

The influence of industrial noise on urinary metanephrine, normetanephrine and vanilmandelic acid levels in workers of textile factory in Yazd, Iran

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ABSTRACT

Noise, as the most common physical agent in workplace, not only causes hearing loss but also leads to various diseases, such as hypertension and cardiovascular disease. Sound is likely to stimulate and increase the secretion of adrenal catecholamines, and may have an indirect role in the pathogenesis of diseases caused by stress. This study was performed to investigate the effect of noise on urinary levels of metabolites of catecholamines of workers in a textile factory in Yazd, Iran. The sound pressure level was measured in different units of the factory, and the mill divided into two sections as noisy ($l_{eq} \geq 85$ db) and quiet ($l_{eq} \leq 70$ db) units. Forty four eligible workers participated in this study (22 as study and 22 as control group) and their urine samples were collected before and after workday shift. Metanephrine and normetanephrine levels were measured using enzyme linked immunosorbent assay (ELISA), and vanilmandelic acid level was measured using chromatography. Results were analyzed using Wilcoxon and Mann-Whitney statistical tests. Urinary levels of metanephrine, normetanephrine and vanilmandelic acid in the study group were significantly higher than control group in both before and after shift samples. In study group level of all three metabolites were significantly increased after workday shift. Although no significant after shift increase was observed for metanephrine and vanilmandelic acid in control group, but the increase in normetanephrine level was significant. It can be concluded that noise, as a stressor factor, may increase the excretion of catecholamines which may increase the risk of stress-related diseases.

Keywords: Noise, Stress, Catecholamines, Metanephrines, Vanilmandelic acid, Textile workers.

Received: 16 August 2016 Accepted: 9 December 2016

Introduction

Nowadays, workers are exposed to many adverse factors in workplaces and these factors can adversely affect their health and performance. Noise as the most common physical

agent in workplace has many known adverse effects on human beings (1). After the presbycusis, hearing loss is the most common hearing impairment. Sliwinska-Kowalska and Davis expressed hearing loss as the most important adverse effect, due to the noise exposure (2). Previous studies estimated that more than 16% of the adult hearing loss was caused by the occupational exposure (3). The noise adverse effect is not only limited to auditory system but other organs of the body may be affected by acute or chronic exposure to noise. Jarup et al. (4) and Tomei et al. (5) have shown that people who are exposed to noise, have higher blood pressure than the control group. Increasing the risk of cardiovascular disease, caused by noise has been reported by researches (6-8). Digestive system disorders (9), speech intelligibility (10), sleep disturbance (11), efficiency reduction (12, 13) and other adverse effects of noise have been shown in many studies that indicate the importance of noise role in occupational related diseases. Some researchers supposed that noise non- auditory effects comes from stress- related hormones secreted due to the noise exposure (14, 15). These hormones, generally called catecholamine (including epinephrine, norepinephrine and dopamine) are part of the sympathetic nervous system and are released from the adrenal gland in response to stress factors (16, 17). These hormones have a short half-life about two minutes in blood and quickly are metabolized to metanephrine, normetanephrine, vanilmandelic acid and excreted by urine. About 50% of urinary catecholamines are metanephrine and normetanephrine and 35% is vanilmandelic acid. Body metabolism is affected by catecholamines. Heart rate, heart muscles contraction force and

peripheral vascular resistance are catecholamine-dependent. A study conducted by Gesi et al. (18) has shown that noise can cause morphological and biochemical effects in norepinephrine and epinephrine cells of adrenal medulla. Ising and Braun observed that short term exposure to the intense noise (105-125 dbA) lead to the increases in cortisol and metanephrine levels (19). In addition, they found the level of norepinephrine and metanephrine in workers who exposed to noise in an industrial plant is higher than the control group. Another study on the subjects exposed to the traffic noise, has shown that norepinephrine level increases significantly due to exposure to noise (20). Sudo and colleagues study also indicate higher urinary levels of epinephrine, norepinephrine and dopamine in workers exposed to high noise levels ($l_{eq} \geq 90\text{db}$) compared with the workers exposed to low-level noise ($l_{eq} \leq 75\text{db}$) (21).

Given the importance of catecholamines role in causing various diseases and the possible effects of noise on these hormones secretion, it seems the sound can stimulate the adrenal glands and increase the catecholamines secretion and in this way plays an indirect role in chain of causation of many diseases. Considering the instability of catecholamines in plasma, this study designed to determine the variability of urinary metanephrine, normetanephrine and vanilmandelic acid as the catecholamines metabolites in workers exposed to the intense noise compared with the control group in a textile factory in Yazd, Iran.

Materials and Methods

This case-control study was performed to investigate the changes in metanephrine, normetanephrine and vanilmandelic acid urinary

levels in 22 male workers who exposed to intense noise ($leq \geq 85$ dbA) compared with 22 male workers in less-noisy environments ($leq \leq 70$ dbA) in the above mentioned textile industry. For this purpose, the noise level of the factory units was measured using Bruel & kjaer 2230 sound level meter and the mill were divided into two sections: as noisy ($leq \geq 85$ db) and quiet ($leq \leq 70$ db) units.

The volunteers were enlisted in the study and the individuals with at least three year of experience, the average hearing loss less than 25 dB at 3-6KH frequencies, were included and the ones with diabetes, hypertension with family history, migraine, cardio-vascular diseases, psychological diseases and smokers were identified using a self-reported check list and those were = used drugs such as phenothiazines and levodopa were excluded from the study.

Obtaining consent from participants, their demographic information was collected by a self-report questionnaire. Approximately most workers did not use earplug. Therefore the situation without ear protectors was the normal work situation. After learning how to properly collect urine samples, participants were asked not to use interventions, such as vanilla, banana and coffee up to the sampling day and as well to avoid heavy exercises.

The first urine sample was collected before the first workday after holidays to determine the basal level of urinary catecholamines. The subjects were asked to void their bladder before sleeping at 23:00 and to collect any urine arising during the night and morning into containers. The containers were chemically prepared to prevent decomposition of the metabolites acidifying with Acetic acid. The same procedure was done on the same workday and any urine arising during the dayshift work was collected into the second

containers. Samples were collected and transported to the laboratory in cool conditions, avoiding heat and sunlight and stored at -20°C until analysis.

Metanephrine and normetanephrine samples were analyzed, using ELISA method (BA10-0500 and BA E85000 Test Kits, LDN CO-Germany) and the levels were measured using ELISA reader (Model DANA3200, Novin Gostar CO, Iran) as $\mu\text{g}/8\text{h}$. Vanilmandelic acid samples were analyzed, using chromatography method (COD11003 test kit, Bio systems CO, Spain) and levels were measured using photometer (Model Clinic II, Tajhiz Sanjesh CO, Iran) as $\text{mg}/8\text{h}$.

Data normalities were tested with Kolmogorov-Smirnov test, using SPSS version 16 and means were compared using Mann-Whitney and Wilcoxon tests. The differences of p value < 0.05 were considered significant.

Results

Demographic characteristics of case and control groups are shown in Table 1. Data were compared using Independent Samples Test. As presented no significant differences was observed in height, weight, body mass index and age between groups. Measuring sound levels of factory and the units with sound level above 85 dbA was defined as noisy and units with noise level lower than 70dbA was defined as quite units.

The mean and SD of pre / post shifts levels of vanilmandelic acid, metanephrine and normetanephrine were shown in Table (2). The comparison results between the case and control groups show that the mean levels of all three metabolites in the case workers, is significantly higher than the control group (P-value < 0.05).

The mean levels of vanilmandelic acid, metanephrine and normetanephrine after

Table 1. The demographic characteristics of case and control groups and statistical comparison between groups

	Job experience (year)		Body mass index		Weight (kg)		Height (cm)		Age (years)	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
Case (N=22)	11.3	7/3	24.2	3/6	70.8	12/2	170.0	5/54	32.4	7.7
Control (N=22)	12.68	6/7	25.81	3/7	75.0	13/07	170.4	7/2	36.2	8.5
P-value	0.62		0.177		0.322		0.473		0.86	

SD= standard deviation

Table 2. Pre and after shift levels of metanephrine, normetanephrine and vanilmandelic acid in case and control groups and statistical comparison between groups

	Vanilmandelic acid (mg/8h)				normetanephrine (µg/8h)				metanephrine (µg/8h)			
	After shift		Pre-shift		After shift		Pre-shift		After shift		Pre-shift	
	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD	mean	SD
Case (N=22)	5.5	1.9	3.7	1.4	65.36	14.4	59.23	7.9	71.5	8.4	66.7	4.2
Control (N=22)	2.5	1.1	2.4	1.06	46.24	13.00	41.07	4.6	37.85	4.47	37.16	3.5
P-value	0/00		0/005		0/00		0/00		0/00		0/00	

SD= standard deviation

exposure to the noise were increased significantly in the noise group (P-value<0.05) while in the control group, significant increase observed only in normetanephrine level after than before shift (P-value<0.05) but the levels of vanilmandelic acid and metanephrine in this group was not significantly changed.

Discussion

Many studies have been conducted in relation to noise effects on hearing loss, blood pressure, heart disease, cardiovascular system and other diseases. But few studies have investigated the direct effect of noise on epinephrine and norepinephrine hormones level and especially their metabolites and so indirect role of these hormones on non-auditory effects of noise.

In Iran this topic has not been done and if done, its results have not been published yet. Also we have not found any study that examined the effect of noise on all three metabolites. As for the important role of

catecholamines in noise related diseases this study has been designed.

The main limitations of this study, includes effects of lifestyle, dietary habits and non-voice stressors on hormones levels and possibility of samples losing during collection and transportation to the lab.

Regarding the collecting of 8-hour urine samples from workers, the reported levels were as µg/8h for metanephrine and normetanephrine and as mg/8h for vanilmandelic acid. Standard levels of these metabolites are reported as µg/day for metanephrine and normetanephrine (respectively 74 - 297 and 105 - 354 µg/day) and mg/day for vanilmandelic acid as 1.4 – 6.5. Also the Similar studies have not been reported the levels of hormone that were investigated. Thus comparison of results with the standard was not allowed.

The results of this study indicate that the vanilmandelic acid, metanephrine and normetanephrine pre-shift levels in workers in

noise group were significantly higher than the control group (29.54 units ($\mu\text{g}/8\text{h}$) in metanephrine, 12.1 units ($\mu\text{g}/8\text{h}$) in normetanephrine and 1.3 units ($\text{mg}/8\text{h}$) in vanilmandelic acid levels). These results are consistent with Ising and Sudo's findings.

Accordingly, we can say that the long-term exposure to the noise can chronically increase the levels of stress hormones secretion. Also, due to increasing the levels of vanilmandelic acid, metanephrine and normetanephrine in end of shift compared to pre-shift (4.8 units ($\mu\text{g}/8\text{h}$) in metanephrine, 6.31 units ($\mu\text{g}/8\text{h}$) in normetanephrine and 1.8 units $\text{mg}/8\text{h}$ in vanilmandelic acid levels), it can be stated that short-term exposure to the noise can lead to the increasing in levels of stress hormones.

Conclusion

Given these results, we can conclude that the workplace noise exposure acts as a stressor, stimulates the adrenal glands and leads to fight-flight reaction in human beings, increasing levels of catecholamines, and subsequently increases the risk of cardiovascular disease, hypertension and other stress-related disorders. So it is recommended that periodic examinations of workers exposed to noise, should include in addition to audiometry exams, these hormone level as a biological indicator of long-term exposure to noise.

Conflict of Interest

The authors declare no conflict of interest.

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