

# Occupational noise exposure and ischemic heart disease: a pooled analysis of six Nordic cohorts

by Claudia Lissåker, PhD,<sup>1</sup> Mattias Sjöström, PhD,<sup>1,2</sup> Andrei Pyko, PhD,<sup>1-3</sup> Anja Olsen, PhD,<sup>4</sup> Annika Rosengren, MD,<sup>5,6</sup> Aslak Harbo Poulsen, PhD,<sup>4</sup> Debora Rizzuto, PhD,<sup>6</sup> Göran Pershagen, MD,<sup>1</sup> Jesse D Thacher, PhD,<sup>7</sup> Jørgen Brandt, PhD,<sup>8</sup> Leo Stockfelt, MD,<sup>9,10</sup> Maria Albin, MD,<sup>1,8</sup> Pekka Jousilahti, MD,<sup>11,12</sup> Pekka Tiittanen, MSc,<sup>13</sup> Steen Solvang Jensen, PhD,<sup>14</sup> Timo Lanki, PhD,<sup>15-17</sup> Mette Sørensen, PhD,<sup>3,18</sup> Jenny Selander, PhD<sup>1,2</sup>

Lissåker C, Sjöström M, Pyko A, Olsen A, Rosengren A, Harbo Poulsen A, Rizzuto D, Pershagen G, Thacher JD, Brandt J, Stockfelt L, Albin M, Jousilahti P, Tiittanen P, Solvang Jensen S, Lanki T, Sørensen M, Selander J. Occupational noise exposure and ischemic heart disease: a pooled analysis of six Nordic cohorts. *Scand J Work Environ Health* – online first.

**Objectives** This study aimed to investigate the impact of occupational noise exposure on incident ischemic heart disease (IHD), including acute myocardial infarction (AMI).

**Methods** We conducted a pooled analysis of six Nordic cohorts with a total of 83 103 participants with individual and area-level confounder information. Occupational codes were matched to a job-exposure matrix developed from Swedish measurements in five categories (LAeq8h): <70, 70–74, 75–79, 80–84, and ≥85 dB(A). Residential address history was used to estimate baseline 1-year average residential road traffic noise exposure. Data on incident IHD and AMI were obtained from national patient registers. We calculated hazard ratios (HR) using Cox proportional hazard models adjusted for socioeconomic and lifestyle factors.

**Results** After adjustment for baseline year, sex, marital status, area-level income, education, smoking, and physical activity, exposure to ≥85 dB(A) of occupational noise was associated with an 10% and 12% increased risk of IHD, excluding angina pectoris, and AMI, respectively. For these outcomes, suggestive exposure–response trends were observed, but risks were only statistically significant for those exposed to ≥85 dB(A) as well as men exposed to ≥75 dB(A). We did not find evidence that combined exposure to both occupational noise and road traffic noise was particularly harmful.

**Conclusion** We found increased risks of IHD and AMI among those exposed to occupational noise ≥85 dB(A). Further studies should investigate whether this association is confounded by other occupational exposures.

**Key terms** acute myocardial infarction; cardiovascular disease; IHD; JEM; job-exposure matrix; occupational exposure; road traffic noise.

<sup>1</sup> Institute of Environmental Medicine, Karolinska Institutet, Stockholm, Sweden.

<sup>2</sup> Center for Occupational and Environmental Medicine, Region Stockholm, Stockholm, Sweden.

<sup>3</sup> Aging Research Center, Department of Neurobiology Care Science and Society, Karolinska Institutet and Stockholm University, Stockholm, Sweden.

<sup>4</sup> Danish Cancer Institute, Copenhagen, Denmark.

<sup>5</sup> Department of Molecular and Clinical Medicine, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden.

<sup>6</sup> Region Västra Götaland, Department of Medicine Geriatrics and Emergency Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden.

<sup>7</sup> Division of Occupational and Environmental Medicine, Lund University, Lund, Sweden.

<sup>8</sup> Department of Environmental Science, Aarhus University, Roskilde, Denmark.

<sup>9</sup> Department of Occupational and Environmental Medicine, Sahlgrenska University Hospital, Gothenburg, Sweden.

<sup>10</sup> Occupational and Environmental Medicine, School of Public Health and Community Medicine, Institute of Medicine, Sahlgrenska Academy, University of Gothenburg, Gothenburg, Sweden.

<sup>11</sup> Department of Public Health, Finnish Institute for Health and Welfare, Helsinki, Finland.

<sup>12</sup> Department of Public Health, University of Helsinki, Helsinki, Finland.

<sup>13</sup> Department of Public Health, Finnish Institute for Health and Welfare, Kuopio, Finland.

<sup>14</sup> Department of Public Health, University of Copenhagen, Copenhagen, Denmark.

<sup>15</sup> Department of Public Health, Finnish Institute for Health and Welfare, Oulu, Finland.

<sup>16</sup> School of Medicine, University of Eastern Finland, Kuopio, Finland.

<sup>17</sup> Department of Environmental and Biological Sciences, University of Eastern Finland, Kuopio, Finland.

<sup>18</sup> Department of Natural Science and Environment, Roskilde University, Roskilde, Denmark.

Ischemic heart disease (IHD) is one of the leading causes of morbidity and mortality in the world, with an estimated 293 million prevalent cases in 2023 (1). Occupational factors have been receiving increased attention as potential contributors to IHD. Indeed, in Europe, cardiovascular diseases have been named as the second leading cause of work-related death, accounting for an estimated 24% of all cases (2), which emphasizes the importance of identifying elements of the work environment that can be targeted with interventions to reduce this burden.

Workplace environments, particularly in industrial, manufacturing, and transportation sectors, are often characterized by elevated noise levels. In 2015, 28% of European workers reported being exposed to excessive occupational noise (3). Several mechanisms have been proposed linking noise exposure to cardiovascular disease. Noise exposure can impair endothelial function and promote inflammation and oxidative stress, while also disrupting autonomic balance by increasing sympathetic activity and suppressing parasympathetic tone (4, 5). Noise-induced sleep disturbance may further amplify these effects through sleep fragmentation and impairment of circadian rhythm (6).

Studies have shown that occupational noise can affect the risk of cardiovascular diseases. One recent meta-analysis estimated that exposure to high occupational noise was associated with a 1.29 [95% confidence interval (CI) 1.15–1.43] fold increased risk of incident IHD (7). This estimate, however, was based on only two cohort studies, and the authors concluded that evidence is limited, further emphasizing the need for additional high-quality longitudinal studies. Three case-control studies were also pooled, with a resulting increased odds of 1.38 (95% CI 0.94–2.02) (7). Other smaller studies not included in these estimates also showed an increased risk of IHD associated with occupational noise (8). An additional limitation of prior literature is that most studies utilized a cutoff of 85 dB(A), a level deemed to be hazardous for hearing impairment, and did not explore whether an elevated risk of IHD might also occur at lower levels.

Environmental noise has been found to be associated with an increased risk of IHD (9). Given that many individuals will be exposed to high noise levels both at home and at work, it is important to explore this relationship further. Few studies, however, have assessed the impact of the combination of road traffic and occupational noise. One Swedish study that did investigate both sources of noise found that those who were exposed to high levels of road traffic and occupational noise had a slightly increased risk of acute myocardial infarction (AMI) compared to those exposed to occupational or road traffic noise alone (10). Further studies to elucidate this relationship are needed.

In this pooled study of six Nordic cohorts, we aimed to investigate the impact of occupational noise exposure on incident IHD and AMI, adjusting for socioeconomic and individual risk factors. We also aimed to explore whether concurrent road traffic noise exposure impacts this relationship.

## Methods

### Study population

Data came from pooled Nordic cohorts from the “Nordic studies on occupational and traffic noise in relation to disease” (NordSOUND) project ([www.nordsound.dk](http://www.nordsound.dk)). For the present study, we included six cohorts with information on occupational exposure: the Diet, Cancer, and Health (DHC) cohort from Copenhagen/Aarhus, Denmark (11); the FINRISK cohort from Helsinki, Vantaa and Turku, Finland (12); the Swedish Primary Prevention Study (PPS) from Gothenburg, Sweden (13); the Malmö Diet and Cancer study (MDCS) from Malmö, Sweden (14); and the Swedish National Study of Ageing and Care in Kungsholmen (SNAC-K) (15) and Stockholm Screening Across the Lifespan Twin Study (SALT) (16) harmonized by the Cardiovascular Effects of Air Pollution and Noise in Stockholm (CEANS) project (17). Information on the cohorts can be found in the supplementary material ([www.sjweh.fi/article/4333](http://www.sjweh.fi/article/4333)), table S1. Data harmonization and checks were done centrally according to a common protocol.

### Occupational noise exposure

To estimate occupational noise, we utilized a job-exposure matrix (JEM), which links occupational codes to exposure estimates, enabling us to estimate exposure levels for large study populations where individual-level measurements are not feasible. A Swedish JEM on noise was linked to the pooled data using occupational codes. The noise JEM estimates annual average of daily 8-hour exposure to A-weighted sound pressure level ( $L_{Aeq8h}$ ) in five exposure classes: <70, 70–74, 75–79, 80–84, and  $\geq 85$  dB(A) in five-year intervals from 1970–2014. Current and historical measurements from clinics, occupational health services, large companies, and other reports throughout Sweden were used in the development of the JEM. The JEM was originally developed for 321 occupational groups coded in the 1983 version of the Nordic Occupational Classification (NYK83) coding system and has been translated into different coding systems (FOB80, ISCO-88, ISCO-08) by an occupational hygienist. A team of occupational hygienists have validated the JEM and found it to have consistent and reliable results (18).

Occupational data in the NordSOUND cohorts were collected from national registers (DCH, FINRISK), or from baseline questionnaires given to participants at time of recruitment (MDCS, PPS, SALT, SNAC-K). From each cohort, we selected participants with available occupational data at baseline or the latest occupation listed in the five years prior to baseline, such as for those who were already of retirement age at recruitment. Individuals with occupational data older than five years prior to baseline were not included in this study. Two cohorts, DCH and FINRISK, had additional longitudinal occupational data after baseline. Different coding systems were used depending on the country and the year of data collection. For each country, occupational data were matched to the JEM in the appropriate coding system (ISCO-88 and ISCO-08 for DCH and FINRISK, and NYK83, FOB80, and FOB85 for MDCS, PPS, SNAC-K, and SALT) and were assigned a noise exposure class based on the year of exposure. Information on part-time work was not available; therefore, we assume full-time work for all those with occupational data.

### Road traffic noise

Exposure to road traffic noise was estimated using participants' address history and the Nordic Prediction method, which accounts for the location of the residence, obstructions from landforms and structures, and data on annual mean daily traffic averages, type of traffic, speed, and the road type for all major roads. Except for the Stockholm cohorts and FINRISK, information on smaller roads was included. A time-weighted mean for the year preceding the baseline was calculated. Details on the exposure assessment for road traffic noise can be found elsewhere (19).

### Outcome

For each cohort, data on IHD and AMI were obtained from national in- and outpatient and mortality registers. The following International Classification of Diseases, ninth and tenth revisions (ICD-9 and ICD-10) codes were used to extract IHD cases: ICD9 codes 410–414 and ICD10 codes I20–I25. To identify the subgroup of myocardial infarction cases, the following codes were used: ICD9 codes 410 and ICD10 codes I21–I23. Because of differences in the composition of IHD cases between the Danish and Swedish data (20), we further selected a subgroup of IHD that excluded angina pectoris using the following codes: ICD9 codes 410, 411, 412, 414 and ICD10 codes I21–I25. Information on angina pectoris was not available for the FINRISK cohort; therefore, this cohort was excluded from all analyses on this outcome. Those with a diagnosis of IHD at or prior to their baseline year were excluded.

### Covariates at baseline

Covariate selection was based on existing literature and availability in the cohorts. From each cohort's individual baseline questionnaire, we obtained data on age, smoking status (current, former, or never), leisure-time physical activity (low: once a month or <1 hour/week, medium: about once a week or approximately 1 hour/week, high:  $\geq 3$  times a week or >2 hours/week), alcohol consumption (daily, weekly, seldom, or never) and body mass index (BMI). Information on alcohol consumption was not available for the PPS cohort. Information on educational level (low: primary school or less; medium: up to secondary school or its equivalent; or high: university or higher) and marital status (single: not married or widowed; married: married or living with a partner) were obtained from questionnaires or national registers. To account for area-level socioeconomic status, mean income was categorized into country-specific quartiles based on register data for small, socioeconomically homogenous areas with approximately 1000–2000 inhabitants.

### Statistical methods

We created Cox proportional hazards models with age as the underlying time scale to calculate hazard ratios (HR) and 95% CI for IHD, IHD excluding angina pectoris, and AMI associated with increasing occupational noise categories compared to <70 dB(A). We checked proportional hazards assumptions by a test of the correlation between scaled Schoenfeld residuals and time. Deviations were detected for sex, educational level, and smoking, which were then all included as strata in the models. Additionally, models were stratified by cohort, which allowed for different baseline hazards. All participants in each cohort were followed from recruitment until emigration, death, loss to follow-up, end of follow-up, or IHD incidence.

Three models were created with increasing adjustments: model 1 was adjusted for age (by design), cohort, baseline year, and sex; model 2 was additionally adjusted for marital status, area-level income, and education; and in model 3, we added adjustments for smoking, and physical activity. Model 3 was then stratified by sex to obtain separate estimates for men and women. Since the DCH and FINRISK cohorts included longitudinal occupational data we were able to assess the time-varying effect of occupational noise in these two cohorts by taking changes in exposure into account. For these cohorts, we also censored after 5 years and 1 year and compared it to the uncensored estimates in these two cohorts.

We also assessed the effect of exposure to both road traffic and occupational noise. To do this, we created a variable by combining categories of occupational noise

[<70, 70–80 and  $\geq 80$  dB(A)] and categories of baseline 1-year average road traffic noise [<55, 55–65 and  $\geq 65$  dB(A)] into nine categories (<70 and <55, <70 and 55–65, <70 and  $\geq 65$ , 70–80 and <55, 70–80 and 55–65, 70–80 and  $\geq 65$ ,  $\geq 80$  and <55,  $\geq 80$  and 55–65,  $\geq 80$  and  $\geq 65$ ). Because of the limited number of exposed cases in some groups, we collapsed the five categories of occupational noise into three.

Several sensitivity analyses were conducted. In our pooled cohort, <1% of those with the highest noise levels reported having high education, which indicates a potential for over-adjustment with the inclusion of this confounder. Therefore, we repeated the three models while excluding education, as well as stratified for the different levels of education. In the stratified model, we were unable to estimate the effects of exposure  $>85$  dB(A); therefore, we combined the two highest categories. We also repeated analyses and included adjustments for both alcohol consumption and BMI, while excluding the PPS cohort due to its lack of information on the former. Lastly, we repeated model 3 for each cohort individually. Individuals with missing data for covariates were excluded. The PPS cohort did not ask

questions regarding alcohol use and FINRISK did not have angina pectoris diagnosis codes; therefore, these cohorts were excluded from analyses needing this data.

All analyses were performed in SAS version 9.4 (SAS Institute, North Carolina, USA) and R (version 3.2.3).

## Results

Our study included 83 103 individuals with occupational data at baseline and complete information on all covariates except for alcohol consumption. Table 1 shows the characteristics of the pooled cohort by occupational noise exposure. Those exposed to  $\geq 85$  dB(A) of noise were more often men, married/cohabiting, living in either the lowest or highest income areas, and had a lower proportion of high education. They were also more likely to report being current smokers, having low leisure-time physical activity, and consuming alcohol daily.

Table 2 shows HR for the association between occupational noise exposure and IHD, IHD excluding angina

**Table 1.** Characteristics of the study population and occupational noise exposure at baseline.

|                                             | Total  | Occupational noise exposure [dB(A)] |      |                  |      |                |      |                |      |              |      |      |
|---------------------------------------------|--------|-------------------------------------|------|------------------|------|----------------|------|----------------|------|--------------|------|------|
|                                             |        | <70 (N=50 775)                      |      | 70–74 (N=14 692) |      | 75–79 (N=8048) |      | 80–84 (N=6234) |      | ≥85 (N=3354) |      |      |
|                                             | N      | %                                   | Mean | %                | Mean | %              | Mean | %              | Mean | %            | Mean |      |
| Age at baseline                             |        |                                     |      | 55.9             |      | 55.9           |      | 55.5           |      | 55.3         |      | 55.6 |
| Sex                                         |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Men                                         | 39 494 | 40.7                                |      |                  | 47.5 |                | 66.1 |                | 56.7 |              | 89.1 |      |
| Women                                       | 43 609 | 59.3                                |      |                  | 52.5 |                | 33.9 |                | 43.3 |              | 10.9 |      |
| Marital status                              |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Single                                      | 22 197 | 27.7                                |      |                  | 25.4 |                | 25.2 |                | 27.1 |              | 21.0 |      |
| Married/cohabiting                          | 60 906 | 72.3                                |      |                  | 74.6 |                | 74.8 |                | 72.9 |              | 79.0 |      |
| Educational level <sup>a</sup>              |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Low                                         | 32 026 | 32.8                                |      |                  | 43.3 |                | 46.0 |                | 56.9 |              | 51.6 |      |
| Medium                                      | 31 872 | 40.8                                |      |                  | 34.2 |                | 35.9 |                | 26.6 |              | 47.5 |      |
| High                                        | 19 205 | 26.4                                |      |                  | 22.5 |                | 18.1 |                | 16.5 |              | 0.9  |      |
| Area-level income <sup>b</sup> (quartiles)  |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Q1                                          | 22 601 | 26.0                                |      |                  | 29.8 |                | 28.4 |                | 28.2 |              | 29.8 |      |
| Q2                                          | 17 390 | 20.8                                |      |                  | 21.1 |                | 20.6 |                | 21.6 |              | 22.3 |      |
| Q3                                          | 16 924 | 20.5                                |      |                  | 19.2 |                | 20.7 |                | 21.7 |              | 19.2 |      |
| Q4                                          | 26 188 | 32.7                                |      |                  | 29.9 |                | 30.3 |                | 28.5 |              | 28.7 |      |
| Smoking Status                              |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Current                                     | 26 993 | 30.1                                |      |                  | 35.0 |                | 36.3 |                | 35.6 |              | 43.0 |      |
| Former                                      | 25 246 | 30.7                                |      |                  | 29.1 |                | 30.0 |                | 30.7 |              | 31.4 |      |
| Never                                       | 30 864 | 39.2                                |      |                  | 35.9 |                | 33.7 |                | 33.7 |              | 25.6 |      |
| Leisure-time physical activity <sup>c</sup> |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Low                                         | 40 024 | 46.0                                |      |                  | 51.0 |                | 52.0 |                | 50.0 |              | 55.7 |      |
| Medium                                      | 20 385 | 25.4                                |      |                  | 22.5 |                | 22.7 |                | 26.1 |              | 22.3 |      |
| High                                        | 22 694 | 28.6                                |      |                  | 26.5 |                | 25.4 |                | 23.9 |              | 20.0 |      |
| Alcohol consumption                         |        |                                     |      |                  |      |                |      |                |      |              |      |      |
| Daily                                       | 14 246 | 17.3                                |      |                  | 16.1 |                | 18.6 |                | 14.6 |              | 20.0 |      |
| Weekly                                      | 41 446 | 52.4                                |      |                  | 50.4 |                | 45.9 |                | 38.2 |              | 40.6 |      |
| Seldom                                      | 17 567 | 20.9                                |      |                  | 23.0 |                | 19.8 |                | 24.2 |              | 15.1 |      |
| Never                                       | 4771   | 5.2                                 |      |                  | 6.0  |                | 6.4  |                | 9.3  |              | 4.7  |      |
| Missing                                     | 5073   | 4.2                                 |      |                  | 4.5  |                | 9.3  |                | 13.7 |              | 19.6 |      |
| Body mass index                             |        |                                     | 25.5 |                  | 26.0 |                | 26.3 |                | 26.1 |              | 26.7 |      |

<sup>a</sup> Low: primary school or less; medium: up to secondary school or equivalent; high: university degree and more at recruitment.

<sup>b</sup> Area-based mean household income at the address where the participant lived at the enrollment, categorized into country-specific quartiles.

<sup>c</sup> Low: once a month or less or <1 hour/week; medium: about once a week or about 1 hour/week; high:  $\geq 3$  a week or >2 hours/week.

**Table 2.** Associations between occupational noise exposure at baseline and ischemic heart disease and myocardial infarction incidence. [HR=hazard ratio; CI=confidence interval.]

| Occupational noise level [dB(A)]                                    | Cases | Model 1 <sup>a</sup> | Model 2 <sup>b</sup> | Model 3 <sup>c</sup> |
|---------------------------------------------------------------------|-------|----------------------|----------------------|----------------------|
|                                                                     | N     | HR (95% CI)          | HR (95% CI)          | HR (95% CI)          |
| <b>Ischemic heart disease</b>                                       |       |                      |                      |                      |
| <70                                                                 | 9071  | ref                  | ref                  | ref                  |
| 70–74                                                               | 2960  | 1.11 (1.06–1.16)     | 1.07 (1.03–1.12)     | 1.05 (1.01–1.09)     |
| 75–79                                                               | 1794  | 1.14 (1.08–1.20)     | 1.09 (1.03–1.14)     | 1.06 (1.01–1.12)     |
| 80–84                                                               | 1434  | 1.13 (1.07–1.20)     | 1.07 (1.01–1.13)     | 1.04 (0.98–1.11)     |
| ≥85                                                                 | 961   | 1.22 (1.14–1.30)     | 1.12 (1.05–1.20)     | 1.08 (1.00–1.15)     |
| <b>Ischemic heart disease excluding angina pectoris<sup>d</sup></b> |       |                      |                      |                      |
| <70                                                                 | 5006  | ref                  | ref                  | ref                  |
| 70–74                                                               | 1616  | 1.11 (1.05–1.17)     | 1.06 (1.00–1.12)     | 1.03 (0.97–1.09)     |
| 75–79                                                               | 1049  | 1.16 (1.09–1.25)     | 1.09 (1.02–1.17)     | 1.07 (1.00–1.14)     |
| 80–84                                                               | 913   | 1.19 (1.11–1.28)     | 1.11 (1.03–1.19)     | 1.07 (1.00–1.16)     |
| ≥85                                                                 | 615   | 1.28 (1.18–1.40)     | 1.16 (1.06–1.26)     | 1.10 (1.01–1.21)     |
| <b>Acute myocardial infarction</b>                                  |       |                      |                      |                      |
| <70                                                                 | 3168  | ref                  | ref                  | ref                  |
| 70–74                                                               | 1007  | 1.12 (1.04–1.20)     | 1.05 (0.98–1.13)     | 1.03 (0.95–1.10)     |
| 75–79                                                               | 690   | 1.16 (1.06–1.26)     | 1.07 (0.99–1.17)     | 1.05 (0.97–1.15)     |
| 80–84                                                               | 640   | 1.18 (1.09–1.29)     | 1.08 (0.99–1.18)     | 1.06 (0.97–1.16)     |
| ≥85                                                                 | 440   | 1.32 (1.19–1.47)     | 1.17 (1.06–1.30)     | 1.12 (1.01–1.24)     |

<sup>a</sup> Adjusted for age (by design), cohort, baseline year, sex.<sup>b</sup> Adjusted for age (by design), cohort, baseline year, sex, marital status, area-level income, education.<sup>c</sup> Adjusted for age (by design), cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity.<sup>d</sup> Information on angina pectoris was not available for the FINRISK cohort.

pectoris, and AMI. After adjusting for age, cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity, exposure to occupational noise levels >70 dB(A) was associated with a 4–8% increased risk of IHD (model 3). For IHD excluding angina pectoris and AMI, we found a weak exposure–response trend in the fully adjusted model 3, with increased risks associated with each increasing category of occupational noise, though the CI overlapped; only exposure to ≥85 dB(A) was statistically significant (HR 1.10, 95% CI 1.01–1.21 for IHD excluding angina pectoris and HR 1.12, 95% CI 1.01–1.24 for AMI). Excluding education from the analysis yielded statistically significant increased HR for all three outcomes from 70 dB(A), with an exposure–response trend for IHD excluding angina pectoris and AMI (supplementary table S3, model 3). When stratified by education, increased risks were observed for those with low and medium education but not high education after adjusting for age, cohort, baseline year, sex, marital status, area-level income, smoking, and physical activity (supplementary table S4). In the analysis excluding the PPS cohort (supplementary table S5), model 3 – which is adjusted for age, cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity – showed similar results to the full cohort; however, with no exposure–response trend. Adjustment for alcohol consumption and BMI (model 5) showed further attenuation in the hazard; however, we still observed increased, albeit non-statistically significant, HR for those exposed to ≥85 dB(A) of occupational noise.

When stratifying by sex, there was an increased risk

among men showing a weak exposure–response trend for both IHD excluding angina, where the increased risk was statistically significant starting at ≥75 dB(A) of occupational noise exposure (table 3). We observed the same pattern for AMI among men; however, only the highest noise category was statistically significant. On the other hand, no clear or consistent increases in risk related to occupational noise exposure and/or exposure–response trends were observed among women.

When assessing longitudinal occupational noise exposure within the DCH and FINRISK cohorts (table 4), exposure ≥85 dB(A) was associated with increased HR for IHD, IHD excluding angina pectoris, and AMI when censoring five years after the last occupational code of 1.11 (95% CI 0.98–1.26), 1.22 (95% CI 1.02–1.45), and 1.16 (95% CI 0.93–1.44), respectively. Censoring after one year attenuated the effect sizes for IHD excluding angina pectoris (HR 1.11, 95% CI 0.89–1.38), and AMI (HR 1.02, 95% CI 0.78–1.35).

Results for the combined exposure to occupational and road traffic noise are shown in table 5. We did not observe any consistent pattern of increased hazards in those with high exposure to both types of noise. Within the <55 dB and 55–65 dB of road traffic noise groups, increased exposure to higher levels of occupational noise was associated with increased HR of IHD, IHD excluding angina pectoris, and AMI when compared to those exposed to the lowest levels of both occupational and road traffic noise. Among those exposed to ≥65 dB of road traffic noise, those exposed to 70–79 dB(A) of occupational noise had the highest risk of all three outcomes.

**Table 3.** Associations between occupational noise exposure at baseline and ischemic heart disease and myocardial infarction incidence stratified by sex. [HR=hazard ratio; CI=confidence interval.]

| Occupational noise level [dB(A)]                              | Men   |                      | Women |                      |
|---------------------------------------------------------------|-------|----------------------|-------|----------------------|
|                                                               | Cases | Model 3 <sup>a</sup> | Cases | Model 3 <sup>a</sup> |
|                                                               | N     | HR (95% CI)          | N     | HR (95% CI)          |
| Ischemic heart disease                                        |       |                      |       |                      |
| <70                                                           | 4849  | ref                  | 4222  | ref                  |
| 70–74                                                         | 1794  | 1.03 (0.98–1.09)     | 1166  | 1.08 (1.01–1.15)     |
| 75–79                                                         | 1427  | 1.08 (1.01–1.14)     | 367   | 1.01 (0.91–1.13)     |
| 80–84                                                         | 1070  | 1.05 (0.98–1.12)     | 364   | 1.03 (0.92–1.15)     |
| ≥85                                                           | 900   | 1.08 (1.00–1.16)     | 61    | 1.03 (0.80–1.33)     |
| Ischemic heart disease excluding angina pectoris <sup>b</sup> |       |                      |       |                      |
| <70                                                           | 2832  | ref                  | 2174  | ref                  |
| 70–74                                                         | 1069  | 1.04 (0.97–1.12)     | 547   | 1.01 (0.92–1.12)     |
| 75–79                                                         | 869   | 1.10 (1.02–1.19)     | 180   | 0.96 (0.83–1.12)     |
| 80–84                                                         | 737   | 1.12 (1.03–1.22)     | 176   | 0.95 (0.81–1.11)     |
| ≥85                                                           | 590   | 1.14 (1.04–1.25)     | 25    | 0.85 (0.57–1.27)     |
| Acute myocardial infarction                                   |       |                      |       |                      |
| <70                                                           | 1917  | ref                  | 1251  | ref                  |
| 70–74                                                         | 705   | 1.03 (0.94–1.12)     | 302   | 1.04 (0.92–1.19)     |
| 75–79                                                         | 584   | 1.08 (0.99–1.19)     | 106   | 0.96 (0.78–1.17)     |
| 80–84                                                         | 531   | 1.09 (0.99–1.21)     | 109   | 0.96 (0.78–1.16)     |
| ≥85                                                           | 426   | 1.15 (1.03–1.28)     | 14    | 0.91 (0.54–1.55)     |

<sup>a</sup> Adjusted for age (by design), cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity.<sup>b</sup> Information on angina pectoris was not available for the FINRISK cohort.**Table 4.** Associations between longitudinal occupational noise exposure and ischemic heart disease and myocardial infarction incidence among the DCH and FINRISK cohorts. [HR=hazard ratio; CI=confidence interval.]

| Occupational noise level [dB(A)]                              | Cases | All participants         | Cases | Censored after 5 years   | Cases | Censored after 1 year    |
|---------------------------------------------------------------|-------|--------------------------|-------|--------------------------|-------|--------------------------|
|                                                               | N     | HR <sup>a</sup> (95% CI) | N     | HR <sup>a</sup> (95% CI) | N     | HR <sup>a</sup> (95% CI) |
| Ischemic heart disease                                        |       |                          |       |                          |       |                          |
| <70                                                           | 4649  | ref                      | 2535  | ref                      | 1601  | ref                      |
| 70–74                                                         | 1889  | 1.02 (0.97–1.08)         | 1038  | 1.03 (0.96–1.11)         | 637   | 1.03 (0.94–1.13)         |
| 75–79                                                         | 969   | 0.99 (0.92–1.06)         | 534   | 0.93 (0.85–1.03)         | 343   | 0.94 (0.83–1.06)         |
| 80–84                                                         | 423   | 0.99 (0.90–1.10)         | 221   | 0.94 (0.82–1.08)         | 132   | 0.93 (0.77–1.11)         |
| ≥85                                                           | 489   | 1.05 (0.95–1.16)         | 294   | 1.11 (0.98–1.26)         | 187   | 1.10 (0.94–1.29)         |
| Ischemic heart disease excluding angina pectoris <sup>b</sup> |       |                          |       |                          |       |                          |
| <70                                                           | 2256  | ref                      | 1124  | ref                      | 699   | ref                      |
| 70–74                                                         | 930   | 1.00 (0.93–1.08)         | 473   | 1.01 (0.90–1.12)         | 285   | 0.99 (0.85–1.14)         |
| 75–79                                                         | 516   | 1.02 (0.92–1.12)         | 268   | 0.96 (0.84–1.11)         | 175   | 0.97 (0.82–1.15)         |
| 80–84                                                         | 201   | 0.95 (0.82–1.10)         | 100   | 0.92 (0.75–1.13)         | 57    | 0.85 (0.65–1.12)         |
| ≥85                                                           | 275   | 1.09 (0.96–1.24)         | 167   | 1.22 (1.02–1.45)         | 100   | 1.11 (0.89–1.38)         |
| Acute myocardial infarction                                   |       |                          |       |                          |       |                          |
| <70                                                           | 1177  | ref                      | 671   | ref                      | 456   | ref                      |
| 70–74                                                         | 473   | 0.96 (0.86–1.07)         | 277   | 0.96 (0.84–1.11)         | 174   | 0.90 (0.76–1.08)         |
| 75–79                                                         | 287   | 0.99 (0.87–1.13)         | 160   | 0.90 (0.75–1.07)         | 112   | 0.90 (0.73–1.11)         |
| 80–84                                                         | 106   | 0.92 (0.76–1.13)         | 61    | 0.91 (0.70–1.19)         | 37    | 0.83 (0.59–1.16)         |
| ≥85                                                           | 161   | 1.11 (0.93–1.31)         | 100   | 1.16 (0.93–1.44)         | 64    | 1.02 (0.78–1.35)         |

<sup>a</sup> Adjusted for age (by design), cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity.<sup>b</sup> Information on angina pectoris was not available for the FINRISK cohort.

## Discussion

In this pooled study of six Nordic cohorts, we observed an increased risk of AMI associated with occupational noise ≥85 dB(A) after adjusting for individual confounders. We also observed an increased hazard of IHD, but only when excluding those with a diagnosis of angina pectoris, a less well validated outcome than AMI. We did not observe any associations when looking at combined exposure to occupational and road traffic noise. When adjusting for education, we observed a significant attenuation of the results.

One recent meta-analysis estimated a relative risk of 1.29 (95% CI 1.15–1.43) for IHD associated with exposure to over 85 dB(A) of occupational noise based on two cohort studies from Sweden and Finland, both using exposure assessment based on JEM (7). However, this was only adjusted for age and systolic blood pressure, which is most comparable to our estimate adjusted for baseline year and sex where we observed a HR of 1.22 (95% CI 1.14–1.30). Systolic blood pressure can be a potential mediator and its inclusion as a confounder may lead to over-adjustment, attenuating the estimates given in the meta-analysis. Of these two studies, only one adjusted for additional covariates and found a

**Table 5.** Associations between combined exposure to occupational noise and road traffic noise at baseline and ischemic heart disease and myocardial infarction incidence. [HR=hazard ratio; CI=confidence interval.]

| Occupational noise level [dB(A)]                              | Road traffic noise, $L_{den}$ (dB) |                          |            |                          |             |                          |
|---------------------------------------------------------------|------------------------------------|--------------------------|------------|--------------------------|-------------|--------------------------|
|                                                               | <55                                |                          | 55–65      |                          | 65 and over |                          |
|                                                               | Cases<br>N                         | HR <sup>a</sup> (95% CI) | Cases<br>N | HR <sup>a</sup> (95% CI) | Cases<br>N  | HR <sup>a</sup> (95% CI) |
| Ischemic heart disease                                        |                                    |                          |            |                          |             |                          |
| <70                                                           | 3363                               | ref                      | 2726       | 1.00 (0.95–1.06)         | 987         | 1.04 (0.97–1.12)         |
| 70–79                                                         | 1892                               | 1.05 (0.99–1.11)         | 1633       | 1.09 (1.03–1.16)         | 565         | 1.07 (0.98–1.17)         |
| ≥80                                                           | 773                                | 1.07 (0.98–1.16)         | 679        | 1.11 (1.02–1.21)         | 204         | 1.03 (0.89–1.19)         |
| Ischemic heart disease excluding angina pectoris <sup>b</sup> |                                    |                          |            |                          |             |                          |
| <70                                                           | 1745                               | ref                      | 1429       | 1.02 (0.95–1.10)         | 532         | 1.07 (0.97–1.18)         |
| 70–79                                                         | 989                                | 1.03 (0.95–1.12)         | 878        | 1.11 (1.02–1.21)         | 323         | 1.14 (1.01–1.29)         |
| ≥80                                                           | 448                                | 1.10 (0.99–1.23)         | 400        | 1.16 (1.04–1.30)         | 125         | 1.12 (0.93–1.35)         |
| Acute myocardial infarction                                   |                                    |                          |            |                          |             |                          |
| <70                                                           | 1080                               | ref                      | 795        | 0.97 (0.89–1.07)         | 298         | 1.05 (0.92–1.19)         |
| 70–79                                                         | 614                                | 1.01 (0.91–1.12)         | 514        | 1.06 (0.95–1.19)         | 191         | 1.15 (0.99–1.35)         |
| ≥80                                                           | 317                                | 1.14 (1.00–1.30)         | 249        | 1.11 (0.96–1.27)         | 73          | 1.02 (0.80–1.29)         |

<sup>a</sup> Adjusted for age (by design), cohort, baseline year, sex, marital status, area-level income, education, smoking, and physical activity<sup>b</sup> Information on angina pectoris was not available for the FINRISK cohort.

non-statistically significant HR of 1.22 for IHD (21). This study, however, did not adjust for education. In our study, without adjustment for education, we found a statistically significant HR of 1.15 for all IHD and 1.20 for IHD excluding angina pectoris (supplementary table S3). A Swedish cohort study based in Stockholm County, one of the areas included in our pooled cohort, found that occupational noise >75 dB(A) was associated with an increased risk of AMI with an odds ratio of 1.17 (95% CI 0.98–1.41) (10). This study also did not adjust for education; therefore, when comparing our findings without education adjustment, we find similar risks.

Care must be exercised when interpreting results adjusted for education as it is closely related to occupational noise levels. In our study, <1% of those exposed to ≥85 dB(A) of noise reported high education. Therefore, we believe the true association lies somewhere between the two estimates, indicating a risk of >12–<24% for myocardial infarction and >10–<20% for IHD excluding angina pectoris. When attempting to stratify for education, we were unable to estimate the risk of occupational noise ≥85 dB(A) for those with high education; however, we observe a tendency for increased risks for myocardial infarction at levels >70 dB(A) for those with low and medium education (supplementary table S4).

A recent study on self-reported occupational noise did not find an association between working in a noisy environment and myocardial infarction (22). However, because of the subjective nature of the exposure, it is possible that this type of exposure classification cannot differentiate between those exposed to some occupational noise as opposed to those exposed to very high noise levels. However, in our study, we observed an indication of an increased, albeit non-statistically significant, association between IHD and occupational noise already starting at 70 dB(A). Other studies using similar self-reported measures did find increased risk for myocardial infarction, and a significant increase for car-

diovascular heart disease associated with self-reported exposure to occupational noise (23, 24).

There is a considerable difference in incident IHD, and in particular AMI, between men and women at the same age, with much lower rates among women (25). When stratifying by sex, we did not find any association between occupational noise and any of the outcomes among women as we did for men. Though few studies investigate women separately, one case-control study did find a non-statistically significant increased risk for AMI among women exposed to 85–94 dB(A) of occupational noise but not for men exposed to the same levels (26). In this study, 11 women were exposed to noise levels >85 dB(A), and of these there were 4 cases, thus creating wide CI and making comparisons difficult. Similarly, our study only had 14 cases of AMI in the highest noise group, suggesting insufficient statistical power in this outcome. Nevertheless, the consistent lack of increased risk across all IHD outcomes we found suggests that women may respond differently to occupational noise than men. This could reflect differences in noise exposure characteristics or patterns that exist both within and between occupational groups which the current version of the JEM cannot capture. Alternatively, IHD appears at a later age compared to men (27), where the effects of occupational noise at earlier ages are less influential than other risk factors. The presumed protective effect of estrogen (28) may further limit the development of disease during working age.

We found no evidence that very recent exposure to noise was associated with IHD. Censoring after five years had the strongest effect on all outcomes. This indicates that there may be a temporal aspect to the association between occupational noise exposure and IHD. However, when censoring after one year, we found a weaker effect for IHD excluding angina pectoris and AMI than the analysis censoring after five years and, for AMI, the uncensored analyses. One other study on

the same cohorts investigating stroke found a stronger association for those exposed to  $\geq 85$  dB(A) of occupational noise after censoring for one year (29). Another study found that among all workers, longer follow-up times resulted in slightly increased risks of coronary heart disease (30). It is important to note that statistical power and comparability was lost when restricting the analysis to the younger individuals in the cohort who were not yet retired at the time of enrollment and had occupational exposure after baseline, which can affect our ability to detect a true association. Therefore, this analysis may be best suited for a larger cohort.

When evaluating the combined effect of road traffic and occupational noise, we did not find any evidence that being exposed to the highest level of both exposures led to an increase in the risk of AMI despite evidence of independent associations due to both occupational noise found in the present study and road traffic noise in a separate study of the same cohorts (20). A previous study of Swedish men and women in Stockholm County found that the combination of occupational and road traffic noise led to a synergistic effect on myocardial infarction risk (10). The discrepancy in results could be because our study includes a wider range of road traffic noise exposures. The study by Selander and colleagues (10) characterized high road traffic noise exposure as being exposed to  $>51.8$  dB(A), whereas we included three categories of noise, where the lowest category was  $<55$  dB  $L_{den}$ . A previous study on this cohort found no increased risk of IHD at noise levels  $<55$  dB  $L_{den}$  and found that there may be differences in both noise exposure distribution and assessment between the cohorts included (20). Additionally, a similar study to ours found no evidence of an interaction between exposure to occupational and road traffic noise for stroke (29).

Several biological mechanisms may underlie the observed associations. Chronic exposure to high-intensity noise activates the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system, leading to increased secretion of stress hormones such as cortisol and catecholamines (5). These hormonal changes can elevate blood pressure, heart rate, and vascular inflammation, thereby promoting atherosclerosis and increasing the risk of ischemic events (4). Noise exposure can also disrupt sleep, further contributing to metabolic and cardiovascular dysregulation (6). While these mechanisms are well documented for environmental noise, evidence for occupational noise remains limited. Our findings of a risk increase primarily at very high exposure levels suggest that intensity and duration are critical determinants of cardiovascular effects.

This study has a few limitations. The use of a JEM implies that exposure is assigned at the occupational group level rather than measured individually. While this approach reduces differential misclassification, it does

not capture within-occupation variability. Additionally, we were unable to adjust for co-occurring exposures such as physical workload, vibration, or shift work. As a result, the estimated associations may reflect a mixture of occupational exposures rather than the isolated effect of noise. We did not have information on working hours, which could lead to nondifferential misclassification as the JEM was developed assuming full-time work. Having exposure categories rather than continuous estimates limited our ability to determine a risk threshold and the lack of sex-specific occupational noise levels in the JEM impairs our ability to accurately estimate sex differences in exposure. We also lacked information on other occupational and environmental exposures that may confound the association. Occupational noise exposure limits in Sweden are harmonized with the European Union (EU) minimum requirement, suggesting that the Swedish JEM is largely applicable to the Nordics and generalizable across the EU member states. However, generalizability is limited beyond the EU, particularly to regions with different noise regulations. Lastly, we cannot rule out potential residual confounding. Though the association between occupational noise and IHD remained after adjusting for lifestyle and socioeconomic covariates, confounding due to other sources, such as other occupational exposures, remain a possibility. However, one study found that the effect of occupational noise on coronary heart disease remained even after adjusting for shift work and physical workload (31), suggesting an independent effect of noise.

Our study has several strengths. We use data from a large number of participants with complete information on lifestyle factors from several countries, allowing for more detailed confounder control while maintaining statistical power. Our study also benefits from the use of an extensive JEM, which covers  $>300$  occupations, and is based on objective measurement reports, and has been validated and shown to have no systematic differences in classification between raters (18). Moreover, the cohorts included high quality, national registers to assess the outcome, which provided accurate information on diagnosis of IHD. Lastly, we were able to assess associations with road traffic noise in addition to occupational noise using validated models with high spatial resolution.

### Concluding remarks

Our results show that exposure to high levels of occupational noise was associated with an increased risk of AMI and IHD excluding angina pectoris, which is corroborated by previous findings. Further studies should investigate the precise levels at which risk increases and whether this association is confounded by other occupational exposures.

## Acknowledgements

This work was supported by NordForsk, grant number 83597. Individual cohort funding is shown in supplementary table S1.

## Disclosure of conflict of interests

All authors declare no conflicts of interest.

## Ethical approval

Data collection was approved by relevant national ethics review boards for each individual cohort.

## References

- Global Burden of Cardiovascular Diseases and Risks 2023 Collaborators. Global, regional, and national burden of cardiovascular diseases and risk factors in 204 countries and territories, 1990–2023. *J Am Coll Cardiol* 2025 Dec;86(22):2167–243. <https://doi.org/10.1016/j.jacc.2025.08.015>.
- European Commission. EU strategic framework on health and safety at work 2021–2027. Brussels, 2021.
- Eurofound WA. Cabrita J et al. 6th European working conditions survey – 2017 update: Publications Office, 2017.
- Münzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. *Eur Heart J* 2014 Apr;35(13):829–36. <https://doi.org/10.1093/eurheartj/ehu030>.
- Münzel T, Sørensen M, Daiber A. Transportation noise pollution and cardiovascular disease. *Nat Rev Cardiol* 2021 Sep;18(9):619–36. <https://doi.org/10.1038/s41569-021-00532-5>.
- Münzel T, Molitor M, Kuntic M, Hahad O, Röösl M, Engelmann N et al. Transportation noise pollution and cardiovascular health. *Circ Res* 2024 Apr;134(9):1113–35. <https://doi.org/10.1161/CIRCRESAHA.123.323584>.
- Teixeira LR, Pega F, Dzhambov AM, Bortkiewicz A, da Silva DT, de Andrade CA et al. The effect of occupational exposure to noise on ischaemic heart disease, stroke and hypertension: A systematic review and meta-analysis from the WHO/ILO Joint Estimates of the Work-Related Burden of Disease and Injury. *Environ Int* 2021 Sep;154:106387. <https://doi.org/10.1016/j.envint.2021.106387>.
- Dzhambov AM, Dimitrova DD. Occupational noise and ischemic heart disease: A systematic review. *Noise Health* 2016;18(83):167–77. <https://doi.org/10.4103/1463-1741.189241>.
- Pershagen G, Pyko A, Aasvang GM, Ögren M, Tiittanen P, Lanki T et al. Road traffic noise and incident ischemic heart disease, myocardial infarction, and stroke: A systematic review and meta-analysis. *Environ Epidemiol* 2025 May;9(3):e400. <https://doi.org/10.1097/EE9.0000000000000400>.
- Selander J, Bluhm G, Nilsson M, Hallqvist J, Theorell T, Willix P et al. Joint effects of job strain and road-traffic and occupational noise on myocardial infarction. *Scand J Work Environ Health* 2013 Mar;39(2):195–203. <https://doi.org/10.5271/sjweh.3324>.
- Tjønneland A, Olsen A, Boll K, Stripp C, Christensen J, Engholm G et al. Study design, exposure variables, and socioeconomic determinants of participation in Diet, Cancer and Health: a population-based prospective cohort study of 57,053 men and women in Denmark. *Scand J Public Health* 2007;35(4):432–41. <https://doi.org/10.1080/14034940601047986>.
- Borodulin K, Tolonen H, Jousilahti P, Jula A, Juolevi A, Koskinen S et al. Cohort Profile: The National FINRISK Study. *Int J Epidemiol* 2018 Jun;47(3):696–696i. <https://doi.org/10.1093/ije/dyx239>.
- Wilhelmsen L, Berglund G, Elmfeldt D, Samuelsson O, Svardsudd K. The Multifactor Primary Prevention Trial in Göteborg, Sweden. Comparison with a previously untreated population sample. *Drugs* 1986;31 Suppl 1:47–51. <https://doi.org/10.2165/00003495-198600311-00009>.
- Berglund G, Elmstahl S, Janzon L, Larsson SA. The Malmö Diet and Cancer Study. Design and feasibility. *J Intern Med* 1993 Jan;233(1):45–51. <https://doi.org/10.1111/j.1365-2796.1993.tb00647.x>.
- Lagergren M, Fratiglioni L, Hallberg IR, Berglund J, Elmstahl S, Hagberg B et al.; The Swedish National Study on Aging and Care (SNAC). A longitudinal study integrating population, care and social services data. The Swedish National study on Aging and Care (SNAC). *Aging Clin Exp Res* 2004 Apr;16(2):158–68. <https://doi.org/10.1007/BF03324546>.
- Magnusson PK, Almqvist C, Rahman I, Ganna A, Viktorin A, Walum H et al. The Swedish Twin Registry: establishment of a biobank and other recent developments. *Twin Res Hum Genet* 2013 Feb;16(1):317–29. <https://doi.org/10.1017/thg.2012.104>.
- Pyko A, Andersson N, Eriksson C, de Faire U, Lind T, Mitkovskaya N et al. Long-term transportation noise exposure and incidence of ischaemic heart disease and stroke: a cohort study. *Occup Environ Med* 2019 Apr;76(4):201–7. <https://doi.org/10.1136/oemed-2018-105333>.
- Sjöström M, Lewné M, Alderling M, Selander J, Gustavsson P. An updated job-exposure matrix for occupational noise: development and validation. *Ann Work Expo Health* 2024 Feb;68(2):146–54. <https://doi.org/10.1093/annweh/wxad074>.
- Persson Å, Pyko A, Stucki L, Ögren M, Åkesson A, Oudin A et al. Long-term exposure to transportation noise and obesity: A pooled analysis of eleven Nordic cohorts. *Environ Epidemiol* 2024 Jul;8(4):e319. <https://doi.org/10.1097/EE9.0000000000000319>.

20. Pyko A, Roswall N, Ögren M, Oudin A, Rosengren A, Eriksson C et al. Long-term exposure to transportation noise and ischemic heart disease: a pooled analysis of nine Scandinavian cohorts. *Environ Health Perspect* 2023 Jan;131(1):17003. <https://doi.org/10.1289/EHP10745>.
21. Eriksson HP, Andersson E, Schiöler L, Söderberg M, Sjöström M, Rosengren A et al. Longitudinal study of occupational noise exposure and joint effects with job strain and risk for coronary heart disease and stroke in Swedish men. *BMJ Open* 2018 Apr;8(4):e019160. <https://doi.org/10.1136/bmjopen-2017-019160>.
22. Michaud DS, Marro L, McNamee JP. Self-reported occupational noise exposure and cardiovascular disease in Canada: Results from the Canadian Health Measures Survey. *J Acoust Soc Am* 2021 Aug;150(2):990–1000. <https://doi.org/10.1121/10.0005588>.
23. Gan WQ, Davies HW, Demers PA. Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999–2004. *Occup Environ Med* 2011 Mar;68(3):183–90. <https://doi.org/10.1136/oem.2010.055269>.
24. Gopinath B, Thiagalingam A, Teber E, Mitchell P. Exposure to workplace noise and the risk of cardiovascular disease events and mortality among older adults. *Prev Med* 2011 Dec;53(6):390–4. <https://doi.org/10.1016/j.ypmed.2011.10.001>.
25. Mosca L, Barrett-Connor E, Wenger NK. Sex/gender differences in cardiovascular disease prevention: what a difference a decade makes. *Circulation* 2011 Nov;124(19):2145–54. <https://doi.org/10.1161/CIRCULATIONAHA.110.968792>.
26. Kersten N, Backé E. Occupational noise and myocardial infarction: considerations on the interrelation of noise with job demands. *Noise Health* 2015;17(75):116–22. <https://doi.org/10.4103/1463-1741.153403>.
27. McSweeney JC, Rosenfeld AG, Abel WM, Braun LT, Burke LE, Daugherty SL et al.; American Heart Association Council on Cardiovascular and Stroke Nursing, Council on Clinical Cardiology, Council on Epidemiology and Prevention, Council on Hypertension, Council on Lifestyle and Cardiometabolic Health, and Council on Quality of Care and Outcomes Research. Preventing and experiencing ischemic heart disease as a woman: state of the science: a scientific statement from the American Heart Association. *Circulation* 2016 Mar;133(13):1302–31. <https://doi.org/10.1161/CIR.0000000000000381>.
28. Moolman JA. Unravelling the cardioprotective mechanism of action of estrogens. *Cardiovasc Res* 2006 Mar;69(4):777–80. <https://doi.org/10.1016/j.cardiores.2006.01.001>.
29. Thacher JD, Roswall N, Lissåker C, Aasvang GM, Albin M, Andersson EM et al. Occupational noise exposure and risk of incident stroke: a pooled study of five Scandinavian cohorts. *Occup Environ Med* 2022 Aug;79(9):594–601. <https://doi.org/10.1136/oemed-2021-108053>.
30. Virkkunen H, Kauppinen T, Tenkanen L. Long-term effect of occupational noise on the risk of coronary heart disease. *Scand J Work Environ Health* 2005 Aug;31(4):291–9. <https://doi.org/10.5271/sjweh.885>.
31. Virkkunen H, Härmä M, Kauppinen T, Tenkanen L. The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. *Occup Environ Med* 2006 Jun;63(6):378–86. <https://doi.org/10.1136/oem.2005.022558>.

Received for publication: 28 April 2026