Brief Report

Assessment of Impact on Health of Children Working in the Garbage Dumping Site in Dhaka, Bangladesh

by Gargy Lahiry, Tania Rahman, A. K. M. Mahbub Hasan, Alak K. Dutta, Md. Arif, *and* Zakir H. Howlader
Department of Biochemistry and Molecular Biology, University of Dhaka, Dhaka-1000, Bangladesh

Correspondence: Zakir H. Howlader, Department of Biochemistry and Molecular Biology, University of Dhaka, Dhaka-1000, Bangladesh. E-mail: <hhzakir@yahoo.com>.

Summary

Waste dumping is one of the major causes of environment pollution in Bangladesh. This study was designed to assess the impact on health of children working in one of the garbage dumping sites in Dhaka. Blood samples were collected from exposed ($n = 20$, aged: 8–15 years, exposed to dumped garbage from 6 months to 6 years) and control subjects ($n = 15$, age matched and never worked in the garbage dumping site). Oxidative stress markers like lipid hydroperoxides, thiobarbituric acid reactive substances and protein carbonyl content were measured. Alkaline comet assay was performed to assess the possible damage in DNA. To check the consequences of possible toxic exposure, we performed liver function tests of the study subjects. Oxidative stress-mediated damage of macromolecules was found to be significantly increased in the exposed children. Liver function tests were found normal. Thus, the children working in garbage dumping site are in severe health risk.

Key words: garbage dumping, environment pollution, health risk, oxidative stress.

Introduction

The solid waste disposal system in Bangladesh is very poor and unorganized. Improper solid waste disposal posses a greater health problem because it leads soil, water and air pollution when dumped and/or burnt openly [1]. World Health Organization (WHO) estimates that environmental pollution causes about a quarter of the diseases in the people, especially in developing countries; most of them acquired such health problems in childhood but demonstrate the symptoms in later stage of their life span [2]. A very common and unavoidable effect of garbage exposure is the oxidative stress due to exposure to the highly contaminated polluted air with toxic metals, such as lead, cadmium, mercury, arsenic, iron, copper and chromium. The metals can cause the production of reactive oxygen and nitrogen species (ROS and RNS) and thus causes the imbalance of pro-oxidant and anti-oxidant homeostasis termed oxidative stress [3]. Cells under oxidative stress display various dysfunctions due to random damage of their macromolecules like protein, lipid, DNA and RNA [4].

The study was designed to assess the health risk of study subjects who were working in the garbage dumping sites in the Matuail area, an open low-land waste disposal site, 3 km west from Dhaka, the capital city of Bangladesh. These children work in the garbage with naked hands and feet and without any mask. They look for some recyclable matters in the garbage like plastics and metals, and at the end of the day they sell it to the brokers to earn some money for their family. There is no system to separate burnable or non-burnable garbage, even the hospital garbage and household garbage are dumped into the same dumping site where the poor children work for their survival with a high health risk.

Subjects and Methods

Study subjects

Children aged 8–15 years ($n = 20$) who were working in the garbage dumping site from 6 months to 6 years were selected as study subjects (exposed group) and children ($n = 15$) of the same age group were selected as control, who never worked in the garbage

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dumping site (control group). The study was approved by the Dhaka University Ethical Review Committee and all participants and their guardians gave the consent.

Analytical methods

Blood samples were analyzed for oxidative stress-induced modifications of lipid, protein and DNA damage along with some routine test of biochemical parameters. Lipid hydroperoxide value was determined by colorimetric method based on the oxidation of ferrous ion [5]. Thiobarbituric Acid Reactive substances (TBARS) value was determined according to the method described by Ohkawa *et al.* [6] using malondialdehyde (MDA) as standard. Serum carbonyl contents were measured using the method described by Levine *et al.* [7]. The extent of DNA damage was measured using a single-cell microgel electrophoresis technique under alkaline conditions also termed comet assay [8]. Comet images were analyzed using a software Computer Assay Software Project (CASP, version 1.2.2; University of Wroclaw, Poland), developed by Konca *et al.* [9]. Alanine amino transferase (ALT) [10], aspartate amino transferase (AST) [10], alkaline phosphatase (ALP; assay kit from Abcam; UK), albumin [11] content in serum was estimated by the method described in the respective references. All the data were analyzed using the statistical package for social sciences (SPSS; version 11.0; Chicago, IL, USA). Student *t*-test (two-tailed) was used to evaluate statistical significances between the two study groups. A $p \leq 0.05$ was the criterion for a statistically significant difference.

Results

Oxidative stress-induced damage of cellular major components like lipid, protein and DNA was evaluated in the exposed group and compared with the control group. The average lipid hydroperoxide in serum, the major initial reaction products of lipid peroxidation were 7.63 ± 0.38 and $12.21 \pm 4.98\,\text{nmol ml}^{-1}$ in control and exposed group, respectively (Table 1; $p \leq 0.01$). Serum TBARS value was determined to measure the secondary products of lipid peroxidation in the exposed group (15.99 ± 4.61 nmol MDA eq ml^{-1}) and that was significantly higher (Table 1; $p \leq 0.001$) compared with the control group (6.37 ± 0.41 nmol MDA eq ml^{-1}). Oxidative stress-mediated ROS can be reactive with protein side groups forming protein carbonyl, a well-known marker of protein damage. In control group, the mean protein carbonyl value was $394.74 \pm 25.56\,\text{nmol mg}^{-1}$ of protein and in exposed group, the value was $951.58 \pm 154.60\,\text{nmol mg}^{-1}$ of protein (Table 1; $p \leq 0.01$). Table 2 shows analytical data of the representative comet images of lymphocytes in two study groups. Almost 95% DNA were concentrated in the head region of the control group, whereas in the exposed group it was 71% ($p \leq 0.001$). The calculated tail moment, a commonly used marker of DNA damage, was 15.6 times higher in the exposed group compared to the control group ($p \leq 0.001$).

TABLE 1
Serum level of markers to assess the oxidative stress induced damage of lipid and protein in study subjects

Test parameter	Control ($n = 15$)	Exposed ($n = 20$)
Lipid hydroperoxide (nmol ml^{-1})	7.63 ± 0.38	12.21 ± 4.98**
TBARS value (nmol MDA eq ml^{-1})	6.37 ± 0.41	15.99 ± 4.61***
Protein carbonyl value (nmol mg^{-1} of protein)	394.74 ± 25.56	951.58 ± 154.60**

Serum level of lipid hydroperoxide, TBARS value and Protein carbonyl content was significantly higher in subjects exposed to garbage ($n = 20$), compared to control ($n = 15$). Data presented here as mean ± SE. Student's *t*-test was performed to analyze data.
**$p < 0.01$, ***$p < 0.001$.

TABLE 2
DNA damage measured by comet assay

	Head DNA (%)	Tail DNA (%)	Tail moment
Control ($n = 15$)	95.23 ± 1.57	04.77 ± 1.09	0.38 ± 0.01
Exposed ($n = 20$)	71.76 ± 1.78***	28.24 ± 1.07***	5.93 ± 0.19***

Comet images were analyzed using CASP, version 1.2.2. Data are presented as mean ± SEM. Student's *t*-test was performed to analyze the data.
***$p < 0.001$.

TABLE 3
Serum level of some biochemical parameters to assess liver functions in the study subjects

Test parameter	Control ($n=15$)	Exposed ($n=20$)	Reference value
Serum bilirubin	0.62 ± 0.09	0.95 ± 0.12, NS	0–1.0 mg dl^{-1}
AST	34.75 ± 5.62	29.80 ± 6.42, NS	10–40 U l^{-1}
ALT	23.50 ± 7.05	24.00 ± 4.42, NS	10–55 U l^{-1}
ALP	183.00 ± 18.14	195.00 ± 12.88, NS	up to 644 U l^{-1}
Serum albumin	4.35 ± 0.81	4.15 ± 0.69, NS	3.5–5.5 g dl^{-1}

Data are presented as mean ± SEM. Student's *t*-test was performed to analyze the data; NS, not significant.

No significant differences were found in the parameters tested to assess liver functions of the two groups of study subjects (Table 3).

Discussion

The waste causes a potential hazard on the human health, environment and the ecosystem when improperly stored, transported, disposed and treated [12] leading to lethal diseases. The continuous and accumulative oxidative stress produced by exposure to dumped garbage causes the damage of cellular components. Increased level of lipid hydroperoxide and TBARS value in the exposed group confirms the presence of high rate of lipid peroxidation, which is an important indicator of lipid moiety damage by oxidative stress. Proteins, which constitute the major components of living cells, are among the main targets of ROS-induced damage [13]. Elevated level of protein carbonyl group (the major marker of protein damage) in exposed group indicates the presence of high rate of protein oxidation and hence high rate of oxidative stress. Protein modification can hamper the functions of some important enzymes like DNA polymerases and DNA damage repair relevant enzymes that could lead the DNA lesions including base and sugar lesions, strand breakage [14]. The effect of exposure to dumped garbage to the damage of DNA, were assessed by comet assay (single-cell gel electrophoresis). Recently, the DNA damage of human subjects working in adverse environment due to their occupation is reported, for example in petrol pump workers [15], due to exposing to electric waste [16] and due to air pollution and smoking [17].

Thus, the children, working in the garbage dumping site are in great health risk. It is observed that exposure to garbage can cause the alterations of the major cellular components to a significant degree. Oxidative DNA damage results the detrimental biological consequences including cell death, DNA mutations and transformation to malignant cells [18]. ROS-mediated oxidative stresses are also linked to atheroscelrosis [19], aging and life span [20]. These oxidative stress-mediated damages may ultimately cause several genetic or neuro-degenerative diseases. As the children work in the dumped garbage with naked hands and feet and the garbage includes all sorts of waste materials including medical wastes, so there is a high risk of serious infections. To check the consequences of possible toxic exposure, we performed liver function tests of the study subjects and found the values within normal reference values. All the damages in cellular components are possibly the consequences of heavy metal exposure. Therefore, estimation of heavy metal concentration in the soil, water and air of dumped site and also in serum of the subjects required to better understand the reason of the consequences in the exposed children.

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