associated with a 3%-5% increased risk of arterial hypertension.[13] Based on the Danish National Cohort, associations with higher T2DM risk were found, with effect sizes increasing when nighttime exposure was accounted for.[14] Exposure to chronic noise may also contribute to adiposity. The Danish National Birth Cohort revealed that children living near busy roads had higher BMI z-scores and an increased risk of overweight by age 7.[15] Similar results have been observed in adults. These findings support the hypothesis that environmental stressors disrupt metabolic regulation via neuroendocrine pathways. The WHO identifies sleep disturbance as the most prevalent and disruptive health outcome of environmental noise.[2] Meta-analyses have shown that aircraft and railway noise are particularly potent disruptors of sleep, leading to increased nocturnal awakenings, sleep stage transitions, and suppressed slow-wave sleep.[16] Sleep fragmentation has cascading effects on blood pressure regulation, glucose metabolism, and emotional resilience. Chronic sleep loss increases cardiovascular disease risk via activation of the HPA axis, increased oxidative stress, and impaired endothelial repair. A systematic review also found associations between noise exposure and depression, anxiety, and suicide risk.[17,18] Several large-scale studies challenge the notion of a "safe" noise level. A study based on the Danish National Cohort observed linear increases in cardiovascular risk starting at 35 dB(A) L_{den}, thus well below current EU thresholds.[19] These findings echo concerns in the air pollution field, where even minimal PM2.5 exposure is associated with increased mortality. Collectively, this underscores the need to re-evaluate regulatory limits.

TRANSLATIONAL EVIDENCE: HUMAN STUDIES LINKING NIGHTTIME TRANSPORTATION NOISE TO VASCULAR INJURY

Controlled exposure studies have provided critical insights into the biological effects of noise in humans. In a landmark study, healthy volunteers exposed to recorded nighttime aircraft noise (maximum 60 events at 60-70 dB) experienced impaired flow-mediated dilation, increased systolic blood pressure, elevated adrenaline levels, and reduced sleep quality.[20] Notably, oral vitamin C administration reversed the endothelial dysfunction, implicating oxidative stress (Figure 2).

Biomarkers such as 3-nitrotyrosine and 8-isoprostane are consistently elevated after noise exposure, indicating activation of NOX2-derived ROS. Proteomic analyses identified upregulation of stress- and inflammation-related pathways after railway noise exposure.[21] Noise timing plays a critical role: exposure during the biological night has greater adverse effects than daytime noise of equal intensity.[24] This is likely due to interaction with circadian gene expression (Bmal1, Cry1), melatonin suppression, and increased vulnerability of vascular endothelium during sleep.

OXIDATIVE STRESS, CIRCADIAN DISRUPTION, AND THE PRIMED HEART: LESSONS FROM NOISE-EXPOSED ANIMALS

Animal models have provided indispensable insights into the biological mechanisms linking transportation noise to cardiovascular and metabolic dysfunction. Among the most influential contributions are those from the Mainz research group, which developed a translational mouse model simulating real-world aircraft noise exposure, with maximum sound pressure levels reaching up to 85 dB(A) and average levels around 72 dB(A). These studies revealed a powerful stress response, mediated through activation of both the HPA axis and the SNS, within just a few hours of exposure.[7,24] This biological cascade is marked by a steep rise in circulating stress mediators such as adrenaline, noradrenaline, angiotensin II, endothelin-1, and aldosterone. Concurrently, there is a pronounced upregulation of vascular NADPH oxidase enzymes, particularly NOX2, which catalyze the production of reactive oxygen species. The endothelial nitric oxide synthase (eNOS), a key enzyme for vascular homeostasis, becomes uncoupled in this oxidative environment, reducing nitric oxide (NO) bioavailability; oxidative stress markers including 3-nitrotyrosine, malondialdehyde, and 8-isoprostane rise substantially. Endothelial function is impaired, as shown by reduced acetylcholine-induced vasodilation, and systolic blood pressure increases significantly—especially when the noise exposure occurs during the nighttime. Strikingly, these adverse vascular changes are almost completely absent in NOX2-deficient mice, underscoring NOX2's central role as a molecular switch in noise-induced vascular dysfunction. In contrast, eNOS-deficient mice display an even greater hypertensive response to noise, highlighting the protective role of intact NO signalling.[24]

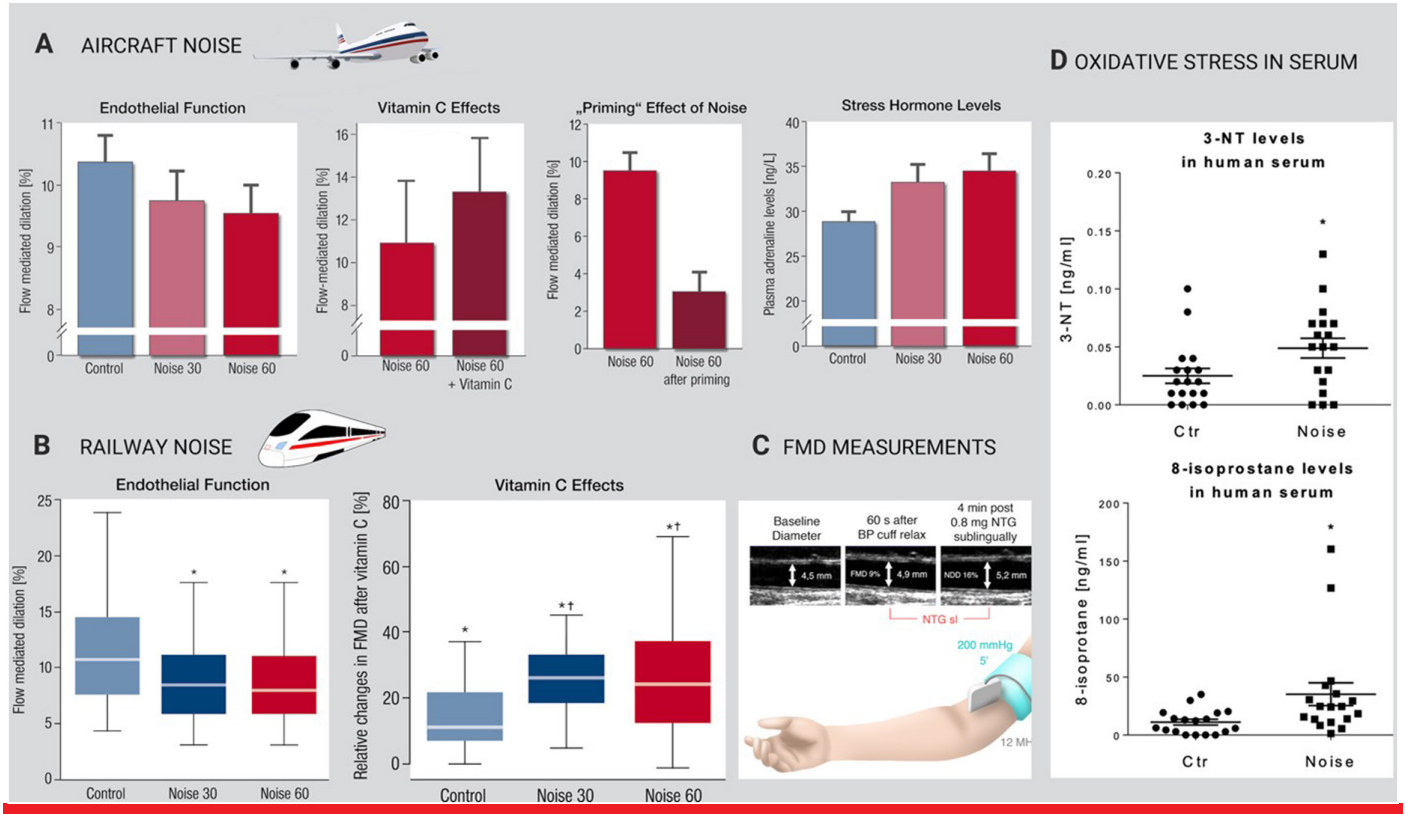

Figure 2. Key effects of noise observed in human field studies. A: effects of 30 and 60 aircraft noise events on flow-mediated dilation (FMD) of the brachial artery (noise 30 and 60) of 70 healthy subjects. Vitamin C effects were assessed in a subgroup of the cohort. A priming effect of aircraft noise on endothelial function was observed, i.e., previous exposure to Noise30 caused Noise60 to have a significantly stronger reduction of flow-mediated dilation.[20] Serum adrenaline levels also increased significantly. B. Effects of 30 and 60 railway noise events on flow-mediated brachial artery dilation in 70 healthy subjects. Vitamin C effects were assessed in a subgroup.[21] C. Methodology of FMD.[22] D. Effects of aircraft noise on oxidative stress markers (3-nitrotyrosine [3-NT] and 8-isoprostane) in serum that were measured in the samples of the aircraft noise study and published in.[23] Adapted from[23] with permission. Copyright ©2018, Oxford University Press.

Additional insights have emerged from the study of circadian rhythms. Noise exposure during the sleep phase (ZT12-ZT24) leads to stronger elevations in stress hormones and oxidative stress compared to daytime exposure. Transcriptomic and proteomic profiling of aortic and cerebral vasculature reveals altered expression of core circadian genes such as Bmal1, Cry1, and Per2, alongside dysregulation of redox-sensitive transcription factors like Nrf2 and NF-κB, as well as metabolic regulators including Foxo3 and Sirt1.[25] These findings provide a mechanistic foundation for the clinical observation that nocturnal noise, rather than daytime exposure, is more strongly linked to adverse cardiovascular outcomes (Figure 3).

Even more compelling is the evidence that noise acts as a cardiovascular "primer." In a murine model of myocardial infarction, animals pre-exposed to aircraft noise for 7 days developed significantly larger infarcts, poorer contractile function, and more extensive cardiac fibrosis compared to controls.[27] Molecular analysis of the infarct border zone revealed increased expression of pro-inflammatory cytokines such as interleukin-6 (IL-6) and IL-1β, elevated monocyte recruitment, and amplified oxidative stress.

THE EXPOSOME IN ACTION: HOW NOISE AND AIR POLLUTION AMPLIFY CARDIOVASCULAR RISK

Noise rarely acts in isolation. In urban environments, transportation noise is almost invariably accompanied by air pollution. As a result, there is growing recognition of the need to understand their individual and joint contributions to cardiovascular risk. While most large-scale cohort studies adjust for particulate matter (PM2.5) when analyzing noise effects, recent findings from the UK Biobank and the Danish National Cohort show that associations between noise and cardiovascular endpoints persist even after rigorous adjustment for air pollutants, suggesting that noise exerts independent effects.[19,28]

Animal studies provide strong biological support for these epidemiological findings. In a pivotal experiment, Kuntic et al[29] exposed mice to both aircraft noise and ultrafine particles, simulating real-world co-exposure scenarios. The findings revealed additive, if not synergistic, effects on vascular health. Endothelial dysfunction was significantly more pronounced, oxidative stress and inflammation were elevated, and monocyte infiltration into vascular tissue increased. Furthermore, co-exposure amplified NOX2

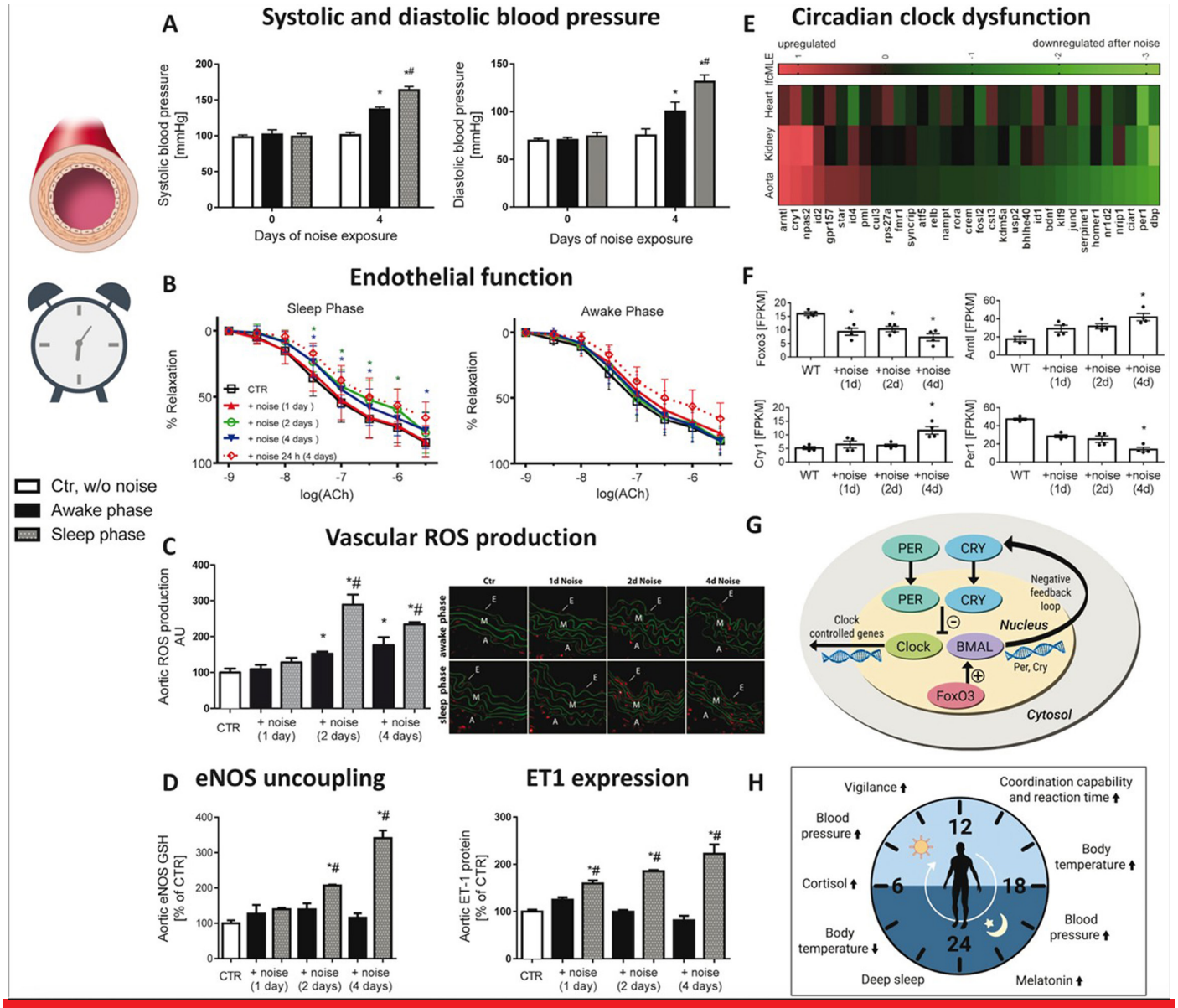

Figure 3. Effects of sleep and awake phase aircraft noise [mean sound pressure level 72 dB(A) for 12 hours per day for 1, 2, and 4 days] on the murine vasculature. Sleep phase noise showed a more pronounced increase in systolic and diastolic blood pressure (A) and caused significant endothelial dysfunction (diminished response to acetylcholine [ACh]) (B). For comparison, the impaired endothelium-dependent relaxation in response to 24 hours noise exposure is shown in a dotted line. Sleep phase noise induced more vascular oxidative stress (red fluorescence staining by oxidized hydroethidium in aortic cryo sections) (C), eNOS uncoupling by immunostaining against S-glutathionylated (= uncoupled) enzyme (eNOS-GSH), and endothelin-1 (ET-1) protein expression by immunohistochemistry (D). Sleep phase noise also caused substantial dysregulation of the expression of circadian clock genes in the aorta and kidney, as revealed by Illumina RNA sequencing (E). Aortic gene expression of the transcription factor Foxo3 (regulates BMAL1) and period-1 (Per1) was down-regulated. In contrast, brain and muscle aryl hydrocarbon receptor nuclear translocator (Arnt)-like (Bmal) 1 and cryptochrome-1 (Cry1) were up-regulated (F). The clock core components consist of the positive regulators CLOCK and BMAL that directly control circadian gene expression and the negative regulators PER and CRY (G). The circadian clock regulates several essential biological functions (H). Adapted from[23] (A-F) and[26] (G-H) with permission. Copyright © 2018, Oxford University Press and Copyright © 2020 by Annual Reviews.

activation and eNOS uncoupling, culminating in severe reductions in NO signaling.[29] These results illustrate how environmental exposures can potentiate each other's harmful effects, reinforcing the importance of studying them in tandem.

Such evidence supports the broader concept of the "exposome," which accounts for the cumulative, lifelong burden of environmental exposures. By adopting an exposomic framework, researchers and policymakers can better identify individuals at risk, design more effective interventions,

and ultimately understand how overlapping environmental stressors shape cardiovascular outcomes over time.[30,31]

Air pollution and noise have been implicated in a broad spectrum of health outcomes beyond cardiovascular disease. They increase the risk of metabolic disorders such as diabetes, cerebrovascular events including stroke, and various forms of mental illness. Co-exposure may be especially deleterious in vulnerable populations: children, the elderly, and individuals with pre-existing heart disease, whose physiological resilience to environmental insults is limited.

NOISE EXPOSURE ACROSS THE LIFE COURSE AND IN VULNERABLE POPULATIONS

The burden of noise is not evenly distributed. Socioeconomically disadvantaged groups, those with lower income, less political power, and fewer housing options, are more likely to reside in noisy environments near highways, rail corridors, or under flight paths. The EEA 2025 report underscores this point clearly, showing that the elderly, people with chronic illness, and low-income households bear a disproportionate share of environmental noise exposure across Europe.[1]

Crucially, noise exposure during critical periods of development can exert long-term consequences. During school age, chronic noise has been linked to cognitive delays, increased cortisol levels, impaired memory consolidation, and disrupted sleep, each of which may cascade into behavioral and physiological problems over time.[32] Among individuals with diagnosed cardiovascular disease, diabetes, or hypertension, nighttime noise represents a particularly insidious threat. Repeated nocturnal arousals drive up nocturnal blood pressure, reduce heart rate variability, and impair endothelial repair mechanisms. Even small shifts in average noise levels can produce outsized effects in these sensitive groups, making them especially vulnerable to additional morbidity and mortality.[33,34]

Mental health vulnerability also intersects with noise exposure. Individuals with anxiety, depression, or sleep disorders report more substantial subjective annoyance and exhibit greater physiological stress responses to the same acoustic stimuli. Conversely, chronic noise exposure itself may trigger or exacerbate psychiatric conditions through the persistent activation of the HPA axis, leading to emotional dysregulation. The interaction between noise and mental illness is thus both bidirectional and self-reinforcing.

TURNING DOWN THE VOLUME: STRATEGIES TO MITIGATE NOISE AND PROTECT CARDIOVASCULAR HEALTH

Given the strength of the evidence and the modifiable nature of the exposure, noise mitigation represents one of the most cost-effective public health strategies available. Technological solutions, behavioral adaptations, and regulatory measures can each contribute to reducing cardiovascular risk, especially if implemented during the sleep phase, when the biological consequences of noise are most severe.

At the level of the source, several measures have proven effective. For road traffic, using low-noise asphalt, promoting quieter tires, and enforcing speed reduction zones can reduce average noise levels (L_{den}) by 3-7 dB(A). In aviation, steeper descent trajectories, optimized flight paths, and bans on night flights, particularly between 23:00 and 06:00, offer clear benefits. Technical improvements, such as quieter jet engines, can reduce noise footprints. On railways, replacing cast-iron brake pads with composite materials, track grinding, and even curfews on freight traffic have all demonstrated success in lowering noise exposure.

Barriers and building insulation are key at the transmission path and receiver level. Façade insulation, window glazing, and bedroom orientation away from traffic sources can reduce nighttime indoor noise by 10 to 20 dB(A). In urban planning, the expansion of green infrastructure, such as parks, vegetated noise buffers, and tree belts, reduces noise and improves air quality and psychological well-being.[35]

Animal studies suggest that lifestyle and pharmacologic interventions may also offer protection. Regular physical activity, intermittent fasting, and pharmacological activation of AMP-activated protein kinase (AMPK) via AICAR administration have all been shown to attenuate vascular dysfunction in noise-exposed mice.[36] While such strategies are not a substitute for structural noise reduction, they may help mitigate damage in high-exposure populations.

On the regulatory front, the EU Environmental Noise Directive currently mandates noise mapping and action plans but stops short of enforcing binding exposure thresholds. Aligning legal standards with WHO guidelines, particularly the ≤45 dB(A) night-time recommendation, would significantly reduce disease burden. Crucially, a health equity lens is essential: prioritizing schools, hospitals, and vulnerable communities for mitigation can yield the most significant public health benefits while reducing inequality.

CLOSING THE GAPS: FUTURE DIRECTIONS IN NOISE AND CARDIOVASCULAR RESEARCH

Although the link between transportation noise and cardiovascular health is now widely accepted, several key gaps remain. First, longitudinal studies that span the entire life course are needed to define critical windows of vulnerability and to quantify cumulative dose effects. Second, current exposure metrics such as L_{den} and L_{night}, while useful, may not fully capture the biological impact of noise. Emerging measures such as NAT55 (number of noise events above 55 dB) or peak levels (L_{max}) may be more relevant to arousal physiology and should be adopted in future studies.

Third, developing robust biomarkers, both of exposure and of early effect, could aid in risk stratification and targeted interventions. Candidate markers include stress hormones (e.g., cortisol), oxidative stress indicators (e.g., 8-isoprostane), inflammatory cytokines (e.g., IL-6), and epigenetic signatures of exposure such as DNA methylation changes. Fourth, interventional trials are needed to test the effects of noise reduction, either via insulation or behavior change, on cardiovascular endpoints in exposed populations.

Finally, economic analyses remain essential. The cost of inaction is high: lost productivity, increased healthcare expenditures, and reduced quality of life. Quantifying the cost–benefit ratio of mitigation, particularly in terms of DALYs averted and QALYs gained, will strengthen the case for urgent and coordinated policy response.

Equally important is integrating noise exposure into clinical decision-making. Incorporating environmental risk into cardiovascular risk scores, electronic health records, and patient counseling would move environmental cardiology from the realm of research into everyday practice.

ON PAR WITH SMOKING: WHY NOISE DESERVES A PLACE IN CARDIOVASCULAR PREVENTION

Transportation noise is a significant and modifiable environmental risk factor for cardiovascular disease. The evidence from epidemiological studies, human exposure experiments, and mechanistic animal models is robust and biologically plausible. Chronic noise exposure induces oxidative stress, inflammation, endothelial dysfunction, and circadian disruption, which are all hallmarks of cardiovascular pathophysiology.

Importantly, noise affects vulnerable groups disproportionately and often co-occurs with other environmental stressors such as air pollution. Mitigation measures are available, cost-effective, and urgently needed, particularly during the sleep period.

The full recognition of environmental noise as a cardiovascular risk factor, on par with hypertension, hyperlipidemia, diabetes, and smoking, is advocated. Health professionals, urban planners, and policymakers must collaborate to reduce exposure and improve cardiovascular outcomes. The next step is integrating noise into public health guidelines, exposome research, and preventive cardiology.

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