Caspian Journal of Health Research
"Caspian J Health Res"

Journal Homepage: https://cjhr.gums.ac.ir

Review Article

Association Between Occupational Noise and Cardiovascular Events: A Literature Review

Elahe Oveisi[1*], Duha Ali[2]

1. Department of Ergonomics, Research Center for Health Sciences, School of Public Health, Hamadan University of Medical Sciences, Hamadan, Iran.
2. Department of Industrial Engineering, Auburn University, Auburn, Alabama, USA.

Use your device to scan and read the article online

Citation Oveisi E, Ali A. Association Between Occupational Noise and Cardiovascular Events: A Literature Review. Caspian Journal of Health Research. 2022; 7(2):97-106. https://doi.org/10.32598/CJHR.7.2.420.1

Running Title Occupational Noise and Cardiovascular Disease

https://doi.org/10.32598/CJHR.7.2.420.1

Article info:
Received: 10 Jan 2022
Accepted: 15 Mar 2022
Published: 01 Apr 2022

ABSTRACT

Background: Occupational noise is an occupational risk factor that might affects the body system.

Objectives: A literature review research was implemented to review the published studies that reported relationships between occupational noise and Cardiovascular Diseases (CVDs).

Materials & Methods: Three databases were searched. A total of eight articles were identified and included.

Results: The results show that noise exposure and high noise levels were associated with an increased CVDs. Additionally, some environmental factors such as temperature imbalance, job strain as well as other social and individual risk factors influence the risk of CVDs.

Conclusion: Evidence shows that occupational noise is a prominent risk factor for CVDs. Therefore, it is suggested that noise levels exposure should be reduced by personal and social protective equipment or revision measures.

Keywords: Occupational noise, Myocardial infarction, Cardiovascular diseases

*** Corresponding Author:**
Elahe Oveisi, MSc.
Address: *Department of Ergonomics, School of Public Health, Hamadan University of Medical Sciences, Hamadan, Iran.*
Tel: *+98 (919) 3617827*
E-mail: *elahe.oveysi1992@gmail.com*

Caspian Journal of Health Research

1. Introduction

Cardiovascular Diseases (CVDs) are currently accounted for nearly half of non communicable diseases and are considered as the leading cause of mortality, responsible for 17.3 million deaths per year, a number that is predicted to increase to higher than 23.6 million by 2030 [1, 2]. In 2004, the World Health Organization (WHO) estimated that 12.2% of worldwide deaths were from ischemic heart disease [3]. Rates of mortality from Ischemic Heart Disease (IHD) have declined in most high-income countries, although CVDs still account for one in three deaths in the US in 2008 [4]. In addition, CVDs are the main cause of mortality in the United States, and are considered one of the costliest chronic diseases. As the population ages, CVDs costs are expected to increase substantially [5]. So, it is imperative to identify CVDs' risk factors and the age groups who are more susceptible to CVDs to be able reduce the rate of CVDs. Many risk factors, which include hypertension, smoking, abdominal obesity, abnormal lipids, and diabetes mellitus, as well as stress and lack of regular physical activity, are significant contributors to cardiovascular mortality, and responsible for more than 90% of all Myocardial Infarctions (MIs) [6, 7]. Some occupational exposures such as noise, air, and radiofrequency transmitters risk factors may result in CVDs [8-10]. Many workers are consistently under occupational noise exposure, and it has been overlooked in many cases while it is one of the most dangerous risk factors in workplaces.

Occupational noise is a common exposure in the work environment [11]. Surveys show that 23.9% of the working population in Germany are often exposed to noise, and 54% feel burdened by noise [12]. The National Health and Nutrition Examination Survey (NHANES), conducted between 1999 and 2004, found that 17% of workers reported hazardous noise exposure in their workplace in the United States [13].

High levels of industrial noise exposure cause a significant risk to health, safety, and hearing acuity [14-16]. In addition to the association on hearing, nonauditory results such as those on the cardiovascular systems are discussed [11], there is physiological evidence from laboratory and field studies showing that the underlying mechanisms of the cardiovascular associations of noise are unspecified biological stress responses in terms of activation of the autonomic system and neuroendocrine pathways [17, 18]. Noise, as a psychosocial stressor, could lead to dyslipidemia, hypertension, and increased blood glucose, the pathological basis of CVDs [19]. Noise-induced dysregulation may promote the atherosclerotic process, which causes hypertension and cardiovascular events [11].

Although several investigations have shown the association between noise and CVDs, several studies have shown contradictory results [11]. For example, Data from the Copenhagen male study could not determine an association between noise and death from IHD [20], although this paper was excluded because it did not classified different cardiovascular diseases especially myocardial infarction and death and noise levels. In addition, a Finnish enumeration study reported a weak associations [21].In contrast, within the Helsinki heart study, the noise was associated with a moderate but statistically considerable increment in Coronary Heart Disease (CHD) risk that remained even after the workers had passed the age of retirement [22] but this study did not consider lower noise effects so it was excluded. In a Canadian follow-up study investigating lumber mill workers, a Relative Risk (RR) of 1.5 in the highest exposure category was found [23]. Furthermore, although there are some review and meta-analysis articles about the association between noise and CVDs, there is no review and meta-analysis article about the association between occupational noise and CVDs. In the present study, we considered all accessible studies to clarify the association of occupational noise exposure on risk of developing CVDs.

2. Materials and Methods

This study reviewed the published articles investigating the association between occupational noise and MI. Relevant articles were searched from three scientific databases: Scopus, Pubmed, and Web of science. Different combinations of strings using the logical operators' AND' and 'OR' were used: ("occupational noise" OR "industrial noise" OR "noise exposure") AND ("MI" OR "coronary heart disease" OR "heart attack" OR "ischemic heart disease" OR "cardiovascular disease" OR "cardiovascular mortality") without time limitation. Review articles, non-English, non-human studies, conference papers and those articles which did not regard "noise level" or " noise duration exposure" were excluded.

The remaining articles were screened by two independent researchers based on the following criteria: (1) Relevance of title to the topic of interest (occupational noise and myocardial infraction); (2) Full-text articles published in peer-reviewed journals; and (3) Written in English. A total of eight articles were included in the final review. PRISMA checklist was used for quality assessment.

3. Results

The initial search identified 517 articles. Titles and abstracts of these articles were screened, and 196 duplicated articles and 207 unrelated articles were excluded. Selected articles were published in English journals. Figure 1 shows the selection process of articles. Eight experimental studies encompassed various aspects of industrial noise. Included articles were categorized into two main groups; 1) articles that solely assessed the association of occupational noise (level and exposure duration) with CVDs adjusted for a variety (Table 1) , and 2) articles that considered both occupational and environmental or social factors with CVDs (Table 2).

In Girard et al. study [24], 8910 retired male workers from the Quebec National Institute of Public Health with at least one audiology test in mobile laboratories with no variable adjustment were assessed using a nested case-control study. They defined exposure in term of years of exposure to occupational noise and noise-induced hearing loss. The investigators concluded that Prolonged duration of noise exposure (≥36.5 years) was associated with an increased risk of MI as compared with a shorter period of noise exposure (<27 years). In Eriksson et al. study [25] follow-up data from 7494 people were collected from men living in Gutenburg. Baseline occupation's data from 1974 to 1977 were considered for classification of occupational noise (Low noise <75 dB(A), Medium noise, 75–85 dB(A), High noise, >85 dB(A) and incidence of CHD and stroke were measured through available data. They revealed that increment in occupational noise increased the risk of CHD, but it does not affect stroke. In Wen Qi et al. study [26], 6307 participants reported exposure to occupational noise and answered health-related questionnaires in a cross-sectional study. In their study, MI prevalence was remarkably higher in people exposed to occupational noise. In Davies et al. [23] cohort study 27,464 workers exposed to occupational noise for more than one year were followed up, and vital status was extracted from the data

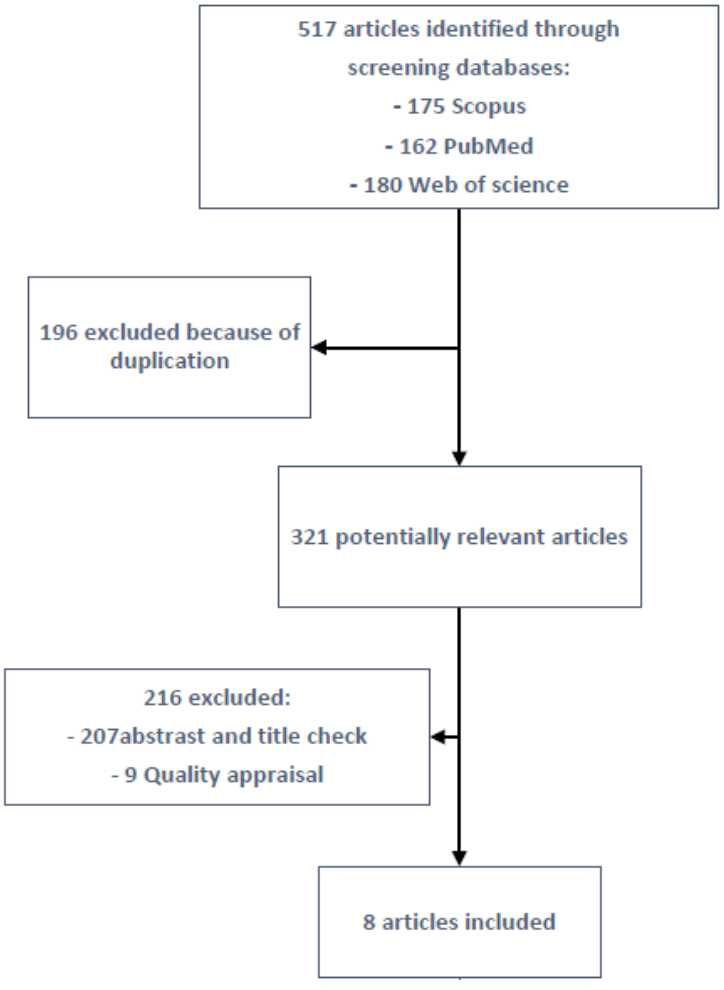

Figure 1. The flowchart of the articles' selection process

Table 1. Articles related to the association between occupational noise (level and exposure duration) and CVDs

Line	Authors/Year	Study Type	Sample Discription	Exposure Definition	Significant Finding	Adjusted Variables
1	Girard et al., 2015 [24]	Nested Case-control	Data was gathered from the Quebec National Institute of Public Health on 8910 retired male workers with audiology tets.	1. Years of exposure to occupational noise: Prolonrged duration= (≥36.5 years) shorter duration= (<27 years) 2. Noise-Induced Hearing Loss (NIHL); Mild <33 dB HL (ref), Moderate 33–51.4 dB HL, Severe ≥51.5 dB HL	OR for CVD in Prolonged compared to shorter noise duration (1.70; 95% CI: 1.10–2.62) OR for CVD in moderate NIHL (1.64; 95% CI: 1.04–2.6) And severe NIHL (1.66; 95% CI: 1.06–2.60) compared to mild NIHL	-
2	Eriksson et al., 2018 [25]	Prospective cohort	Follow-up data from 7494 people were collected from men living in Gutenburg. levels of occupational noise was collected from baseline data on occupation from 1974 to 1977.	Noise level: ≤75 dB(A) (ref), 76-85 dB (moderate noise), >85 (high noise)	Risk for CHD: Moderate noise (HR= 1.15, 95% CI: 1.01-1.31) High noise (HR= 1.27, 95% CI: 0.99 to 1.63)	Age in years, body mass index, diabetes, smoking, and cholesterol
3	Gan, 2011 [26]	Cross-sectional	6307 participants reported exposure to occupational noise and answered health-related questionnaires.	Self-reported exposure to loud noise in the workplace.	OR (95% CIs) for angina pectoris, CHD and isolated diastolic hypertension were 2.91 (1.35 to 6.26), 2.04 (1.16 to 3.58) and 2.23 (1.21 to 4.12)	Age, sex, race/ethnicity, BMI, educational level, physical activity, diabetes, annual family income, pack-years of cigarette smoking, passive smoking in the workplace or at home, alcohol drinking, waist circumference and total cholesterol.
4	W. Davies, 2005 [23]	Cohort	27,464 workers exposed to occupational noise for more than one year were followed up.	In the highest exposed group of 1.5 (95% confidence interval = 1.1-2.2)	A Strong association between MI and subgroup without hearing protection was observed. In addition, duration of exposure and noise level have significant associations on increasing MI.	Age and calendar year
5	Gopinath, 2011 [27]	Cross-sectional	2942 participants reported noise exposure in their workplace.	Exposure to severe workplace noise for less than 1 to 5 years versus no exposure	OR for incident stroke 3.44 (95% CI: 1.11–10.63)	Age, sex, occupational prestige, BMI, mean arterial blood pressure, physical activity, total dietary fat consumption and self-reported poor health.

OR: Odds Ratio; HR: Hazard Ratio; CI: Confidence Interval; BMI: Body Mass Index; MI: Myocardial Infarction; CHD: Coronary Heart Disease; CVDs: Cardiovascular Diseases; ISCO: International Standard Classification of Occupation.

Table 2. Articles related to the association of environmental, social and occupational factors and CVDs

Line	Authors/Year	Study Type	Sample Description	Exposure Definition	Environmental or Social Factor	Significant Finding	Adjusted Variables
1	Pettersson et al., 2020 [28]	Cohort	Data was collected from 194,501 workers in the Swedish construction industry between 1971 and 1993	Noise level: ≤75 dB(A) (ref), 76-85 dB (moderate noise), >85 (high noise)	Temperature of living region into: cold (reference), colder and coldest	MI mortality: Moderate (RR=1.01, 95%CI:1.01-1.19) and high noise exposure (RR=1.13, 95%CI:1.03-1.23) Stroke: Moderate (RR=1.15, 95%CI:1.01-1.32) and high noise exposure(RR=1.19, 95%CI:1.03-1.38). Interaction was found between different region and noise exposure on MI but not o stroke	Age, BMI, smoking habits, and region
2	Selander, 2013 [29]	Case-control	3050 Participants answered a self-report questionnaire and implemented a physical test.	Noise exposed if subjects had an occupational noise exposure >75 dBA for >1 year.	Road traffic noise and job strain	Increased odds of MI among exposed subjects (OR 1.17, 95% CI: 0.98–1.41). Odds ratio of MI among Participants exposed to occupational and traffic noise and job strain: 2.27, 95% CI: 1.41–3.64,	Age, sex, hospital catchment area, physical inactivity, smoking, air pollution and socioeconomic position
3	Kersten, 2015 [11]	Case-control	4113 people exposed to noise for more than 10 years participated in this study, and their jobs were categorized according to ISCO-88.	Sound values were categorized into four categories [46-61 dB, 62-84 dB, 85-94 dB, and 95-124 dB]	Job complexity	OR for myocardial infarction in the highest noise range (95-124 dB(A): 2.18, 95%CI: 1.17-4.05. Different association of occupational noise with CVDs according to job demand	Current employment status, shift work, work >40 h per week

OR: Odds Ratio; CI: Confidence Interval; MI: Myocardial Infarction; CVDs: Cardiovascular Diseases.

bank. They reported a strong association between MI and subgroup without hearing protection. In addition, duration of exposure and noise level had significant effects on increasing MI. In Gopinath et al. [27] cross-sectional study 2942 participants reported noise exposure in their workplace by answering a questionnaire about the duration of exposure and mortality from cardiovascular mortality was confirmed by Australian National Death Index.

In Pettersson et al. study [28] Noise exposure was characterized on a job-exposure matrix based on a survey of the working conditions carried out during the mid-1970s. They also assessed the temperature of living region of the participants into reference (Götaland), colder (Svealand), and coldest (Norrland) region and found an interaction between different region and noise exposure on MI but not o stroke. In Selander, 2013 study [29] a population based case-control study conducted on 3050 Participants in Stockholm County and a mixture of residential road traffic noise, job strain and occupational noise were assessed as exposure. MI events was defined as outcome and were checked from hospital discharge registry from 1975 onwards. They found that exposure to traffic and occupational noise simultaneously increased the risk of MI. They reported that mortality from MI and stroke was higher in those with more prolonged exposure to occupational noise. In Kersten et al. [11] study, 4113 people exposed to noise for more than 10 years participated and their jobs were categorized according to ISCO-88. The daily sound exposure was assessed with the help of the machine's catalogs, and sound levels were into four categories stratified by job complexity according to International Standard Classification of Occupation. They found different association of occupational noise with CVDs in various job demand.

4. Discussion

Studies showed that occupational noise has some considerable association on increasing risk of CVDs. Other factors, such as environmental conditions and personal and social characteristics, affected risk of CVDs when combined with occupational noise.

Epidemiological studies have shown contradictory associations between noise and CVDs. Copenhagen's male study data could not detect an association between noise and mortality from IHD [20]. On the other hand, a Canadian follow-up study investigating lumber mill workers found a Relative Risk (RR) of 1.5 within the highest exposure category [23]. The discrepancies derive from differences in noise level, duration of exposure, exposure assessment method, study design, and sample size in various studies [26].

Some studies showed that the duration of exposure to industrial noise has a strong association with MI. In a study from Gopinath, mortality from MI increases when workers are exposed to prolonged periods of occupational noise [27]. in a case-control study conducted by Girard et al., the investigation demonstrated a strong relationship between the duration of exposure to occupational noise and the risk of MI [24]. This result was supported by another study by Qi Gan, where the results show that more prolonged exposure to occupational noise increases the risk of MI [26]. In Davis's study, results showed an incremented risk of MI in workers without hearing protection, especially in those workers who had longer exposures [23].

Six studies have investigated noise level association with and the risk of MI. In a study of acute MI survivors in Berlin [31], subjects self-reported questionnaire shows that high noise levels have a high relative risk with acute MI (3.8), with a positive exposure-response relation. The same conclusion was supported in another study where an association between Moderate and high noise with increasing the risk of MI was observed [32]. Erikson et al. [33] and Virkkunen et al.'s [31] studies were significantly homogenous to be combined in a quantitative meta-analysis. Based on these workers exposed to ≥85 dBA were found to have a 29% higher risk of acquiring IHD, when compared with workers exposed to <85 dBA (RR=1.29, 95% CI 1.15–1.43, 2 studies, about 11,758 participants, I2=0%) [34]. Nonetheless, occupational noise increases the risk of coronary heart diseases; it has been observed that it does not affect heart stroke and MI [25]. A chronic character's stress-related physiological transformations probably lead to atherosclerosis, hypertension, and ischemic heart disorder [33]. High-level occupational noise may be regarded as a potent external stressor, similar to sudden emotional stress [34] and physical exertion [35], to activate the sympathetic nervous system and endocrine system [36], leading to spasm and subsequent partial or complete coronary occlusion [37-39].

Some factors affect MI combined with noise, such as environmental factors and temperature-related factors. Peterson studied Swedish workers in three main regions exposed to noise and coldness simultaneously; his study showed that the prevalence of MI is higher in colder areas. In a study done by Liu [40], results showed that chronic exposure to environmental risk factors, through sustained changes in the environment, may also predispose to the genesis of CVDs, particularly via potentiation of risk factors such as diabetes and hypertension. In contrast, in the Marchant study, the environmental temperature affects the MI irrespective of the time of the year [41]. In colder situations, Heart rate and blood pressure tend to rise, increasing the myocardial oxygen demand. Hematological variables are also affected, as reflected by increases in thromboglobulin and platelet factor 4, enhancing platelet aggregation [42].

Personal and social factors, such as psychological, physiological, and job characteristics, can affect MI's prevalence when combined with noise. Selander's study showed that MI increases in workers exposed to noise and job strain compared to those exposed to noise only [29]. Following this, in a study conducted by Knutsson, MI risk was associated with shiftwork in both genders, men (Odds Ratio [OR]: 1.3, 95% Confidence Interval [95% CI]: 1.1 to 1.6) and women (OR: 1.3, 95% CI: 0.9 to 1.8) [34]. In contrast, in the Marchant study, the environmental temperature affects the MI irrespective of the time of the year [43]. In a study done in Brazil, smoking cigarette per day (OR: 4.90, P<0.00001); glucose waist/hip ratio ≥0.94 (OR: 2.45, P<0.00001); family history of CAD, hypertension (OR: 2.09, P<0.00001) were observed in MI patients [44]. Nine risk factors were associated with MI in women and men. A study performed by Marchant [41] showed that Hypertension, diabetes, moderate alcohol consumption, and physical activity were more extremely associated with MI among women than men. Risk factors associations were normally stronger among the youngsters than older women and men. Many adverse emotions, such as anger, tension, and sadness, resulted in myocardial ischemia [45].

Noise exposure is one of the major risk factors in industries, and in addition, MI is one of the most dominant CVDs among workers. Recognizing risk factors would be essential to reduce the risk of MI. Occupational noise

may have some detrimental associations on the heart; therefore, understanding its impact and limitation can be beneficial to deal with this problem. It is suggested that further research would be implemented to assess other environmental factors' associations on MI.

5. Conclusion

Occupational noise exposure may increase the risk of CVDs, particularly in higher levels and higher duration of exposures. There are also other environmental factors sush as cold temperature, personal and social factors such as job strain, diabetes, smoking, etc., that could interacted with the effect of occupational noise on risk of CVDs. To decrease the risk of CVDs, the noise level and duration of exposure should be minimized to an acceptable level. Furthermore, workers' health and habits and job demands should be considered by health organization authorities. Although we considered all possible criteria, some deficiencies may be related to the limited number of English studies about the associations of industrial noise on MI and different data collecting methods in the included studies. Additionally, many intervening factors were not considered in many articles.

Ethical Considerations

Compliance with ethical guidelines

This article is a review article with no human or animal sample.

Funding

This research did not receive any grant from funding agencies in the public, commercial, or non-profit sectors.

Authors' contributions

Conceptualization and Supervision: Elahe Oveisi; Methodology: Elahe Oveisi; Investigation, Writing – original draft, and Writing – review & editing: All authors; Data collection: Elahe Oveisi; Data analysis: Elahe Oveisi and Duha Ali. All authors equally contributed to preparing this article.

Conflict of interest

The authors declared no conflict of interest.

Acknowledgments

Thanks to Hamadan University of Medical sciences and Auburn University.

References

[1] Mendis S, Puska P, Norrving B, World Health Organization, World Heart Federation. Global atlas on cardiovascular disease prevention and control. Geneva: World Health Organization; 2011. https://apps.who.int/iris/handle/10665/44701

[2] Smith SC Jr, Collins A, Ferrari R, Holmes DR Jr, Logstrup S, McGhie DV, et al. Our time: A call to save preventable death from cardiovascular disease (heart disease and stroke). Eur Heart J. 2012; 33(23):2910-6. [DOI:10.1093/eurheartj/ehs313] [PMID]

[3] Chepkirui C, Ochieng PJ, Sarkar B, Hussain A, Pal C, Yang LJ, et al. Antiplasmodial and antileishmanial flavonoids from Mundulea sericea. Fitoterapia. 2021; 149:104796. [DOI:10.1016/j.fitote.2020.104796] [PMID]

[4] Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, et al. Executive summary: Heart disease and stroke statistics--2012 update: A report from the American Heart Association. Circulation. 2012; 125(1):188-97. [PMID]

[5] Nelson S, Whitsel L, Khavjou O, Phelps D, Leib A. Projections of cardiovascular disease prevalence and costs: 2015–2035. North Carolina: Research Triangle; 2016. https://www.heart.org/-/media/Files/Get-Involved/Advocacy/CVD-Predictions-Through-2035.pdf

[6] Dzau VJ, Antman EM, Black HR, Hayes DL, Manson JE, Plutzky J, et al. The cardiovascular disease continuum validated: Clinical evidence of improved patient outcomes: Part I: Pathophysiology and clinical trial evidence (risk factors through stable coronary artery disease). Circulation. 2006; 114(25):2850-70. [PMID]

[7] Yusuf S, Hawken S, Ôunpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): Case-control study. Lancet. 2004; 364(9438):937-52. [DOI:10.1016/S0140-6736(04)17018-9]

[8] Münzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. Eur Heart J. 2014; 35(13):829-36. [DOI:10.1093/eurheartj/ehu030] [PMID] [PMCID]

[9] Damen JA, Hooft L, Schuit E, Debray TP, Collins GS, Tzoulaki I, et al. Prediction models for cardiovascular disease risk in the general population: Systematic review. BMJ. 2016; 353:i2416. [DOI:10.1136/bmj.i2416] [PMID] [PMCID]

[10] Wilkins E, Wilson L, Wickramasinghe K, Bhatnagar P, Leal J, Luengo-Fernandez R, et al. European cardiovascular disease statistics. Brussels: European Heart Network; 2017. https://www.ehnheart.org/images/CVD-statistics-report-August-2017.pdf

[11] 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. [PMID] [PMCID]

[12] Rohrbach-Schmidt D. The BIBB/IAB-and BIBB/BAuA-surveys of the working population on qualification and working conditions in Germany. Bonn: The Federal Institute for Vocational Education and Training; 2009. https://www.bibb.de/dienst/veroeffentlichungen/de/publication/show/6526

[13] Tak S, Davis RR, Calvert GM. Exposure to hazardous workplace noise and use of hearing protection devices among US workers-NHANES, 1999-2004. Am J Ind Med. 2009; 52(5):358-71. [PMID]

[14] World Health Organization (WHO). Occupational noise: Assessing the burden of disease from work-related hearing impairment at national and local levels. Geneva: World Health Organization; 2004. https://www.who.int/publications/i/item/occupational-noise-assessing-the-burden-of-disease-

[15] Picard M, Girard SA, Simard M, Larocque R, Leroux T, Turcotte F. Association of work-related accidents with noise exposure in the workplace and noise-induced hearing loss based on the experience of some 240,000 person-years of observation. Accid Anal Prev. 2008; 40(5):1644-52. [DOI:10.1016/j.aap.2008.05.013] [PMID]

[16] Rabinowitz PM. The public health significance of noise-induced hearing loss. In: Le Prell CG, Henderson D, Fay RR, Popper AN, editors. Noise-induced hearing loss. New York: Springer; 2012. [DOI:10.1007/978-1-4419-9523-0_2]

[17] Babisch W. Stress hormones in the research on cardiovascular effects of noise. Noise Health. 2003; 5(18):1-11. [PMID]

[18] McEwen BS, Stellar E. Stress and the individual: Mechanisms leading to disease. Arch Intern Med. 1993; 153(18):2093-101. [DOI:10.1001/archinte.1993.00410180039004]

[19] Zhang K, Jiang F, Luo H, Liu F. Occupational noise exposure and the prevalence of dyslipidemia in a cross-sectional study. BMC Public Health. 2021; 21(1):1258. [DOI:10.1186/s12889-021-11274-x] [PMID] [PMCID]

[20] Suadicani P, Hein HO, Gyntelberg F. Occupational noise exposure, social class, and risk of ischemic heart disease and all-cause mortality-a 16-year follow-up in the Copenhagen Male Study. Scand J Work Environ Health. 2012; 38(1):19-26. [DOI:10.5271/sjweh.3200] [PMID]

[21] Virtanen SV, Notkola V. Socioeconomic inequalities in cardiovascular mortality and the role of work: A register study of Finnish men. Int J Epidemiol. 2002; 31(3):614-21. [PMID]

[22] 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; 63(6):378-86. [PMID] [PMCID]

[23] Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology. 2005; 16(1):25-32. [PMID]

[24] Girard SA, Leroux T, Verreault R, Courteau M, Picard M, Turcotte F, et al. Cardiovascular disease mortality among retired workers chronically exposed to intense occupational noise. Int Arch Occup Environ Health. 2015; 88(1):123-30. [DOI:10.1007/s00420-014-0943-8] [PMID]

[25] 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; 8(4):e019160. [DOI:10.1136/bmjopen-2017-019160] [PMID] [PMCID]

[26] 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; 68(3):183-90. [DOI:10.1136/oem.2010.055269] [PMID]

[27] 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; 53(6):390-4. [DOI:10.1016/j.ypmed.2011.10.001] [PMID]

[28] Pettersson H, Olsson D, Järvholm B. Occupational exposure to noise and cold environment and the risk of death due to myocardial infarction and stroke. Int Arch Occup Environ Health. 2020; 93(5):571-5. [DOI:10.1007/s00420-019-01513-5] [PMID]

[29] 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; 39(2):195-203. [DOI:10.5271/sjweh.3324] [PMID]

[30] Ising H, Babisch W, Kruppa B, Lindthammer A, Wiens D. Subjective work noise: A major risk factor in myocardial infarction. Soz Praventivmed. 1997; 42(4):216-22. [PMID]

[31] 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; 31(4):291-9. [DOI:10.5271/sjweh.885] [PMID]

[32] Teixeira LR, Pega F, Dzhambov AM, Bortkiewicz A, da Silva DTC, de Andrade CAF, 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; 154:106387. [PMID]

[33] Babisch W. Cardiovascular effects of noise. Noise Health. 2011; 13(52):201-4. [DOI:10.4103/1463-1741.80148] [PMID]

[34] Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005; 352(6):539-48. [PMID]

[35] Mittleman MA, Maclure M, Tofler GH, Sherwood JB, Goldberg RJ, Muller JE. Triggering of acute myocardial infarction by heavy physical exertion. Protection against triggering by regular exertion. Determinants of Myocardial Infarction Onset Study Investigators. N Engl J Med. 1993; 329(23):1677-83. [PMID]

[36] Remme WJ. The sympathetic nervous system and ischaemic heart disease. Eur Heart J. 1998; 19 (Suppl F):F62-71. [PMID]

[37] Pupita G, Maseri A, Kaski JC, Galassi AR, Gavrielides S, Davies G, et al. Myocardial ischemia caused by distal coronary-artery constriction in stable angina pectoris. N Engl J Med. 1990; 323(8):514-20. [DOI:10.1056/NEJM199008233230804] [PMID]

[38] Maseri A, Davies G, Hackett D, Kaski JC. Coronary artery spasm and vasoconstriction. The case for a distinction. Circulation. 1990; 81(6):1983-91. [PMID]

[39] Mohri M, Koyanagi M, Egashira K, Tagawa H, Ichiki T, Shimokawa H, et al. Angina pectoris caused by coronary microvascular spasm. Lancet. 1998; 351(9110):1165-9. [DOI:10.1016/S0140-6736(97)07329-7]

[40] Liu C, Ying Z, Harkema J, Sun Q, Rajagopalan S. Epidemiological and experimental links between air pollution and type 2 diabetes. Toxicol Pathol. 2013; 41(2):361-73. [PMID]

[41] Marchant B, Ranjadayalan K, Stevenson R, Wilkinson P, Timmis AD. Circadian and seasonal factors in the pathogenesis of acute myocardial infarction: The influence of environmental temperature. Br Heart J. 1993; 69(5):385-7. [DOI:10.1136/hrt.69.5.385] [PMID]

[42] Kawahara J, Sano H, Fukuzaki H, Saito K, Hiroucht H. Acute effects of exposure to cold on blood pressure, platelet function and sympathetic nervous activity in humans. Am J Hypertens. 1989; 2(9):724-6. [DOI:10.1093/ajh/2.9.724] [PMID]

[43] Knutsson A, Hallquist J, Reuterwall C, Theorell T, Akerstedt T. Shiftwork and myocardial infarction: A case-control study. Occup Environ Med. 1999; 56(1):46-50. [PMID]

[44] Piegas LS, Avezum Á, Pereira JCR, Neto JMR, Hoepfner C, Farran JA, et al. Risk factors for myocardial infarction in Brazil. Am Heart J. 2003; 146(2):331-8. [DOI:10.1016/S0002-8703(03)00181-9]

[45] Chi JS, Kloner RA. Stress and myocardial infarction. Heart. 2003; 89(5):475-6. [PMID]

This Page Intentionally Left Blank