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REVIEW

Long-Term Exposure to Traffic Noise and Risk of Incident Cardiovascular Diseases: a Systematic Review and Dose-Response Meta-Analysis

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Abstract While noise pollution from transportation has become an important public health problem, the relationships between different sources of traffic noise and cardiovascular diseases (CVDs) remain inconclusive. A comprehensive meta-analysis was therefore conducted to quantitatively assess the effects of long-term exposure to road traffic, railway, and aircraft noise on CVDs and relevant subtypes. We systematically retrieved PubMed, Embase, and Web of Science for articles published before April 4, 2022. Summary relative risks (RRs) and 95% confidence intervals (CIs) were estimated by the fixed- or random-effects models. In total, 23 articles were included in our meta-analysis. The risk of CVDs increased by 2% (RR 1.020, 95% CI 1.006–1.035) and 1.6% (RR 1.016, 95% CI 1.000–1.032) for every 10 dB increment of road traffic and aircraft noise. For CVD subtypes, the risk increased by 3.4% (1.034, 1.026–1.043) for stroke and 5% (1.050, 1.006–1.096) for heart failure with each 10 dB increment of road traffic noise; the risk of atrial fibrillation increased by 1.1% (1.011, 1.002–1.021) with each 10 dB increment of railway noise; and the risk increased by 1% (1.010, 1.003–1.017) for myocardial infarction, 2.7% (1.027, 1.004–1.050) for atrial fibrillation, and 2.3% (1.023, 1.016–1.030) for heart failure with each 10 dB increment in aircraft noise. Further, effects from road traffic, railway, and aircraft noise all followed positive linear trends with CVDs. Long-term exposure to traffic noise is positively related to the incidence risk of cardiovascular events, especially road traffic noise which significantly increases the risk of CVDs, stroke, and heart failure.

Keywords Traffic noise · Cardiovascular diseases · Cohort study · Dose-response

Highlights
1. Long-term exposure to traffic noise could increase the risk of CVDs.
2. Effects on CVDs of traffic noise from road, rail, and aircraft followed positive linear trends.

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Introduction

Cardiovascular diseases (CVDs) constitute the leading cause of death worldwide [1], with 18.5 million people dying from CVDs in 2019 and roughly one-third of all deaths globally [2, 3]. Given the prevalence of CVDs, the identification of modifiable risk factors so as to reduce the burden of CVDs is essential. Cumulative studies have suggested that environmental factors may facilitate the progression of CVDs [4]. Traffic noise has become an important public concern due to rapid urbanization and economic growth. The World Health Organization (WHO) alleges that more than one million healthy life years are lost in Europe each year because of traffic noise [2]. Besides, it is estimated that chronic exposure to environmental noise causes 12,000 premature deaths and 48,000 new cases of ischemic heart disease (IHD) annually in European territory [5].

Noise, as a stressor, may trigger the release of stress hormones [6, 7], causing inflammation [8, 9] and oxidative stress [10, 11] and influencing the cardiovascular system. Increasing epidemiological studies indicate that chronic exposure to traffic noise is associated with an increased risk of CVDs [12–15], but the relationship is still inconclusive because of inconsistent, controversial findings in studies [16–19]. Previous meta-analyses have concluded that traffic noise is linked to an increased risk of coronary heart disease [20, 21]. While one meta-analysis [22] published in 2014 reported a nonsignificant relationship between traffic noise and CVDs, it included only cross-sectional studies and failed to quantitatively estimate the risk of CVDs. More recently, several large-scale longitudinal studies have been published on traffic noise and CVDs [14, 15, 23–26]. To date, no meta-analysis examining the correlation between transportation noise and heart failure (HF) has been published. Additionally, the strength and shape of the dose-response correlations between traffic noise from roads, rail, and aircraft sources and CVDs remain unclear. We therefore carried out a comprehensive systematic review and meta-analysis of all available cohort studies to synthesize the evidence regarding the relationships between CVDs, stroke, myocardial infarction, atrial fibrillation, and HF morbidity and traffic noise from roads, railways, and aircraft and to investigate the dose-response relationships involved.

Method

Search Strategy

PubMed, Embase, and Web of Science were retrieved for relevant English-language reports published before 4 April 2022. The search strategy of merging MeSH terms and free-text terms included *noise, transportation noise, traffic noise, environmental noise, cardiovascular diseases, and cohort study*. Details are shown in Supplementary Material Table 1. We also manually retrieved the bibliographies of included articles and relevant review articles for additional studies. Our meta-analysis was performed and reported in alignment with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) [27] and registered on the International Prospective Register of Systematic Reviews (PROSPERO) with the registration number CRD42022321347.

Selection Criteria

Studies were considered to be eligible if they were (1) cohort studies in which participants were 18 years or older at baseline; (2) studies that reported long-term traffic noise exposure from roads, railways, or aircraft, which were measured or model estimated; (3) studies that assessed the incidence of CVDs, IHD, stroke, myocardial infarction (MI), atrial fibrillation (AF), and HF; and (4) studies that provided odds ratios (ORs), relative risks (RRs), or hazard ratios (HRs) with 95% confidence intervals (CIs). If there were multiple studies exploring the same outcome in the same cohort population, we selected the study with the most information or the largest sample size or the longest follow-up duration.

Data Extraction and Quality Assessment

Two reviewers (X.F. and L.W.) separately collected data from the studies. Information collected included the first author, publication year, study country, data source, sample size, follow-up duration, participants' characteristics at baseline (sex and age), traffic noise sources and assessment methods, outcome ascertainment, fully adjusted ORs, RRs or HRs and 95% CIs for each noise level, and adjusted confounding factors.

The two reviewers adopted the Newcastle Ottawa Scale (NOS) to evaluate the quality of retained studies [28]. Studies were classified as low, medium, and high quality based on NOS scores, with 0–3, 4–6, and 7–9 points ascribed, respectively. Possible contradictions arising from data extraction and quality assessment were discussed with the third author (D.H.).

Data Synthesis and Analysis

The included studies applied different noise metrics to describe the effects from traffic noise on CVDs, metrics such as L_{den}, L_{Aeq24h}, L_{Aeq16h}, and L_{night} (definitions are shown in Supplementary Material Table 2). We selected the European Union standard L_{den} as a unified noise metric. When it was not available, we prioritized the extraction of noise data in the following order: L_{Aeq24h}, L_{Aeq16h}, and L_{night}. Before entering into the analysis, various noise metrics were converted to L_{den} using these criteria: L_{Aeq24h}+3.6 dB, $L_{Aeq(6–22h)}$+2.3 dB, $L_{Aeq(7–23h)}$+2.4 dB, and $L_{night(8h)}$+8.3 dB for road traffic noise [29]. We assigned the intermediate noise level of each interval as the mean exposure corresponding to the risk estimate. In the case of open intervals, the range was assumed to be the same as the neighboring one [30].

If different subtypes of CVDs or different sources of traffic noise were reported in an article, we regarded them as independent studies. If an article only reported subtypes of stroke, we adopted the fixed-effects model to combine the risk estimates. RRs (95% CI) were considered as the unified risk estimate for the included studies. For studies reporting HRs (95% CI), it was assumed that HRs were equal to RRs [31]. Since the incidence rates of outcomes in the general population were less than 10% [32], we considered that the ORs reported in the original studies were approximate to the RRs [33]. If the specific number of cases or participants for a group was missing, it was calculated according to the corresponding risk estimates and total cases or participants [34].

The study-specific dose-response association for per 10 dB increment of traffic noise was evaluated using generalized least-squares regression [35]. Pooled RRs (95% CI) of different outcomes (CVDs, stroke, MI, AF, and HF) for the highest versus lowest level in the dichotomous analysis and per 10 dB increment were assessed with a fixed-effects model when I^2 statistic was less than 50%; otherwise, a random-effects model was utilized. Additionally, the nonlinear trends were evaluated by modeling traffic noise using a restricted cubic spline, with three knots placed at the 25th, 50th, and 75th percentiles of the distribution [36]. Only studies reporting at least three levels of noise were used to estimate the potential linear or nonlinear trends between traffic noise and different types of cardiovascular events.

Heterogeneity was examined using Cochran Q and I^2 statistics [37]. $P<0.1$ was regarded as statistically significant for the Q statistic, while the I^2 values less than 50% represented low heterogeneity. Subgroup analysis was conducted according to sex, region, follow-up years, sample size, study quality, noise metrics, and adjustments (age, income, smoking, drinking, air pollution, and other noise exposure). The P value for the heterogeneity between subgroups was calculated with meta-regression [38]. Sensitivity analysis was undertaken by excluding one study at a time to evaluate the steadiness of the results in the analysis that included more than 5 studies. Publication bias (8 or more studies) was evaluated by funnel plot and Egger's test [39].

$P<0.05$ was regarded as statistically significant, and all statistical tests were two-sided and conducted with Stata 14.0 (Stata Corp, College Station, TX, USA).

Results

Literature Search and Study Characteristics

In brief, 6593 articles were searched from PubMed, Embase, and Web of Science and the reference lists of related articles. After removing duplicates (n=1661), potentially relevant articles that met the inclusion criteria were screened from 4932 articles by reading the titles and abstracts, with the final remaining 52 articles evaluated by reviewing the full text. Of these 52 articles, we further excluded 29 due to lack of relevant exposure, outcome, or risk estimate data (n=9), duplicate data (n=10), conference or editorial articles (n=4), and review or meta-analysis papers (n=5), while one article was unavailable. Consequently, 23 articles (including 57 studies) were included in the meta-analysis (Fig. 1).

Included articles covered a total of 18,503,167 participants, the sample size ranging from 420 to 8,610,000, with only 4 articles referencing less than

10,000 participants. Among the 23 included articles, 21 [12, 14–19, 23, 24, 26, 40–50] were from European countries and 2 [25, 51] from Canada. Four articles [15, 17, 23, 24] reported results for road traffic, railway, and aircraft noise simultaneously, while others only assessed one or two sources of traffic noise. All noise exposures were estimated by the model except for one article where the noise exposure was actually measured [40]. Sixteen articles [12, 14–18, 23, 24, 41, 42, 44, 46–50] adopted the European Union standard L_{den} as the noise metric, while the remaining 7 articles [19, 25, 26, 40, 43, 45, 51] selected other noise metrics (L_{Aeq24h}, L_{Aeq16h}, or L_{night}). The characteristics of relevant studies are listed in Supplementary Material Table 3. The quality assessment of included studies yielded a mean NOS score of 8.04 (Supplementary Material Table 4).

Traffic Noise and CVDs

Road Traffic Noise and CVDs

We included 16 studies [14, 15, 17–19, 23–25, 40, 42, 47, 48, 51] in dichotomous analysis. Compared with the lowest noise level, the pooled risk of CVDs was 1.068 (95% CI 1.023–1.115, I^2=93.5%, $P_{heterogeneity}$<0.001, Fig. 2) for the highest noise level. Egger's test and funnel plot did not detect publication bias (P=0.742, Supplementary Material Fig. 1). Sensitivity analysis showed consistent results.

In total, the dose-response analysis contained 29 studies [12, 14–19, 23–26, 40–42, 44–51], with elevated road traffic noise increasing the risk of CVDs (RR 1.020, 95% CI 1.006–1.035, I^2=90.6%, $P_{heterogeneity}$<0.001, Fig. 3). The Egger's test (P=0.800) and funnel plot showed no publication bias (Supplementary Material Fig. 2a). Sensitivity analysis showed a similar association. As 13 studies only reported road traffic noise as a continuous variable, 16 studies [14, 15, 17–19, 23–25, 40, 42, 47, 48, 51] were included to fit the dose-response relationship curve. Finally, a positive linear relationship between road traffic noise and CVDs was observed ($P_{nonlinearity}$=0.339, Fig. 4a). We found evidence of heterogeneity among subgroups stratified by adjustment for age (P=0.009) and income (P=0.019), with a stronger relationship for studies with adjustment for age and income (Table 1).

Railway Noise and CVDs

For railway noise, the pooled RR for CVDs was 1.065 (95% CI 1.016–1.116, I^2=29.7%, $P_{heterogeneity}$=0.234, Fig. 2) in the dichotomous analysis of 4 studies [12, 17, 23, 24]. In total, 8 studies [12, 14, 15, 17, 23, 24,

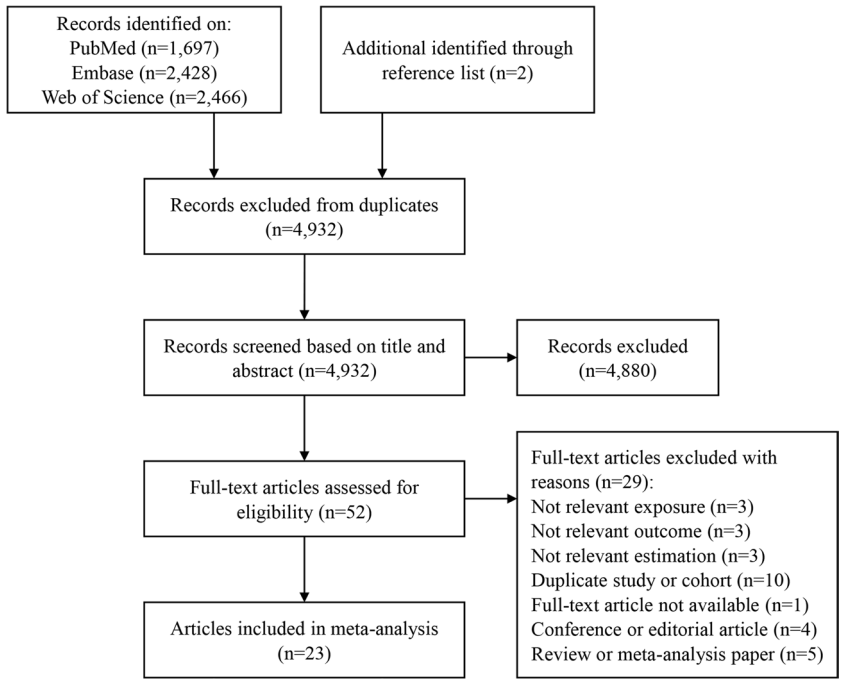

Fig. 1 Flowchart of article selection

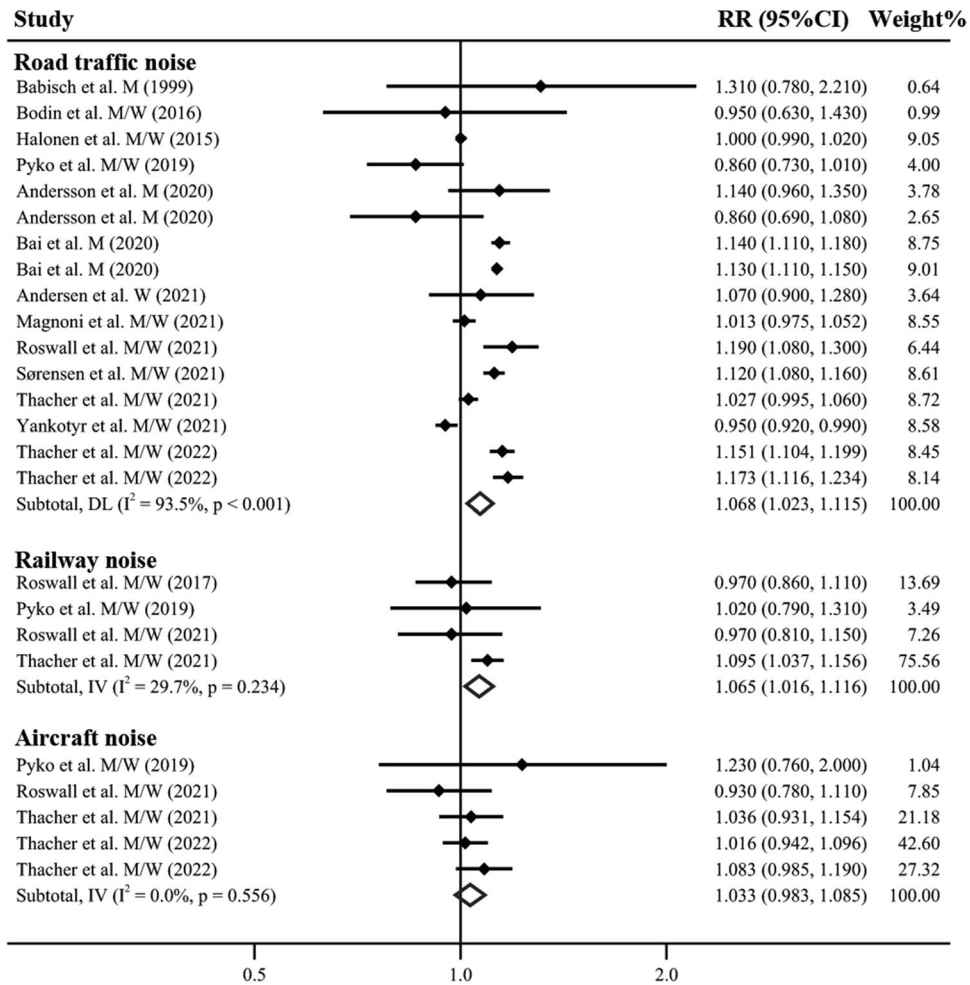

Fig. 2 Forest plot of pooled relative risks for cardiovascular disease morbidity with road traffic, railway, and aircraft noise (highest versus lowest levels). RR, relative risk; CI, confidence interval; M, men; W, women

44] were included in the dose-response analysis. The pooled RR was 0.986 (95% CI 0.968–1.004, I^2=83.4%, $P_{heterogeneity}$<0.001, Fig. 3) for each 10 dB increment. Egger's test (P=0.472) and funnel plot showed no publication bias (Supplementary Material Fig. 2b). Sensitivity analysis showed consistent results. Because 4 studies only reported railway noise as a continuous variable, 4 studies [12, 17, 23, 24] were finally used to fit the dose-response relationship curve. We identified a positive linear relationship between railway noise and CVDs ($P_{nonlinearity}$=0.759, Fig. 4b). The associations between railway noise and CVDs were not significant among most subgroups, but the heterogeneity reduced among the subgroups stratified by sex and adjustment for air pollution (Table 1).

Aircraft Noise and CVDs

For aircraft noise, the pooled RR for CVDs was 1.033 (95% CI 0.983–1.085, I^2 =0.0%, $P_{heterogeneity}$=0.556, Fig. 2) in the dichotomous analysis of 5 studies [15, 17, 23, 24]. We included 8 studies [15, 17, 23, 24, 45] in the dose-response analysis which showed that the risk of CVDs increased by 1.6% (RR 1.016, 95% CI 1.000–1.032, I^2=72.7%, $P_{heterogeneity}$=0.001, Fig. 3) for each 10 dB increase. Egger's test (P=0.612) and funnel plot indicated no evidence of publication bias

	No of studies	No of participants	No of cases	Pooled RR (95% CI)	Heterogeneity I^2 (P value)	Egger's test P value
CVDs						
Road	29	18,503,167	1,331,508	1.020 (1.006-1.035)	90.6% (<0.001)	0.800
Railway	8	6,291,239	673,675	0.986 (0.968-1.004)	83.4% (<0.001)	0.472
Aircraft	8	6,244,842	480,802	1.016 (1.000-1.032)	72.7% (0.001)	0.612
Stroke						
Road	8	13,860,116	279,519	1.034 (1.026-1.043)	0.0% (0.478)	0.703
Railway	2	3,752,484	195,580	0.978 (0.964-0.993)	0.0% (0.457)	-
Aircraft	2	101,479	7,740	1.011 (0.939-1.089)	0.0% (0.989)	-
MI						
Road	10	6,332,126	181,022	1.019 (0.987-1.051)	91.0% (<0.001)	0.890
Railway	2	2,589,139	79,228	0.984 (0.960-1.008)	66.5% (0.084)	-
Aircraft	2	2,538,815	76,843	1.010 (1.003-1.017)	0.0% (0.387)	-
AF						
Road traffic	5	3,893,091	279,781	1.000 (0.995-1.005)	34.0% (0.195)	-
Railway	2	3,655,210	272,448	1.011 (1.002-1.021)	12.7% (0.285)	-
Aircraft	1	3,604,968	269,756	1.027 (1.004-1.050)	-	-
HF						
Road traffic	5	3,807,029	179,754	1.050 (1.006-1.096)	92.9% (<0.001)	-
Railway	1	2,538,395	79,358	0.988 (0.970-1.005)	-	-
Aircraft	1	2,538,395	79,358	1.023 (1.016-1.030)	-	-

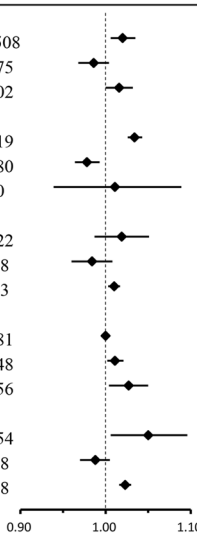

Fig. 3 Summary relative risks for per 10 dB increment on road traffic, railway, and aircraft noise with cardiovascular disease and its subtypes. RR, relative risk; CI, confidence interval; MI, myocardial infarction; AF, atrial fibrillation; HF, heart failure

(Supplementary Material Fig. 2c). Sensitivity analysis showed consistent results. We found a positive linear relationship between aircraft noise and CVDs (n=4 [14, 18, 19, 23], $P_{\text{nonlinearity}}$=0.592, Fig. 4c) and evidence of heterogeneity among subgroups stratified by sex (P=0.015) (Table 1).

Traffic Noise and Stroke

Road Traffic Noise and Stroke

Four studies [14, 18, 19, 23] were included in the dichotomous analysis for stroke. The pooled RR was 1.090 (95% CI 1.039–1.144, I^2=75.7%, $P_{\text{heterogeneity}}$=0.006, Supplementary Material Table 5). We included 8 studies [14, 16, 18, 19, 23, 26, 45] in the dose-response analysis, and the incidence risk of stroke rose by 3.4% (RR 1.034, 95% CI 1.026–1.043, I^2=0.0%, $P_{\text{heterogeneity}}$=0.478; Fig. 3) for each 10 dB increment. Employing Egger's test and funnel plot, we failed to detect publication bias (P=0.703, Supplementary Material Fig. 3a). Sensitivity analysis gave similar results. Four studies [14, 18, 19, 23] were included to model the restricted cubic splines, with a nonlinear correlation between road traffic noise and stroke observed ($P_{\text{nonlinearity}}$=0.001, Supplementary Material Fig. 4a). The curve rose rapidly until road traffic noise reached 62 dB and then flattened out.

Railway, Aircraft Noise, and Stroke

For railway noise, the pooled RR was 0.970 (95% CI 0.810–1.150) for the dichotomous analysis which included 1 study [23] (Supplementary Material Table 5), while the pooled RR was 0.978 (95% CI 0.964–0.993, I^2=0.0%, $P_{\text{heterogeneity}}$=0.457, Fig. 3) for every 10 dB increment in the dose-response analysis of 2 studies [14, 23]. For aircraft noise, we included 1 study [23] in the dichotomous analysis, obtaining a pooled RR of 0.930 (95% CI 0.780–1.110, Supplementary Material Table 5). The dose-response analysis involving two studies [23, 45] showed a summary risk estimate of 1.011 (95% CI 0.939–1.089, I^2=0.0%, $P_{\text{heterogeneity}}$=0.989, Fig. 3) with each 10 dB increment.

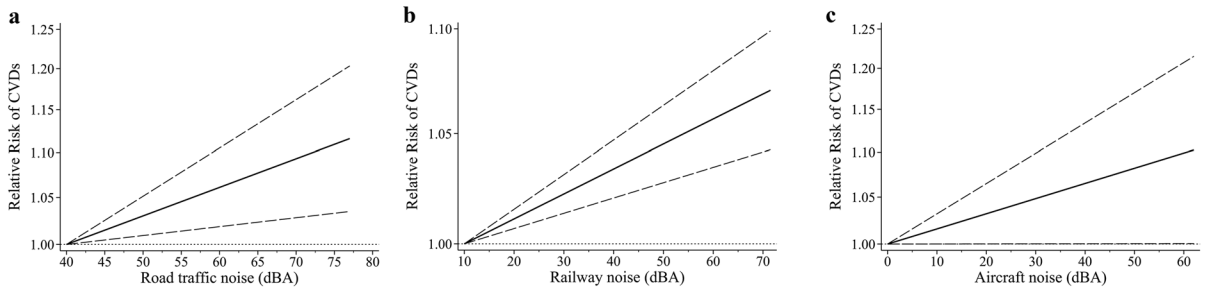

Fig. 4 Dose-response associations of **a** road traffic noise, **b** railway noise, and **c** aircraft noise on risk of cardiovascular diseases by the generalized least-squares regression. RR, relative risk; CI, confidence interval

Traffic Noise and Myocardial Infarction

Road Traffic Noise and Myocardial Infarction

Six studies [15, 18, 25, 42, 43, 51] were used in the dichotomous analysis with the pooled RR of 1.037 (95% CI 0.949–1.133, I^2=92.7%, $P_{heterogeneity}$<0.001, Supplementary Material Table 5). Sensitivity analysis gave consistent results. Although a potential linear relationship between road traffic noise and MI was identified (n=6 [15, 18, 25, 42, 43, 51], $P_{nonlinearity}$=0.081, Supplementary Material Fig. 4b), the pooled RR was nonsignificant (RR 1.019, 95% CI 0.987–1.051, I^2=91.0%, $P_{heterogeneity}$<0.001, Fig. 3) for each 10 dB increment in the dose-response analysis which included 10 studies [12, 15, 18, 25, 26, 42, 43, 45, 50, 51]. Egger's test and funnel plot detected no publication bias (P=0.890, Supplementary Material Fig. 3b). Sensitivity analysis gave consistent results. The correlation between road traffic noise and MI was not significant among most subgroups, except for studies citing nighttime noise levels, with adjustment for age and income and without adjustment for air pollution (Supplementary Material Table 6).

Railway, Aircraft Noise, and Myocardial Infarction

For railway noise, the pooled RR was 0.970 (95% CI 0.860–1.110) for the dichotomous analysis included 1 study [12] (Supplementary Material Table 5). The pooled RR was 0.984 (95% CI 0.960–1.008; I^2=66.5%, $P_{heterogeneity}$=0.084, Fig. 3) for each 10 dB increase in the dose-response analysis of 2 studies [12, 15]. For aircraft noise, we included 1 study [15] in the dichotomous analysis, and the pooled RR was 1.071 (95% CI 0.977–1.174, Supplementary Material Table 5). The dose-response analysis covering 2 studies [15, 45] showed a pooled RR of 1.010 (95% CI 1.003–1.017, I^2=0.0%, $P_{heterogeneity}$=0.387, Fig. 3) with each 10 dB increment.

Traffic Noise and Atrial Fibrillation

Road Traffic Noise and Atrial Fibrillation

We included 4 studies [24, 43, 47, 48] in the dichotomous analysis on AF. The pooled RR was 1.016 (95% CI 0.987–1.045, I^2=44.3%, $P_{heterogeneity}$=0.146, Supplementary Material Table 5). Although a potential linear trend between road traffic noise and AF was observed (n=4 [24, 43, 47, 48], $P_{nonlinearity}$=0.203, Supplementary Material Fig. 4c), the pooled RR was nonsignificant (RR 1.000, 95% CI 0.995–1.005, I^2=34.0%, $P_{heterogeneity}$=0.195, Fig. 3) for every 10 dB increment in the dose-response analysis which included 5 studies [24, 43, 44, 47, 48].

Railway, Aircraft Noise, and Atrial Fibrillation

For railway noise, the pooled RR was 1.095 (95% CI 1.037–1.156) for the dichotomous analysis included 1 study [24] (Supplementary Material Table 5). The pooled RR was 1.011 (95% CI 1.002–1.021, I^2=12.7%, $P_{heterogeneity}$=0.285, Fig. 3) with every 10 dB increment in the dose-response analysis from 2 studies [24, 44]. For aircraft noise, 1 study [24] was included in the

Table 1 Dose-response subgroup analyses of the risk of cardiovascular disease morbidity from road traffic, railway, and aircraft noise (per 10 dB increment)

Subgroup	Road traffic noise (per 10 dB increment)					Railway noise (per 10 dB increment)					Aircraft noise (per 10 dB increment)				
	N	RR (95% CI)	I^2 (%)	P^1	P^2	*N*	RR (95% CI)	I^2 (%)	P^1	P^2	*N*	RR (95% CI)	I^2 (%)	P^1	P^2
All studies	29	1.020 (1.006–1.035)	90.6	<0.001		8	1.014 (0.968–1.063)	92.1	<0.001		8	1.016 (1.000–1.032)	72.7	0.001	
Sex					0.945					0.814					0.015
Men	10	1.016 (0.976–1.057)	82.2	<0.001		2	1.013 (1.000–1.026)	0.0	0.811		1	0.900 (0.783–1.034)	–	–	
Women	10	1.029 (1.001–1.057)	51.4	0.029		2	1.019 (1.006–1.033)	0.0	0.986		1	1.250 (1.088–1.437)	–	–	
Both	16	1.026 (1.006–1.043)	91.9	<0.001		6	0.979 (0.966–0.992)	52.1	0.064		7	1.016 (0.999–1.032)	76.4	0.001	
Region					0.263					–					–
Europe	26	1.016 (1.002–1.031)	82.9	<0.001		8	1.014 (0.968–1.063)	92.1	<0.001		8	1.016 (1.000–1.032)	72.7	0.001	
America	3	1.039 (0.989–1.092)	98.3	<0.001		0	–	–	–		0	–	–	–	
Follow-up (years)					0.197					0.264					0.489
<10	12	1.009 (0.987–1.030)	87.9	<0.001		2	0.974 (0.949–1.000)	81.8	0.019		5	1.013 (0.993–1.033)	82.9	<0.001	
≥10	17	1.028 (1.006–1.051)	92.3	<0.001		6	0.993 (0.974–1.012)	68.6	0.007		3	1.026 (1.005–1.048)	0.0	0.892	
Sample size					0.511					–					0.685
<10,000	6	0.977 (0.870–1.096)	36.2	0.165		0	–	–	–		3	1.111 (0.736–1.679)	0.0	0.434	
≥10,000	23	1.021 (1.006–1.037)	92.4	<0.001		8	1.014 (0.968–1.063)	92.1	<0.001		5	1.016 (1.000–1.032)	83.2	<0.001	
Study quality					0.117					–					0.685
High	25	1.022 (1.007–1.037)	91.8	<0.001		8	1.014 (0.968–1.063)	92.1	<0.001		5	1.016 (1.000–1.032)	83.2	<0.001	
Medium	4	0.920 (0.823–1.028)	0.0	0.796		0	–	–	–		3	1.111 (0.736–1.679)	0.0	0.434	
Noise metrics					0.142					–					0.685
L_{den}	19	1.020 (1.003–1.037)	87.3	<0.001		8	1.014 (0.968–1.063)	92.1	<0.001		5	1.016 (1.000–1.032)	83.2	<0.001	
L_{Aeq24h}	6	0.990 (0.981–1.000)	0.0	0.889		0	–	–	–		3	1.111 (0.736–1.679)	0.0	0.434	
L_{Aeq16h}	2	1.004 (0.989–1.018)	0.0	0.440		0	–	–	–		0	–	–	–	
L_{night}	2	1.063 (1.054–1.073)	15.1	0.278		0	–	–	–		0	–	–	–	
Adjustment															
Age					0.009					0.601					0.670
Yes	24	1.030 (1.014–1.046)	90.9	<0.001		7	0.985 (0.966–1.004)	85.7	<0.001		7	1.016 (0.999–1.032)	76.4	<0.001	
No	5	0.986 (0.968–1.005)	19.0	0.294		1	1.010 (0.933–1.093)	–	–		1	1.040 (0.940–1.150)	–	–	
Income					0.019					0.603					0.609
Yes	11	1.036 (1.016–1.056)	96.3	<0.001		5	0.983 (0.960–1.006)	90.3	<0.001		4	1.016 (0.999–1.032)	87.2	<0.001	
No	18	1.002 (0.988–1.016)	11.1	0.322		3	0.996 (0.976–1.017)	0.0	0.749		4	1.044 (0.947–1.151)	0.0	0.623	
Smoking					0.156					0.603					0.609
Yes	20	1.006 (0.985–1.028)	35.1	0.062		3	0.996 (0.976–1.017)	0.0	0.749		4	1.044 (0.947–1.151)	0.0	0.623	
No	9	1.030 (1.010–1.050)	97.0	<0.001		5	0.983 (0.960–1.006)	90.3	<0.001		4	1.016 (0.999–1.032)	87.2	<0.001	

Table 1 (continued)

Subgroup	Road traffic noise (per 10 dB increment)				Railway noise (per 10 dB increment)				Aircraft noise (per 10 dB increment)			
	N	RR (95% CI)	I^2 (%)	P^1	P^2	*N*	RR (95% CI)	I^2 (%)	P^1	P^2	*N*	RR (95% CI)	I^2 (%)	P^1	P^2
Drinking					0.725					0.603					0.609
Yes	14	1.015 (0.984–1.047)	32.1	0.119		3	0.996 (0.976–1.017)	0.0	0.749		4	1.044 (0.947–1.151)	0.0	0.623	
No	15	1.022 (1.005–1.040)	95.0	<0.001		5	0.983 (0.960–1.006)	90.3	<0.001		4	1.016 (0.999–1.032)	87.2	<0.001	
Air pollution					0.821					0.028					0.903
Yes	20	1.019 (1.004–1.033	88.1	<0.001		4	0.975 (0.961–0.989)	54.8	0.084		1	1.011 (0.939–1.089)	–	–	
No	9	1.029 (1.001–1.057)	71.4	<0.001		4	1.009 (1.001–1.018)	0.0	0.472		7	1.016 (1.000–1.033)	76.6	<0.001	
Other noise exposure					0.284					0.601					0.609
Yes	7	1.030 (1.009–1.052)	95.0	<0.001		7	0.985 (0.966–1.004)	85.7	<0.001		4	1.016 (0.999–1.032)	87.2	<0.001	
No	22	1.011 (0.989–1.034)	87.3	<0.001		1	1.010 (0.933–1.093)	–	–		4	1.044 (0.947–1.151)	0.0	0.623	

N number of studies, *RR* relative risk, *CI* confidence interval

P^1: *P* value for heterogeneity within each subgroup

P^2: *P* value for heterogeneity between subgroups with meta-regression analysis

dichotomous analysis, and the pooled RR was 1.036 (95% CI 0.931–1.154, Supplementary Material Table 5). The risk of AF increased by 2.7% (RR 1.027, 95% CI 1.004–1.050, Fig. 3) in the dose-response analysis included 1 study [24].

Traffic Noise and Heart Failure

Road Traffic Noise and Heart Failure

For road traffic noise, 3 studies [15, 43, 51] were included in the dichotomous analysis. The pooled RR was 1.134 (95% CI 1.115–1.153, I^2=7.9%, $P_{heterogeneity}$=0.337, Supplementary Material Table 5). In the dose-response analysis included 5 studies [15, 43, 46, 49, 51], we observed a positive linear relationship between road traffic noise and HF (*n*=3 [15, 43, 51], $P_{nonlinearity}$=0.977, Supplementary Material Fig. 4d). The pooled RR was 1.050 (95% CI 1.006–1.096, I^2=92.9%, $P_{heterogeneity}$<0.001, Fig. 3) with each 10 dB increment.

Railway, Aircraft Noise, and Heart Failure

For railway noise, the pooled RR for each 10 dB increment was 0.988 (95% CI 0.970–1.005, Fig. 3) in the dose-response analysis from 1 study [15]. For aircraft noise, 1 study [15] was included in the dichotomous analysis, and the pooled RR was 1.071 (95% CI 0.977–1.174, Supplementary Material Table 5). For each 10 dB increment of aircraft noise, the pooled RR was 1.023 (95% CI 1.016–1.030, Fig. 3) in the analysis included 1 study [15].

Discussion

We quantitatively evaluated the relationships between traffic noise from roads, railways, and aircraft sources and cardiovascular events (CVDs, stroke, MI, AF, and HF), finding a 2%, 3.4%, and 5% increase for the risk of CVDs, stroke, and HF, respectively, with per 10 dB increment in road traffic noise. For each 10 dB increment in railway noise, the risk of AF increased by 1.1%. In addition, the risk increased by 1.6% for CVDs, 1% for MI, 2.7% for AF, and 2.3% for HF for each 10 dB increment in aircraft noise. We also found positive linear associations between road traffic, railway, and aircraft noise sources and risk of CVDs, between road

traffic noise and risk of HF, and a nonlinear correlation between road traffic noise and risk of stroke.

We found a positive correlation between road traffic noise and CVDs which was inconsistent with a previous meta-analysis that reported nonsignificant results [22]. The previous study, assessing the relationship between total transportation noise and CVDs, only included cross-sectional studies from 1980 to 2010. Health effects arising from traffic noise, however, could vary according to its sources, characteristics, and intensity [7]. Our meta-analysis included cohort studies with a large number of participants, thus providing sufficient statistical power and increasing the reliability of the risk estimates. Although road transport is the main source of road traffic noise and air pollution, road traffic noise still increased the risk of CVDs after adjusting for air pollution in subgroup analyses, suggesting that road traffic noise is a risk factor for CVDs independent of air pollution. Moreover, we found that age and income might be important confounding factors in the association of road traffic noise and CVDs, possible explanations being that hearing ability reduces with age and that socioeconomic status partly determines the type of house construction and the quality of sound insulation in those houses, all of which could affect the perception of noise and perhaps decrease the annoyance caused by noise [14, 52].

A 2018 WHO review concluded that road traffic noise could increase the incidence risk of stroke [53]. Our meta-analysis reported a similar positive risk estimate, contrary to previous meta-analyses [26, 54]. The meta-analysis published in 2022 combined both morbidity and mortality risk estimates [26], possibly obscuring the true relationship between road traffic noise and stroke. Previously, Dzhambov used a categorical meta-analysis to evaluate a nonlinear trend in the relationship between road traffic noise and stroke [54]. This approach requires that included studies be similar in the fields of exposure assessment, noise metrics, and reference levels. Given that few studies meet this requirement, the results need to be considered with caution. We fully integrated available studies to evaluate the shape of dose-response relationships for stroke with restricted cubic splines, finding a similar nonlinear curve for road traffic noise and stroke with a leveling-off point at about 62dB, echoing a previous study [14].

We found that the relationships between road traffic noise and MI and AF were not significant, possibly because most studies in the analysis adjusted for air pollution; yet road transport is a source of both road traffic noise and air pollution. These are highly correlated, making it difficult to disentangle their effects on MI and AF. Further, sleep disturbance is thought to be a critical risk factor for AF [24, 55]. While L_{den} might not therefore be the most optimal metric for noise estimation, most studies included in our analysis used this noise metric. It is anticipated that future studies will adopt a more targeted metric to explore the correlation between traffic noise and AF. We also synthesized the evidence of the correlation between road traffic noise and HF, finding that the incidence risk of HF increased with increments in road traffic noise, in alignment with previous studies [46, 51]. Additionally, the effect of road traffic noise followed a linear trend, with the risk of HF increasing from the low noise level. Nevertheless, the results should be interpreted cautiously owing to the high heterogeneity and small number of studies.

This is the only meta-analysis to date that quantitatively analyzes the relationships between railway and aircraft noise and all CVDs. We found the correlation between railway noise and CVDs was not significant, which may be due to the fact that railway noise is usually perceived as less irritating than road traffic noise at equivalent sound levels [56], emphasizing the importance of the annoyance related to traffic noise. Additionally, there was a linear dose-response trend between aircraft noise and CVDs, with an increased risk of CVDs at lower noise levels. These results should be interpreted with great caution, first because the lower limit of the 95% CI for aircraft noise was approximate to the reference and second because CVDs contain many subtypes and the number of studies that we included in the analysis was relatively limited.

Currently, there are several principal ways in which noise affects the cardiovascular system. First, noise acts as a stressor that could trigger stress reactions via activation of the hypothalamic-pituitary-adrenal and sympathetic adrenal medullary axes, causing the release of stress hormones such as adrenaline, noradrenaline, and cortisol [7, 57], subsequently inducing inflammation [8, 9] and oxidative stress [10, 11, 58]. Annoyance caused by noise may disrupt the endocrine system, which also induces the release of stress hormones, ultimately leading to elevations in heart rate, blood pressure, lipids, and glucose, for instance [59–61]. Moreover, noise could also influence the cardiovascular system through sleep disturbance by increasing awakenings throughout the

night [62, 63]. These factors may result in endothelial dysfunction, oxidative stress, and inflammation, all of which are linked to the etiology of cardiovascular diseases [7, 64]. In addition, long-term exposure to traffic noise has been related to known cardiovascular risk factors like obesity [65], diabetes [66], and hypertension [53].

The main strength of this meta-analysis is that we comprehensively investigated the relationships between chronic exposure to different sources of traffic noise and cardiovascular events and quantitatively evaluated the dose-response relationships of traffic noise and all CVDs, road traffic noise, and HF. In addition, currently eligible cohort studies were included in this meta-analysis, possibly providing adequate statistical power and minimizing recall bias. There are certainly some limitations to be noted. First, given the limited number of studies, we did not assess the potential linear or nonlinear trends for railway and aircraft noise and stroke, MI, AF, and HF such that even the associations between railway and aircraft noise and CVD subtypes should be interpreted with caution. Second, the study showed high heterogeneity in most analyses, but we performed subgroup analysis and meta-regression to explore the latent source of heterogeneity, finding potential evidence of heterogeneity in some analyses. Further, all studies were from Europe except for two Canadian studies. More studies outside Europe should be conducted to understand the effects on cardiovascular events from traffic noise across the world. Finally, even though the extracted risk estimate was adjusted for a series of variables, the results of this meta-analysis might be influenced by other confounding factors.

Conclusion

Long-term exposure to traffic noise is positively linked with the incidence risk of cardiovascular events in adults, especially road traffic noise which significantly increases the risk of all CVDs, stroke, and heart failure. Consequently, high-quality prospective studies are urgently required to elucidate the effects of different sources of traffic noise on stroke, myocardial infarction, atrial fibrillation, and heart failure for better prevention of CVDs into the future.

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Author Contribution Xueru Fu conceived, designed, and performed the work; Longkang Wang analyzed the data; Lijun Yuan, Huifang Hu, Tianze Li, Jinli Zhang, Yamin Ke, Mengmeng Wang, Yajuan Gao, Weifeng Huo, Yaobing Chen, Wenkai Zhang, Yang Zhao, Jiong Liu, Zelin Huang, Fulan Hu, Ming Zhang, Yu Liu, Xizhuo Sun, and Dongsheng Hu revised the manuscript.

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