Original Article

Zahedan Journal of Research in Medical Sciences

Journal homepage: www.zjrms.ir

Exposure to Noise Pollution and Its Effect on Oxidant and Antioxidant Parameters in Blood and Liver Tissue of Rat

Asghar Dehghani,[1] Mohammad Ranjbarian,*[1] Ali Khavanin,[2] Mansour Rezazade-Azari,[1] Shahram Vosooghi[1]

1. Department of Professional Health, Faculty of Health, Shahid Beheshti University of Medical Science, Tehran, Iran
2. Department of Professional Health, Tarbiat Modares University, Tehran, Iran

Article information

Article history:
Received: 2 Mar 2012
Accepted: 22 May 2012
Available online: 18 Nov 2012
ZJRMS 2013; 15(5): 13-17

Keywords:
Noise pollution
Oxidative stress
Reduced Glutathione and
Malondialdehyde

*Corresponding author at:
Department ShahidBeheshti
university of medical science
E-mail:
mohammad_rangbarian@yahoo.com

Abstract

***Background*:** Noise is a known physical stress in workplace, which induces alterations of various physiological responses in exposed individuals. Aim of this study was to explore the status of oxidant and antioxidant parameters in blood and liver tissue of rats exposed to noise.

***Materials and Methods*:** Twenty-one adult albino wistar male rats seven in each of the following groups were used (N=7): 1- Control group, 2- Noise exposure (100 dB, 700-5700 Hz, 8 h/day, 8 days), 3- Noise exposure (100 dB,700-5700 Hz, 8 h/day, 14 days). The animals were anesthetized by CO_2 and after decapitation, blood and liver samples were collected and processed for estimation of biochemical parameters (MDA and GSH levels) in control and exposed groups.

***Results*:** The present research findings showed significant decrease in liver weight and liver/body weight ratio in noise exposed groups compared to the control group (p<0.05). Fourteen days noise exposure caused a statistically significant increase in MDA level in serum and liver tissue and also statistically significant decrease in GSH level in serum (p<0.05), however, the level of GSH in the liver tissue showed no significant change. 8-days exposure just caused a statistically significant increase in MDA level in serum, and there was no significant change in the other parameters. Moreover, the levels of changes in biochemical parameters in noise exposed groups were exposure time-dependent.

***Conclusion*:** The results of present study showed that exposure to noise is toxic to blood and liver tissue. Further research for exploring the toxicity of noise in occupationally exposed groups is recommended.

Introduction

Today, noise pollution is a global problem (in particular for industrial workers) and damage resulted from noise is amongst the first 10 harmful damages reported [1, 2]. Harmful effect of loud noise has been recognized as an occupational stress inducing physiological and mental alternations in human for ages [3]. By industrialization of the societies, the number of individuals exposed to noise has increased. Noise is considered the most wide spread physical harmful factor in workplaces and environment so that it is estimated that over 600 million people are exposed to harmful noise existing in their workplaces among whom 50 to 60 million are in European and North American countries [4]. National Institute for Occupational Safety and Health (NIOSH) estimates that 14% of the workers is exposed to risky noise over 90 dBA [5]. The harmful effects of noise, in particular the production of free radicals, are not limited to hearing organ but they damage other tissues [6-11]. Exposure to noise results in some complications such as hearing loss, cardiovascular diseases, high blood pressure, mortality risk increase, serious physiological effects, headache, anxiety and nausea [8-12]. According to Mbulgiwe's study, noise in wood industry (developing countries) is over 90 dB [13]. And, also cross-sectional study of the 200 Danish wood and furniture industries reported equivalent noise level as 90.5 dBA [14]. Exposure to any kind of noise over 90 dB is recognized as stress source [10]. As chronic exposure, acute exposure can lead to the production of extra free radicals like superoxidase, catalase, and glutathione peroxidase [15].

Naturally, there is an approximate balance between the production of compounds derived from oxygen (oxidants) and the amount of antioxidant defense system activity. If the balance is broken for the compounds derived from oxygen, oxidative stress will be induced and results in biological damage [16-19]. Probable harmful effects of oxygen reactive species are neutralized through antioxidant defense system of cell including enzymes such as catalase and superoxide desmutase. Also, glutathione as an antioxidant results in the increase of toxins solubility and expulsion through kidneys [7]. Since noise is considered as dangerous physical contaminant in workplace and since final goal of occupational health is to prevent from effects resulted from work, and the labor and employees of many industries (including wood industry, textile industry and occupations like soldiers,

drivers and etc.) are also exposed to this risky factor, present study is aimed at an examination of antioxidant (GHS) and lipid peroxidant (MDA) parameters as a result of noise exposure.

Materials and Methods

The study was conducted empirically. 21 heads of male albino Wistar rats in 200±10 g weight range provided by Pasteur Research Institute (Tehran-Iran) were used. Animals were kept in animal house of Neuroscience Research Center of Shahid Beheshti University of Medical Sciences under light/dark cycle, 12-12 h (light from 7 am to 7 pm), temperature 25±2˚C and free access to animals' food and water. In this study, ethical codes of working with laboratory animals approved by Ethics Committee of Shahid Beheshti University of Medical Sciences were followed.

Based on toxicological studies classification, the study was carried out in semi-acute form. Based on the type of exposure, animals were randomly divided into three groups as follow: 1) noise exposure 100 dB (8 days and 8 h daily), 2) noise exposure 100 dB (14 days and 8 h daily), 3) control group with no noise pollution exposure. Exposure chamber was designed so that 7 rat heads can simultaneously be placed there. Output capacity of the transparent plexiglass container (interior volume 60l) was arranged as 12 times ventilation in an hour. Thiobarbitoric acid (TBA), trichloroacetic acid (TCA), 5, 5-dithiobis (2-nitrobanzoic acid) (DTNB) were prepared from Sigma Co. (America) and ethanol, NACL and EDTA were prepared from Merk Co. (Germany). Shimadzo UV 3100 (Australia) was used for measuring MDA and GSH.

Noise exposure: 100-dB noise level and desirable band width is 700-5700 Hz (combination of three Octavian noises with central frequencies of 1000-2000-4000 Hz) so that the exposure conditions are close to real conditions of workplace. Noise with desirable frequency combination was made by Signal Software. Noise files made by computer were run in Cool edit software. The run noise in the software was boosted by an amplifier and sent to loudspeakers fitted in chamber's ceiling (30 cm height). During the exposure, noise intensity was measured at 4 points inside the chamber using calibrated B & K noisemeter device (MODEL 2238) and regularly monitored.

Surgery and preparing specimens: Upon anesthesia by CO_2, rats were decapitated on ninth and fifteenth days (to prevent from acute effect of noise exposure on eighth and fourteenth days). After separating liver tissue and preparing it for biochemical analysis, specimens were washed by phosphate buffer (pH=7). Liver tissue of all groups was weighed.

Measuring GSH level in tissues of liver and blood serum: GSH level of blood serum and GSH level of liver were measured respectively using Beutler method [20] and Ellman's reagent by Sedlak and Lindsay method [21]. Accordingly, surface solution was mixed with Tris buffer (2%) containing EDTA (0.02 M, pH=8.9) and DTNB (0.01 M) and immediately absorbed at 412 nm wavelength against blank without homogenate.

Measuring MDA level in tissues of liver and blood serum: In this study, TBA reagent was used for measuring MDA of liver tissue and blood serum [22]. MDA+TBA reactive solution absorption at 535 nm wavelength was read using spectrometer (Shimadzo-3100) (in blank specimens, phosphate buffer of which homogenate was prepared used instead of homogenate) and MDA level was calculated using its molar extinction coefficient (ε=1.56*$10^5 M^{-}cm^{-}$) [23].

Statistical Method: in this study, SPSS-16 software was applied to analyze the data. To determine mean and standard deviation of the data, descriptive statistics was used. Kroskal-Wallis and Mann-Whitney *U* tests were used to detect significant effect of noise. Statistical level of the test was considered less than 0.05.

Results

Results of measuring rats' weight and their liver tissue weight: In this study, weight of rats and their livers were examined in groups under study to examine the effect of noise. Rats' weights were recorded on the first day of research and also before surgery, that is, on ninth and fifteenth days. As seen in table 1, mean weight before and after exposure has no significant difference (p>0.05) between control and exposure group based on Kroskal-Wallis test, but comparing liver/body weight ratio in the exposure groups (comparing to control group) it is seen that it has a significant reduction (p<0.05). However, it is shown that liver/body weight ratio reduction in noise exposure groups does not depend on exposure duration (p>0.05).

Results of measuring MDA and GHS of liver tissue: based on the results from table 2 and Mann-Whitney test, MDA level of liver tissue has a significant increase in 14-day noise exposure group comparing to control group (p<0.001). It must be noted that MDA level increase in exposure groups depends on exposure duration (p<0.05). However, GSH level of liver tissue has no significant decrease in intervention groups comparing to control group.

Results of measuring MDA and GHS of blood serum: Based on the results of table 3, MDA level of blood serum in the intervention groups has significant increase comparing to control group (p<0.05). It must be noted that MDA level increase in noise exposure groups depends on exposure duration (p<0.001). GSH level of blood has a significant decrease in 14-day noise exposure group and also 8-day exposure (p<0.05).

Discussion

Since weight is considered as one of the major toxicological parameters, results of the present study have not shown any significant differences in pre- and post-exposure mean weights in the groups under study, but liver weight and liver/body weight ratio in the intervention groups has had a significant decrease compared to control group. And, also results of the study

demonstrated that noise pollution leads to an increase in the amount of MDA in blood serum in the intervention groups comparing to control group. It must be noted that blood MDA level has had a significant decrease in the intervention group (14-day noise exposure) comparing to control group and also 8-day noise exposure group.

Table 1. Basic weight, final weight of rat and liver/body weight ratio in groups under study (N=7)

Variable	Group	Mean±SD	*p*-Value
Pre-exposure weight (g)	Control	201.49±21.44	-
	8-day noise	191±17.85	0.318
	14-day noise	193.34±16.92	0.209
Post-exposure weight (g)	Control	224.47±16.93	-
	8-day noise	215.77±19.67	0.383
	14-day noise	221.77±23.19	0.620
Liver/body weight ratio	Control	0.048±0.002	-
	8-day noise	0.037±0.002	0.001
	14-day noise	0.034±0.002	0.001

p-Value of each variable is displayed comparing to control group

Table 2. MDA and GHS levels of liver tissue in groups under study (N=7)

Variable	Group	Mean±SD	*p*-Value
MDA (nmol/g)	Control	0.67±0.174	-
	8-day noise	0.82±0.142	0.097
	14-day noise	1.68±0.182	0.001
GSH (µmol/g)	Control	6.78±0.597	-
	8-day noise	6.71±0.618	0.710
	14-day noise	6.66±0.614	0.318

p-Value of each variable is displayed comparing to control group

Table 3. MDA and GHS concentrations of blood serum in groups under study (N=7)

Variable	Group	Mean±SD	*p*-Value
MDA (nmol/l)	Control	0.12±0.021	-
	8-day noise	0.16±0.019	0.038
	14-day noise	0.26±0.018	0.001
GSH (mg/dl)	Control	38.87±2.51	-
	8-day noise	37.21±2.57	0.318
	14-day noise	32.22±2.70	0.038

p-Value of each variable is displayed comparing to control group

Based on the studies done, noise exposure leads to an increase in metabolic activities and creation of oxygen reactive species. Noise-exposure-induced reactive oxygen species play a pivotal role in damaging the body, as well [24] which the results corresponded with the Monsefi study where 100 dBA-noise-exposed rats on 15th and 30th days showed no significant difference comparing to control group [25], in Cappaert study also no significant differences were observed in noise exposure group and ethyl benzene group comparing to control group [26]. But in Manikandan study lasted for 30 days, body weight loss was observed in the noise exposure group [15] that the results are in conformity to those of various studies where animals were exposed to harmful factors [27, 28]. Moreover, it was shown that liver weight loss and liver/body weight ratio loss in the noise exposure groups do not depend on the duration of the exposure. Since noise is recognized as one of the most harmful physical factors [4] and its harmful effects on living entities and biological systems are reported in systematic form as well as inducing hearing loss. Like the increase of free radicals resulting in oxidative stress and adverse effects on heart, liver, blood, and reproductive system [6-12]. Nevertheless, results of different studies have shown that glutathione enzyme system serves as a defense mechanism against noise-induced hearing loss. Studies have demonstrated that if the amount of glutathione increases in cochlea tissue after noise exposure, the extent of damage will decrease in Corti organ of auditory-sensory tissue of hearing [1, 18].

Noise-induced acoustic trauma results in the formation of superoxide anion radical in guinea pig inner ear [29]. Free radical species (FRS) lead to hearing loss resulted from noise and chemical compounds. However, antioxidants play protective role in these conditions [30, 31]. Other studies also indicate that noise exposure results in ischemia [32] and long-term hypoxia together with ischemia lead to the formation of FRS and lipid peroxidation [33]. Since blood circulation system results in the transmission of oxidant and antioxidant parameters to different tissues, so studying the relationship between these changes in blood and liver tissue can demonstrate indirect effects of noise.

Based on the results of the present study, noise has also led to the increase of MDA amount in blood serum in the intervention groups compared to control group. It must be noted that blood MDA level increase in the noise exposure groups depends on exposure duration. Also, blood GSH level in the 14-day noise exposure group comparing to the control group and 18-day noise exposure group has had a significant decrease. In the results of Demirel study where rats had 4-h (20 days) daily exposure to 100-dB white sound, it was observed that MDA, NO level and GSH-Px activity has significantly increased in noise-exposed group which of course in control group no significant changes were observed between mean parameters under study at the beginning and end of the period but MDA level increase was observed in blood and liver tissue of noise-exposed group [12].

In the study of Ohlemoller carried out on the rats exposed to heavy noise with a wide band, it was observed that reactive oxygen species increased in cochlea after 1 to 2h of exposure and it was also revealed that blood MDA level which is the final product of the free radicals from lipid peroxidation increases [31] which corresponds with the results of the present studyIn the study of Ising et al., it is also cited that acute and chronic noise exposure leads to an increase of hormones and consequently an increase of oxygen free radicals and showed that levels of these oxidative factors can be measured in other organs like liver [34].

In the present study, MDA level of liver tissue has only increased in 14-day exposure group comparing to control group. Also, it was seen that liver tissue MDA level increase depends on exposure duration in the exposure groups. Of course, liver tissue GSH level in the intervention group has not significantly decreased comparing to control group. Janaco et al. also concluded

that with the increase of glutathione and catalase, damage level of capillary cells in Corti organ decreases and considered enzyme system and glutathione as a protective mechanism against hearing loss, also the amount of blood serum glutamine decreased as a result of exposure [35] which resembled the results of the present study.

Moreover, in a study conducted by Derekoy (corresponding to results of the present study), rabbits were exposed to a 100-dB noise with wide frequency band. Reduction of glutathione and increase of MDA level in the exposed groups were demonstrated [19].

Generally, results of biochemical measurements showed that noise leads to an increase of oxidation metabolites in blood and liver tissue which correspond with the results of the studies mentioned above and the increase in oxidation metabolites had led to the decrease of the antioxidants. However, at the presence of other harmful factors, the changes may increase their effects. So, oxidation effect of the noise stress in work and living environments on tissues other than hearing tissue must be taken serious.

It must be noted that in the study, the exposure is of semi-acute types, but the exposure in workplaces is chronic and to determine the effect which can be generalized to workplace, an exposure of over 90 days is required. Regarding the efficiency of the antioxidants in preventing from oxidative damage resulted from noise, it is recommended that studies be designed and conducted concerning the use of antioxidant substances to protect against harmful effects like noise pollution. And, also, further studies are suggested to examine the toxicity of noise in societies occupationally exposed to this factor.

Acknowledgements

The present study is a result of Mr. Asghar Dehghani's MS thesis approved by honorable research deputy of Shahid Beheshti University of Medical Sciences with agreement No. p/25/11/2517. The authors declare that there is no conflict of interests.

Authors' Contributions

All authors had equal role in design, work, statistical analysis and manuscript writing.

Conflict of Interest

The authors declare no conflict of interest.

Funding/Support

Shahid Beheshti University.

References

1. Karlidag T, Yalsin S, Ozturk A, et al. The role of free oxygen radicals in noise induced hearing loss: Effects of melatonin and metylprednisolone. Auris Nasus Larynx 2002; 29(2): 147-52.
2. Ohinataab Y, Yamasobae T, Schachta J and Millera JM. Glutathione limits noise induced hearing loss. Hear Res 2000; 146(1-2):28-34.
3. Rom WN. Environmental and occupational medicine. 3rd ed. Philadephia: Lippincotte-Raven Press; 1998: 1345-49.
4. Alberti PW. Noise the most ubiquitous pollutant. Noise Health 1998; 1(1): 3-5.
5. Rees T, Duckert L. Hearing loss and other otic disorders. 1st ed. In: Rosenstock L, Mark R. Textbook of clinical occupational and environmental medicine. Toronto: WB Saunders; 1994. 98-142.
6. Pinar T, Atli AK, Alacam H, et al. The effects of noise on oxidative and antioxidative balance in human erythrocytes. Inter J Hematol Oncol 2011; 21(1): 27-37.
7. Mates JM, Perez-Gomez C, Nunez de Castro I. Antioxidant enzymes and human diseases. Clin Biochem 1999; 32(8): 595-603.
8. Abbate C, Concetto G, Fortunato M, et al. Influence of environmental factors on the evolution of industrial noise-induced hearing loss. Environ Monit Assess 2005; 107(1-3): 351-61.
9. Lenzi P, Frenzilli G, Gesi M, et al. DNA damage associated with ultrastructural alterations in rat myocardium after loud noise exposure. Environ Health Perspect 2003; 111(4): 467-71.
10. Ravindran RD, Samson J, Senthilvelan M. Noise-stress-induced brain neurotransmitter changes and the effect of ocimum sanctum (Linn) treatment in albino rats. J Pharmacol Sci 2005; 98(4): 354-60.
11. Stansfeld SA, Matheson MP. Noise pollution: non-auditory effects on health. Br Med Bull 2003; 68: 243-57.
12. Demirel R, Mollaoglu H, Yeşilyurt H, et al. Noise induces oxidative stress in rat. Eur J General Med 2009; 6(1): 20-24.
13. Mbuligwe SE. Levels and influencing factors of noise pollution from small-scale industries (SSIs) in a developing country. Environ Manage 2004; 33(6): 830-39.
14. Vinzents P, Laursen B. A national cross-sectional study of the working environment in the Danish wood and furniture industry air pollution and noise. Ann Occup Hyg 1993; 37(1): 25-34.
15. Manikandan S, Srikumar R, Parthasarathy NJ and Devi RS. Protective effect of Acorus Calamus Linn on free radical scavengers and lipid peroxidation in discrete regions of brain against noise stress exposed rat. Biol Pharm Bull 2005; 28(12): 2327-30.
16. Abuja PM, Albertini R. Methods for monitoring oxidative stress, lipid peroxidation and oxidation resistance of lipoproteins. Review. Clin Chim Acta 2001; 306(1-2): 1-17.
17. Elsayed MN. Antioxidant mobilization in response to oxidative stress: A dynamic environmental nutritional interaction. Nutrition 2001; 17(10): 827-34.
18. Yamasoba T, Harris C. Influence of intense sound exposure on glutathione synthesis in the cochlear. Brain Res 1998; 804(1): 72-78.
19. Derekoy FS, Koken T, Yilmaz D, et al. Influence of noise exposure on antioxidant system and TEOAEs in rabbits. Eur Arch Otorhinolaryngol 2001; 285(10): 518-22.
20. Beutler E, Duron O, Kelly BM. Improved method for the determination of blood glutathione. J Lab Clin Med 1963; 61: 882-88.
21. Sedlak J, Lindsay RH. Estimation of total protein bound and non-protein sulphydryl groups in tissue with Ellman's reagent. Anal Biochem 1968; 25(1): 192-205.
22. Wills ED. Lipid peroxide formation in microsomes: General consideration. Biochem J. 1969; 113(2): 315-24.

23. Buege JA, Aust SD. Microsomal lipid peroxidation. Methods Enzymol 1978; 52: 302-11
24. Wu WT, Sha SH, Schacht J. Recent advances in understanding aminoglycoside ototoxicity and its prevention. Audiol Neurootol 2002; 7(3): 171-74.
25. Monsefi M, Bahoddini A, Nazemi S and Dehghani GA. Effects of noise exposure on the volume of adrenal gland and sSerum levels of cortisol in rat. Iran J Med Sci 2006; 31(1): 5-8.
26. Cappaert NL, Klis SF, Muijser H, et al. Simultaneous exposure to ethyl benzene and noise: Synergistic effects on outer hair cells. Hear Res 2002; 162(1-2): 67-79.
27. Ozen OA, Yaman M, Sarsilmaz M, et al. Testicular zinc, copper and iron concentrations in male rats exposed to subacute and subchronic formaldehyde gas inhalation. J Trace Elem Med Biol 2003; 17(1): 6.
28. Ozen OA, Yaman M, Sarsilmaz M, et al. Testicular zinc, copper and iron concentrations in male rats exposed to subacute and subchronic formaldehyde gas inhalation. J Trace Elem Med Biol 2002; 16(2): 119-22.
29. Yamane H, Nakai Y, Takayama M, et al. Appearance of free radicals in the guinea pig inner ear after noise-induced acoustic trauma. Eur Arch Otohinolaryngol 1995; 252(8): 504-08.
30. Henderson D, McFaden SL, Lio CC, et al. The role of antioxidants in protection from impulse noise. Ann NY Acad Sci 1999; 884: 368-80.
31. Ohlemiller KK, Wright JS, Dugan LL. Early elevation of cochlear reactive oxygen species following noise exposure. Audiol Neurootol 1999; 4(5): 229-36.
32. Lamm K, Arnold W. Noise induced cochlear hypoxia is intensity dependent, correlates with hearing loss and precedes reduction of cochlear blood flow. Audiol Neurootol 1996; 1(3): 148-60.
33. McCord JM. Mechanism of disease, oxygen-derived free radicals in post – ischemic tissue. N Engl J Med 1965; 312: 159-63.
34. Ising H. [Acute and chronic stress hormone increases in noise exposure] German [Abstract]. Schriftenr Ver Wasser Boden Lufthyg 2000; 106: 169-77.
35. Jacono AA, Hu B, Kopke RD, et al. Changes in cochlear antioxidant enzyme activity after sound conditioning and noise exposure in the chinchilla. Hear Res 1998; 117(1-2): 31-38.

Please cite this article as: Dehghani A, Ranjbarian M, Khavanin A, Rezazade-Azari M, Vosooghi S. Exposure to noise pollution and its effect on oxidant and antioxidant parameters in blood and liver tissue of rat. Zahedan J Res Med Sci (ZJRMS) 2013; 15(5): 13-17.