Recurrent Indoor Environmental Pollution and Its Impact on Health and Oxidative Stress of the Textile Workers in Bangladesh

Environmental Health Insights Volume 14: 1-7 © The Author(s) 2020 Article reuse guidelines: sagepub.com/journals-permissions DOI: 10.1177/1178630220938393 (S)SAGE

Tania Rahman¹, Ar-Rafi Md. Faisal¹, Tahura Khanam² and Hossain Uddin Shekhar¹

¹Department of Biochemistry and Molecular Biology, Faculty of Biological Sciences, University of Dhaka, Dhaka, Bangladesh. ²Qtex Solutions Limited, 1st ISO 17020:2012 Accredited Environmental inspection body in Bangladesh.

ABSTRACT: Perennial indoor environmental pollution in the textile industrial area is a potential health hazard for workers engaged in this line of work, resulting in mental aberration to severe health risks. This study was designed to investigate the indoor environmental quality of textile industries and correlate its effect on the occupational health and well-being of the textile workers by measuring plasma oxidative stress status in textile workers and healthy control subjects. Environmental samples were collected from 15 textile industries located in Dhaka division, and 30 volunteer textile workers and 30 volunteer office workers (control) aged 18 to 57 years participated in the study. The concentration of plasma ascorbic acid (P-ASC), plasma malondialdehyde (P-MDA), and plasma conjugated diene (P-CD) was measured in both groups. The noise level (78.0 \pm 0.68 dB) and the formal dehyde level (141.80 \pm 4.47 μ g/m³) were found to be significantly higher in the indoor environmental area compared with those in the control area (70.17 ± 0.25 dB and 108.0 ± 0.76 µg/m³, respectively). Furthermore, the daily average concentration of suspended particulate matters (PMs), that is, $PM_{2.5}$ (322.2 ± 13.46 µg/m³) and PM_{10} (411.0 ± 17.57 µg/m³), was also found to be significantly higher in the indoor environmental air compared with that in the control area (78.59 ± 1.66 and $174.0 \pm 2.33 \mu g/m^3$, respectively). The levels of P-MDA (0.37 ± 0.03 nmol/L) and P-CD (14.74 ± 0.61 nmol/L) were significantly increased, whereas the level of P-ASC level (0.46 ± 0.04 mg/dL) was markedly decreased in the textile workers compared with the healthy control subjects (0.18±0.01 nmol/L of P-MDA, 10.04±0.44 nmol/L of P-CD, and 1.29 ± 0.06 mg/dL of P-ASC). The textile plants were found to have significantly elevated levels of indoor environmental pollutants compared with those in the control area, and the textile workers were significantly exposed to oxidative stresses compared with the control subjects. The use of noise pads and high-efficiency air filters is perhaps highly instrumental to put an end to this prevailing situation. Moreover, to overcome the oxidative stresses among workers, supplementation of antioxidant vitamins (ie, ascorbic acid and/or vitamin E) may be beneficial. In addition, to prevent serious health-related issues, proper precautions should be taken to protect the occupational health of the textile workers.

KEYWORDS: Indoor environmental pollution, textile worker, occupational health, oxidative stress

RECEIVED: January 27, 2020. ACCEPTED: June 2, 2020.

TYPE: Original Research

FUNDING: The author(s) received no financial support for the research, authorship, and/or publication of this article

DECLARATION OF CONFLICTING INTERESTS: The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this

CORRESPONDING AUTHOR: Hossain Uddin Shekhar, Department of Biochemistry and Molecular Biology, Faculty of Biological Sciences, University of Dhaka, Dhaka 1000, Bangladesh. Email: hossainshekhar@du.ac.bd

Introduction

The textile industry in Bangladesh plays an increasingly important role in the economic development of the country by exporting apparels to the United States and European countries. Although the industry is the most important contributor to the national reserve, it is associated with a wide range of environmental stressors including air pollutants and noise in the working environment. Approximately 10 million people are engaged in the textile (including garments and others) industry, where around 90% of workers are women and children.¹ Hence, it is imperative to maintain a congenial and safe working environment in textile industries. In the textile industry, different types of instruments and chemicals are used to increase the product quality seemingly, which has essentially many disadvantages. Old machineries/malfunctioned instruments often create exceedingly high level of noise and get involved in air pollution. High levels of formaldehyde, carbon monoxide, carbon dioxide, and suspended particulate matters (PMs) are other common yet dangerous contaminants prevalent in their working place.²

Noise is considered the most widely prompted physical harmful factor in the workplace as well as in the environment.³ It is accounted that more than 600 million people are exposed to high sound level existing in their workplaces, among which 50 to 60 million are from Europe and North America.^{4,5} The World Health Organization (WHO) has declared the optimum level of noise as 45 dB by day and 35 dB by night to maintain a healthy environment.⁶ Any sound level that exceeds these levels is marked as noise which has serious impact on human health.⁷⁻⁹ The harmful effect of high sound/noise has been recognized as occupational stress particularly among industrial workers, inducing physiological and mental aberration.⁷ Exposure to any kind of noise above 90 dB is recognized as a great source of oxidative stress.¹⁰

Formaldehyde, a chemical compound which acts as a carcinogen, is found in some indoor air.¹¹ Formaldehyde unconstrained from external sources enters body via either inhalation or ingestion of substances.12 Long-term exposure of formaldehyde has been shown to decrease white blood cell number and possibly lower platelet and hemoglobin counts.13

 $(\mathbf{\hat{n}})$

Creative Commons Non Commercial CC BY-NC: This article is distributed under the terms of the Creative Commons Attribution-NonCommercial 4.0 License (https://creativecommons.org/licenses/by-nc/4.0/) which permits non-commercial use, reproduction and distribution of the work without further permission provided the original work is attributed as specified on the SAGE and Open Access pages (https://us.sagepub.com/en-us/nam/open-access-at-sage). Particulate matter, a key component of polluted air, is estimated to kill more than 500000 people each year.¹⁴ Worldwide, epidemiological studies showed a consistent increase in cardiac and respiratory morbidity and mortality from exposure to PM.^{15,16} Thicker fine particles (PM₁₀) are effectively removed from the upper respiratory tract; however, PM less than PM₁₀ (PM₅, PM_{2.5}, PM_{1.0}, PM_{0.5}, PM_{0.3}) cannot be efficiently removed.^{17,18} These ultrafine particles penetrate the respiratory tract and get deposited on the bronchi walls. Recent data have demonstrated that PM_{2.5} and PM_{0.1} can cross the pulmonary and systemic circulations directly, affecting heart and blood vessels.¹⁹

Occupational exposure of these environmental pollutants, such as noise and air pollutants including PM and formaldehyde, unevenly damages natural oxidative balance, resulting in disproportionate amount of free radicals and reactive oxygen species (ROS) production.²⁰ Outnumbered ROS species result in oxidative stress, triggering an imbalance in body's antioxidant level.²¹ Plasma ascorbic acid (P-ASC), nitric oxide, plasma conjugated diene (P-CD), and plasma malondialdehyde (P-MDA) are some of the oxidative stress markers usually used to assess the index of redox potential in diverse environments.^{18,22,23} Ascorbic acid (ASC), an antioxidant, has shown to play a significant role in oxidative stress management caused by ROS molecules, thereby protecting the cell against ROSinduced damage, whereas P-CD has been demonstrated as a primary product of a free-radical-mediated attack on polyunsaturated fatty acids (PUFA).^{22,24} Polyunsaturated fatty acid peroxide products often react to form MDA, a secondary product of lipid peroxidation, and are one of the frequently used biomarkers to evaluate oxidative stress.²⁵ Therefore, the study aims to shed light on the state of the textile plants in Bangladesh by monitoring the indoor environmental air quality in terms of noise and air pollutants in the working space. As a result, 15 textile industries located in different areas of Dhaka division were randomly selected as sample sites to evaluate the level of pollution. Furthermore, to correlate the effect of these environmental stressors on the occupational health and well-being of the textile workers, certain biomarkers were used to assess the oxidative stress status in their bodies.

Material and Methods

Site selection and environmental data collection

Forty-one different locations of textile plants (working area) and office areas (area of same textile industry was used as control area) of 15 textile industries located in various areas of Dhaka division were randomly chosen for the collection of environmental data such as noise level and presence of air pollutants including PM and formaldehyde in the air. Noise was measured using a sound level meter (Pulsar Models 91), whereas formaldehyde was measured using a formaldehyde meter (PCE-HFX205). Moreover, particle counter (PCE-PCO 1 machine) was used to measure the PMs in indoor air of selected textile industries.

Study subjects

A total of 30 nonsmoker textile workers (sample group) with age range between 18 and 48 years were included in the study, who had been in the profession for the last 5 years and working an average of 6 hours/day and 6 days per week. The control group (from the same area) consisted of 30 healthy nonsmoker office workers (unexposed to industrial environment), age ranging between 19 and 57 years. The ethical committee of Dhaka University approved this study, and all participants provided written informed consent before participation (20150106/ERC_biosciencedu).

Blood sample collection

For biochemical analysis, blood samples were collected from textile workers (n=30) and healthy control subjects (n=30). The blood samples were collected in clean, sterile, screw-capped tubes containing k-EDTA. The whole blood samples from each subject were centrifuged at 2000 rpm at 4°C for 10 minutes and plasma was then separated.²⁶ The plasma samples were stored at -20°C until the day of analysis.

Estimation of oxidative stress markers

Estimation of P-ASC. To measure the concentration of P-ASC, modified Roe and Kuether²⁷ described method was used. In this method, an aliquot of 0.2 mL of plasma was taken in a 5-mL test tube and 0.8 mL of 5% trichloroacetic acid (TCA) solution was added. The mixture was then centrifuged at 3000 rpm for 10 minutes. An aliquot of 0.6 mL of supernatant was separated and 0.25 mL of dinitrophenylhydrazine-thiourea-copper sulfate solution was added. The tube was then incubated at 60°C for 1 hour in water bath. After incubation, the test tube was chilled in an ice cool water bath. One milliliter of sulfuric acid was added dropwise and mixed well. The mixture was then incubated at room temperature for 30 minutes. Then, 0.2 mL of standard ASC solution (Loba Chemie, Colaba, India) was taken and treated according to the previously described method. The absorbance of both samples and standards was taken at 520 nm. The P-ASC concentration was determined using the following formula, and the result is expressed in milligram per deciliter:

$$ASC(mg/dL) = \left(\frac{Absorbance of the sample}{Absorbance of the standard}\right)$$

× Standard concentration

Determination of P-MDA

The thiobarbituric acid reactive substance (TBARS) assay, an important oxidative stress marker assay, was performed to measure the lipid peroxidation level in the textile workers and the healthy control subjects. The modified TBARS method described by Satoh was used to determine the P-MDA level.²⁸

In this method, thiobarbituric acid (TBA) reagent was prepared with 15 g of TCA, 0.375 g of TBA, 25 mL of HCl, and 40 mg of benzotriazole (BHT) and was made to a volume of 100 mL with distilled water. Then, 2 mL of TBA reagent, 1 mL of plasma sample (100 μ L plasma + 900 μ L saline), and 30 μ L of 50 mM BHT were added in a tube and the tube was kept in incubated in boiling water bath for 15 minutes. After incubation, it was kept for 10 minutes to cool down. Then, it was centrifuged for 10 minutes at 2000 rpm, and after that the supernatant was collected. The absorbance of the collected supernatant was measured at 535 nm. The MDA equivalents of the samples were calculated using an extinction coefficient of $1.56 \times 10^5 \text{ M}^{-1} \text{ cm}^{-1}$.

Determination of P-CD

The modified Spranger et al²⁹ described method was used in the determination of P-CD concentration. Plasma was treated with 3 mL of chloroform:methanol (2:1) solution followed by vigorous mixing. The mixture was then centrifuged at 2000 rpm for 10 minutes. The upper layer was discarded along with the proteins, whereas the lower chloroform layer was dried under a stream of nitrogen at 45°C. The residue obtained was dissolved in cyclohexane (Merck, Darmstadt, Germany), and the absorbance was taken at 233 nm against a blank that contains cyclohexane. Here, in this assay, 1 optical density (OD) represented 37.5 nmol of conjugated diene.

Statistical analysis

Statistical analyses were performed using analysis of variance and Student *t* test, and data were presented as mean \pm standard error of mean (SEM). GraphPad prism version 8.0.1 was used to perform the analyses, and the level of significance was assumed for difference with values of P < 0.05.

Results

Analysis of demographic data

A cross-sectional study was conducted with 30 healthy control subjects and 30 textile workers of age range 19 to 57 years and 18 to 48 years, respectively (Table 1). The mean age of textile workers and healthy control subjects was 30 ± 1.55 years and

 25 ± 1.53 years, respectively. Of a total of 30 textile workers, 5 were female and 25 were male workers. On the contrary, among 30 control subjects, 2 individuals were women. Demographic data of textile workers and healthy control subjects are shown in Table 1.

Analysis of environmental pollutant data

Results of environmental pollutants such as noise and air pollutants (PM and formaldehyde) for both working area and control area are shown in Table 2. The levels of indoor noise $(78.0 \pm 0.68 \,\text{dB})$ and formaldehyde $(141.80 \pm 4.47 \,\mu\text{g/m}^3)$ in the working area were significantly higher than those in the control area $(70.17 \pm 0.25 \,\text{dB}$ and $108.0 \pm 0.76 \,\mu\text{g/m}^3$, respectively) (Figures 1 and 2). Moreover, significant increase in the daily average concentrations of PM_{2.5} and PM₁₀ (322.2 ± 13.46 and $411.0 \pm 17.57 \,\mu\text{g/m}^3$, respectively) was observed in the indoor air of the working area compared with the daily average concentration in the control area ($78.59 \pm 1.66 \,\text{and} 174.0 \pm 2.33 \,\mu\text{g/m}^3$, respectively) (Figures 3 and 4).

Analysis of biochemical data

In this study, levels of P-MDA $(0.37 \pm 0.03 \text{ nmol /L})$ and P-CD $(14.74 \pm 0.61 \text{ nmol /L})$ were significantly increased, whereas the level of P-ASC $(0.46 \pm 0.04 \text{ mg/dL})$ was considerably decreased in the textile workers compared with the healthy control subjects $(0.18 \pm 0.01 \text{ nmol/L})$ of P-MDA, $10.04 \pm 0.44 \text{ nmol/L}$ of P-CD, and $1.29 \pm 0.06 \text{ mg/dL}$ of P-ASC) (Figures 5-7).

 Table 1. Demographic data of textile workers and healthy control subjects.

PARAMETERS	TEXTILE WORKERS (N=30)	HEALTHY CONTROL SUBJECTS (N=30)
Age range	30 ± 1.55 years 18-48 years	25 ± 1.53 years 19-57 years
Gender	Male=25 Female=5	Male=28 Female=2

Table 2. Summary of all parameters of environmental pollutants in control and working area.

PARAMETERS	CONTROL AREA (MEAN \pm SEM) N=41	WORKING AREA (MEAN \pm SEM) N=41	P VALUE	REFERENCE STANDARD AMBIENT VALUE
Noise, dB	70.17 ± 0.25	78.0 ± 0.68	<.001	70 ³⁰
Formaldehyde, $\mu g/m^3$	108.0 ± 0.76	141.80 ± 4.47	<.001	10012
$PM_{2.5}$, $\mu g/m^3$ per day	78.59 ± 1.66	322.2 ± 13.46	<.001	65 ³⁰
PM_{10} , $\mu g/m^3$ per day	174.0±2.33	411.0 ± 17.57	<.001	150 ³⁰



Figure 1. Noise level in the working area was significantly higher compared with the control area, reflecting significant noise level in textile plants.



Figure 2. Indoor formaldehyde level in the working area was significantly higher compared with the control area, which infers significant formaldehyde level in textile plants.



Figure 3. Level of $PM_{2.5}$ in the working area was significantly higher compared with the control area, indicating significant $PM_{2.5}$ level in textile plants. PM indicates particulate matter.

Discussion

Today, environmental pollutants severely affect human health as important stress factors. With industrialization, more individuals have been exposed to these pollutants, causing various types of health problems.² Our study attempted to monitor the indoor environmental quality of different textile plants and correlate its effect on the occupational health and well-being of the textile workers through measuring plasma oxidative stress status in textile workers



Figure 4. Level of PM_{10} in the working area was significantly higher compared with the control area, revealing significant PM_{10} level in textile plants. PM indicates particulate matter.



Figure 5. Effect of noise and air pollutant exposure on plasma malondialdehyde level (nmol/L) in the sample group compared with controls. MDA indicates malondialdehyde.



Figure 6. Effect of noise and air pollutant exposure on plasma conjugated diene level (nmol/L) in the sample group compared with controls.

and healthy control subjects. Our study indicated that indoor noise level (78.0 \pm 0.68 dB) in the working area was significantly higher than in the control area (70.17 \pm 0.25 dB) (Figure 1). However, it has been reported that exposure to any kind of noise above 90 dB is recognized as a great source of oxidative stress.¹⁰ But long-time exposure (as each textile worker included in the study was in the profession for 5 years and working an average of 6 hours/day and 6 days per





week) to these elevated level of noise may affect the health of the textile workers. Moreover, earlier reports demonstrated that noise-induced hearing loss had occurred in textile workers as well.^{31,32} Epidemiological study showed that noise is associated with increased arterial hypertension, myocardial infarction, and brain stroke owing to increased hormonal imbalance by oxidative stress.³³ So, textile workers may become prone to different types of oxidative damage and noise-induced hearing loss.³

Formaldehyde, a chemical compound which works as a carcinogen, is found in the indoor and outdoor air of textile industries. Our study revealed that indoor formaldehyde level (141.80 \pm 4.47 µg/m³) in the working area was significantly higher compared with the control area (108.0 \pm 0.76 µg/m³) (Figure 2). Moreover, it was found that the working area had a formaldehyde level much higher than the value set by the WHO guideline.¹² A study conducted in China demonstrated that 66 workers in the chemical industry exposed occupationally to formaldehyde were found to suffer from congestion in the cornea, nasal membrane, and pharynx.³⁴ So, elevated level of formaldehyde exposure may result in acute mucus membrane irritation, leading to dry skin, dermatitis, tearing eyes, sneezing, and coughing.³⁵

Exposure to PM is one of the most crucial concerns in modern-day public health, as it is one of the key constituents of air pollution, and corroboration continues to increase citing PM as one of the most harmful pollutants.^{36,37} Particulate matter has been specially linked with oxidative stress and various types of cardiovascular conditions, including myocardial infarction, hypertension, atherosclerosis, heart rate variability, and coronary heart disease.³⁸⁻⁴³ Our study found that the daily average concentration of both $PM_{2.5}$ (322.2 ± 13.46 µg/m³) and PM_{10} $(411.0 \pm 17.57 \,\mu\text{g/m}^3)$ was significantly higher in the indoor air of the working area compared with the daily average concentration in the control area $(78.59 \pm 1.66 \text{ and } 174.0 \pm 2.33 \mu \text{g})$ m³, respectively) (Figures 3 and 4). Moreover, these levels were far much higher than the value set by the guideline of the Ministry of Environment and Forests, Bangladesh.³⁰ So, our study indicated significant level of environmental pollution

(noise, formaldehyde, and PM) in the working area compared with the control area, because exposure of textile workers to these environment pollutants may lead to oxidative stress and various health complications.

So, to measure the oxidative stress status of the textile workers and the control subjects, we targeted P-MDA and P-CD, 2 biomarkers of lipid peroxidation. Lipid molecules are highly susceptible to oxidation by ROS, and oxidation of lipoproteins includes peroxidation of PUFA that produces high amounts of lipid peroxidation products such as conjugated dienes. Moreover, breakdown of these products also produces aldehydes, such as MDA.^{22,44} As a result, levels of both P-MDA and P-CD are directly proportional to lipid peroxidation level, and therefore to the oxidative stress condition.²² Previously, in 1 study that was done in Turkey, MDA levels were found to be significantly higher in textiles workers than in controls.⁴⁵ In another cohort study, increased concentrations of P-MDA level was found in the workers of battery manufacturing company compared with the control.46 In this study, the level of P-MDA $(0.37 \pm 0.03 \text{ nmol/L})$ was determined to be significantly higher in textile workers compared with the control group $(0.18 \pm 0.01 \text{ nmol/L})$ (Figure 5). Moreover, the level of P-CD $(14.74 \pm 0.61 \text{ nmol /L})$ was also found to be significantly higher in textile workers compared with the control group $(10.04 \pm 0.44 \text{ nmol/L})$ (Figure 6). Increased levels of P-MDA and P-CD in textile workers may be due to the exposure to environmental pollutant, which may result in the elevated production of free radicals. If the free radicals produced are not balanced by an antioxidant defense system, then they may lead to various damages in tissues. So, it is possible that the raised P-MDA and P-CD may be due to decreased antioxidant activity.47,48

Ascorbic acid, an important antioxidant, protects cells from free radicals and ROS and thus prevents the oxidative damage of important biomolecules.^{49,50} In a study on 28 male cement workers, significantly lower P-ASC (35%) levels were observed in cement workers than in the control population.⁵¹ Similarly, in our study, we found significantly lower P-ASC level $(0.46 \pm 0.04 \text{ mg/dL})$ in textile workers than in control subjects $(1.29 \pm 0.06 \text{ mg/dL})$ (Figure 7). Deficiency of ASC is known to cause oxidant-induced injuries to the erythrocyte membrane, showing the critical role in maintaining cell integrity.^{52,53} However, it was reported earlier that P-ASC showed a tendency to decrease between 3 and 24 hours after exercise; as textile workers were continually involved in physical activities compared with control subjects, it was possible to possess a lower level of P-ASC than the control subjects.⁵⁴ So, the increased level of P-MDA and P-CD together with the decreased level of P-ASC in textile workers in this study supports the hypothesis that indoor environmental pollution in the textile plants leads to a greater oxidative burden and depletion of antioxidant defense system. It was mentioned earlier that approximately 90% of workers are women and children in the textile industries (including garments and others) in

Bangladesh.¹ However, most of the subjects in this study were men. This holds certain limitations, albeit the study provided salient information shedding light on the state of the textile plants as well as those working in it.

Conclusion

In conclusion, the present findings indicated that textile workers were exposed to significant level of indoor environmental pollutions as evidenced by significantly higher level of noise, formaldehyde, PM2,5, and PM10. Moreover, increased level of P-MDA and P-CD along with lower P-ASC in textile workers compared with the control subjects supports the opinion that environmental pollutants cause oxidative stress. To overcome oxidative stresses, the study suggests that vitamins (like ASC and vitamin E) can be supplied to the textile workers as a source of antioxidants.^{52,55} It was reported that supplementation with ASC protects against increased oxidative damage.56 Again, supplementation of ASC was found to be effective in reducing oxidative stress among shift workers of Tehran Shahid Tondgoyan oil refinery.⁵⁷ In addition, the textile workers need to take better precautions to protect them against serious health issues in their later life. Moreover, to get over the indoor environmental pollutions, use of noise pads and carpets for absorption of sounds is suggested. It is highly recommended that the textile workers use masks and earplugs and those working in the industries with noisy machineries use noise absorbents and lubricants. Furthermore, increased ventilation and use of high-efficiency air filters in the textile plants may be highly instrumental to get over this condition.

Author Contributions

All the authors actively participated during the conception of the research work; TK performed the data collection and all the authors actively took part in analyzing and interpreting the results. TR, AMF and HUS wrote the manuscript. HUS supervised the overall research work. All the authors read and approved the final manuscript.

ORCID iD

Tania Rahman (D) https://orcid.org/0000-0002-6398-3214

REFERENCES

- Yunus M, Yamagata T, Chousakenkyu Houkokusho I-J. In: Fukunishi T, ed., Dynamics of the Garment Industry in Low Income Countries: Experience of Asia and Africa, Interim Report, ChousakenKyu, Huokokusho, Japan: IDE-JETRO series; 2012: 29.
- Abdul-Wahab SA, En SCF, Elkamel A, Ahmadi L, Yetilmezsoy K. A review of standards and guidelines set by international bodies for the parameters of indoor air quality. *Atmos Pollut Res.* 2015;6:751-767.
- Münzel T, Sørensen M, Schmidt F, et al. The adverse effects of environmental noise exposure on oxidative stress and cardiovascular risk. *Antioxid Redox Signal*. 2018;28:873-908.
- 4. Alberti PW. Noise, the most ubiquitous pollutant. Noise Health. 1998;1:3-5.
- Munzel T, Sorensen M, Gori T, et al. Environmental stressors and cardio-metabolic disease: part I-epidemiologic evidence supporting a role for noise and air pollution and effects of mitigation strategies. *Eur Heart J.* 2017;38:550-556.
- World Health Organization. The World Health Report 2000: Health Systems: Improving Performance. Geneva, Switzerland: World Health Organization; 2000.

- 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;107:351-361.
- Karlidag T, Yalcin S, Ozturk A, et al. The role of free oxygen radicals in noise induced hearing loss: effects of melatonin and methylprednisolone. *Auris Nasus Larynx*. 2002;29:147-152.
- 9. Ohinata Y, Yamasoba T, Schacht J, Miller JM. Glutathione limits noise-induced hearing loss. *Hear Res.* 2000;146:28-34.
- da Costa KVT, de Andrade KCL, di Cavalcanti ME, Frizzo ACF, Carnaúba ATL, de Lemos Menezes P. Hearing loss at high frequencies and oxidative stress: a new paradigm for different etiologies. 2018:95–110.
- Dingle P, Tapsell P, Hu S. Reducing formaldehyde exposure in office environments using plants. *Bull Environ Contam Toxicol*. 2000;64:302-308.
- Nielsen GD, Larsen ST, Wolkoff P. Re-evaluation of the WHO (2010) formaldehyde indoor air quality guideline for cancer risk assessment. *Arch Toxicol.* 2017;91:35-61.
- Tang X, Bai Y, Duong A, Smith MT, Li L, Zhang L. Formaldehyde in China: production, consumption, exposure levels, and health effects. *Environ Int.* 2009;35:1210-1224.
- Raskin P. Global Environment Outlook Scenario Framework: Background Paper for UNEP's Third Global Environment Outlook Report (GEO-3), Vol 6. London, England: UNEP/Earthprint; 2004.
- Dockery DW, Pope CA, 3rd Xu X, et al. An association between air pollution and mortality in six U.S. *Cities. N Engl J Med.* 1993;329:1753-1759.
- Samet JM, Dominici F, Curriero FC, Coursac I, Zeger SL. Fine particulate air pollution and mortality in 20 U.S. cities, 1987-1994. N Engl J Med. 2000;343:1742-1749.
- Brook RD, Franklin B, Cascio W, et al. Air pollution and cardiovascular disease: a statement for healthcare professionals from the Expert Panel on Population and Prevention Science of the American Heart Association. *Circulation*. 2004;109: 2655-2671.
- 18. Sim J-Y. Nitric oxide and pulmonary hypertension. *Korean J Anesthesiol.* 2010; 58:4.
- Nelin TD, Joseph AM, Gorr MW, Wold LE. Direct and indirect effects of particulate matter on the cardiovascular system. *Toxicol Lett.* 2012;208: 293-299.
- Abuja PM, Albertini R. Methods for monitoring oxidative stress, lipid peroxidation and oxidation resistance of lipoproteins. *Clin Chim Acta*. 2001;306:1-17.
- 21. Elsayed NM. Antioxidant mobilization in response to oxidative stress: a dynamic environmental-nutritional interaction. *Nutrition*. 2001;17:828-834.
- Ayala A, Muñoz MF, Argüelles S. Lipid peroxidation: production, metabolism, and signaling mechanisms of malondialdehyde and 4-hydroxy-2-nonenal. Oxid Med Cell Longev. 2014;2014:360438.
- Chatterjee I, Nandi A. Ascorbic acid: a scavenger of oxyradicals. *Indian J Biochem Biophys.* 1991;28:233-236.
- 24. Kurutas EB. The importance of antioxidants which play the role in cellular response against oxidative/nitrosative stress: current state. *Nutr J.* 2015;15:71.
- Yoshida Y, Umeno A, Shichiri MJ. Lipid peroxidation biomarkers for evaluating oxidative stress and assessing antioxidant capacity in vivo. J Clin Biochem Nutr. 2012:12-112.
- Jung M, Klotzek S, Lewandowski M, Fleischhacker M, Jung K. Changes in concentration of DNA in serum and plasma during storage of blood samples. *Clin Chem*. 2003;49:1028–1029.
- Roe JH, Kuether CA. The determination of ascorbic acid in whole blood and urine through the 2, 4-dinitrophenylhydrazine derivavative of dehydroascorbic acid. *J Biol Chem.* 1943;147:399-407.
- 28. Kei S. Serum lipid peroxide in cerebrovascular disorders determined by a new colorimetric method. *Clin Chim Acta*. 1978;90:37-43.
- Spranger T, Finckh B, Fingerhut R, Kohlschütter A, Beisiegel U, Kontush A. How different constituents of human plasma and low density lipoprotein determine plasma oxidizability by copper. *Chem Phys Lipid*. 1998;91:39-52.
- Bangladesh Climate Change Strategy and Action Plan. Ministry of Environment and Forests. 2009. https://www.iucn.org/downloads/bangladesh_climate_ change_strategy_and_action_plan_2009.pdf
- Shakhatreh FM, Abdul-Baqi KJ, Turk MM. Hearing loss in a textile factory. Saudi Med J. 2000;21:58-60.
- 32. Oleru U. Comparison of the hearing levels of Nigerian textile workers and a control group. *Am Ind Hyg Assoc J.* 1980;41:283-287.
- Schell LM, Gallo MV, Denham M, Ravenscroft J. Effects of pollution on human growth and development: an introduction. J Physiol Anthropol. 2006;25: 103-112.
- 34. Speit G, Schütz P, Weber I, et al. Analysis of micronuclei, histopathological changes and cell proliferation in nasal epithelium cells of rats after exposure to formaldehyde by inhalation. *Mutat Res.* 2011;721:127-135.
- Rovira J, Roig N, Nadal M, Schuhmacher M, Domingo JL. Human health risks of formaldehyde indoor levels: an issue of concern. J Environ Sci Health A Tox Hazard Subst Environ Eng. 2016;51:357-363.

- Brook RD, Rajagopalan S, Pope CA III, et al. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation*. 2010;121:2331-2378.
- Sun Q, Hong X, Wold LE. Cardiovascular effects of ambient particulate air pollution exposure. *Circulation*. 2010;121:2755-2765.
- Allen RW, Criqui MH, Roux AVD, et al. Fine particulate matter air pollution, proximity to traffic, and aortic atherosclerosis. *Epidemiology*. 2009;20:254.
- Cavallari JM, Fang SC, Eisen EA, et al. Time course of heart rate variability decline following particulate matter exposures in an occupational cohort. *Inhal Toxicol.* 2008;20:415-422.
- Ibald-Mulli A, Stieber J, Wichmann H-E, Koenig W, Peters A. Effects of air pollution on blood pressure: a population-based approach. *Am J Public Health*. 2001;91:571.
- Peters A, Dockery DW, Muller JE, Mittleman MA. Increased particulate air pollution and the triggering of myocardial infarction. *Circulation*. 2001;103:2810-2815.
- 42. Simkhovich BZ, Kleinman MT, Kloner RA. Particulate air pollution and coronary heart disease. *Curr Opin Cardiol.* 2009;24:604-609.
- Sullivan J, Sheppard L, Schreuder A, Ishikawa N, Siscovick D, Kaufman JJE. Relation between short-term fine-particulate matter exposure and onset of myocardial infarction. *Epidemiology*. 2005;16:41-48.
- 44. Trevisan M, Browne R, Ram M, et al. Correlates of markers of oxidative status in the general population. *Am J Epidemiol.* 2001;154:348-356.
- 45. Yildirim I, Kilinc M, Okur E, et al. The effects of noise on hearing and oxidative stress in textile workers. *Indus Health*. 2007;45:743-749.
- Singh Z, Chadha P, Sharma S. Evaluation of oxidative stress and genotoxicity in battery manufacturing workers occupationally exposed to lead. *Toxicol Int.* 2013;20:95.

- Esterbauer H. Cytotoxicity and genotoxicity of lipid-oxidation products. *Am J Clin Nutr.* 1993;57:779S-786S.
- Ferrari R, Ceconi C, Curello S, et al. Oxygen free radicals and myocardial damage: protective role of thiol-containing agents. *Am J Med.* 1991;91:S95-S105.
- 49. Cerutti PA. Prooxidant states and tumor promotion. *Science*. 1985;227: 375-381.
- Yen G-C, Duh P-D, Tsai H-L. Antioxidant and pro-oxidant properties of ascorbic acid and gallic acid. *Food Chem.* 2002;79:307-313.
- Aydin S, Aral I, Kilic N, Bakan I, Aydin S, Erman F. The level of antioxidant enzymes, plasma vitamins C and E in cement plant workers. *Clinica Chimica Acta*. 2004;341:193-198.
- 52. Bendich A, Machlin L, Scandurra O, Burton G, Wayner D. The antioxidant role of vitamin C. *Adv Free Rad Biol Med.* 1986;2:419-444.
- Wefers H, Sies H. The protection by ascorbate and glutathione against microsomal lipid peroxidation is dependent on vitamin E. *Eur J Biochem*. 1988;174: 353-357.
- Schneider M, Niess AM, Rozario F, et al. Vitamin E supplementation does not increase the vitamin C radical concentration at rest and after exhaustive exercise in healthy male subjects. *EurJ Nutr.* 2003;42:195-200.
- McCay PB. Vitamin E: interactions with free radicals and ascorbate. Annu Rev Nutr. 1985;5:323-340.
- Tamari Y, Nawata H, Inoue E, et al. Protective roles of ascorbic acid in oxidative stress induced by depletion of superoxide dismutase in vertebrate cells. *Free Radic Res.* 2013;47:1-7.
- Khajehnasiri F, Akhondzadeh S, Mortazavi SB, et al. Are supplementation of omega-3 and ascorbic acid effective in reducing oxidative stress and depression among depressed shift workers. *Int J Vitam Nutr Res.* 2016;10:1-12.