Effect of Acute Noise Exposure on Salivary Cortisol: A Randomized Controlled Trial

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Abstract- Cardiovascular adverse effects are interesting aspects of occupational noise exposure. One possible mechanism of these effects is an alternation in hypothalamic-pituitary-adrenal axis. Our aim was to measure salivary cortisol response to relatively high-intensity noise exposure in a controlled randomized trial study. We exposed 50 male volunteers to 90 dBA noise for 20 minutes and compared their level of salivary cortisol with 50 non-exposed controls. Salivary samples obtained before and after exposure. Before intervention means (SD) salivary cortisol level were 3.24 $(0.47)^{ng/ml}$ and 3.25 $(0.41)^{ng/ml}$ for exposed and non-exposed groups respectively. Mean salivary cortisol level increased to 4.17 ng/ml after intervention in exposure group. This increment was statistically significant (P=0.00). Mean salivary cortisol level of the non-exposed group had statistically non-significant decrement after this period (0.2 ng/ml). The difference between salivary cortisol level of non-exposed and exposed groups after the intervention was statistically significant. Noise exposure may affect the hypothalamic-pituitary-adrenal axis activity, and this may be one of the mechanisms of noise exposure cardiovascular effects.

Introduction

Hearing loss and heart-related problems are significant adverse health effects of noise exposure (1). Cardiovascular effects of noise exposure can be ranged from increased heart rate, systolic and diastolic blood pressure to increase the risk of myocardial infarction (2-5). At present, regardless of some negative studies (6-7), there are significant evidence in agreeing with the existence of noise-induced cardiovascular effects, but some remaining questions are a magnitude, modifying factors and mechanisms of these effects (8). Previous studies indicated that lower intensity of noise can affect the cardiovascular system if subjects have activities such as conversation, concentration, or recreation (9).

Many studies related to cardiovascular effects of noise have been conducted on subjects during sleep or activities with the need to concentrate on a mental task. So these studies indirectly have supported noise annoyance hypothesis of noise-induced cardiovascular effects (8,10-11). Some other studies have found different findings. Y. Aydin and M. Kaltenbach didn't find a correlation between subjective noise perception and blood pressure indices in one subgroup of their study (12).

Possible involved mechanism of noise-induced cardiovascular problems is alternation in hypothalamic-pituitary-adrenal (HPA) axis (13-15). Measurement of cortisol as a steroid end-product of HPA axis may be used for studying cardiovascular response to noise-induced stress (16). So many studies used the measurement of stress hormones including cortisol to evaluate noise induced cardiovascular effects (17-21). Plasma free cortisol is the active component of blood cortisol that needs invasive sampling. But salivary cortisol is a reliable indicator of plasma free cortisol with non-invasive sampling and convenient laboratory analytical method (22-27).

Our aim was to measure salivary cortisol response to relatively high-intensity noise exposure in a controlled randomized trial.

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Materials and Methods

In a randomized controlled, single-blind trial, we invited middle-aged (20-40 years), healthy male volunteers to participate in our study. The study design and intervention protocol were approved by ethical committee of Tehran University of Medical Sciences. All participants underwent a physical examination, and medical history was taken. Subjects with any complaint related to hearing, history of an ear problem, hypertension or any other cardiovascular diseases, diabetes mellitus, oral diseases, using any drug that can alter corticosteroid secretion and shift workers, detected by medical history or physical examination, were excluded. 109 volunteer assessed for eligibility, 7 of them excluded for not meeting inclusion criteria, and 2 of them declined to participate in the study. Finally, 100 paid, middle-aged (20-40 years), healthy male have entered the study. All participants filled and signed written informed consent.

Participants abstained from any vigorous physical activity during 24 hours before sampling, previous night bad sleep, alcohol consumption during the test day and eating, drinking, smoking, chewing gums or brushing teeth within 30 minutes before sampling. Participants were divided into two equal groups using balanced blocked randomization. Each group consists of 50 participants. The experimental sessions commenced between 2:30 and 4:30 PM lasted for approximately 90 minutes. In each session, two blokes of participants consisting of one exposed block and one control block were tested. The study was conducted in an isolated comfortable room. All subjects rested in sitting position without physical activity for 10 minutes before baseline saliva sampling. At the end of this period, blood pressure and heart rate were measured. To measure cortisol level alternation in response to stress and to avoid any possible pitfalls, three milliliters of un-stimulated total saliva collected via passive drooling into the centrifuge glass tube (28-29). After centrifuge, saliva samples stored at -20° C. After saliva sampling, exposure group was exposed to 90 dB recorded industrial noise for 20 minutes. Then, they had 10 minutes rest without exposure prior to second saliva sampling.

This timing of measurements and exposure was based on observed salivary cortisol alternation in response to stress in previous studies (29-32). The control group had the same experience but without noise exposure and they had 30 minutes rest between two saliva samplings (Figure 1). It was impractical to design a placebo for the non-exposed group. So they were not blind, but members of the research team were blind to samples. For determination of salivary cortisol levels, samples assayed by ELISA method using *DIAMETRA (Italy) DKO020 Cortisol Saliva ELISA* kits with 0.12 ng/ml sensitivity and 0.5–100 analytical range. The significance level was set at the 0.05.
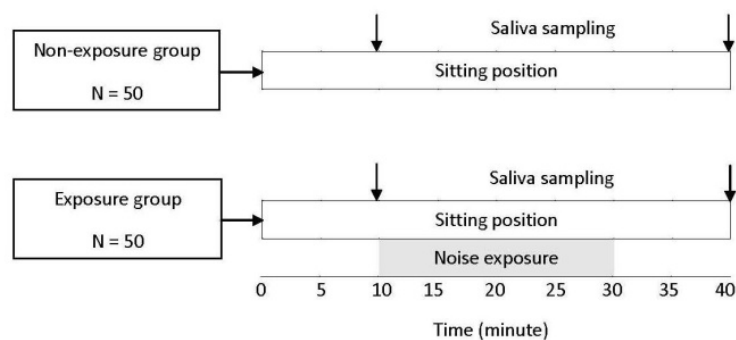

Figure 1. Timing of noise exposure and saliva sampling in exposure and non-exposure groups (↓time of saliva sampling)

Results

Mean (SD) age of exposed and non-exposed participants were 30.64 (6.1) and 30.56 (6.5) years respectively. The mean heart rate of exposure group before the study was 74.5 bit per minute and this measure for the non-exposed group was 74.2, and there was not any significant difference between two groups. Also, there was not any significant difference between diastolic and systolic blood pressure of two groups before intervention (Table 1).

Table 1. Systolic and diastolic blood pressure of subjects before intervention

	Mean (SD)		P.value
	Exposure group (N=50)	Non-exposed group (N=50)	
SBP (mmHg)	117.7 (5.1)	118.6 (5.2)	0.38
DBP (mmHg)	75.5 (4.9)	77.2 (4.3)	0.07

Using Kolmogrov-Smirnov Test, distribution of salivary cortisol level for both exposed and non-exposed groups before and after the intervention was normal. Mean (SD) of salivary cortisol level before intervention for exposure group was 3.24 (0.47)$^{ng/ml}$ and for the non-exposed group was 3.25 (0.41)$^{ng/ml}$. Using t-test, there was not any statistically significant difference between mean salivary cortisol levels of groups before intervention (*P*=0.92). Mean (SD) salivary cortisol level of exposure group after intervention raised to 4.17 (1.66)$^{ng/ml}$ and there was 0.93$^{ng/ml}$ increment in average. This increment was statistically significant (*P*=0.00 CI95% =0.44-1.43). Mean (SD) salivary cortisol level of the non-exposed group after this period (without noise exposure) decreased to 3.05 (0.99)$^{ng/ml}$, and there was 0.2$^{ng/ml}$ decrement in average. This decrement was not statistically significant (*P*=0.11 CI95%= -0.45-0.05). We also compared mean salivary cortisol level of exposed and non-exposed groups after the intervention, using independent sample t-test (Table 2).

Table 2. Salivary cortisol level comparison between two groups after intervention

	Mean (SD)	Difference of mean (ng/ml)	P.value
Exposure group (N=50)	4.17 (1.66)	1.12 (CI 95%= 0.58–1.66)	0.00
Non-exposure group (N=50)	3.05 (0.99)		

Discussion

We found statistically significant difference between salivary cortisol levels in exposed and non-exposed groups after intervention. Comparison of mean age, heart rate, blood pressure and salivary cortisol level of two groups before intervention showed that two groups were similar and the finding of study cannot be explained by the difference between two groups before the intervention, indicating the activation of the hypothalamic-pituitary-adrenal axis by noise exposure. Our finding is consistent with the study of Jasmin Wagner *et al.*, (23). They exposed twenty participants to binaurally recorded 75 dB (LA,eq) traffic noise for 20 minutes without a control group. They collected saliva samples immediately before and after exposure and found the statistically significant rise of salivary cortisol after exposure to noise. Salivary cortisol increment in their study was lower than our study.

It may be due to higher noise exposure in our study, the timing of sampling or other difference between settings of two studies. Our result also is consistent with, but not similar to, the study of Batmanabane Gitanjali and Ramachandran Ananth (20). They found statistically significant elevation of serum cortisol in the morning after exposure to noise (20). Jenny Selander *et al.*, find statistically significant salivary cortisol elevation in noise-exposed female subgroup participants of their study population (18). Contrary to expectation, their study did not find any significant difference between salivary cortisol level of exposed and non-exposed male subgroup.

A possible explanation for this might be that they calculated 24 h noise exposure using environmental noise map and they did not consider occupational noise exposure in their noise exposure calculation which could be a most important source of noise exposure in men.

Limitation of this study is that our study has only examined reaction to "brief" "high intensity" noise exposure in "resting" position. So our result should be generalized with caution to occupational and environmental settings. However, our study provides some support for non-auditory adverse effects of noise exposure. Alternation of the hypothalamic-pituitary-

adrenal axis activity by noise exposure may contribute to hypertension and other adverse cardiovascular effects of occupational and environmental noise exposure.

Acknowledgment

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