Relationship between Exposure to Industrial Noise and Serum Lipid Profile

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Abstract- Aim of our study was to investigate the effects of exposure to industrial noise on serum lipid profile among workers who are exposed to noise at work. In a historical cohort study, we recruited 154 and 146 male workers as high and low level noise exposure groups respectively. We defined workers with at least one year exposure to noise level more than 90 dB as high exposure group, and those with exposure to less than 80 dB as low exposure group. Afterwards, in the fasting blood specimens of participants we measured serum Triglyceride (TG), total Cholesterol (TC), high and low density lipoprotein (HDL and LDL). Mean of TG, TC, HDL and LDL for low exposure group were 148, 189, 38 and 103 mg/dl and for high exposure group were 237, 189, 37 and 104 mg/dl respectively. Mean serum TG between two groups was different. Even after adjustment for age, BMI, smoking and work hours per week, serum TG among high exposure group was 89 mg/dl higher than low exposure group and this difference was statistically significant (P=0.00). There was no significant difference between two groups in TC, LDL and HDL levels. This study did not find a statistically significant relationship between exposure to noise and serum TC, LDL and HDL, but TG in two groups was different and this difference was statistically significant.
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Acta Medica Iranica, 2011; 49(11): 725-729.

Introduction

Nowadays, in progress industrialism leads to significant increase of occupational noise. Undoubtedly noise is considered as one of the most hazardous physical factors in work place. Noise exposure is associated with several adverse pathophysiological effects such as acute and chronic hearing loss, hypertension and cardiovascular diseases (1).

Exposure to noise is unavoidable part of many industries, therefore, the vast majority of labor forces suffer from its side effects (2). Most of previous studies have focused on association between chronic noise exposure and hypertension. In other word, rising blood pressure, especially diastolic, in presence of occupational noise exposure have been the most favorite theory in many studies (1,2).

On the other hand, there were a few studies focusing on the effects of noise exposure on the serum lipoproteins (3). The study by Melamed *et al.,* on 1455 men and 624 women, exposing to more than 80 dB noise for long time, revealed that there was a significant increase in total serum cholesterol (TC) and TC/HDL ratio (P=0.03) in men under 45 year. In contrast, there was no statistical evidence of such a result in terms of women and men over 45 year old (4).

In a study by Prabhakaran *et al.,* on acute hearing stress in mice, a significant relationship between noise and TC (not triglyceride) was revealed (5). Marth *et al.,* showed that individuals with exposure to airport noise (over 105 dB) had prominent rise in their TC and blood sugar, but their triglyceride decreased (6). In another study, Sroczynskiy *et al.,* reported a significant increase in phospholipids, beta lipoproteins and total lipids (not TC) in subjects with exposure to industrial noise and localized vibrations (7). Furthermore, Virkkunen *et al.,* in his integrated cohort study on 6005 labor forces in Finland, in 18 year period of time, reported drastic increased risk of coronary artery diseases continuing in retirement duration (8).

Our study was designed to determine the association between occupational noise and serum lipoproteins in work forces facing industrial noise. Raising occupational health and preventative medicine were the major goals.

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Materials and Methods

Using a historical cohort study, we recruited 154 and 146 male workers from Raja Passenger Train Company, as high and low level noise exposure groups respectively. This company is located in Tehran and manages domestic and international train transportation since 1996. More than 2000 employees are working in this company that nearby half of them are active in the wagon maintenance sector which is a noisy workplace. Workers in this sector were not shift worker.

We defined workers with at least one year exposure to noise level more than 90 dB as high exposure group, and those with exposure to less than 80 dB considered as low exposure group. By this definition, 438 of them were located in high exposure group, in contrast with 579 workers in low exposure group. Afterward with simple random sampling, we derived 154 subjects from high exposure group and 146 from low exposure group and invited them to participate in our study.

This study adhered to the tenets of the Declaration of Helsinki. We emphasized the voluntary nature of our study and asked the participants to fill in an informed consent sheet. The data gathering sheets and research protocol were approved by the ethics committee of Tehran University of Medical Sciences.

At least one year work in high or low exposure group was the only inclusion criterion. Exclusion criteria were: presence of diabetes mellitus, familial hyperlipidemia, thyroid dysfunction (hyper and hypothyroidism), chronic renal failure, nephrotic syndrome, recent using of medications (serum lipid reducing agents, β-blockers, thiazids, isotretinoids, glucocorticoids and anabolizing steroids), past history of myocardial infarction, cerebrovascular attacks, sever trauma and prominent changes in nutritional habits through last month.

We used company`s data, achieving from the annual measurements in various units, to estimate the exposure level for each participant. For this purpose the company had divided work place to different subunits. Then, every subunit was subdivided to 2×2 m^2 Square scales. Take placing a sound detector station in the centre of each scale, sound pressure levels were measured. The company had used sound level meter type 2 (Lutron SL-4011) to measure sound pressure level in each unit. Afterwards the sound pressure levels in several sound frequencies were determined with the analyzer device.

We met the participants at workplace and a trained member of our research team helped them to fill a data collection sheet. In these sheets we recorded personal information, namely: age, work duration, work hours per week and alcohol and cigarette consume. Then, the participant's height and weight were measured and body mass index (BMI) was determined with Quetelet formula, [weight (kg)/ height (m)^2].

Regarding main variables measurement, we explained the process of our study to all participants; then, requested them to be in fasting condition (12 hours fasting period) for next day. Early morning at the next day, our trained team obtained participant`s fresh blood specimens at work site. We used enzymatic methods to estimate serum Triglyceride (TG), total Cholesterol (TC) and high density lipoprotein (HDL) levels (9,10). TC and TG were determined with Cho-PAP and GPO-PAP enzymatic methods respectively and phosphotungstate enzymatic method was used regarding HDL measurement. Low density lipoprotein (LDL) levels were determined with Fridewald Formula (9).

After gathering data, using SPSS software version 16 we analyzed our data to find an association between exposure to noise and lipid profile among this group of workers. We firstly compared demographic data and work related variables between two groups. We then used Mann-Whitney U test to compare mean of serum lipoproteins between low and high stress groups. For multivariate analysis, we considered TC, TG, HDL and LDL as dependent variable and demographic and work related variables as independent variables to examine if there is a relationship between exposure to noise and each of these lipid profiles after adjustment for other demographic and work related variables.

Results

Table 1 demonstrates mean and standard deviation (SD) of demographic and work related variables (age, weight, height, BMI, work duration and work hours per week) in low and high exposure groups. Difference of mean for age, work duration and work hours per week was statistically significant between two groups. 19.5% of high exposure group and 7.5% of low exposure group were shift workers (*P*=0.00). Moreover, 20.1% of high exposure group and 15.8% of low exposure group were smoker (*P*=0.32).

Table 2 compares mean of serum lipoproteins between high and low exposure groups. Mean of serum TG among high exposure group was 89 mg/dl higher than low exposure group and this difference was statistically significant (*P*=0.00). Serum HDL, LDL and TC levels did not demonstrate statistically significant differences between two groups.

Table 1. Comparison of means and standard deviations (SD) of demographic data between low exposure (n=146) and high exposure (n=154) groups.

variable	Unit	High exposed	Low exposed	Diff	*P*-value
Age	year	42 (10)	37 (8)	5	0.00*
Weight	kg	79 (12)	78 (13)	1	0.44
Height	cm	173 (7)	173 (7)	0	0.61
BMI	kg/m^2	26.5(3)	26 (4)	0.5	0.31
Work duration**	year	17 (10)	10 (8)	7	0.00*
Work hours	hour/week	71 (13)	60 (13)	11	0.00*

* Indicates statistically significant difference

** Years of Job experience in this company

Table 2. Comparison of mean (SD) of lipoproteins between low exposure (n=146) and high exposure (n=154) groups.

Variable	Unit	High exposed	Low exposed	Diff	*P*-value
TG	Mg/dl	237(79)	148(105)	89	0.00*
TC	Mg/dl	189 (38)	189(34)	0	0.98
HDL	Mg/dl	37(9)	38(8)	1	0.73
LDL	Mg/dl	104(33)	103(26)	1	0.81
TC/HDL**		5.3(1.6)	5.2(1.5)	0.1	0.71
LDL/HDL		2.0(0.90)	2.8(0.85)	0.8	0.80

* Indicates statistically significant difference

** Total cholesterol/HDL ratio

Table 3. Multiple linear regression analysis for serum TG adjusted for some personal and work related variables

Variable	B	SE	Beta	*P*-value	95% CI for B
Exposure to noise	74.49	11.65	0.36	0.000*	(51.57-97.41)
Age	0.29	0.63	0.03	0.647	(-0.95-1.53)
BMI	2.95	1.48	0.11	0.046*	(0.05-5.86)
Hour/week	0.99	0.42	0.14	0.022*	(0.14-1.80)
Smoking	6.79	14.13	0.03	0.63	(-21.02-34.60)
Constant	1.77	43.91			

* Indicates statistically significant difference

Table 4. Lipid profile changes across a few studies

study	TG	Total C
(6)Marth E, *et al.*	↓	↑
(4) Melamed S, *et al.*	↑	↑
(5) Prabhakaran K, *et al.*	↓	↑
(7) Sroczynski J, *et al.*	↑	→

↓: Decrease

↑: Increase

→: No change

Considering serum TG as dependent variable in linear multiple regression and adjustment for other variables revealed that difference of serum TG between high and low exposure groups decreased from 89 to 74, however this difference remained statistically significant after adjustment for age, BMI, smoking and work hours per week (Table 3). Linear regression analysis showed that other than working in a place with high exposure to noise, increasing working hours per week, will increase serum TG among this group of workers. BMI as a well

known affecting factor on serum lipids was related to serum TG in our participants (Table 3).

Discussion

Noise is common burden of our life, especially in large cities and at industrial work sites (2). While sound is one of the most important environmental pollutants, many recent studies place a particular emphasis on several adverse pathophysiological effects of that, namely: hyperlipidemia, hypertension, cardiovascular diseases and hearing loss.

Our study was designed in order to assess the association between chronic industrial noise exposure (over than 80 dB) and serum lipid profile level, which have potentially important impacts on risk of ischemic heart diseases. Previous studies about relationship between noise and hyperlipidemia have produced various results, some showed an increased level of serum lipid profile and others did not find such a relationship. Table 4 demonstrates lipid profile changes across a few of these studies.

Melamed et al., studied 1455 male and 624 female workers with chronic exposure to sound pollution (> 80 dB) and demonstrated a significant increase in TG (P=0.001) and TC (P=0.02) levels as well as TC/HDL ratio (P=0.03) in younger than 45 year old male workers but this relation was not observed for female and elder than 45 year male workers (4). Malamed and colleagues also assessed noise annoyance with Tamopolsky criteria and reported a relation between noise annoyance and TC and TG levels in young men and also with TC, TG and HDL levels in women (4). They also observed additive effects of noise exposure on serum TC level. No interaction was detected between noise annoyance and amount of sound exposure (4). Sroczynski et al., studied 265 workers who were exposed to industrial noise and local vibrating sounds and the level of total lipids, phospholipids, beta lipoproteins and TC in the serum was determined. The level of all those substances, except TC, was found to be increased (7). The study of Prabhakaran et al., showed a significant relationship between noise and TC (not TG) in mice with acute hearing stress (5).

Marth et al., focused on the effect of stress caused by aircraft noise. They chose 14 female and 11 male volunteers, who were exposed to an aircraft simulator that stimulated a maximum noise level of 105 dB for 3 sec. They reported an increase level of TC and a decrease of serum TG in the serum (6).

The results of Melamed et al., and Sroczynski et al., demonstrate the increasing level of TG in presence of chronic noise burden (4,7). In contrast, in Marth et al., and Prabhakaran et al., studies there are a decreasing trend for TG (5.6). TC level in all of these studies, except Sroczynski`s study, were significantly increased. Similarly, we observed increased levels of TG, but not TC in our study.

Several findings of our study focused on the relationship between exposure to chronic noise and serum lipoproteins level, but undeniably, its damaging consequences for ischemic heart diseases is more important. In fact, the relationship between noise and cardiovascular diseases has been one of the most controversial issues in recent decades and many studies have been designed to investigate it (11,12).

Davies et al., in their cohort study identified 27,464 blue-collar workers from 14 lumber mills in British Columbia who worked at least 1 year between 1950 and 1995 and who were followed up over the same period and quantitatively assessed the cumulative noise exposure (13). During the follow-up period, 2,510 circulatory disease deaths occurred. In his study relative risks for acute myocardial infarction mortality were elevated in the full cohort. There was an exposure-response trend, with a relative risk in the highest exposed group of 1.5 (95% confidence interval=1.1-2.2). They concluded that chronic exposure to noise levels, typical of many workplaces, was associated with excess risk for acute myocardial infarction death (13).

Lee et al., in their study focused on the effect of chronic noise exposure on blood pressure among male workers (14). In their cohort study, five hundred thirty male workers at a metal manufacturing factory in Busan, Korea, were enrolled. They were monitored with annual health check-ups for nine consecutive years from 1991 to 1999. The subjects were divided into four groups which were determined by noise level categories (NLCs) according to the exposure to noise. After controlling the possible confounders, they determined that chronic noise exposure increases systolic blood pressure independently, among male workers (14).

Chang et al., in their study in 2005, focused on the effects of hyperlipidemia in noise-induced hearing loss (15). In a case control study, they collected the laboratory data from routine health examinations administered to workers who were exposed to noise greater than 85 dB over a one-year period. Then they analyzed the relationships of TC and TG levels with noise induced hearing loss (NIHL). A total of 4,071 cases were analyzed. After adjusting for age and gender,

hypertriglyceridemia was found to be related to NIHL (odds ratio=1.281; 95% CI, 1.09-1.51), but hypercholesterolemia was not (odds ratio=0.951; 95% CI, 0.79-1.14) (15).

Mac Namee *et al.* also the predictive validity of a retrospective measure of noise exposure to investigate the effects of long term exposure to excessive noise on the risk of ischemic heart disease (16).

In our study exposure to industrial noise did not made noticeable changes in TC, LDL and HDL levels, so we could not find mounting evidence about impacts of noise exposure on the cardiovascular disorders via TC, LDL and HDL levels. After adjustment for some confounding factors, difference of TG between two groups remained statistically significant.

A limitation for our study was the measurement of nutritional habits and physical activities in participants which can play a role as confounding factors. However the workers in both groups have similar socioeconomic status (income, education levels and family size) and they eat the same meals for breakfast and lunch at work place. In conclusion, while our study did not find a statistically significant relationship between exposure to noise and serum TC, LDL and HDL, it demonstrated convincingly that level of serum TG in the two groups was different. More studies should be designed in order to highlight the effects of exposure to noise at work on the ischemic heart diseases.

Acknowledgement

References

1. Melamed S, Kristal-Boneh E, Froom P. Industrial Noise Exposure and Risk Factors for Cardiovascular Disease: Findings from the CORDIS Study. Noise Health1999;1(4):49-56.
2. Willich SN, Wegscheider K, Stallmann M, Keil T. Noise burden and the risk of myocardial infarction. Eur Heart J 2006;27(3):276-82.
3. Melamed S, Harari G, Green MS. Type A behavior, tension, and ambulatory cardiovascular reactivity in workers exposed to noise stress. Psychosom Med 1993;55(2):185-92.
4. Melamed S, Froom P, Kristal-Boneh E, Gofer D, Ribak J. Industrial noise exposure, noise annoyance, and serum lipid levels in blue-collar workers: the CORDIS Study. Arch Environ Health 1997;52(4):292-8.
5. Prabhakaran K, Suthanthirarajan N, Namasivayam A. Biochemical changes in acute noise stress in rats. Indian J Physiol Pharmacol 1988;32(2):100-4.
6. Marth E, Gallasch E, Fueger GF, Mose JR. Aircraft noise: changes in biochemical parameters. Zentralbl Bakteriol Mikrobiol Hyg B 1988;185(4-5):498-508.
7. Sroczynski J, Wegiel A, Wieja A. Effect of vibration and noise on the fat balance in exposed workers. Med Pr 1979;30(1):49-54.
8. 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.
9. Akuyam A, Isah HS, Ogala WN. Serum lipid profile in malnourished Nigerian children in Zaria. Niger Postgrad Med J 2008;15(3):192-6.
10. Ribas SA, Silva LC. Dyslipidemia in schoolchildren from private schools in Belem. Arq Bras Cardiol 2009;92(6):412-7, 29-34, 46-51.
11. Yuan J, Yang M, Yao H, Zheng J, Yang Q, Chen S, Wei Q, Tanguay RM, Wu T. Plasma antibodies to heat shock protein 60 and heat shock protein 70 are associated with increased risk of electrocardiograph abnormalities in automobile workers exposed to noise. Cell Stress Chaperones 2005;10(2):126-35.
12. McNamee R, Burgess G, Dippnall WM, Cherry N. Occupational noise exposure and ischaemic heart disease mortality. Occup Environ Med 2006;63(12):813-9.
13. 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.
14. Lee JH, Kang W, Yaang SR, Choy N, Lee CR. Cohort study for the effect of chronic noise exposure on blood pressure among male workers in Busan, Korea. Am J Ind Med 2009;52(6):509-17.
15. Chang NC, Yu ML, Ho KY, Ho CK. Hyperlipidemia in noise-induced hearing loss. Otolaryngol Head Neck Surg 2007;137(4):603-6.
16. McNamee R, Burgess G, Dippnall WM, Cherry N. Predictive validity of a retrospective measure of noise exposure. Occup Environ Med 2006;63(12):808-12.