

Noise Effect on Blood Pressure and Heart Rate - Regression Analysis in Service of Prediction

Fikret Veljovic¹, Senad Burak¹,
Edin Begic², Izet Masic³

¹Faculty of Mechanical Engineering,
University of Sarajevo, Sarajevo, Bosnia
and Herzegovina

²Department of Cardiology, General
Hospital «Prim.dr. Abdulah Nakas»,
Sarajevo, Bosnia and Herzegovina

³Academy of Medical Sciences in Bosnia
and Herzegovina, Sarajevo, Bosnia and
Herzegovina

Corresponding author: Professor Fikret Veljovic,
PhD. Faculty of Mechanical Engineering,
University of Sarajevo, Sarajevo, Bosnia and
Herzegovina. E-mail: veljovic@mef.unsa.ba.
ORCID ID: <https://orcid.org/0000-0002-3722-2542>

doi: 10.5455/aim.2019.27.162-166

ACTA INFORM MED. 2019 SEP 27(3): 162-166

Received: Jul 05, 2019 • Accepted: Aug 08, 2019

© 2019 Fikret Veljovic, Senad Burak, Edin Begic,
Izet Masic

This is an Open Access article distributed under the
terms of the Creative Commons Attribution Non-
Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Introduction: Investigations have shown that noise is one of the etiologic factors that leads to a risk of cardiovascular incidence. **Aim:** To present effect of noise on arterial tension and heart rate of workers who work on machine press during period of three-years. **Methods:** The study had a prospective character and included 30 subjects (n = 30) who were monitored over a three-year period (36 months). The respondents worked at the factory "Cimos" on machine press (Zenica, Bosnia and Herzegovina). Ten machine presses were monitored, and three workers worked on each press. Approximately every worker was affected by a wide range of noise between 65 and 110 dB in the workplace (via isohypse). MATLAB (version 9.4, MathWorks, Natick, Massachusetts, United States of America (USA)) software was used to estimate the possible damage caused by noise in factories that produce noise in their work. **Results:** During the three-year period, arterial tension in the subjects increased, and at the end of the observed period, they were considered as patients with a diagnosis of arterial hypertension grade I. The tension depends on the strength of the produced noise, and the values also depend on position of the respondent on the machine press. **Conclusion:** Noise prevention has become a problem of modern medicine. The result of our work allows estimation of arterial hypertension in specified time in case of exposure to a certain strength of noise. Prevention of noise, daytime noise prevention as well as better equipment for work and preventive equipment are imposed as imperative in such or similar conditions, with the need of development of national strategies for this issue in countries where they are not present.

Keywords. noise, cardiovascular risk, arterial hypertension, heart rate.

1. INTRODUCTION

Noise (unwanted sound) is divided into industrial and ambient noise and can be continuous, variable, intermittent or impulsive (1). It represents the etiologic factor of many pathologies, and has implications on many organic systems, inducing hearing impairment, hypertension and ischemic heart disease, annoyance, sleep disturbances, and reduced school performance (2). A person can hear sounds in a frequency range of 20 hertz (Hz) to 20 kHz. The sound of 0 decibel (dB) is at the threshold of the sound. Long-term, high-intensity noise exposure has a negative impact on hearing, and can cause hearing loss. Noise above 90 dB can lead to chronic hearing damage if people are exposed to them every day or all the time. Hearing becomes uncomfortable if the sound pressure level

is above 110 dB (threshold of discomfort), and it becomes painful above 130 dB (threshold of pain) (3).

2. AIM

The aim of this study was to present effect of noise on arterial tension and heart rate of workers who work on machine press during period of three-years assessed by use of MATLAB Software package.

3. METHODS

The study had a prospective character and included 30 subjects (n = 30) who were monitored over a three-year period (36 months). The respondents worked on machine press at the factory "Cimos" (Zenica, Bosnia and Herzegovina). Ten machine presses were monitored, and three workers worked on each press. Approximately every worker was af-

Place of worker	Parameter	Press 1	Press 2	Press 3	Press 4	Press 5	Press 6	Press 7	Press 8	Press 9	Press 10
1	Height	178	176	178	180	184	179	181	183	182	181
2	Weight	76	80	79	82	84	81	83	80	70	81
3	Age	24	26	25	24	26	27	25	28	26	27

Table 1. Anthropological parameters of respondents (average values for 10 workers)

Place of worker	at the beginning (mmHg)	after 6 months (mmHg)	after 12 months (mmHg)	after 18 months (mmHg)	after 24 months (mmHg)	after 30 months (mmHg)	after 36 months (mmHg)
1	120/81	125/85	130/88	136/90	145/91	151/92	155/95
2	125/80	126/82	130/85	135/87	147/91	147/91	150/95
3	122/82	124/84	131/87	134/77	141/91	148/93	158/96

Table 2. Average measurement of arterial pressure in respondents compared to place of work (average values for 10 workers)

Place of worker	at the beginning (beats per minute)	after 6 months (beats per minute)	after 12 months (beats per minute)	after 18 months (beats per minute)	after 24 months (beats per minute)	after 30 months (beats per minute)	after 36 months (beats per minute)
1	78	83	85	89	91	93	96
2	80	82	87	89	91	93	95
3	75	79	82	84	87	89	91

Table 3. Average heart rate measurements for respondents in relation to the workplace (average values for 10 workers)

ected by a wide range of noise between 65 and 110 dB in the workplace (via isohypse). Physical examinations were performed every six months. Criteria for inclusion in the study: voluntary consent for participation, absence of elevated blood pressure, heart rate at optimal physiological limits (60-80 beats per minute), normal echocardiographic finding. Study exclusion criteria were: declined participation, diagnosis of arterial hypertension or information about tachycardia in anamnestic data, verified systolic or diastolic dysfunction by echocardiography, or valvular defect in patient medical records. During the three years patients did not take any antihypertensive therapy. During the realization of this study the identity and all personal data of the patients were permanently protected in accordance with the protection regulations of the identification data. For each person, an identification number was used for the purpose of protecting the personal data used in the statistical processing of the data. Regression analysis was used for statistical analysis and MATLAB (version 9.4, MathWorks, Natick, Massachusetts, United States of America (USA)) software was used (hypertension values were entered, based on which regression analysis was performed that could be very reliable in determining the value of tension at any time interval, and ultimately, it can be used to estimate the possible damage caused by noise in factories that produce noise in their work) and Microsoft Excell (version 11, Microsoft Corporation, Redmond, Washington, USA).

4. RESULTS

Table 1 shows the anthropological parameters of the respondents in relation to the press on which they work.

The average height of all workers was 180 cm, weight was 79.6 kg, and age was 25.8 years. In order to estimate and predict the shape of response values (dependent variable) over a range of input parameter values (independent variable) we use a polynomial regression analysis (curve fitting). In general, if we have *n* pairs of values, like we have in above tables, then they can be modeled with a polynomial of order *k* by the following approximation:

$$y = a_0 + a_1x + a_2x^2 + \dots + a_kx^k \text{ (Eq. 1)}$$

where *x* is independent variable (in our case it is time interval in months) and *y_i* is predicted outcome value for the polynomial model (in our case it is a blood pressure or heart rate measurements). Regression analysis aims to determine predictions for dependent variable *y* for any possible value of independent variable *x*. Parameters *a₀*, *a₁*, *a₂*, ...*a_k* are called regression coefficients and they need to be determined mathematically in such a way that they minimize the sum of squares or residual

$$R^2 = \sum_{i=1}^n [y - (a_0 + a_1x + a_2x^2 + \dots + a_kx^k)]^2 \text{ (Eq. 2)}$$

Equation (2) can be written in the matrix form

$$\mathbf{X} \mathbf{a} = \mathbf{Y} \text{ (Eq. 3)}$$

where

$$\mathbf{X} = \begin{bmatrix} n & \sum_{i=1}^n x_i & \dots & \sum_{i=1}^n x_i^k \\ \sum_{i=1}^n x_i & \sum_{i=1}^n x_i^2 & \dots & \sum_{i=1}^n x_i^{k+1} \\ \dots & \dots & \dots & \dots \\ \sum_{i=1}^n x_i^k & \sum_{i=1}^n x_i^{k+1} & \dots & \sum_{i=1}^n x_i^{2k} \end{bmatrix} \text{ (Eq. 4)}$$

a is a vector of regression coefficients and vector of dependent variables *Y* is given by

$$\mathbf{Y} = \begin{bmatrix} \sum_{i=1}^n y_i \\ \sum_{i=1}^n x_i y_i \\ \dots \\ \sum_{i=1}^n x_i^k y_i \end{bmatrix} \text{ (Eq. 5)}$$

For *k* <= 2, the above system can be solved analytically by manual substitution or determinant method using Cramer's rule. In case *k* > 2 we rather write a computer program or use predefined functions from systems like Microsoft Excel. To do this, we can use an inversion method

which allows us to efficiently determine regression coefficients all at once.

Multiplying equation (3) by X^{-1} from the left hand side $X^{-1}Xa = X^{-1}Y$ (Eq. 6) and taking into account that $X^{-1}X = I$, we get

$$a = X^{-1}Y = \begin{bmatrix} a_0 \\ a_1 \\ \dots \\ a_k \end{bmatrix} \text{ (Eq. 7)}$$

Using Excel's built-in functions MINVERSE and MMULT the above method can be easily solved in an interactive manner. In this particular case we use Excel's capability to perform a multiple regression analysis using a third order polynomial (cubic curves) by adding a trendline and scatter plot graphical tool.

The results are shown in Figure 1, 2 and 3 with calculated coefficients of determination R^2 , which is a statistical measure of how close the data are fitted to the regression line. As it can be seen, those coefficients are in all case very close to 1, which proves that the models fits the measured data very well.

5. DISCUSSION

Acute noise exposure can cause increases in blood pressure, heart rate, and cardiac output, likely mediated by the release of stress hormones such as catecholamines (4, 5). Umemura et al. and Björ et al. showed that the type and presence of noise, respectively, could affect heart rate results. (6, 7). Additionally, Lee et al. reported that heart rate results were affected by the level of noise, as well as on the acceleration of inflammatory processes (inflammation is the connection between atherosclerosis and arterial thrombosis and plays an important role in progression, plaque vulnerability and thrombus formation) (8). The engineering control or personal control by wearing hearing protection device should be used to decrease noise exposure levels lower than 85 dB for 8 hours. Moreover, if the exposure level reaches 85 dB for 8 hours, the employer needs to implement a hearing conservation program in the workplace (9, 10). Interventions in the work environment could be a preventable and controllable manner for reducing the incidence of cardiovascular disease (11). Occupational noise exposure was associated with higher levels of systolic blood pressure and diastolic blood pressure, and the risk of hypertension (11). Bluhm et al. found connection between residential road traffic noise and hypertension (12). Kalantari et al. found that noise can increase the heart rate of workers (13). Heart rate is an important factor that is widely used in determining the health of an individual, especially overall cardiovascular health. There is some evidence that demonstrate that elevated resting heart rate is associated with a greater risk of developing sustained hypertension and with increased cardiovascular morbidity and mortality (14, 15). Persistent tachycardia of any form can cause tachycardia-mediated cardiomyopathy (TMC), and can precipitate heart failure (16). Exposure to the noise may lead to the onset of hypertension and the consequences of hypertension itself. 90-95% of all patients with hypertension suffer from primary hypertension (essen-

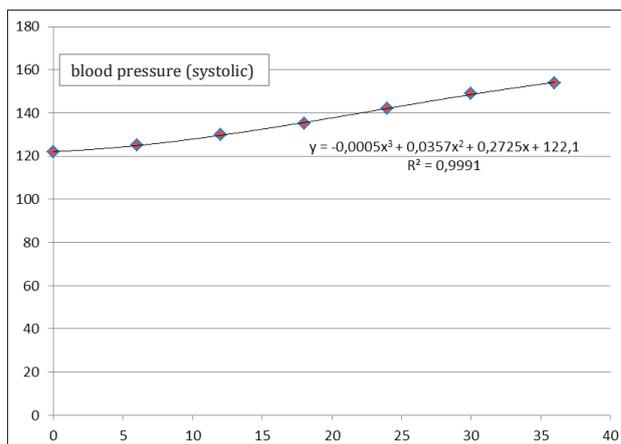


Figure 1. Systolic blood pressure – regression analysis estimation (cubic curves)

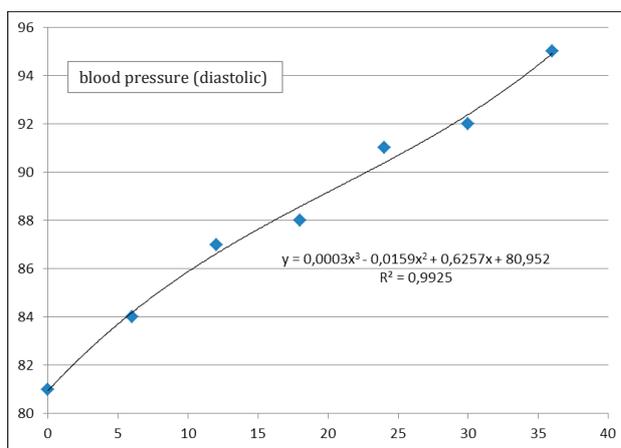


Figure 2. Diastolic blood pressure – regression analysis estimation (cubic curves)

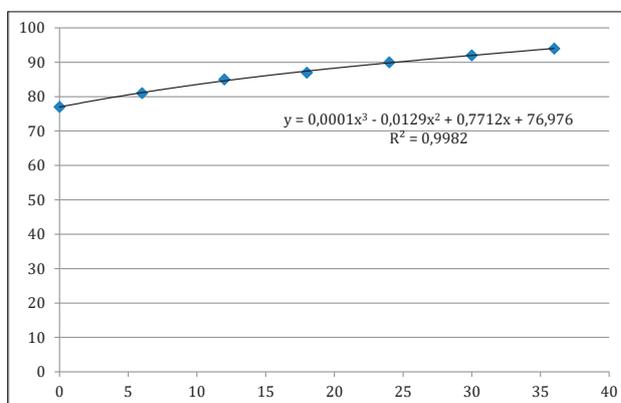


Figure 3. Heartbeat values – regression analysis estimation (cubic curves)

tial) and that represents hypertension of unknown origin (17). Hypertension is defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) values ≥ 90 mmHg (18). Some progress has been made in understanding the epidemiology, pathophysiology, and risk associated with hypertension, and there are a lot of evidences that demonstrate that lowering blood pressure can reduce premature morbidity and mortality. Elevated blood pressure was the leading global contributor to premature death in 2015, with almost 10 million deaths and over 200 million disability-adjusted life years. It is important to note that despite advances in diagnosis and treatment over the past 30 years, the disability-adjusted

life years attributable to hypertension have increased by 40% since 1990 (18). The heart muscle can be burdened in two ways, with pressure (systolic) or volume (diastolic load). Volume excretion causes a proportional increase in the volume of myofibrils and the volume of mitochondria (eccentric hypertrophy develops over time). Due to the pressure load, the volume of myofibrils is increased (aortic stenosis, isometric load, arterial hypertension, pulmonary hypertension) and is characterized by concentric hypertrophy. Concentric hypertrophy will increase myocardial stiffness. With this kind of hypertrophy, myocardial stiffness is increased. Increased workload leads to early development of heart failure and coronary heart disease (atherosclerosis) or myocardial infarction (19). High blood pressure damages also the cerebrovascular system and presents the etiologic factor of kidney damage (19, 20).

In long-lasting untreated arterial hypertension, the fibrosis and hyalinization of the blood vessels will make changes in the flow and flow control in the kidney. Gradually in the kidneys parenchyma cells will decay (19,20). Patients with mild or severe hypertension usually have no subjective problems (the only symptom that is described is morning's occipital headache) (20). Symptoms include dizziness, numbness in the mouth, insecure walking. Hypertension is the major risk factor for coronary heart disease, left ventricular hypertrophy and heart failure. Left ventricular hypertrophy in hypertension is an independent predictor of morbidity and mortality, and the condition is predisposed to the occurrence of cardiac insufficiency (20). In hypertrophy of the heart in hypertension, coronary flow is normal at rest, but in the effort coronary vasodilatory reserve will be insufficient (20).

Blood supply is not insufficient even in the absence of coronary artery disease, leading to sub-endocardial ischemia under the conditions of increased myocardial oxygen demand. Sub-endocardial ischemia and cardiovascular fibrosis exacerbate diastolic regression, leading to dyspnea and diastolic heart failure (20). Although significant success in treatment has been achieved, hypertension is the most common cause of diastolic heart failure and is a major factor for acute coronary syndrome and indirectly for systolic heart failure (20). Although we do not know the cause of hypertension, prevention is imperative. Most commonly, the lack of physical activity or obesity is the cause of hypertension, and physical activity is the first thing to prevent hypertension (21, 22). Many authors have shown that working conditions, as well as stress at work itself, lead to an increased cardiovascular risk (23-32). This research has proven that prevention must still be thought of in much broader sense. In our case, noise has increased heart rate and blood pressure over time and present etiological factor that should be prevented. The hypothesis that exposure to excessive noise could be associated to high blood pressure was described by Babisch (4), and later the thesis was confirmed also by other researchers (5-14). Liu et al. made connection between hypertension and hearing loss caused by noise in Chinese coal miners. The occupational noise was an independent risk factor for hypertension and could in-

crease the values of SBP and DBP (33). De Souza showed that exposure is independently associated to hypertension (34). Chang et al. showed that both systolic and diastolic blood pressure were significantly higher among workers exposed to noise levels ≥ 85 dB (35). Powazka et al. observed an average increase in the systolic blood pressure of 5 mmHg among workers at a steel factory exposed to 89 dB (36). Meta-analysis investigated the association between cardiovascular abnormalities and chronic occupational exposure to noise (37). Cayir et al. have revealed that occupational noise exposure is associated with higher risk of elevated blood pressure and hypertension in young adults, and demonstrated an interaction of noise with gender and BMI (38). Daniell et al. confirmed that most companies gave limited or no attention to noise controls and relied primarily on hearing protection to prevent hearing loss; yet 38% of employees did not use protectors routinely. Protector use was the highest when hearing loss prevention programs were completed, indicating that use of protection was attributable to incomplete or inadequate company efforts. (39, 40)

6. CONCLUSION

Noise prevention has become a problem of modern medicine. The result of our work allows estimation of arterial hypertension in specified time in case of exposure to a certain strength of noise. Prevention of noise, daytime noise prevention as well as better equipment for work and preventive equipment are imposed as imperative in such or similar conditions, with the need of development of national strategies for this issue in countries where they are not present.

- **Declaration of patient consent:** The authors certify that they have obtained all appropriate patient consent forms.
- **Author's contribution:** F.V., S.B., E.B. and I.M. gave substantial contribution to the conception or design of the work and in the acquisition, analysis and interpretation of data for the work. F.V, E.B. and I.M. had role in drafting the work and revising it critically for important intellectual content. Each author gave final approval of the version to be published and they agree to be 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.
- **Conflicts of interest:** There are no conflicts of interest.
- **Financial support and sponsorship:** Nil.

REFERENCES

1. Dornic S, Laaksonen T. Continuous noise, intermittent noise, and annoyance. *Percept Mot Skills*. 1989 Feb; 68(1): 11-18.
2. Passchier-Vermeer W, Passchier WF. Noise exposure and public health. *Environ Health Perspect*. 2000;108 (1): 123-131.
3. Fink DJ. What Is a Safe Noise Level for the Public?. *Am J Public Health*. 2017; 107(1): 44-45.
4. Babisch W. Cardiovascular effects of noise. *Noise Health*. 2011; 13: 201-204.
5. Münzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. *Eur Heart J*. 2014; 35(13): 829-836.
6. Björ B, Burström L, Karlsson M, Nilsson T, Näslund U, Wiklund U. Acute effects on heart rate variability when exposed to hand transmitted vibration and noise. *Int Arch Occup Environ Health*. 2007; 81:

- 193-199.
7. Umemura M, Honda K. Influence of music on heart rate variability and comfort - a consideration through comparison of music and noise. *J Hum Ergol (Tokyo)* 1998; 27: 30-38.
 8. Lee GS, Chen ML, Wang GY. Evoked response of heart rate variability using short-duration white noise. *Auton Neurosci*. 2010; 155: 94-97.
 9. Sriopas A, Chapman RS, Sutammasa S, Siriwong W. Occupational noise-induced hearing loss in auto part factory workers in welding units in Thailand. *J Occup Health*. 2017; 59(1): 55-62.
 10. Wu X, Yang D, Fan W, Fan C, Wu G. Cardiovascular risk factors in noise-exposed workers in china: Small area study. *Noise Health*. 2017; 19(91): 245-253.
 11. Chen S, Ni Y, Zhang L, et al. Noise exposure in occupational setting associated with elevated blood pressure in China. *BMC Public Health*. 2017; 17(1): 107.
 12. Leon Bluhm G, Berglund N, Nordling E, Rosenlund M. Road traffic noise and hypertension. *Occup Environ Med*. 2007; 64(2): 122-126.
 13. Kalantary S, Dehghani A, Yekaninejad MS, Omid L, Rahimzadeh M. The effects of occupational noise on blood pressure and heart rate of workers in an automotive parts industry. *ARYA Atheroscler*. 2015; 11(4): 215-219.
 14. Gillum RF, Makuc DM, Feldman JJ. Pulse rate, coronary heart disease, and death: the NHANES I epidemiologic follow-up study. *Am Heart J*. 1991; 121: 172-177.
 15. Kannel WB, Kannel C, Paffenbarger RS Jr, Cupples LA. Heart rate and cardiovascular mortality: the Framingham study. *Am Heart J*. 1987; 113: 1489-1494.
 16. Gopinathannair R, Olshansky B. Management of tachycardia. *F1000Prime Rep*. 2015; 7: 60.
 17. Bolívar JJ. Essential hypertension: an approach to its etiology and neurogenic pathophysiology. *Int J Hypertens*. 2013; 2013: 547809.
 18. Williams B, Mancia G, Spiering W, Agabiti Rosei E, Azizi M, Burnier M, et al; ESC Scientific Document Group. 2018 ESC/ESH Guidelines for the management of arterial hypertension. *Eur Heart J*. 2018 Sep 1; 39(33): 3021-3104.
 19. Smaljcelj A, Mahocak I. Poremecaji tlaka i protoka krvi. U: Gamulin S, Marusic M, Kovac Z i sur. *Patofiziologija, knjiga prva, sedmo izdanje*. Medicinska naklada, Zagreb, 2011. 931-935.
 20. Cvetkovic Matic D, Ivanaovic B, Simic VD. Primarna (esencijalna) arterijska hipertenzija. In: Ostojic M, Kanjuh V, Beleslin B. *Kardiologija*. Zavod za udzbenike, Beograd, 2011. 503-508.
 21. Diaz KM, Shimbo D. Physical activity and the prevention of hypertension. *Curr Hypertens Rep*. 2013; 15(6): 659-668.
 22. Stewart J, Manmathan G, Wilkinson P. Primary prevention of cardiovascular disease: A review of contemporary guidance and literature. *JRSM Cardiovasc Dis*. 2017; 6:2048004016687211.
 23. Sara JD, Prasad M, Eleid MF, Zhang M, Widmer RJ, Lerman A. Association Between Work-Related Stress and Coronary Heart Disease: A Review of Prospective Studies Through the Job Strain, Effort-Reward Balance, and Organizational Justice Models. *J Am Heart Assoc*. 2018; 7(9): e008073.
 24. Siegrist J, Peter R, Junge A, Cremer P, Seidel D. Low status control, high effort at work and ischemic heart disease: prospective evidence from blue-collar men. *Soc Sci Med*. 1990; 31: 1127-1134.
 25. Alterman T, Shekelle RB, Vernon SW, Burau KD. Decision latitude, psychologic demand, job strain, and coronary heart disease in the Western Electric Study. *Am J Epidemiol*. 1994; 139: 620-627.
 26. Szerencsi K, van Amelsvoort L, Prins M, Kant I. The prospective relationship between work stressors and cardiovascular disease, using a comprehensive work stressor measure for exposure assessment. *Int Arch Occup Environ Health*. 2014; 87: 155-164.
 27. Netterstrom B, Kristensen TS. Psychosocial factors at work and ischemic heart disease [in Danish]. *Ugeskr Laeger*. 2005; 167: 4348-4355.
 28. Kivimaki M, Virtanen M, Elovainio M, Kouvonen A, Vaananen A, Vahtera J. Work stress in the etiology of coronary heart disease: a meta-analysis. *Scand J Work Environ Health*. 2006; 32: 431-442.
 29. Backe EM, Seidler A, Latza U, Rosnagel K, Schumann B. The role of psychosocial stress at work for the development of cardiovascular diseases: a systematic review. *Int Arch Occup Environ Health*. 2012; 85: 67-79.
 30. Szerencsi K, van Amelsvoort LG, Viechtbauer W, Mohren DC, Prins MH, Kant I. The association between study characteristics and outcome in the relation between job stress and cardiovascular disease: a multilevel meta-regression analysis. *Scand J Work Environ Health*. 2012; 38: 489-502.
 31. Eaker ED, Sullivan LM, Kelly-Hayes M, D'Agostino RB Sr, Benjamin EJ. Does job strain increase the risk for coronary heart disease or death in men and women? The Framingham Offspring Study. *Am J Epidemiol*. 2004; 159: 950-958.
 32. Laszlo KD, Ahnve S, Hallqvist J, Ahlbom A, Janszky I. Job strain predicts recurrent events after a first acute myocardial infarction: The Stockholm Heart Epidemiology Program. *J Intern Med*. 2010; 267: 599-611.
 33. Liu J, Xu M, Ding L, et al. Prevalence of hypertension and noise-induced hearing loss in Chinese coal miners. *J Thorac Dis*. 2016; 8(3): 422-429. doi:10.21037/jtd.2016.02.59.
 34. de Souza TC, Périssé AR, Moura M. Noise exposure and hypertension: investigation of a silent relationship. *BMC Public Health*. 2015; 15: 328.
 35. Chang TY, Hwang BF, Liu CS, Chen RY, Wang VS, Bao BY, et al. Occupational noise exposure and incident hypertension in men: a prospective cohort study. *Am J Epidemiol*. 2013; 177(8): 818-825.
 36. Powazka E, Pawlas K, Zahorska-Markiewicz B, Zejda JE. A cross-sectional study of occupational noise exposure and blood pressure in steelworkers. *Noise & Health*. 2002; 5: 8.
 37. Tomei G, Fioravanti M, Cerratti D, Sancini A, Tomao E, Rosati MV, et al. Occupational exposure to noise and the cardiovascular system: a meta-analysis. *Sci Total Environ*. 2010; 408(4): 681-9. doi: 10.1016/j.scitotenv.2009.10.07.
 38. Cayir A, Barrow TM, Wang H, Liu H, Li C, Ding N, et al. Occupational noise exposure is associated with hypertension in China: Results from project ELEFANT. *PLoS ONE* 2018; 13(12): e0209041
 39. Daniell WE, Swan SS, McDaniel MM, Camp JE, Cohen MA, Stebbins JG. Noise exposure and hearing loss prevention programmes after 20 years of regulations in the United States [published correction appears in *Occup Environ Med*. 2006 Jun; 63(5): 343-351.
 40. Veljovic F, Burek I, Begic E, Masic I. Redesign of Work Space in Order to Reduce Noise Health Effects.. *Mater Sociomed*. 2019 Jun; 31(2): 135-140. Doi: 10.5455/msm.2019.31.135-140.