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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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TRAFFIC NOISE EXPOSURE AND THE RISK OF HYPERTENSION AND CARDIOVASCULAR DISEASES: A REVIEW

Natalia Kruszewska *(Corresponding Author, Email: nkruszewska14@gmail.com)*
Provincial Hospital in Poznań, Poznań, Poland
ORCID ID: 0009-0001-5297-3846

Urszula Borucińska
Wolski Hospital, Warsaw, Poland
ORCID ID: 0009-0008-7441-9011

Hanna Pietruszewska
Medical University of Łódź, Łódź, Poland
ORCID ID: 0009-0000-7626-2996

Oliwia Sędziak
Faculty of Medicine, University of Opole, Opole, Poland
ORCID ID: 0009-0003-2128-0662

Sabina Skrzynecka
University Clinical Hospital in Opole, Opole, Poland
ORCID ID: 0009-0008-8383-0785

ABSTRACT

Introduction: Environmental noise originating from road, rail, and air traffic is one of the most significant environmental stressors in urbanized areas. In recent years, growing evidence has indicated that long-term exposure to noise constitutes an independent risk factor for arterial hypertension and cardiovascular diseases. The aim of this review is to provide an in-depth analysis of the biological and social mechanisms underlying these associations, to examine health inequalities related to noise exposure, and to present opportunities for the use of modern noise-monitoring technologies in socio-medical research.
Methods: A systematic review of the scientific literature from 2015 to 2025 was conducted, including randomized controlled trials, observational studies, meta-analyses, and reports from the World Health Organization (WHO) and the European Environment Agency (EEA). The review focused on studies assessing the effects of long-term environmental noise exposure on the development of arterial hypertension and the risk of cardiovascular events.
Results: Long-term exposure to environmental noise levels exceeding 55 dB significantly increases the risk of hypertension, sleep disturbances, ischemic heart disease, and stroke. The primary pathophysiological mechanisms include activation of the hypothalamic–pituitary–adrenal axis, increased cortisol secretion, oxidative stress, and sleep disruption.
Conclusions: Noise is a social determinant of health. Individuals with lower socioeconomic status are more likely to live in areas with high traffic density and poor acoustic insulation, leading to cumulative adverse health effects. In recent years, advanced noise-monitoring technologies such as IoT-based sensors, mobile applications, and geographic information systems (GIS) have been developed to support public health policy. The findings of this review highlight the need for integrated health, environmental, and social strategies aimed at reducing the impact of environmental noise on population health.

KEYWORDS

Environmental Noise, Traffic Noise, Hypertension, Cardiovascular Diseases, Public Health

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Introduction

Cardiovascular diseases (CVD) remain the leading cause of death worldwide, accounting for approximately 18 million deaths annually (World Health Organization [WHO], 2023). Although traditional risk factors, such as smoking, poor diet, hypertension, and lack of physical activity, are well understood, increasing attention is being paid to environmental determinants of cardiovascular health. Among these, environmental noise, particularly traffic noise, is becoming a significant, though often underestimated, public health problem.

Progressive urbanization and the expansion of transport infrastructure mean that a significant portion of the population is constantly exposed to high levels of noise from road, rail, and air traffic. The European Environment Agency (EEA) estimates that over 100 million Europeans are exposed to noise levels exceeding 55 dB (EEA, 2023), which surpasses the WHO recommendations for cardiovascular protection.

In the HYENA study and prospective analyses from the UK Biobank, traffic noise levels above 60 dB were shown to significantly increase the risk of myocardial infarction (MI) and stroke, independently of air pollution levels (Münzel et al., 2021). Long-term exposure to such noise levels is associated with increased blood pressure, endothelial dysfunction, sleep disturbances, oxidative stress, and inflammation, all of which contribute to the development of hypertension and other cardiovascular diseases (Münzel et al., 2018; Sørensen et al., 2020).

Despite extensive evidence linking environmental noise exposure to negative cardiovascular outcomes, the causal relationship remains a subject of debate due to methodological differences between studies and the influence of confounding factors such as air pollution, socioeconomic status, and lifestyle (Dzhambov et al., 2020; Feng et al., 2023). Nevertheless, the biological foundations and consistency of epidemiological findings support the hypothesis that traffic noise is an independent risk factor for cardiovascular disease (Hahad et al., 2023).

The aim of this review is to provide a synthetic presentation of current scientific data regarding the link between exposure to traffic noise and the risk of hypertension and cardiovascular disease, and to discuss the biological and social mechanisms underlying these associations. This integrated approach is crucial for developing effective preventive strategies, incorporating noise issues into public health policy, and promoting equitable access to a healthy acoustic environment.

Methodology

The review covers publications from the years 2015–2025, retrieved from the PubMed, Scopus, and Google Scholar databases. Keywords used were: environmental noise, traffic noise, hypertension, cardiovascular disease, myocardial infarction, stroke, noise monitoring.

A systematic search was conducted, including cohort studies, meta-analyses, review articles, and reports from the World Health Organization (WHO) and the European Environment Agency (EEA). Studies focusing on the effects of long-term (chronic) environmental noise exposure on arterial hypertension and the risk of cardiovascular events were included. Studies primarily focusing on occupational noise or *in vitro* molecular mechanisms lacking human epidemiological context were excluded. Two authors independently screened titles and abstracts, resolving conflicts through discussion to ensure the objectivity of the selection.

Results

1.1 Sympathetic Nervous System (SNS) Activation Induced by Noise

Chronic noise exposure is a powerful activator of the Sympathetic Nervous System (SNS), which plays a key role in the development of arterial hypertension (Münzel et al., 2018). Noise acts as a stressor, stimulating the Hypothalamic-Pituitary-Adrenal (HPA) axis and the SNS. This activation leads to the release of catecholamines, such as adrenaline and noradrenaline, which cause vasoconstriction, increased heart rate, and elevated blood pressure (Münzel et al., 2025).

1.2 The Role of Endothelial Dysfunction in Noise-Induced Hypertension

Chronic noise exposure induces oxidative stress and inflammation, which are key mechanisms underlying cardiovascular damage (Münzel et al., 2025). Cortisol and adrenaline initiate a cascade of physiological reactions, including the activation of NADPH oxidase and mitochondrial dysfunction, resulting in the production of Reactive Oxygen Species (ROS), which degrade NO and impair vasodilation. Experimental studies in humans and animals have shown that elevated ROS levels contribute to endothelial dysfunction, a precursor to atherosclerosis and other cardiovascular diseases (Münzel et al., 2021). Furthermore, noise exposure is associated with increased levels of pro-inflammatory cytokines, such as Interleukin-6 (IL-6) and Tumour Necrosis Factor-alpha (TNF-α), which further exacerbate vascular damage (Münzel et al., 2018). Studies have shown that individuals exposed to high noise levels exhibit reduced flow-mediated dilation (FMD), an indicator of endothelial dysfunction (Hahad et al., 2023).

1.3 Amygdala Hyperactivity and Arterial Inflammation

Stress induced by noise leads to increased activity of the amygdala, which is associated with arterial inflammation and an increased cardiovascular risk. Positron Emission Tomography with glucose (18F-FDG-PET) has shown that individuals exposed to chronic noise exhibit elevated metabolic activity in the amygdala, even in the absence of established cardiovascular disease risk factors, which correlates with increased arterial inflammation and the risk of Major Adverse Cardiovascular Events (MACE) (Osborne et al., 2020). This pathway involves a neurovascular mechanism where excessive amygdala activity triggers the release of stress hormones and inflammatory cytokines, such as Interleukin-6 (IL-6) and Tumour Necrosis Factor-alpha (TNF-α) (Münzel et al., 2021).

1.4 Noise-Induced Epigenetic and Gene Expression Changes

Increasing evidence suggests that noise exposure induces epigenetic modifications and changes in gene expression that contribute to hypertension and cardiovascular diseases (Münzel et al., 2025). Noise has been shown to alter the expression of genes involved in oxidative stress, inflammation, and vascular function. For example, studies have indicated an increase in the expression of pro-inflammatory genes and a decrease in the expression of genes responsible for antioxidant defense in individuals exposed to chronic noise (Münzel et al., 2025). Epigenetic changes, such as DNA methylation and histone modifications, are also linked to the effect of noise on the cardiovascular system. These modifications can lead to long-term activation of inflammatory pathways and impairment of vascular repair mechanisms. For instance, exposure to noise is associated with increased methylation of the endothelial nitric oxide synthase (eNOS) gene, leading to reduced NO production and endothelial dysfunction (Hahad et al., 2023).

1.5 Night Noise Exposure and Sleep-Related Hypertension Risk

Crucially, night-time noise exposure has been shown to intensify SNS activation because it disrupts sleep and prevents the nocturnal blood pressure dip (Basner & McGuire, 2018; Münzel et al., 2021). Studies using 24-hour ambulatory blood pressure monitoring have demonstrated that individuals exposed to night noise experience a reduced blood pressure dip, which is associated with a higher risk of hypertension and cardiovascular incidents (Sørensen et al., 2022). A meta-analysis conducted by the World Health Organization (WHO) indicated that night-time noise exposure is linked to a 5% increase in hypertension risk for every 10dB increase in noise level (Basner & McGuire, 2018). This risk is heightened in vulnerable groups, such as the elderly and individuals with pre-existing cardiovascular conditions, who are less resilient to sleep disturbances. Additionally, sleep disturbances, shortening of the REM phase, and sleep fragmentation are observed, which promote the development of metabolic disorders and insulin resistance (Münzel et al., 2018).

1.6 Synergistic Effect of Noise and Air Pollution on Cardiovascular Risk

Although noise exposure alone constitutes a significant risk factor for hypertension and cardiovascular diseases, its effects are often exacerbated by concurrent exposure to air pollution (Hahn et al., 2022). Both environmental stressors share common pathophysiological pathways, including oxidative stress, inflammation, and endothelial dysfunction. Studies have shown that individuals living near major roads experience higher levels of both noise and air pollution, leading to an additive or even synergistic increase in cardiovascular risk (Davies et al., 2020). For example, particulate matter (PM2.5) and noise have been shown to independently activate the HPA axis and the SNS, but their combined exposure results in a more pronounced stress response (Münzel et al., 2021). Furthermore, air pollution exacerbates noise-induced vascular damage by increasing

inflammation and oxidative stress (Hahn et al., 2022). Interestingly, the highest risk of hypertension is observed in individuals exposed to both high levels of noise and elevated air pollution, suggesting an additive or even synergistic effect (Davies et al., 2020; Feng et al., 2023).

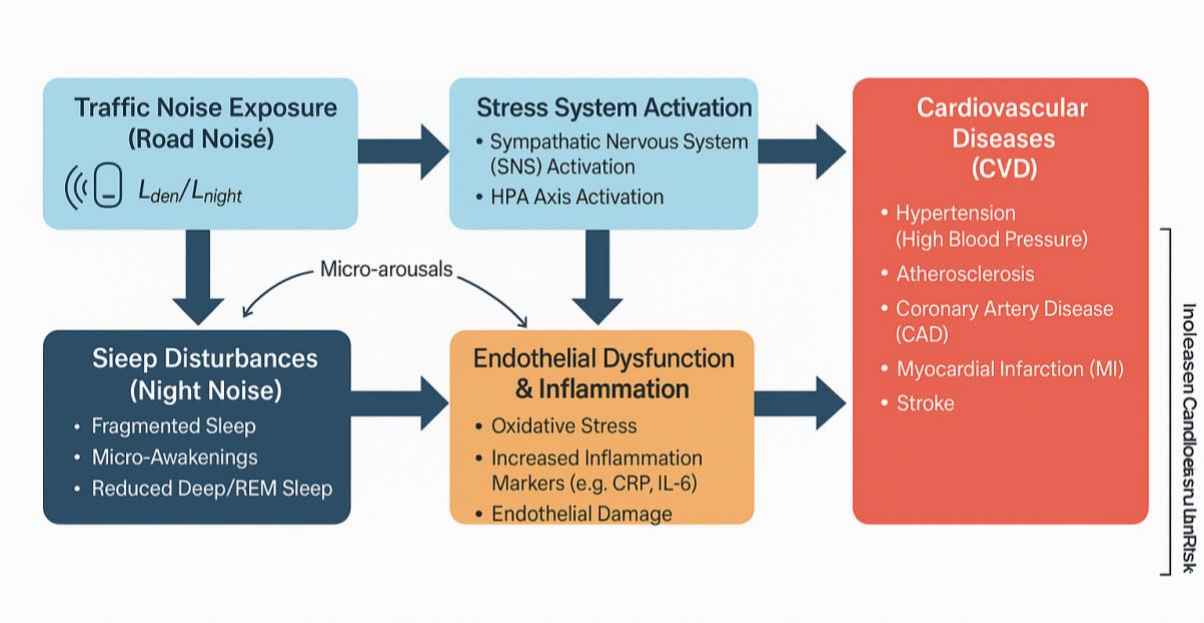

Fig. 1. *Pathophysiological Cascade Schema: Traffic Noise → CVD*

Discussion

Epidemiological studies and meta-analyses consistently show that long-term exposure to road, rail, and air traffic noise significantly increases cardiovascular risk (Dzhambov et al., 2020; Feng et al., 2023). These associations are quantitatively summarized in Table 1, which presents the findings of key epidemiological and experimental studies published between 2015 and 2025, confirming the impact of noise levels exceeding 50–55 dB on hypertension and cardiovascular incidents.

The analyzed publications indicated that individuals with a lower socioeconomic status are more frequently exposed to high levels of traffic noise due to living near high-traffic roads or in areas with poorer acoustic infrastructure (Lee et al., 2021). These environmental inequalities translate into a greater burden of cardiovascular disease in lower-income populations, which constitutes a significant public health issue. Moreover, vulnerable populations, such as the elderly, individuals with pre-existing cardiovascular conditions, and socioeconomically disadvantaged groups, are disproportionately affected by this problem, underscoring the need for targeted interventions. Urban planning and infrastructure development play a crucial role. For example, designing roads to minimize congestion, investing in quieter vehicles, and constructing acoustic barriers can significantly reduce exposure levels (Engelmann et al., 2024; Guski, 2021). Furthermore, public awareness campaigns can educate the public about the health risks associated with traffic noise and encourage exposure-limiting behaviors, such as using white noise generators or moving bedrooms away from street-facing windows. By considering these factors, public health initiatives can significantly reduce the incidence of noise-induced hypertension and improve cardiovascular outcomes at the population level.

Table 1. Summary of epidemiological and experimental studies assessing the relationship between traffic noise exposure, hypertension, and cardiovascular diseases (2015–2025).

Author / Year	Population (Country)	Noise Level (dB, Lden)	Main Findings	Study Type	Reference
Sørensen et al., 2022	420,000 adults (Denmark)	>58 dB	10–15% increased risk of hypertension	Cohort study	BMJ, 2022;376:e067664
Hahad et al., 2023	Meta-analysis (24 studies)	>50 dB	5% higher hypertension risk per +5 dB increase	Systematic review / Meta-analysis	Eur Heart J, 2023;44(12):1031–1042
Münzel et al., 2021	10,000 adults (Germany)	>60 dB	Higher incidence of MI and stroke; increased oxidative stress markers	Cohort study	Eur Heart J, 2021;42(10):1022–1036
Basner & McGuire, 2018	15 countries (WHO data)	45–70 dB	Sleep fragmentation, elevated cortisol, stress response	Systematic review	Int J Environ Res Public Health, 2018;15(3):519
de Kluizenaar et al., 2020	25,000 adults (Netherlands)	>55 dB	Strong association between noise exposure and hypertension	Prospective cohort	Environ Res, 2020;182:109101
EEA, 2023	EU (27 countries)	>55 dB	~48,000 new CVD cases and 12,000 premature deaths annually	Environmental report	European Environment Agency, 2023
WHO, 2018 / 2023	Global	>53 dB	Noise as independent cardiovascular risk factor	Guidelines / Global report	WHO Environmental Noise Guidelines for the European Region, 2018; WHO, 2023
Daiber et al., 2019	Animal / experimental	60–70 dB	HPA activation, oxidative stress, endothelial dysfunction	Experimental study	Eur Heart J, 2019;40(30):2421–2428
Lee et al., 2021	150,000 adults (South Korea)	50–65 dB	Stronger effect among low-income groups and urban dwellers	Cohort study	Environ Int, 2021;146:106253
Vienneau et al., 2020	Swiss National Cohort	>55 dB	Increased CVD mortality linked to long-term exposure	Population-based cohort	Int J Hyg Environ Health, 2020;226:113491

Conclusions

Chronic exposure to traffic noise is a significant, modifiable risk factor for hypertension and cardiovascular diseases (CVD) (Münzel et al., 2025). The study results confirm the rationale for integrating environmental noise assessment into cardiovascular disease prevention strategies and public health programs. The World Health Organization (WHO) Environmental Noise Guidelines (2018) strengthened the link between transport noise and cardiovascular outcomes, recommending exposure limits of 53dB for road traffic noise and 45dB for night noise to minimize health risks (Basner & McGuire, 2018).

This review highlights compelling evidence linking chronic street noise exposure to an increased risk of hypertension and cardiovascular diseases (CVD), including ischemic heart disease, myocardial infarction, stroke, and heart failure (Hahad et al., 2023). Furthermore, the synergistic effect of noise and air pollution compounds these risks, underscoring the need for integrated strategies to counteract co-exposure to these environmental stressors (Davies et al., 2020; Hahn et al., 2022). The findings emphasize the urgent need for public health policies and strategies to mitigate traffic noise exposure. Public awareness campaigns and individual-level interventions, such as soundproofing homes and relocating bedrooms away from noise sources, can also play a significant role in reducing exposure. Future research should focus on the combined impact of noise and air pollution, as well as longitudinal studies to establish causal relationships and evaluate the effectiveness of mitigation strategies. By addressing these issues, it is possible to alleviate the negative impact of street noise on health and improve cardiovascular outcomes at the population level.

Disclosure

Author Contributions

The authors confirm contribution to the paper as follows:

- **Conceptualization:** Natalia Kruszewska, Oliwia Sędziak
- **Methodology:** Natalia Kruszewska, Oliwia Sędziak
- **Software:** Not applicable
- **Check:** Urszula Borucińska, Hanna Pietruszewska
- **Formal analysis:** Urszula Borucińska, Oliwia Sędziak
- **Investigation:** Hanna Pietruszewska, Sabina Skrzynecka
- **Resources:** Urszula Borucińska, Hanna Pietruszewska
- **Data curation:** Sabina Skrzynecka, Oliwia Sędziak
- **Writing - rough preparation:** Hanna Pietruszewska, Oliwia Sędziak
- **Writing - review and editing:** Natalia Kruszewska, Sabina Skrzynecka
- **Visualization:** Urszula Borucińska, Oliwia Sędziak
- **Supervision:** Natalia Kruszewska, Sabina Skrzynecka
- **Project administration:** Natalia Kruszewska, Hanna Pietruszewska

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