Pol. J. Environ. Stud. Vol. 22, No. 5 (2013), 1547-1551

Short Communication

Occupational Noise Exposure May Induce Oxidative DNA Damage

Syed Kashif Nawaz[1, 2]*, Shahida Hasnain[2, 3]

[1]Department of Biological Sciences, University of Sargodha, Sargodha, Pakistan
[2]Department of Microbiology and Molecular Genetics, University of the Punjab, Lahore, Pakistan
[3]School of Biological Sciences, University of the Punjab, Lahore, Pakistan

Received: 17 January 2012
Accepted: 21 November 2012

Abstract

Occupational noise enhances the risk of several cardiovascular and non-cardiovascular diseases. The reason behind the drastic effects of noise exposure is the excessive generation of reactive oxygen species molecules. The present study investigates oxidative stress due to occupational noise exposure in 192 male smoking workers exposed to normal noise (sound level ≤80 dBA), low noise (sound level 81-94 dBA), and loud noise (sound level ≥95 dBA). University-aged volunteers were selected for the normal sound exposed group. Drivers and conductors were selected for the low-noise exposed group, and workers of power loom factories were chosen for the high-noise exposed group. Oxidative stress was estimated using 8 OHdG as a biomarker for oxidative DNA damage. Serum aldosterone and serum cortisol level was estimated using the enzyme immunoassay method. Results indicated that 8 OHdG level was significantly different in different exposure groups. It was highest in the low-noise exposed group (0.370±0.017 ng/ml) and lowest in the normal-sound exposed group (0.22±0.01 ng/ml). Level of 8 OHdG in the high-noise exposed group was 0.29±0.00. There was no significant variation of aldosterone levels among different groups. Cortisol levels of both noise groups was higher than that of the normal sound group. It can be concluded that noise exposure induces stress in the Pakistani population. This stress leads to oxidative DNA damage.

Keywords: 8 OHdG, oxidative DNA damage, noise-induced oxidative stress, aldosterone serum, cortisol serum

Introduction

Some occupations involve noise exposure for longer duration for workers. Excessive noise exposure may enhance the risk of high blood pressure, myocardial infarction, and coronary artery disease [1-4]. About 600 million workers face occupational noise worldwide [1]. The importance of genetic factors in raising the chances of noise-induced health problems have been realized [3]. In addition to genetic factors, other factors may affect the drastic outcomes of noise exposure. For pointing out those factors, it will be essential to understand the biochemical basis of noise induced health damage. Current knowledge of this field reveals the importance of reactive oxygen species (ROS) [5, 6]. ROS production happens due to physiological processes of normal aerobic living cells. These have a critical role in normal body functions. Nevertheless, their excessive production may cause oxidative damage to proteins, DNA, and lipids of cellular membranes [7]. Decreased catalase activity of growing lymphocytes was observed due to noise exposure [8].

Effects of noise not only depend on oxidative stress but some genetic variations may also play their role. For example, ACE ID and ACE G2350A polymorphism was

*e-mail: kashifnawazshabbir@yahoo.com

observed to modulate the association of noise with high blood pressure [2]. Nevertheless, no association of ACE polymorphism with increased blood pressure in the local population of Pakistan was observed in either case of noise exposure [9]. Several exogenous factors are known to increase the generation of ROS. These include exposure of UV, gamma radiation, tobacco smoke, benzene, styrene, asbestos, silica, chromium, arsenic, cobalt and polycyclic aromatic hydrocarbons [10]. There exists a balance between endogenous oxidants and numerous antioxidant mechanisms in normal physiological condition. When imbalance happens, oxidants cause extensive oxidative damage. In the case of DNA damage, mechanisms involving different enzymes play their role for DNA repair for normal cell functioning. Failure of DNA repair may result in mutations leading to the aging, carcinogenesis, shock, ischemia, reperfusion, or injury [11, 12]. Oxidative DNA damage has extensively been studied using 8-hydroxy deoxyguanosine (8OHdG) as an oxidative DNA damage biomarker. This molecule is produced due to the interaction of hydroxyl radicals with guanine (nucleobase of DNA). On the basis of increased 8 OHdG level, Van-Campen et al. [13] observed noise-induced oxidative DNA damage in rats. Noise-induced DNA damage in human beings has not been studied in detail. In the present study, it was investigated if the noise exposure of 80 dB causes the excessive production and release of ROS molecules leading to oxidative DNA damage. 8-hydroxy-2-deoxyguanosine (8OHdG) was used as a biomarker of oxidative DNA damage. Blood from workers and volunteers (exposed to specific sound level ranges) were used as a sample for the estimation of oxidative DNA damage. The effect of noise exposure on serum aldosterone and cortisol level also was analyzed.

Materials and Methods

Study Sample

All procedures were in compliance with the declaration of Helsinki and the study protocol was approved by the university ethics committee of Punjab Lahore, Pakistan. Occupational sites and workers residing in these sites for eight hours per day and seven days per week were selected as already described [2]. Initially 389 samples were selected. Out of these 389 samples, workers with similar exposure duration and smoking habits were finally selected for the estimation of 8OHdG level. Samples showing positive results for HIV, HCV, HBSAg, diabetes, and deafness were excluded from the study. The subjects were divided in three groups on the basis of noise level at their work site; normal sound exposed group: Volunteers exposed to ≤80 dBA; low-noise exposed group: workers exposed to 81-94 dBA; high-noise exposed group: workers exposed to ≥95 dBA. The normal-sound exposed group consisted of volunteers from university and colleges. The low-noise exposed group consisted of drivers and conductors. The high-noise exposed group consisted of workers in power loom factories.

Detection of 8 OHdG

DNA of each sample was extracted by using Fermentas kit (#K0512) according to manufacturer's protocols. Pretreatment of the samples and estimation of 8 OHdG was performed according to manufacturer's protocol for highly sensitive ELISA kit (JaICA, KOGHS 040914E).

Detection of Cortisol Serum and Aldosterone Serum

Quantitative determination of cortisol in serum was performed using enzyme immunoassay as per instructions of manufacturer (NOVATEC, Product No. DNOV001). Serum aldosterone level was estimated using enzyme immunoassay as per manufacturer instructions (NOVATEC, Product No. DNOV012).

Statistical Analysis

Levels of 8 OHdG, serum aldosterone level, age, and body mass indices were compared using Duncan's test. Matrix plots were explained in terms of Pearson's correlation.

Results

A total of 192 samples were collected on the basis of inclusion criteria. Forty-six samples belonged to the normal sound group, 69 samples were from the low-noise group and 77 samples were from the high noise group. Baseline characteristics of the groups are shown in Table 1. Comparison of age of different groups indicates that the low-noise group had significantly higher values as compared to the other groups (normal sound and high noise). Weight, height, and body mass index of low noise groups were also higher in the low noise group as compared to other groups. Considering these differences, the estimations were made after adjusting data for the confounding variables accordingly.

8OHdG concentration was highest in the low-noise group and lowest in the normal sound group. The concentration of 8OHdG in high noise group was lower than that of the high-noise group but higher than that of the normal sound group. Serum aldosterone level was not significantly different in all the groups. Serum cortisol level of both noise (low and high) groups was higher than that of the normal sound group. Fig. 1 shows the matrix plot for different variables. A significant relationship was seen in the case of age vs 8OHdG and age vs. serum aldosterone. The linear relationship between weight vs. height and weight vs. cortisol was also recorded as significant. Height and 8OHdG showed the relationship with change in cortisol level.

Discussion

The present research was conducted to find epidemiological evidence showing oxidative DNA damage due to

Table 1. Characteristics of groups.

	Normal Sound (46)	Low noise (69)	High noise (77)
Age	38.35±0.45[b]	42.42±0.41[a]	38.99±0.47[b]
Weight	77.67±1.74[b]	82.45±1.42[a]	64.62±1.59[c]
Height	1.63±0.01[b]	1.67±0.01[a]	1.59±0.01[c]
Body mass index	29.17±0.49[a]	29.74±0.48[a]	25.49±0.55[b]
8 OHdG Concentration	0.22±0.01[c]	0.37±0.01[a]	0.29±0.00[b]
Serum aldosterone level (pg/ml)	55.65±2.54[a]	49.57±1.83[a]	63.77±8.01[a]
Serum cortisol (ng/ml)	25.39±1.87[b]	35.16±1.86[a]	38.95±1.67[a]

(Mean ± S.E.M)
c<b<a ($p<0.01$)

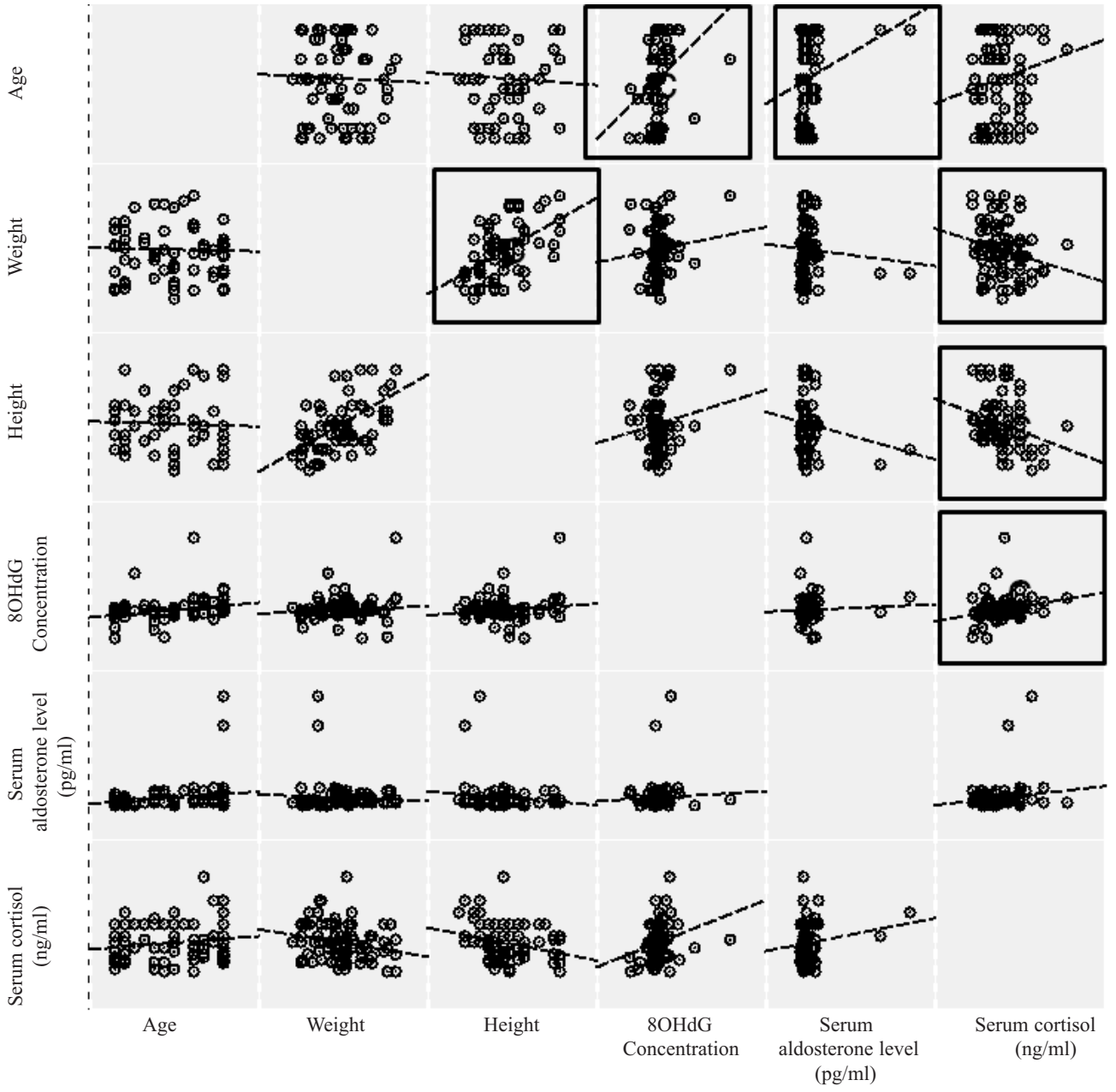

Fig. 1. Matrix plots for different variables.

noise in the different groups defined on the basis of noise exposure. The present study shows the positive correlation between age and 8OHdG level. The reason for this relationship might be explained in terms of accumulation of ROS in the aging body. Increased levels of 8OHdG in noise-exposed groups as compared to that of normal sound group indicates the role of noise in raising oxidative stress. Similar oxidative stress due to noise was detected in textile workers of Turkey [5]. Comparison of 8OHdG between low noise and high noise groups showed that 8OHdG was higher in the low-noise group as compared to that of the high-noise group. The reason for this difference may be described in terms of different exposure conditions of each group. The low-noise exposed group was composed of drivers and conductors. They were not only exposed to noise but also exposed to air pollution (benzene and other polyaromatic hydrocarbons) [14]. The additive effects of noise and air pollution might have increased the oxidative DNA damage. In the case of the high-noise group, all the samples were power loom workers. They were exposed only to extreme noise pollution. Therefore, oxidative DNA damage level was low in them as compared to the low-noise group.

The level of serum cortisol increased in noise-exposed workers whereas no significant variation was seen for the levels of aldosterone. Our findings are in agreement with the information presented by Fouladi et al., which shows the variation of cortisol levels due to noise exposure in blue-collar industrial workers [15]. Similar findings were reported by Sjödin et al. while studying the stressful effects of noise on preschool employees [16]. Positive correlation of cortisol with that of 8OHdG indicates the relationship between these two different stress markers. Recently Joergensen and coworkers also have reported similar observations while studying the phenomenon of aging [17]. According to them, cortisol might be the reason to increase oxidative DNA damage in aging cells. Cortisol level was having negative correlation with that of weight and height. That means a higher level of cortisol is responsible for lower values of weight or height. In this way, the present finding supports the hypothesis that cortisol and body mass index are inversely related. Similar observations were recorded by Daniel et al. while studying obesity in blue-collar women [18].

An increased level of aldosterone is found to be associated with the increased risk of renal complications, inflammation, and oxidative stress. Fibrosis in a number of tissues and metabolic syndrome are also related to elevated levels of aldosterone. Increased risk of cardiovascular diseases, including hypertension, directly and indirectly to elevated levels of aldosterone by increasing the risk of obesity and renal complications have also been reported by several research groups [19-21]. Positive correlation was observed between age and serum aldosterone level. This indicated that samples having higher age were having higher values of serum aldosterone level, which indirectly indicates higher chances of cardiovascular diseases [20].

The strength of the present study may be defined in terms of the use of occupational noise exposure, which remains stable through the whole working day. Selection of such working sites grants maximum possibility of getting the workers exposed with defined sound level. Additionally, the selection of age matched and similar smoking habits of samples also helped in avoiding potential confounding effects.

Conclusions

The noise exposure induces stress in workers exposed to noise. The impact of this stress causes oxidative DNA damage. A strong relationship between 8OHdG and cortisol points toward the role of cortisol in influencing the level of 8OHdG.

Acknowledgements

The present research work was completed using funds of indigenous scholarship awarded by the Higher Education Commission (HEC) of Pakistan for Syed Kashif Nawaz during his Ph.D. We are thankful to the HEC for providing this financial support.

References

1. ALBERTI P. W. Noise-the most ubiquitous pollutant. Noise Health **1**, 3, **1998**.
2. NAWAZ S.K., HASNAIN S. Noise induced hypertension and prehypertension in Pakistan. Bosnian Journal of Basic Medical Sciences, **10**, 239, **2010**.
3. NAWAZ S.K., HASNAIN S. Effect of ACE polymorphisms on the association between noise and hypertension in Pakistani population. Journal of the Renin-Angiotensin-Aldosterone System, **12**, (4), 516, **2011**.
4. DAVIES H.W., TESCHKE K., KENNEDY S.M., HODGSON M.R., HERTZMAN C., DEMERS P.A. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology, **16**, 25, **2005**.
5. YILDIRIM I., KILINC M., OKUR E., INANC TOLUN F., KILIÇ M.A., KURUTAS E.B., EKERBIÇER H.C. The effects of noise on hearing and oxidative stress in textile workers. Ind. Health, **45**, 743, **2007**.
6. FETONI A.R., GARZARO M., RALLI M., LANDOLFO V., SENSINI M., PECORARI G., MORDENTE A., PALUDETTI G., GIORDANO C. The monitoring role of otoacoustic emissions and oxidative stress markers in the protective effects of antioxidant administration in noise-exposed subjects: a pilot study. Med Sci Monit, **15**, PR1-8, **2009**.
7. SAMSON J., SHEELADEVI R., RAVINDRAN R. Oxidative stress in brain and antioxidant activity of Ocimum sanctum in noise exposure. Neurotoxicology, **28**, 679, **2007**.
8. NAWAZ S.K., HASNAIN S. Effects of noise exposure on catalase activity of growing lymphocytes. Bosnian Journal of Basic Medical Sciences, **11**, 219, **2011**.
9. NAWAZ S.K., HASNAIN S. Association of ACE ID and ACE G2350A polymorphism with increased blood pressure in persons exposed to different sound levels in Pakistan. Int. Arch. Occ. Env. Hea., **84**, 355, **2011**.

10. PILGER A., RÜDIGER H.W. 8-Hydroxy-2'-deoxyguanosine as a marker of oxidative DNA damage related to occupational and environmental exposures. Int. Arch. Occ. Env. Hea., **80**, 1, **2006**.
11. VALAVANIDIS A., VLACHOGIANNI T., FIOTAKIS C. 8-hydroxy-2' -deoxyguanosine (8-OHdG): A critical biomarker of oxidative stress and carcinogenesis. J. Environ. Sci. Heal. C., **27**, 120, **2009**.
12. CHERUBINI A., RUGGIERO C., POLIDORI M.C., MECOCCI P. Potential markers of oxidative stress in stroke. Free Radical Bio. Med., **39**, 841, **2005**.
13. VAN-CAMPEN L.E., MURPHY W.J., FRANKS J.R., MATHIAS P.I., TORAASON M.A. Oxidative DNA damage is associated with intense noise exposure in the rat. Hearing Res., **164**, 29, **2002**.
14. ARAYASIRI M., MAHIDOL C., NAVASUMRIT P., AUTRUP H., RUCHIRAWAT M. Biomonitoring of benzene and 1,3-butadiene exposure and early biological effects in traffic policemen. Sci. Total Environ, **408**, (20), 4855, **2010**.
15. FOULADI D.B., NASSIRI P., MONAZZAM E.M., FARAHANI S., HASSANZADEH G., HOSEINI M. Industrial noise exposure and salivary cortisol in blue collar industrial workers. Noise Health, **14**, 184, **2012**.
16. SJÖDIN F., KJELLBERG A., KNUTSSON A., LANDSTRÖM U., LINDBERG L. Noise and stress effects on preschool personnel. Noise Health, **14**, 166, **2012**.
17. JOERGENSEN A., BROEDBAEK K., WEIMANN A., SEMBA R.D., FERRUCCI L., JOERGENSEN M.B., POULSEN H.E. Association between urinary excretion of cortisol and markers of oxidatively damaged DNA and RNA in humans. PLoS One, **6**, e20795, **2011**.
18. DANIEL M., MOORE D.S., DECKER S., BELTON L., DEVELLIS B., DOOLEN A., CAMPBELL M.K. Associations among education, cortisol rhythm, and BMI in blue-collar women. Obesity (Silver Spring), **14**, 327, **2006**.
19. VYSSOULIS G.P., KARPANOU E.A., TZAMOU V.E., KYVELOU S.M., MICHAELIDIS A.P., GIALERNIOS T.P., COKKINOS D.V., STEFANADIS C.I. Aldosterone levels and stroke incidence in essential hypertensive patients. Int. J. Cardiol., **144**, (1), 171, **2009**.
20. VOGT B., BURNIER M. Aldosterone and cardiovascular risk. Current Hypertension Reports, **11**, 450, **2009**.
21. KOTLYAR E., VITA J.A., WINTER M.R., AWTRY E.H., SIWIK D.A., KEANEY J.F., JR-SAWYER D.B., CUPPLES L.A., COLUCCI W.S. SAM F. The relationship between aldosterone, oxidative stress, and inflammation in chronic, stable human heart failure. J. Card. Fail., **12**, 122, **2006**.