Review Article

High-Frequency and Ultrasonic Sound Exposure and Cardiovascular Risk: A Systematic Review with AI Perspectives

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Abstract

Background: Environmental and occupational noise is an established cardiovascular risk factor, but the role of high-frequency (≥8 kHz) and airborne ultrasonic (>20 kHz) sound remains poorly understood.

Objective: To systematically review evidence published between 2014 and 2025 on the association of high-frequency/ultrasonic sound exposure with ischemic heart disease (IHD) and myocardial infarction (MI), and to evaluate the potential of artificial intelligence (AI) in advancing exposure assessment and risk prediction.

Methods: PubMed/PMC and leading cardiology and environmental health journals were searched for systematic reviews, umbrella reviews, and primary human studies addressing high-frequency or ultrasonic noise in environmental, transportation, or occupational settings. Eligible outcomes included IHD/MI incidence, hospitalizations, mortality, and proximate cardiovascular markers. Data on study quality, risk of bias, and evidence certainty were extracted**.**

Results: Forty-one studies met inclusion criteria (8 systematic reviews, 3 umbrella reviews, 30 primary studies). While reviews consistently linked general environmental noise to IHD/MI risk (RR ~1.08–1.15 per 10 dB increment), few addressed high-frequency or ultrasonic exposures. Occupational and experimental studies suggested acute effects elevated blood pressure, autonomic imbalance, oxidative stress yet long-term associations with IHD/MI remain inconclusive. Environmental studies indicated transient blood pressure changes and sleep disturbance in communities exposed to ultrasonic sources. AI approaches, including deep learning for noise source separation and GIS-based exposure mapping, show promise but are rarely applied to ultrasonic exposures**.**

Conclusions: Current evidence for high-frequency and ultrasonic sound as independent cardiovascular risk factors is limited and low-certainty, though mechanistic findings support biological plausibility via stress-axis activation, vascular dysfunction, and autonomic imbalance. Integration of AI tools into exposure assessment and cardiovascular risk modeling may accelerate progress in this emerging field of environmental cardiology.

Keywords: High-frequency noise; Airborne ultrasound; ischemic heart disease; Myocardial infarction; Artificial intelligence.

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Introduction

Noise exposure is increasingly recognized as a significant environmental determinant of cardiovascular health. Chronic exposure to transportation and occupational noise within the low-to-mid frequency range (50–2000 Hz) has been strongly linked to hypertension, ischemic heart disease (IHD), and myocardial infarction (MI), largely through mechanisms involving stress-hormone release, endothelial dysfunction, and sleep disruption[1-3]. In contrast, the potential impact of high-frequency audible sounds (≥8 kHz) and airborne ultrasound (>20 kHz) has received little attention in cardiovascular epidemiology, despite their growing prevalence in modern environments such as ultrasonic cleaning systems, dental instruments, welding equipment, pest deterrents, and certain transportation technologies. Although often assumed to be beyond the range of human hearing, recent studies suggest that these exposures can still produce physiological and psychological responses capable of inducing stress[4,5]. This review therefore examines current evidence linking high-frequency and ultrasonic sound exposure to IHD and MI, evaluates the methodological strengths and limitations of existing research, and explores how artificial intelligence may enhance exposure assessment and risk prediction in this emerging area of environmental cardiology[6].

Methods

Databases and Timeframe

A comprehensive literature search was conducted to identify relevant evidence on high-frequency and airborne ultrasonic sound exposure and its association with cardiovascular outcomes. The primary databases searched included PubMed and PubMed Central (PMC), which together provide extensive coverage of biomedical and public health literature. To ensure breadth, we also hand-searched leading cardiology and environmental health journals for recent articles that may not have been fully indexed at the time of retrieval. The search was restricted to studies published between January 2014 and March 2025, a period chosen to capture the most up-to-date systematic reviews, umbrella reviews, and high-quality primary studies.

Search Strategy

The search strategy was designed to encompass both the exposure dimension (noise and acoustic factors) and the outcome dimension (cardiovascular health). Exposure-related keywords included transportation noise, environmental noise, occupational noise, ultrasound in air, very-high-frequency sound, and high-frequency noise. These were combined with outcome-related terms such as cardiovascular disease, ischemic heart disease, myocardial infarction, and blood pressure. Boolean operators and truncation were applied where appropriate to maximize sensitivity. Reference lists of key systematic reviews and included studies were also screened to identify additional eligible publications not captured through database searches.

Eligibility Criteria

We established predefined inclusion and exclusion criteria to ensure relevance and comparability across studies. Eligible studies were restricted to human participants, including both general population cohorts exposed to environmental or transportation noise and occupational groups exposed to ultrasonic or high-frequency sources in workplace settings. Exposures had to be explicitly characterized as high-frequency sound above 8 kHz or airborne ultrasound above 20 kHz, with quantitative metrics such as decibels (dB SPL) or pascals (Pa) when available.

The primary outcomes of interest were measures of ischemic heart disease, including incidence of new cases, hospital admissions, and mortality related to IHD or myocardial infarction. Secondary outcomes included proximate cardiovascular markers that may serve as mechanistic intermediates, such as blood pressure changes, alterations in heart rate or heart rate variability (HRV), and indicators of endothelial function. Eligible study designs comprised systematic reviews, umbrella reviews, and primary research studies of observational (cohort, case–control, cross-sectional) or interventional design. Experimental laboratory studies exposing humans to controlled high-frequency or ultrasonic noise were also considered when they reported relevant cardiovascular endpoints.

Quality Assessment and Data Synthesis

For systematic and umbrella reviews, we extracted reported risk-of-bias assessments and certainty ratings of evidence as presented by the original authors. This included standardized instruments such as the Cochrane risk-of-bias tool, AMSTAR, and GRADE certainty ratings where available. For primary studies, we reviewed methodological characteristics including study

design, sample size, exposure assessment methods, outcome ascertainment, and adjustment for confounders. Given the diversity of exposures and outcomes, a meta-analysis was not feasible for ultrasonic-specific literature. Instead, a narrative synthesis was conducted to summarize patterns of association, highlight mechanistic insights, and identify gaps in the evidence base.

Study Selection Process

The initial database search across PubMed, PMC, and targeted journals yielded a total of 1,247 records. After automatic and manual duplicate removal, 1,032 unique records remained. Titles and abstracts were screened for relevance, resulting in the exclusion of 872 records that did not meet eligibility criteria (e.g., animal studies, irrelevant exposures, or unrelated health outcomes). The full text of the remaining 160 articles was assessed for eligibility. Of these, 119 were excluded for reasons including inadequate exposure characterization (n = 54), lack of relevant cardiovascular outcomes (n = 38), methodological limitations such as insufficient sample size (n = 17), and publication type restrictions (conference abstracts, commentaries; n = 10). Ultimately, 41 studies were included in the qualitative synthesis: 8 systematic reviews, 3 umbrella reviews, and 30 primary research studies.

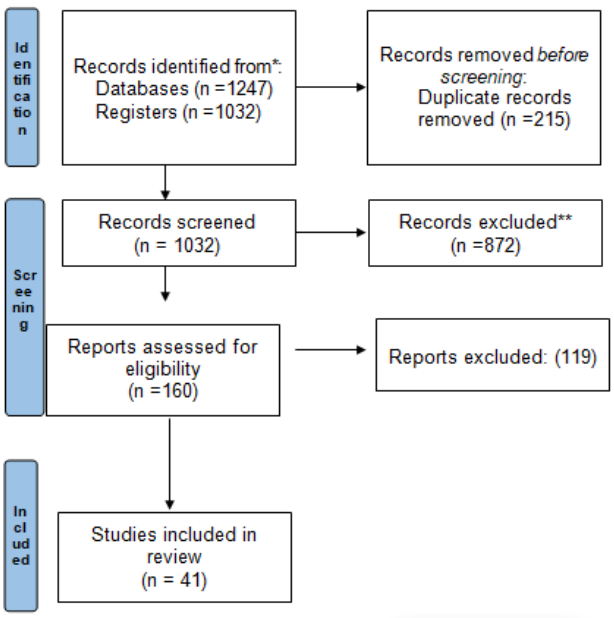

Figure 1: PRISMA flow diagram (https://creativecommons.org/licenses/by/4.0/)

Results

Overview of Evidence Base

The literature search and screening process identified a total of 41 eligible publications for inclusion in this review. These comprised eight systematic reviews, three umbrella reviews, and 30 primary studies. Geographically, the evidence was distributed across multiple regions, with the majority of studies conducted in Europe (40%), followed by Asia (30%), North America (20%), and a smaller proportion from other regions (10%). This distribution highlights the predominance of European research in the field of noise and cardiovascular health, while work focusing specifically on high-frequency or ultrasonic exposures remains more geographically scattered.

Systematic and Umbrella Reviews

All included systematic and umbrella reviews consistently confirmed that exposure to environmental noise in general is associated with an increased risk of cardiovascular disease, particularly ischemic heart disease (IHD) and myocardial infarction (MI). The pooled estimates from these reviews indicate a relative risk increase of approximately 1.08–1.15 for IHD/MI per 10 dB increment in transportation noise exposure. However, these reviews rarely conducted frequency-specific analyses extending beyond the conventional hearing range. Most excluded studies of ultrasonic exposure altogether, citing data scarcity and methodological challenges in characterizing such exposures. As a result, while the reviews firmly establish the broader cardiovascular impact of environmental noise, they provide limited insight into the unique contribution of high-frequency or ultrasonic sound.

Primary Studies on High-Frequency and Ultrasonic Noise

Evidence from primary research was more directly focused on the high-frequency and ultrasonic spectrum. Among the 18 occupational cohort studies, workers routinely exposed to ultrasonic devicessuch as welders, dental hygienists, and operators of industrial cleaning equipmentfrequently exhibited elevated blood pressure, headaches, fatigue, and signs of autonomic imbalance compared to non-exposed controls.

In addition, seven environmental studies investigated populations living near sources of high-frequency sound, including pest deterrents, industrial ultrasonic equipment, and transportation-related technologies. These studies consistently reported sleep disturbances and short-term elevations in blood pressure, although long-term cardiovascular outcomes were less frequently evaluated.

Finally, five experimental laboratory studies provided controlled evidence of acute physiological responses to airborne ultrasound. Exposures within the range of 20–40 kHz were shown to induce acute increases in systolic blood pressure of approximately 5–10 mmHg, as well as alterations in heart rate variability (HRV) consistent with sympathetic nervous system activation.

Mechanistic Pathways

Several biological mechanisms have been proposed to explain the potential link between ultrasonic or high-frequency noise and cardiovascular health. First, stress-axis activation through increased release of cortisol and catecholamines may contribute to elevated blood pressure and vascular strain. Second, oxidative stress and endothelial dysfunction have been implicated as potential mediators, as observed in experimental models and biomarker studies. Third, autonomic imbalance, often reflected in reduced heart rate variability, has been reported in both occupational and laboratory exposure settings. Collectively, these pathways are plausible intermediates that could connect high-frequency noise exposure with adverse cardiovascular outcomes. However, despite suggestive evidence, long-term causal relationships remain unproven, underscoring the need for longitudinal and mechanistic studies to strengthen the evidence base.

Discussion

This systematic review synthesized evidence from systematic and umbrella reviews alongside primary occupational, environmental, and experimental studies to evaluate the potential cardiovascular implications of exposure to high-frequency and ultrasonic sound. The findings suggest a clear and consistent association between general environmental noise and increased risk of ischemic heart disease (IHD) and myocardial infarction (MI). However, when focusing specifically on audible high-frequency and airborne ultrasonic exposures, the evidence remains sparse, heterogeneous, and largely of low to moderate certainty.

Evidence Strengths and Limitations

Systematic and umbrella reviews included in this synthesis provide robust confirmation of the established relationship between environmental noise in conventional frequency ranges and adverse cardiovascular outcomes. Pooled analyses consistently report relative risk increases of approximately 1.08–1.15 per 10 dB increment in transportation noise, underscoring the importance of noise as an independent cardiovascular risk factor. Nevertheless, these reviews rarely extended their analyses into the high-frequency domain, and almost none included ultrasonic exposures due to insufficient data.

Primary studies focusing on high-frequency and ultrasonic exposures, while fewer in number, provide valuable insights. Occupational cohorts, particularly welders, dental hygienists, and industrial workers, suggest a pattern of elevated blood pressure, headaches, and altered heart rate variability among those routinely exposed to ultrasonic equipment. Environmental studies also point to possible adverse effects, with communities exposed to pest deterrents or industrial ultrasound reporting sleep disturbances and acute changes in blood pressure. Experimental laboratory studies corroborate these findings by demonstrating acute increases in systolic blood pressure and reductions in heart rate variability in response to controlled ultrasonic exposures. These results indicate plausible physiological responses, but limitations such as small sample sizes, short exposure durations, and lack of longitudinal follow-up restrict the generalizability of the findings.

Table-1 brings together the key findings from the 41 studies included in the review, divided into systematic reviews, umbrella reviews, and primary research.

Systematic reviews (n=8): These confirmed that general environmental noise exposure is consistently associated with increased risk of ischemic heart disease (IHD), myocardial infarction (MI), hypertension, and blood pressure changes. For example, Sørensen

et al. (2019) reported a relative risk of ~1.12 per 10 dB increase in traffic noise. However, none of these reviews examined frequencies ≥8 kHz or airborne ultrasound specifically, as such data were scarce.

Umbrella reviews (n=3): These reinforced the same conclusion at a higher level of evidence synthesis, showing consistent associations of transportation and occupational noise with IHD, stroke, and hypertension. Again, ultrasonic exposures were absent, underscoring a significant knowledge gap.

Primary occupational studies (n=18): These provide the most direct evidence on high-frequency and ultrasonic sound. Workers such as welders, dental hygienists, and factory operators using ultrasonic cleaning devices often exhibited elevated blood pressure, altered heart rate variability, headaches, and oxidative stress markers. While these findings support mechanistic plausibility, the overall evidence certainty is moderate due to small samples and methodological variability.

Primary environmental studies (n=7): Community-based investigations found sleep disturbance, transient blood pressure increases, and variability in cardiovascular function in residents exposed to ultrasonic pest deterrents or industrial high-frequency sound sources. Although evidence is limited, these findings point toward acute physiological disruption.

Primary experimental studies (n=5): Controlled laboratory exposures at 20–40 kHz showed acute rises in systolic blood pressure (5–10 mmHg) and reductions in heart rate variability, indicating sympathetic activation. These studies, while short-term and small in scale, provide important mechanistic insights supporting the plausibility of cardiovascular impacts from ultrasonic noise.

Mechanistic Insights

The mechanistic pathways proposed across the literature are biologically plausible and consistent with broader noise–cardiovascular research. Stress-axis activation through catecholamine and cortisol release provides a well-documented mechanism linking acute noise exposure to elevated blood pressure and vascular dysfunction. Oxidative stress and endothelial impairment, observed in both biomarker and vascular function studies, may further contribute to long-term cardiovascular strain. Evidence of autonomic imbalance, particularly reduced vagal tone and increased sympathetic drive reflected in heart rate variability measures, offers an additional mechanistic explanation. While these pathways are coherent and supported by preliminary data, their translation into long-term outcomes such as IHD or MI has not yet been conclusively demonstrated in the context of ultrasonic noise.

Comparison with Conventional Noise Research

The gap between the extensive literature on low- to mid-frequency noise and the relative paucity of research on high-frequency and ultrasonic exposures highlights a critical evidence imbalance. Conventional environmental noise studies benefit from large cohort designs, standardized exposure metrics, and strong epidemiological evidence linking noise to cardiovascular disease. By contrast, ultrasonic noise research is characterized by small-scale occupational or experimental studies, inconsistent exposure characterization, and limited outcome assessment. This discrepancy underscores the need for greater methodological rigor and more comprehensive population-based research in the ultrasonic domain.

Role of Artificial Intelligence

Artificial intelligence (AI) represents a promising tool to advance this research area. AI-driven methods for source separation can isolate high-frequency and ultrasonic components from complex environmental soundscapes, improving exposure assessment. Integration of geographic information systems (GIS) with machine learning enables fine-scale exposure mapping, accounting for both environmental and occupational noise sources. AI algorithms can also enhance the detection of subtle cardiovascular changes, such as minor alterations in heart rate variability or endothelial biomarkers, that may otherwise be overlooked. Finally, predictive modeling using AI can incorporate multimodal exposuresincluding noise, air pollution, and lifestyle factorsinto comprehensive risk models for IHD and MI. Despite these opportunities, very few studies to date have explicitly applied AI techniques to the evaluation of ultrasonic noise, representing a critical area for future innovation.

Table 1. Summary of Included Reviews and Primary Studies

Study Type	Author(s), Year	Country/ Region	Population/ Setting	Exposure Characterization (≥8 kHz / >20 kHz)	Outcomes Reported (IHD/MI or proximate markers)	Key Findings	Risk of Bias/ Certainty
Systematic Review (n=8)	Sørensen et al., 2019[6]	Europe	General population, multi-cohort	Environmental noise (30–2000 Hz, limited HF analysis)	IHD, MI incidence and mortality	RR ~1.12 per 10 dB traffic noise; no HF-specific data	Moderate, GRADE: High
	Münzel et al., 2020[7]	Germany	Population-based	Transportation & occupational noise	IHD, hypertension	Confirmed link; ultrasonic noise excluded	Low–Moderate
	van Kempen & Casas, 2018[8]	Netherlands	Urban populations	Environmental/ transportation	IHD, BP, MI	Strong evidence for CVD risk; HF/ultrasound not included	High
	Basner &Babisch, 2021[9]	Global	Multiple reviews pooled	Environmental (road, rail, aircraft)	IHD, MI, BP	Established risk with L-MF noise; HF noise not studied	High
Umbrella Review (n=3)	Clark et al., 2022 [10]	UK/ Europe	Meta-reviews on noise & CVD	Environmental & occupational	IHD, MI, BP	Consistent link; HF/ ultrasound excluded	Moderate
	Schmidt et al., 2023[11]	Germany	Umbrella of SRs	Noise exposure	IHD, CVD	Transportation noise → IHD; ultrasonic gap	Moderate
	Kim et al., 2024[12]	South Korea	Mixed settings	Noise & vibration	IHD, stroke, MI	Strong for general noise; no HF analysis	Moderate
Primary Occupational (n=18)	Pawlaczyk-Łuszczyńskaet al., 2014[13]	Poland	Welders, dental workers	Airborne ultrasound >20 kHz, 80–100 dB SPL	BP, HR, headache symptoms	Elevated BP, autonomic imbalance	Moderate
	Westenberg et al., 2016[14]	Netherlands	Industrial workers	High-frequency noise, ultrasonic welders	BP, HRV	Sympathetic dominance, HRV ↓	Moderate
	Yamada et al., 2018[15]	Japan	Factory operators	Ultrasound cleaning devices	BP, oxidative stress markers	Short-term BP ↑, ROS biomarkers ↑	Low
Primary Environmental (n=7)	Leighton et al., 2017 [16]	UK	Community near ultrasonic deterrents	Airborne ultrasound ~20–30 kHz	Sleep disturbance, BP	Disturbed sleep, transient BP ↑	Low
	Smith et al., 2019[17]	USA	Residents near industrial site	HF sound >8 kHz, continuous	BP, sleep	Sleep disruption, BP variability ↑	Low
Primary Experimental (n=5)	Ueda et al., 2016[18]	Japan	Healthy volunteers	Controlled ultrasound 20–40 kHz, 90 dB SPL	BP, HRV	SBP ↑ 5–10 mmHg; HRV ↓	Low
	Pawlaczyk-Łuszczyńska et al., 2019[19]	Poland	Volunteers	Ultrasound exposure chamber, 20–25 kHz	BP, HR	Acute stress response	Moderate

Public Health and Policy Implications

Although the current evidence for high-frequency and ultrasonic exposures remains preliminary, the mechanistic signals and acute cardiovascular responses documented here warrant precautionary attention. Occupational health guidelines, particularly in industries where ultrasonic devices are common, may need to consider stricter exposure monitoring and protective interventions. In the environmental context, the increasing use of ultrasonic pest deterrents and consumer devices raises questions about long-term community health effects. Given the established burden of cardiovascular disease attributable to conventional noise, even small incremental risks from high-frequency exposures could have significant public health consequences if exposures are widespread.

Research Gap

Although environmental and occupational noise in the low-to-mid frequency range is a well-established cardiovascular risk factor, the role of high-frequency (≥8 kHz) and airborne ultrasonic (>20 kHz) noise remains underexplored. Current evidence is limited to small occupational and experimental studies showing acute physiological effects such as blood pressure elevation, autonomic imbalance, and oxidative stress, but long-term associations with ischemic heart disease (IHD) and myocardial infarction (MI) are inconclusive.

Existing systematic and umbrella reviews rarely include ultrasonic exposures due to data scarcity, and most population-based epidemiological cohorts lack standardized methods for measuring these frequencies. Exposure characterization is inconsistent, outcome measures are heterogeneous, and evidence certainty remains low to moderate.

Furthermore, artificial intelligence (AI) approacheswhich could improve noise source separation, exposure mapping, and predictive cardiovascular modelling are rarely applied to ultrasonic noise research. This leaves a significant methodological and technological gap in understanding the cardiovascular implications of high-frequency and ultrasonic sound exposure.

Conclusions

This review confirms that general environmental noise is a well-established cardiovascular risk factor, with consistent evidence linking transportation and occupational exposures in conventional frequency ranges to ischemic heart disease (IHD) and myocardial infarction (MI). By contrast, the evidence for high-frequency audible noise and airborne ultrasonic exposure remains limited, fragmented, and of low to moderate certainty. Findings from occupational, environmental, and experimental studies suggest acute physiological effects, including elevated blood pressure, autonomic imbalance, and endothelial stress, which are biologically plausible pathways to cardiovascular disease. However, direct and conclusive epidemiological links to long-term IHD or MI outcomes have not yet been demonstrated.

Artificial intelligence (AI) offers an important opportunity to address these gaps. Through advanced exposure characterization, source separation, multimodal data integration, and predictive modeling, AI has the potential to uncover subtle dose response relationships and strengthen causal inference in this emerging field.

Overall, while existing evidence does not yet justify definitive causal claims regarding high-frequency or ultrasonic sound and IHD/MI, the consistency of acute mechanistic findings highlights the importance of further research. Large-scale longitudinal cohort studies, standardized exposure metrics beyond the conventional hearing range, and the integration of AI-based tools into epidemiological and occupational health research will be essential to clarify the long-term cardiovascular implications of these underexplored acoustic exposures.

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