



The effects of occupational noise exposure on the cardiovascular system: a review

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Abstract: The effects of occupational noise exposure on health are increasingly recognized. It is accepted that, in the absence of any protective measures, occupational noise can adversely affect both auditory and non-auditory systems. Beyond noise-induced hearing loss (HL), occupational noise can also precipitate sleep disturbance, feelings such as annoyance, and a higher incidence of occupational accidents. In addition, epidemiological and experimental studies have demonstrated that occupational noise contributes to hypertension and to other cardiovascular diseases (CVDs). These associations may exist because occupational noise is an environmental stressor that stimulates the sympathetic and endocrine systems, and triggers release of stress hormones. This review describes the impact of occupational noise exposure on the cardiovascular system and calls for a mitigation strategy for occupational noise reduction.

Keywords: Occupational noise; cardiology; hypertension

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Introduction

The World Health Organization (WHO) reported that approximately 300,000 Europeans die and 4% of the gross domestic product is lost due to poor working conditions annually. The substandard conditions may be responsible for 1.6% of total disease burden, and include injuries, noise, carcinogens, airborne particulate matter and ergonomic hazards. Noise risks accounted for 22% of the occupational disease burden. The magnitude of the noise issue is substantial if it is considered that around 22 million workers in the United States of America are exposed to hazardous occupational noise each year (1).

Noise is a major occupational hazard at many different types of industrial sites (2). In developing countries, occupational noise is a graver problem because of poor planning and inadequate building construction. The level of occupational noise is often regarded as an indicator in

hazard evaluation at industrial sites. In addition to the intensity of the noise, the health effects of occupational noise relate to the duration of exposure and the nature of the noise (e.g., continuous, impulsive, low or high frequency), as well as individual sensitivity.

It is established that a noisy environment can produce adverse health effects when humans are exposed to an intensity that exceeds their body's ability to adapt. The National Institute for Occupational Safety and Health [1998] reported the maximum noise equivalent sound level as 85 dB(A) for industrial sites. Adverse health effects are associated with noise exposure above this recommended standard. Employees who are directly exposed to a noisy environment often ignore the potential damaging health effects of noise as it is a long-term continuous process. Occupational noise can produce specific health effects involving both auditory and non-auditory systems (3). In

addition to hearing loss (HL), noise can also cause feelings such as annoyance (4), cognitive impairment (5,6), sleep disorders (7), gastrointestinal disorders (8), endocrine problems (9), psychophysiological effects (10), and cardiovascular disease (CVD) (11).

The term CVD encompasses disorders of the heart and/or blood vessels. It includes coronary heart disease (CHD) such as myocardial infarction (heart attack), cerebrovascular disease such as stroke, increased blood pressure (hypertension), peripheral artery disease, rheumatic heart disease, congenital heart disease, and heart failure (12). Mortality data from 2013 indicated that more than 2,200 Americans died from CVD daily, being an average of one death every 40 seconds (13). High lipid concentrations (hyperlipidemia), diabetes mellitus, obesity, an unhealthy diet, cigarette smoking, and physical inactivity are recognized contributors to CVD. In addition, both observational and experimental studies provide evidence that high-intensity occupational noise also contributes to CVD (14).

The WHO is committed to reducing morbidity and mortality of CVD via global reduction in risk factors. This review focuses on the effects of occupational noise exposure on the cardiovascular system, and calls for practical action and strategies to limit and control occupational noise to help prevent CVD.

Cardiovascular effects

Investigators have repeatedly proven that occupational noise exposure can produce adverse effects on the cardiovascular system (15) including hypertension, ischemic heart disease (IHD) and stroke. Occupational noise exerts its effects on physical health via direct and indirect mechanisms (16,17). Effects on the auditory system and on sleep are result from direct interactions with synapses. Indirectly, noise causes annoyance through emotional and cognitive responses. Occupational noise is an environmental stressor that can stimulate autonomic nervous and endocrine systems, disrupting homeostasis. This can result in development of risk factors such as changes in blood pressure, lipid concentrations and blood viscosity that can lead to CVD.

Hypertension

The clinical impact of occupational noise exposure on blood pressure has been most widely studied after the effect on hearing. Most studies showed that long-term exposure to

occupational noise significantly raised systolic and diastolic blood pressure (SBP and DBP, respectively) and increased the prevalence of hypertension.

In one study, noise-exposed sanitary fixture industry workers had significantly greater mean SBP and DBP, and a higher frequency of hypertension (all $P < 0.05$) compared with controls (18). One concern is that the noise level detected in the external environment may not reflect the true intensity of inner ear noise perception. In male workers, high-frequency HL was used as a biomarker for occupational noise exposure, and the relationships between the noise and hypertension explored. The prevalence of hypertension was significantly higher in the high-frequency HL (43.5%; $P = 0.021$) and median-frequency HL (42.1%; $P = 0.029$) groups than in the low-frequency HL group (33.2%). The high and median-frequency HL workers had 1.48-fold [95% confidence interval (CI): 1.02–2.15; $P = 0.040$] and 1.46-fold (95% CI: 1.03–2.05; $P = 0.031$) higher risks of hypertension relative to the low-frequency HL workers. Employment duration was significantly and positively correlated with hypertension among workers with mean bilateral hearing loss values (HLVs) ≥ 15 dB at 4 kHz ($P < 0.001$) and 6 kHz ($P < 0.001$) (19). A cross-sectional study of Chinese coal miners showed that, after adjustment for age, body mass index, smoking status and alcohol consumption, workers exposed to occupational noise had a higher prevalence ($P = 0.012$) and increased risk of hypertension [age-adjusted odds ratio (OR): 1.52; 95% CI: 1.07–2.15] compared with controls. Mean SBP and DBP in the noise-exposed groups were significantly higher than in the control group ($P = 0.006$ and $P = 0.002$, respectively) (20). A recent cross-sectional survey including 1,874 Chinese workers using self-reported noise exposure and audiometrically measured HL performed by Zhou *et al.* showed that occupational noise-exposed subjects had significantly higher blood pressure levels than nonexposed subjects [SBP: 123.18 $\pm$ (standard deviation) 12.44 *vs.* 119.80 $\pm$ 12.50 mmHg; DBP: 77.86 $\pm$ 9.34 *vs.* 75.49 $\pm$ 8.73 mmHg]. Noise exposure (any) was associated with an increase in the prevalence of hypertension (OR: 2.03; 95% CI: 1.15–3.58) (21). National Health Interview Survey data from 2014 were examined to estimate the associations of occupational noise exposure, hearing difficulty and cardiovascular conditions with occupational noise exposure within US industries and occupations. Results indicated that hypertension, elevated cholesterol, and hearing difficulty are more prevalent among noise-exposed workers (22).

In addition to this cross-sectional evidence, many cohort

studies show a causal relationship between occupational noise and hypertension. A 10-year follow-up study of 578 male workers in Taiwan associating exposure to ≥ 85 A-weighted decibels [dB(A)] with a 1.93-fold (95% CI: 1.15–3.22) increased risk of hypertension compared with lower exposure [< 80 dB(A)]. There was also a significant exposure-response pattern ($P=0.016$) between the risk of hypertension and the stratum of noise exposure (23). This work provided evidence for a link between occupational noise and high blood pressure, and also for dose-response relationship between the two. A cohort study of 10,872 sawmill workers in British Columbia also showed a monotonic increase in hypertension incidence with cumulative exposure. The greatest relative risk (RR) was 1.5 in workers exposed to 85 dB(A) for more than 30 years (24).

A meta-analysis of articles published between year 1950 and 2008 included 15 studies that associated occupational noise exposure with hypertension. For each 5-dB(A) increase in occupational noise exposure, the RR for hypertension increased by 1.14 (95% CI: 1.01–1.29) (25). A systematic critical literature review of 12 prospective studies also provided strong associations between workplace noise and hypertension. Exposure to noise at work was consistently and positively associated with hypertension [hazard ratio (HR): 1.68; 95% CI: 1.10–2.57] (26).

However, a study of Japanese male blue-collar workers found inconsistent evidence. A significantly lower prevalence of hypertension was found in the noisy workplace group than in the non-noisy group (16.9% *vs.* 34.7%, respectively; $P<0.01$). Logistic regression analysis with adjustments for confounding factors also showed a significant inverse association between hypertension and workplace noise conditions (OR: 0.48; 95% CI: 0.28–0.81) (27). The conflicting findings of this research may relate to the use of hearing protection devices (HPDs). Employees in a noisy workplace may gradually develop awareness for the potential impacts of noise on health after receiving health education. This may contribute to increased use of HPDs and health management. Therefore, although occupational noise has a strong connection with high blood pressure, the effects can be prevented with proper and continuous use of HPDs.

IHD and CHD

Approximately 15.5 million adult Americans have IHD, which was the major cause of premature death in modern societies between 2009 and 2012 (28). For this reason,

cardiovascular disorders such as IHD have been a primary focus of epidemiological noise research. However, evidence of a relationship between occupational noise exposure and IHD is limited and inconsistent. This may be because the effects of occupational noise on IHD appear small relative to other occupational risks. It is also difficult to accurately assess the contribution of occupational noise to IHD due to challenges with characterizing exposure, insufficient control of confounding factors, and a lack in-depth study of the dose-response relationship.

One cross-sectional study provided evidence that chronic occupational noise exposure may be associated with an increased risk of IHD. This study included 6,307 participants of at least 20 years of age from the National Health and Nutrition Examination Survey (NHANES) of 1999–2004. Participants who were chronically exposed to occupational noise had a 2–3-fold increased prevalence of angina pectoris and CHD compared with non-exposed individuals. After adjustment for various potential confounders, the ORs (95% CIs) for angina pectoris and CHD were 2.91 (1.35–6.26) and 2.04 (1.16–3.58), respectively. There were clear exposure-response relationships for the observed associations, with the links being particularly strong for participants aged < 50 years, men and current smokers. Therefore, chronic exposure to occupational noise is strongly associated with the prevalence of CHD, especially in younger male smokers (29).

One cohort of 27,464 blue-collar workers from 14 lumber mills in British Columbia provided evidence that chronic noise exposure was associated with an increased risk of death from acute myocardial infarction. There was an exposure-response trend, with a RR of 1.5 (95% CI: 1.1–2.2) in the group with highest exposure. The highest RRs (2.0–4.0) were observed during the subjects' working years (30).

One meta-analysis based on just four studies quantifying the risk of IHD reported that there was an RR of 1.06 (95% CI: 0.95–1.18) among workers exposed to > 70 –85 dB(A) (31). Another meta-analysis of 15 studies suggested that occupational noise was a risk factor for IHD morbidity. Some evidence suggested a higher risk of IHD among workers exposed to objectively measured noise > 75 –80 dB(A) for less than 20 years based on five studies. Out of four studies, a higher mortality risk was suggested by one moderate-quality study relying on self-rated exposure and one high-quality study using objective measures (32).

In contrast, one 16-year longitudinal study of 2,998 men aged 53–75 years did not relate long-term occupational noise exposure to risk of IHD. The HR for IHD mortality

was 0.97 (95% CI: 0.71–1.33) and the HR for all-cause mortality was 1.01 (95% CI: 0.89–1.15) in comparison with unexposed men and after adjusting for potential confounders (33). McNamee *et al.*, (34) reported that the OR for increased IHD risk among noise-exposed nuclear power workers was not higher compared with unexposed men and after adjustment for confounding factors.

The evidence for occupational noise effects on IHD/CHD in workers exposed to high levels of occupational noise is limited and the associations are weak. However, the possible small risk conferred by occupational noise deserves attention because of the large number of people exposed to high noise levels and the high prevalence of IHD.

Atherosclerosis

Literature on the relationship between occupational noise and atherosclerosis is scant. One population-based cohort study indicated that nighttime traffic noise was independently associated with subclinical atherosclerosis (35). Further research is needed to clarify whether there is a link between occupational noise and atherosclerosis.

Hyperlipidemia is one of the most common risk factors for atherosclerosis. Noise is an environmental stressor that is believed to activate the endocrine system. Elevated stress hormone concentrations may stimulate an increase in blood lipids suggesting a possible mechanism for noise exposure to cause atherosclerosis (36). Workers exposed to noise greater than 90 dB(A) without ear protection appear to have increased triglyceride level (37). One historical cohort study included male workers in high-level (n=154) and low-level (n=146) noise exposure groups and found a significant relationship between noise exposure and triglyceride concentrations in the two groups (38). However, study findings are not consistent. One cross-sectional study of Danish industrial (n=460) workers, with financial workers (n=69) as a reference, reported no relationship between occupational noise exposure and serum lipid concentrations after adjusting for HPD use, body mass index and smoking status (39). This may indicate that the link between occupational noise exposure and atherosclerosis, if there is one, is not mediated via serum lipids.

Electrocardiograph (ECG) abnormalities

One cross-sectional study that included 72 paper industry

workers exposed to noise and two control groups not exposed to noise found that the noise-exposed group had a higher prevalence of ECG abnormalities (40). In the previously described study on sanitary fixture industry workers, there was also a higher frequency of ECG abnormalities among exposed versus unexposed workers ($P<0.05$) (18).

Heart rate

Some evidence shows that occupational exposure to noise may lead to increased heart rate in workers. One study of workers in the automotive parts industry revealed significant differences in mean heart rate changes ($P<0.001$) between the noise-exposed and unexposed groups (41). Noise exposure was moderately and positively correlated with heart rate (Spearman's $\rho=0.46$; $P<0.001$). A mixed-effects linear regression model indicated that a 1-dB increase in noise exposure was associated with a 0.17 beats per minute increase in heart rate ($P=0.01$) after controlling for work activities, age, smoking, perceived stress and unfavorable physical working conditions (42). A further study of workers exposed to occupational noise above (n=100) or below (n=50) 80 dB found blood concentrations of insulin, glucose and homocysteine, and blood pressure and heart rate to be significantly greater in the high noise level group. Thus, these results indicate that industrial occupational noise could contribute to an increased heart rate (43).

Stroke

It remains unknown whether noise exposure causes stroke (44). A study on the association between aircraft noise and CVD near Heathrow airport in London showed that high levels of aircraft noise in the community might increase the risk of stroke (45). A population-based cohort study of 57,053 subjects showed a relationship between long-term exposure to traffic noise and increased risk of stroke. The incidence rate ratio (IRR) for stroke was 1.14 (95% CI: 1.03–1.25) per 10 dB increase in road traffic noise (L_{DEN} , day-evening-night-level, a measure of noise which summarizes average noise exposure, see *Table 1*) among subjects older than 64.5 years (17). Although the levels of occupational noise are a magnitude higher than community aircraft noise, a dose-response relationship between occupational noise and stroke was not found in epidemiological studies. A cohort study of industrial

Table 1 Noise metrics and their definition

Noise metrics	Definition
SPL	Sound pressure level is a logarithmic measure of the effective pressure of a sound relative to a reference value. It is measured in decibels (dB, see below) higher than a reference level. The reference sound pressure in air is 20 μ Pa (2×10^{-5} Pa), which is thought to be the human hearing threshold at a sound frequency of 1,000 Hz
dB scale	A logarithmic scale to measure sound pressure level. A two-fold increase in sound energy (e.g., two identical jackhammers instead of one) will cause the sound pressure level to increase by 3 dB. A ten-fold increase in sound energy (10 jackhammers) will cause the sound pressure level to increase by 10 dB, which is perceived as about twice as loud
L_{Aeq}	The energy-equivalent average A-weighted sound pressure level expressed in decibels is the most commonly used noise exposure metric reflecting (energetically) averaged noise exposure over a certain time period. The A-weighting (represented by the A in L_{Aeq}) accounts for the different sensitivity of the human ear at different sound frequencies. The filter and time period used for averaging are often indicated in subscript—e.g., L_{Aeq} 8 h, L_{Aeq} 23–7 h, or L_{night}
L_{DEN}	Day-evening-night-level, the 24 h L_{Aeq} with a 5 dB penalty for the evening (usually 6 p.m. to 10 p.m. or 7 p.m. to 11 p.m.) and a 10 dB penalty for the night (usually 10 p.m. to 6 a.m. or 11 p.m. to 7 a.m.). The penalties are introduced to indicate people's extra sensitivity to noise during the evening and the night

(n=116,568) and financial (n=47,679) workers linked to Danish registries between 2001 and 2007 showed that the industrial workers had a 27% higher stroke risk although the result was not related to noise levels. The findings suggested that a higher noise level and longer exposure duration among industrial workers were unrelated to the risk of stroke (46).

Personal protective equipment (PPE)

Engineering measures may be unable to reduce noise due to constraints in costs, technology, and the production process. In these circumstances, failure to meet safety standards means that the use of PPE is advised. PPE to mitigate noise exposure, also called HPDs, includes devices such as earplugs and earmuffs. The National Institute for Occupational Safety and Health [1998] requires workers to wear HPDs when exposure to noise in the workplace is over 80 dB(A). In noisy environments, workers should be equipped with HPDs and they should be widely distributed. However, very few workers are willing to wear HPDs or choose to wear them irregularly. This generally results from discomfort caused by wearing the HPDs for a long duration, their negative influence on communication, and on their ability to reduce environmental awareness leading to an increased risk of accidents. While it is clear that new devices need to be developed that have greater user acceptability, workers should improve their protection awareness and continue using HPDs to attenuate high-intensity sounds in the meantime.

Conclusions

Most evidence shows a strong association between occupational noise exposure and elevated SBP and DBP, and an increased prevalence of hypertension. For other CVDs, existing research suggests only a weak association with limited evidence for occupational noise effects. More studies are needed to investigate the relationship between occupational noise and the cardiovascular system in addition to the effects of hypertension.

Use of hearing protection equipment can help mitigate the adverse effects of occupational exposure to high noise levels. Worker awareness should be established about the harmful effects of noise on hearing and on other physiological systems via compulsory education and training programs. Employers are advised to control occupational noise by managing the environment, duties, health examinations and the health education of their workers.

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Footnote

Conflicts of Interest: All authors have completed the ICMJE

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Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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