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Environmental and Occupational Lead Exposure Among Children in Cairo, Egypt

A Community-Based Cross-Sectional Study

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Abstract: The aim of this study was to assess childhood lead exposure in a representative sample of Cairo, and to investigate the possible risk factors and sources of exposure.

This cross-sectional study was conducted from November 2014 through April 2015. The target population was children aged 6 to 18 years, recruited into 4 groups, garbage city, moderate-living standard area, urban and suburban schools, and workshops in the city of Cairo. Blood lead levels (BLLs) and hemoglobin (Hb) concentrations were measured. Also, potential local environmental sources were assessed for hazardous lead contamination.

Analysis on 400 participants has been carried out. A total of 113 children had BLLs in the range 10 to 20 μg/dL. Smoking fathers, housing conditions, playing outdoors, and exposure to lead in residential areas were significantly correlated with high BLLs. The mean values of hemoglobin were inversely correlated with BLLs. Children involved in pottery workshops had the highest BLLs and the lowest Hb values with a mean of (43.3 μg/dL and 8.6 g/dL, respectively). The mean value of environmental lead in workshop areas exceeded the recommended levels. Also, those values measured in dust and paint samples of garbage city were significantly high. Moreover, the mean lead levels in the soil samples were significantly higher in urban schools ($P = 0.03$) than the suburban ones.

Childhood lead poisoning accounts for a substantial burden in Egypt, which could be preventable. Development of national prevention programs including universal screening program should be designed to reduce incidence of lead toxicity among children.

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Abbreviations: BLLs = blood lead levels, CBC = complete blood count, CDC = Centers for Disease and Prevention, EDTA = ethylenediaminetetraacetic acid, Hb = hemoglobin, IRB = Institutional Review Board, mL = milliliter, NISTSRM = National Institute of Standards and Technology Standard Reference Material, QC = quality control, SD = standard deviation, USA = United States of America.

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INTRODUCTION

Lead exposure remains a major public health problem, especially in developing countries, whereas regulations and policies are missing.[1] In Africa, children, especially those living in the vicinity of industrial areas, are exposed to the highest levels of lead from different sources, such as petrol, lead released by burning of paper products, discarded rubber, battery casings, and painted wood for cooking and heating.[2] Children are more at risk than adults to the toxic effects of lead. This correlates with their ability to walk, their hand-mouth behavior, and spending more time on dusty floors.[3] Also, physiological functions of their blood–brain barrier and liver detoxification systems are biologically immature.[4]

Childhood lead toxicity presents a great challenge for health care providers.[5] During the last 40 years, the Centers for Disease Control and Prevention (CDC) have taken down the threshold for blood lead levels (BLLs) in children from 60 to 10 μg/dL.[6] Recent data have shown that there is no cutoff value for elevated BLLs, and even children with blood lead concentration <10 μg/dL are at risk for reduced cognitive development and functioning.[7] Consequently, the CDC eliminated the terminology ''level of concern.''[8] Therefore, the present study aimed to asses childhood lead exposure in a representative sample of Cairo, and to investigate the possible risk factors and sources of exposure.

METHODOLOGY

Study Design and Setting

We used a community-based, cross-sectional survey design, with a convenience sample collected from the city of Cairo, the main sector of Greater Cairo, between November 2014 and April 2015. A team of 2 professional epidemiologists, 2 undergraduate medical students, trained social worker, and 1 laboratory technician participated in the selection of sites and data collection for the study.

The city of Cairo is located on the east bank of the Nile, extending south to Helwan, north to Shubra, and northeast to Heliopolis and Nasr City. The estimated population is 7,800,000 with 1,7600,016 school students studying in 3281 schools. To ensure a comprehensive coverage of the entire area of Cairo, we aimed to recruit 400 children aged 6 to 18 years from 4 different entities during the study period. The sampled population was categorized into 4 groups, 100 for each. The Group I was collected from Old Cairo district that lies 50 to 100 m away from the highway (Nile Corniche) and characterized by the moderate living standard.[9] The Group II was recruited from

Manshiyat Naser which is considered one of the biggest slum settlements in Cairo Governorate. It is located at the base of ''Mokattam hill''; so it contains some dangerous areas because of collapsing in hillsides. It is famous for ''Garbage Area'' as its economy is based on collecting and recycling garbage.[10] The sample of the Group III (School group) was collected from different schools of the city of Cairo. Four schools were chosen by simple random sampling to represent urban and suburban students. These children present different types of schools; either government or private. The Group IV (Workshop group) was selected from 6 occupations namely automobile repair, car batteries, smelters, radiators, pottery workshops, as well as garbage collection. For inclusion in the study as a worker, the child was granted for that work, or if the yield of this work was bound for the market.[11]

Children who had major health disorders, chronic illness associated with anemia, and neuropsychological changes were excluded from the study.

Questionnaire

Data were collected using a pretested, structured interview questionnaire at a household level. Children's guardians were asked about sociodemographic characteristics, daily activities, and potential environmental and household sources of exposure to lead (e.g., child working, parental occupation, water drinking sources, dwelling, and basic housing conditions such as house painting and plumbing pipes). In our study, painting was considered ''old'' if it was $\geq$25-year old and ''recent'' if it was $<$25-year old.

After designing the questionnaire, we conducted a small-scale pilot study in 2 Cairo's subdistricts. After this pretest, the questionnaire was modified (the content of some questions was changed). The questionnaire was created originally in Arabic and later translated into English.

Laboratory Analysis

Blood samples of approximately 10 milliliter (mL) were drawn from each participating child to measure blood lead and complete blood picture (CBC). All materials used were new and disposable. The blood sample was split into 2 separate pipes for each participant, lead-free and containing ethylenediaminetetraacetic acid (EDTA) as an anticoagulant. During the sampling, all samples were kept in coolers with ice packs. One of the tubes of each participant was immediately transported to a local laboratory for CBC analysis. The other tube was transported to the Occupational Health Department Laboratory at Cairo University to determine lead levels. Concentrations of blood lead were determined through atomic absorption spectroscopy using the method described by Subramanian with matrix modifiers (DMH-Triton X-100 in nitric acid at 0.2%). The equipment used was a 3110 Perkin Elmer spectrometer with a graphite furnace .[12] Quality control (QC) measures included analysis of initial calibration verification standard, procedural blanks, duplicate samples, spiked samples, and analysis of a certified reference material (National Institute of Standards and Technology Standard Reference Material (NISTSRM) 955b: bovine blood for lead) to monitor for contamination, accuracy, and recovery rates. Recovery rates for lead in QC and spiked samples were 90% to 115%, and the precision was measured as % relative standard deviation (SD), with a result of less than 5% for lead. The limits of detection for lead were 0.2 μg/dL. The results were given as the average of 5 replicate measurements.

Environmental Assessment of Lead

Samples collected from the environment of the study areas were analyzed for lead level. Drinking water samples were taken from tap water for detection of lead level using the atomic absorption spectrophotometer (Graphite Furnace, Perkin Elmer HGA-600, Waltham, Massachusetts), a 1 L from each source. Dust samples were gathered from 1 square feet (sq ft) indoor and outdoor using dust wipes. Superficial soil was sampled from the residential area of the study as well as playground of each sampled school not exceeding 2 cm according to recommendations of CDC.[13,14] Dust, soil, and paint lead levels were determined by atomic absorption spectroscopy.[15] In terms of setting, we collected a limited number of air, water, and soil samples from workshop areas. We started from the interior of the source point and at different distances (30, 60, 90, and 120 m), following an enlarging circumferential pattern.

Guidelines, recommendations, and standard operating procedures for sample collection, preparation, and analysis were being followed. All environmental samples were gathered from homes corresponding to those children with the BLL levels $\geq$5 μg/dL and analyzed in the National Research Institute Laboratory, Dokky, Cairo, Egypt.

All study participants were volunteers. Ethical approval was obtained from the Institutional Review Board (IRB) of the New Children's Hospital (Abou el Reesh), Paediatric Specialized Hospital of Cairo University before the study started. Informed consent was obtained from all parents or children's legal guardians before they were enrolled in the study. Verbal assent was obtained from patients $\geq$7 years. The study objectives and procedures were explained and guardians were reassured about the confidentiality of the study data.

No cases of acute lead poisoning were reported during the study period. All subjects with BLL $\geq$5 μg/dL ($n = 208$) were thoroughly examined and evaluated for manifestations of lead toxicity. Children with BLL $\geq$20 μg/dL ($n = 116$) were referred to Toxicology Center, Kasr-el-Aini Hospital for proper assessment and treatment. The results of these analyses and treatment are presented elsewhere.

Statistical Analysis

Data were statistically described in terms of mean $\pm$ stanstandard deviation ($\pm$ SD), median and range, or frequencies (number of cases) and percentages when appropriate. Comparison of numerical variables between the study groups was done using the Student *t* test for independent samples in comparing 2 groups when normally distributed and Mann Whitney *U* test for independent samples when not normally distributed. Comparison of normally distributed numerical variables between more than 2 groups was done using Kruskal Wallis test. For comparing categorical data, χ^2 test was performed. Exact test was used instead when the expected frequency is less than 5. *P* values less than 0.05 were considered statistically significant. All statistical calculations were done using computer program SPSS (Statistical Package for the Social Science; SPSS Inc, Chicago, IL) release 15 for Microsoft Windows (2006).

RESULTS

In the initial phase of the study, it became apparent that the workshop children should be separately assessed to delineate the risk and potential sources of lead exposure.

Sociodemographic characteristics of the study participants are shown in Table 1. Over half of the studied children were boys (60.5%), with girls accounting for the remaining 39.5%,

TABLE 1. Sociodemographic Characteristics of the Participants

	Group I (Old Cairo)	Group II (Manshyt Naser)	Group III (School Children)		Group IV (Workshop)	*P* Value
			Suburban	Urban		
Number of the subjects	100	100	70	30	100	
Age, yr*	10.2 ± 3.04					
6–11	63	59	26	18	33	
12–19	37	41	44	12	67	<0.001
Sex						
Male	41	52	46	6	97	–
Female	59	48	24	24	3	
Smoking fathers						
Currentsmokers	55	52	49	19	69	0.039
Nonsmokers	45	48	21	11	31	
Old houses with:						
chipping paint	9	13	23	3	14	<0.001
lead pipes	17	7	11	5	19	0.006

* Mean ± standard deviation.

giving a male-to-female ratio of 1.5:1. The children's mean age was 10.2 years (SD = 3). The group of children aged 6 to 11 years and the other group aged 12 to 19 years, were almost equally represented (49.7%, 50.3%, respectively). The majority of children's fathers were smokers (61%, n = 244), predominantly in the workers' sample (Table 1).

Table 2 provides the results of the mean BLL for the overall individuals studied (except workshop group). Generally, BLLs exceeded the limit of quantification, with a mean of 10.7 (SD = 3.2) μg/dL. In terms of geographic area, the risk of lead toxicity was the highest in Manshyat Naser district (Group II) and the lowest in the Old Cairo district (Group I) with a mean of [15.8 (SD = 4.4) vs. 5.6 (SD = 1.8) μg/dL, respectively].

After stratification according to sex, age, residential duration, smoking habits of children's fathers, and outdoors playing activities, children of smoking fathers were more likely to have a higher mean BLL ($P = 0.001$) than those whose fathers were not smoking. Housing conditions (painting age and painting status) (Table 1), playing outdoors, and duration of exposure to lead in residential areas were positively correlated with high BLL ($P < 0.05$) (Table 2). Though males were more likely to have a higher BLL than females [10.9 (SD = 3.3) vs.10.4 (SD = 3)], yet this was not statistically significant ($P = 0.1$) (Table 2).

The mean values of hemoglobin (Hb%) were inversely correlated with BLLs in the 1st 3 groups, particularly Group II.

TABLE 2. Mean Blood Lead Level (μg/dL) in a Representative Sample of Cairo (Old Cairo District, Manshyat Nasser District, Urban and Suburban School Children)

	Group I (n = 100) Mean ± SD*	Group II (n = 100)	Group III		P value
			Suburban (n = 70)	Urban (n = 30)	
Total	5.6 ± 1.8	15.8 ± 4.4	10.4 ± 3.3	10.8 ± 3.2	<0.001
Sex					
Male	5.6 ± 1.9 (2.7–12.3)	16.4 ± 5.1 (6.4–30.8)	10.3 ± 3.2 (4.3–15.7)	11.1 ± 3.1 (5.2–16.5)	0.126
Female	5.5 ± 1.7 (2.8–10.1)	15.2 ± 3.7 (4.8–28.6)	10.4 ± 3.3 (4.5–16.2)	10.4 ± 3.2 (3.5–15.7)	
Age					
6–11	5.9 ± 1.8 (3.4–12.3)	16.7 ± 5.4 (7.2–30.8)	10.7 ± 3.2 (5.3–16.2)	9.1 ± 4.3 (3.5–16.5)	0.521
12–19	4.8 ± 1.2 (2.7–9.2)	14.8 ± 3.3 (4.8–25.6)	10.2 ± 3.3 (4.3–14.8)	11.8 ± 1.9 (6.2–14.9)	
Residential duration (yr)	10.05 ± 3.4	12 ± 6.1	9.4 ± 5.7	8 ± 4.2	<0.001
Smoking fathers					
Current smoker	5.9 ± 1.6 (n = 55)	15.9 ± 4.4 (n = 52)	10.8 ± 3.2 (n = 49)	11.7 ± 1.2 (n = 19)	0.001
Nonsmokers	5.3 ± 1.4 (n = 45)	15.6 ± 4.5 (n = 48)	9.5 ± 3.3 (n = 21)	9.6 ± 3.8 (n = 11)	
Outdoor playing activities	5.7 ± 1.3 (n = 14)	15.7 ± 1.6 (n = 79)	9.4 ± 3.1 (n = 46)	9.9 ± 1.3 (n = 8)	<0.001

* Mean ± standard deviation.

TABLE 3. Mean Values of Hb in Relation to Blood Lead Levels in a Representative Sample of Cairo (Old Cairo District, Manshyat Nasser District, Urban and Suburban School Children)

	Group I		Group II		Group III			
					Suburban		Urban	
Mean BLL (μg/dL)	Number	Hb% gm/dL Mean (± SD) *	Number	Hb% gm/dL Mean (± SD)*	Number	Hb% gm/dL mean (± SD)*	Number	Hb% gm/dL mean (± SD)*
<10	94	11.8 ± 0.9 (8.7–14.3)	8	11.0 ± 2.5 (7.9–13.8)	52	11.5 ± 2.6 (9.2–13.4)	17	11.2 ± 2.7 (9.4–14.6)
≥10–20	6	11.7 ± 0.9 (10.5–12.6)	76	10.9 ± 2.9 (7.5–11.7)	18	11.0 ± 2.0 (8.6–13.7)	13	10.8 ± 1.9 (7.4–15.3)
>20	–	–	16	10.6 ± 2.5 (7.2–13.6)	–	–	–	–

* Mean ± standard deviation.

A total of 113 children had BLLs in the range 10 to 20 μg/dL. Only 5.3% (n = 16) of the study population had BLL ≥20 μg/dL. All of them were extracted from Manshyat Naser district (Table 3).

Table 4 presents the analysis of blood samples collected from children working in 6 different occupations. Those involved in pottery workshops had the highest BLLs and the lowest Hb values with a mean of (43.3 μg/dL and 8.6 g/dL, respectively). The least BLL value in this group was those for children working in garbage collection with a mean of 30.9 (SD = 2.5) μg/dL.

The mean lead levels in samples collected in the environment of the 1st 3 groups are presented in Table 5. Overall, results of lead measured in potential local sources, such as paints and relevant media: water, soil, and dust were within the recommended WHO threshold values. Although the mean environmental lead levels in water, dust, and soil samples were higher in Manshyat Naser (Group II) than other groups, yet this difference was not statistically significant. Also, the mean lead levels in the soil samples were significantly higher in urban schools [72.7 (± 10.5) μg/dL, $P < 0.001$] than the other groups (Table 5).

Table 6 shows the distribution of environmental lead in different workshop areas. The lead concentration in environmental samples of most of these areas exceeded the WHO recommended limits.[16] The highest degree of air and soil pollution was detected in the city of Helwan (industrial area) with a mean of [4.2 (SD = 1.3) μg/m^3, 448.5 (SD = 29.7) μg/g]. The mean lead content in the water samples reached the highest levels in Al-Wayli district (lead smelter area), and the lowest in Al-Basatin district (lime and pottery area) [6.3 (SD = 0.4) μg/L and 2.5 (SD = 0.4) μg/L, respectively].

DISCUSSION

Lead is a common environmental contaminant and its toxic effects are a particular concern for child health.[17] Similar to the results of other studies,[18,19] more than half of our study children (57.2%) had BLLs ≥10 μg/dL. In our study, the mean BLLs were at least 3-folds higher than those reported in the United States (USA) and most of the European countries.[20,21] Similar findings were observed in studies carried out in Egypt[22] as well as in Pakistan.[23] Another Egyptian study conducted in Sharkya governorate reported higher mean BLLs than our results.[24] This difference could be attributed to the condition seen in most developing countries, where implementation of legal restrictions and interventions to minimize lead exposure could not be properly applied.

We also found that the mean BLLs of the workshop group were higher than those reported in similar studies from developing countries such as Lebanon and Pakistan.[25,26]

Although the mean BLLs in Cairo were not statistically different between sexes, the prevalence of males having an elevated BLL was higher than females. Similar results were seen in studies conducted in Egypt,[18] Pakistan,[27] Thailand,[28] and Bangladesh.[17] Traditional male and female roles in our society with increased outdoor activities for boys have been involved in this sex-related difference; however, the exact reason is still not clear.[29–31]

Consistent with other studies from Mexico,[32] Bangladesh,[33] and Russia,[34] the present survey also found that the mean BLL was higher in children aged 6 to 11 years than the other age group, though this difference had no statistical significance. Children in this age group are more likely to play outdoors, with increased environmental exposure to lead poisoning, such as automobile exhaust, smoking, and a variety of toys.[35]

TABLE 4. Mean Blood Lead Levels and Hemoglobin (Hb) in Working Participants

Workshop	Number	BLL (μg/dL) Mean ± SD*	Hb (g/dL) Mean ± SD*
Automobile repair	28	32.7 ± 4.3 (22.5 – 36.5)	9.8 ± 0.7 (8.2–11.5)
Car batteries	24	40.8 ± 1.5 (39.4–43.5)	8.7 ± 0.4 (7.5–10.3)
Smelters	17	33.1 ± 5.9 (21.1–40.8)	9.5 ± 0.8 (8–12.6)
Pottery	14	43.3 ± 13.4 (20.9–51.9)	8.6 ± 0.8 (7.6–11.4)
Radiators	10	31.4 ± 2.6 (27.9–33.6)	8.8 ± 1.2 (8–11.5)
Garbage	7	30.9 ± 2.5 (28.5–33.3)	9.6 ± 0.7 (6.9–11.8)

* Mean ± standard deviation.

TABLE 5. Mean Concentration of Environmental Lead Measured in Different Media of Investigated Study Sites

Sample Media Mean and (SD)*	Number of Samples in Each Group	Group I	Group II	Group III		P value
				Suburban	Urban	
Water Pb (μg/L)	5	2.2 ± 0.6 (0.3–4.4)	2.1 ± 0.5 (0.16–4.2)	2.2 ± 0.6 (1.4–5.2)	2.5 ± 0.3 (0.41–4.8)	0.645
Dust (indoor)Pb†	3	10.3 ± 6.2 (3.1–21)	21.8 ± 6.4 (5.6–44.6)	14.0 ± 3.8 (4.2–28.6)	16.4 ± 4.2 (4.9–336)	0.133
Dust (outdoor) Pb†	3	26.3 ± 6.2 (20.2–27.6)	42.5 ± 9.0 (32.7–44)	28.6 ± 6.5 (22–31.7)	37.6 ± 5.8 (28.8–41.5)	0.067
Paint Pb†	3	0.06 ± 0.01 (0.005–0.53)	0.10 ± 0.03 (0.0009–0.9)	0.08 ± 0.03 (0.0007–0.73)	0.09 ± 0.02 (0.0008–0.82)	0.281
Soil Pb†	6	42.5 ± 10.3 (4.8–81.5)	68.3 ± 16.9 (7.6–131)	44.5 ± 11.6 (5.1–85.4)	72.7 ± 10.5 (8.3–139.5)	<0.001

*Mean ± standard deviation.
†Pb is measured in part per million (ppm).

Consistent with previous studies from China [36] and Australia,[37] children with smoking fathers were at higher risk of having elevated BLLs than those with nonsmoking fathers. This provides evidence that parental smoking itself may contribute to a child's lead burden.[31] This highlights the importance of parental awareness of making their homes safe for children.[38]

The results of this study confirm previous reports that exposure to lead paint is a risk factor for lead poisoning either directly through ingestion of flakes of chipping paint or indirectly through renovation of old homes with subsequent contamination of the surrounding environment.[39] Also, we reported significant relation between the BLL and lead pipes in houses ($P < 0.001$). In contrast to previous studies,[6,40] we found no relation between mean BLL and the age of the house.

Duration of exposure to lead in a residential area is consistently found to be a strong predictor of high BLLs,[27,41,42] a finding reported in this study. This reflects contamination of the environment by lead and its compounds from nearby industrial and occupational sources.

Several previous studies have found that anemia is associated with increased risk of high BLLs,[18,19,43] a finding reproduced in our study. However, Froom et al as well as other studies found no significant correlation between anemia and elevated BLLs.[18,44]

The industrial areas and garbage city (Manshyat Naser) of Cairo were identified as the worst affected areas among the study population. Similar findings were observed in a study carried out in Greater Cairo, Egypt[22] as well as in studies conducted in some developing countries.[17]

We found a significant difference in mean BLLs between urban and suburban school children, indicating that the urban environmental pollution is a major risk factor for lead poisoning. A previous Egyptian study found children living in Alexandria city (an urban area) had a significantly higher mean BLL relative to those living in Kafr Al-Sheikh (a suburban and rural area), with a higher proportion of children in Alexandria having BLLs >20 μgLdL than Kafr Al-Sheikh (56.7% vs. 6.7%), respectively.[45] Cairo, with a fleet of more than a million vehicles circulating on its streets, industrial activity of the Cairo city, and its encompassing urban areas and resuspension of street lead bearing dust were imperative sources of lead contamination.[41]

In our study, 100% of all workshop children had BLLs above the reference value of 5 μg/dL.[8] Children working in pottery production were the main victims. Our findings are consistent with the results of a study in Equador, where the mean BLLs for children working in the ceramic tile industry were 70 μg/dL.[46] Children in the pottery industry are potentially exposed to lead when the molded-clay items are glazed with lead salts and the ceramics are fired in furnaces with subsequent saturating the neighborhood air with dust and fumes.[46] A study in Alexandria, Egypt, reported a higher risk of lead toxicity among children working in battery workshops.[47] Another study from India reported blood lead concentrations of 24.3 to 62.4 μg/dL among those who worked as car mechanics.[48] Moreover, a study conducted in a ship repair yard reported a mean BLL of 26 μg/dL among painters, fitters, and other ship repair workers.[49] Children engaged in these activities are also at risk of nutritional deficiencies, which enhance the adverse effects and increase the absorption of lead.[50] Furthermore, our results support the fact that children working at waste disposal sites face many health hazards and risks.[51] A Nicaraguan study reported higher BLLs in working children compared with the nonworking groups.[52] These findings highlight

TABLE 6. Mean Distribution of Environmental Lead in Air, Water, and Soil in Workshop Areas

District	Population	Pb in Air*	Pb in Water†	Pb in Soil‡
Helwan	649.571	4.2 ± 1.3 (1.6–9.7)	5.6 ± 0.5 (0.8–26.1)	448.5 ± 29.7 (170.4–1009.3)
Shubra	71.118	3.5 ± 0.3 (2.1–5.4)	4.8 ± 0.3 (0.7–20.9)	385 ± 24.8 (252.9–586.8)
Al-Basatin	822.513	1.8 ± 0.7 (0.6–3.8)	2.5 ± 0.4 (0.4–16.1)	132.4 ± 21.03 (42.4–286)
Al-Wayli	77.649	1.06 ± 0.1 (0.4–2.6)	6.3 ± 0.4 (0.9–28.6)	336.6 ± 24.04 (181.2–485.4)
Al-Darb al-Ahmar	60.488	2.7 ± 0.4 (1.4–3.9)	3.9 ± 0.6 (0.6–15.2)	140.7 ± 15.3 (89.2–204.9)

* WHO recommended value 0.5 μg/m^3.
† WHO recommended value 7 μ/L.
‡ WHO recommended value 320 μg/g.[16]

the demand for policy development and the implementation of more rigorous health and safety standards in such workplaces.

Because there is a lack of distinction between toxic lead exposure of children at home, work, and external environment,[53] it should be emphasized that we could not establish the actual source of lead in the 1st 3 groups; where the measured lead levels in environmental samples did not exceed the maximum permissible WHO values. However, our study had shown significant exposure to high levels of lead in all environmental samples of workshop areas, especially in residential–industrial regions. Studies from Egypt,[54] Bangladesh,[17] and China [55] found similar results.

These findings are an important reminder that health authorities should investigate local occupational hygiene infrastructures, sanitary facilities, and practices that could reduce exposures in children to lead as well as to many other contaminants.

IMPLICATIONS

Lowering the childhood lead exposure should be an important public health objective and a priority in Egypt. This study reinforced the severity of the problem of lead poisoning in Egyptian children. Although the removal of children from occupational exposure to lead is the cornerstone in management, however, low socioeconomic status and poverty as well as weak enforcement of the current Child's Laws in Egypt are barriers that limit the achievement of such a goal.[56] Thus, measurements to recognize, control, and possibly remove environmental sources of lead will be very effective in minimizing exposure to lead, particularly with recurrent downward adjustment of ''safe'' thresholds of BLLs.[57] There is a lot to be done in terms of control to reduce children's exposure to lead such as promoting the use of unleaded petrol all over Egyptian governorates, removal of lead from paint, strict implementation of environmental laws, rigorous examination and approval of food and packaged goods, including toys containing lead, health education, and promotion. Moreover, early blood lead screening of children in heavily lead polluted areas helps to identify asymptomatic children with elevated BLLs who would benefit from further intervention.[58]

LIMITATION

To interpret the study results, we should mention some of its limitations.

One of the limitations was nonrandom sampling design which is subject to potential weakness of bias. Moreover, the sample may not be sufficient to interpret the real extent of the problem in Egypt; as all subjects were allocated from 1 governorate. However, the study population was chosen to reflect the majority of Egyptian children aged 6 to 18 years. The lead concentrations were measured only once, making it hard to think about the extent to which our results reflect the circumstances of lead exposure throughout the entire time of childhood period. We did not assess the levels of other environmental pollutants, for example, mercury, cadmium, which may act synergistically to enhance lead toxicity. Furthermore, we were restricted in our ability to assess how childhood exposure, particularly occupational exposure, to lead and/or other toxic elements/compounds could affect the status of essential elements and/or vitamins resulting in their deficiency. Finally, our study was a relatively small study; thus, a larger study is needed to verify our results.

CONCLUSION

On the basis of the findings of this study, we need to promote lead regulatory strategies to reduce lead emissions from numerous lead-related industries. Implementation of standardized lead screening program for children, especially those living in lead-contaminated area, should be done. Establishment of mass lead education that targets public health authorities, pediatricians, as well as parents to increase the awareness of the problem and altering the attitudes related to prevention.

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REFERENCES

1. Fewtrell L, Kaufman R, Prüss-Üstün A. Lead: Assessing the Environmental Burden of Diseases at National and Local Levels Geneva: World Health Organization; 2003.
2. Nriagu JO, Blankson ML, Ocran K. Childhood lead poisoning in Africa: a growing public health problem. *Sci Total Environ.* 1996;181:93–100.
3. Ziegler EE, Edwards BB, Jensen RL, et al. Absorption and retention of lead by infants. *Pediatr Res.* 1978;12:29–34.
4. Woolf AD, Goldman R, Bellinger DC. Update on the clinical management of childhood lead poisoning. *Pediatr Clin North Am.* 2007;54:271–294.
5. Ngueta G, Ndjaboue R. Blood lead concentrations in sub-Saharan African children below 6 years: systematic review. *Trop Med Int Health.* 2013;18:1238–1291.

6. Centers for Disease Control and Prevention. Laboratory Quality Assurance and Standardization Programs. Atlanta, GA:US Department of Health and Human Services, 2014. http://www.cdc.gov/labstandards/.
7. Liu J, Lewis G. Environmental toxicity and poor cognitive outcomes in children and adults. *Environ Health.* 2014;76:130–138.
8. Centers for Disease Control and Prevention. Low Level Lead Exposure Harms Children: A Renewed Call for Primary Prevention Atlanta, GA: Advisory Committee on Childhood Lead Poisoning Prevention, Centers for Disease Control and Prevention; 2012.
9. Kondolf GMM, Mozingo L, Marzion R, et al. Connecting Cairo to the Nile Renewing Life and Heritage on the River. Berkeley: Institute of Urban and Regional Development; 2011.
10. Mekawy HS, Yousry AM. The predicament of a fragmented metropolis. *JUR.* 2012;9:38–51.
11. Basu K. Child labour: cause, consequence, and cure, with remarks on international standards. *J Econ Lit.* 1999;37:1083–1119.
12. Subramanian KS. Performance of diammonium hydrogen phosphate-nitric acid-graphite furnace atomic absorption spectrometric method for urinary cadmium determination. *Clin Chem.* 1987;33:1298.
13. Center for Disease Control and Prevention. Preventing Lead Poisoning in Young Children. Atlanta, GA: US Department of Health and Human Services; 2005.
14. American Academy of Pediatrics Committee on Environmental Health. Lead exposure in children: prevention, detection, and management. *Pediatrics.* 2005;116:1036–1046.
15. Standard Methods for the Examination of Water and Wastewater. Available at: http://www.mwa.co.th/download/file_upload/SMWW_10900end.pdf (accessed October 10, 2014).
16. Commission of the European Communities. Update of 5-years EIA report, review of the implementation of Directive 85/337/EEC, covering the time-period from 1990 to the end of 1996, and undertaken on the Commission's own initiative. Brussels: Commission of the European Communities, Directorate General XI; 1997.
17. Mitra AK, Haque A, Islam M, et al. Lead poisoning: an alarming public health problem in Bangladesh. *Int J Environ Res Public Health.* 2009;6:84–95.
18. Hegazy AA, Zaher MM, Abd el-hafez MA, et al. Relation between anemia and blood levels of lea, copper, zinc and iron among children. *BMC Res Notes.* 2010;3:133.
19. Jain NB, Laden F, Guller U, et al. Relation between blood lead levels and childhood anemia in India. *Am J Epidemiol.* 2005;161:968–973.
20. Centers for Disease Control and Prevention (CDC). Fourth National Report on Human Exposure to Environmental Chemicals, NCEH Pub. Updated tables, No. 05-0570, National Center for Environmental Health, Atlanta, GA, 2010.
21. Schulz C, Angerer J, Ewer U, et al. Revised and new reference values for environmental pollutants in urine or blood of children in Germany derived from the German Environmental Survey on Children 2003–2006 (GerESIV). *Int J Hyg Environ Health.* 2009;212:637–647.
22. Shakour A, El-Taieb N. Effect of atmospheric lead exposure on urban children. *Egyptian J Occup Med.* 1994;18:37–47.
23. Rahbar MH, White F, Agboatwalla M, et al. Factors associated with elevated blood lead concentrations in children in Karachi, Pakistan. *Bull World Health Organ.* 2002;80:769–775.
24. Attia WMK, Abdel-Kareem FM, Mohamed AA, et al. Screening tools detection of children at risk for lead exposure. *Zagazig Univ Med J.* 1996;11:144–153.
25. Nuwayhid IA, Usta J, Makarem M, et al. Health of children working in small urban industrial shops. *J Occup Environ Med.* 2005;62:86–94.
26. Ahmad T, Mumtaz A, Ahmad DI, et al. Lead exposure in children living around the automobile and battery repair workshops. *Biomedica.* 2009;25:128–132.
27. Khan DA, Qayyum S, Saleem S, et al. Lead exposure and its adverse health effects among occupational worker's children. *Toxicol Ind Health.* 2010;26:497–504.
28. Neesanan N, Kasemsup R, Ratanachuaeg S, et al. Preliminary study on assessment of lead exposure in Thai children aged between 3–7 years old who live in Umphang District, Tak Province. *J Med Assoc Thai.* 2011;94:113–120.
29. Wang H, Liu X, Liu Y, et al. Incidence and risk factors of non-fatal injuries in Chinese children aged 0–6 years: a case-control study. *Injury Int J Care Injured.* 2011;42:521–524.
30. Thein MM, Lee BW, Bun PY. Childhood injuries in Singapore: a community nationwide study. *Singapore Med J.* 2005;46:103–105.
31. Tuakuila J, Lison D, Mbuyi F, et al. Elevated blood lead levels and sources of exposure in the population of Kinshasa, the capital of the Democratic Republic of Congo. *J Expo Sci Environ Epidemio.* 2013;23:81–87.
32. Romieu I, Palazuelos E, Meneses F, et al. Vehicular traffic as a determinant of blood lead levels in children: a pilot study in Mexico City. *Arch Environ Health.* 1992;47:246–249.
33. Kaiser R, Henderson AK, Daley WR, et al. Blood lead levels of primary school children in Dhaka, Bangladesh. *Environ Health Perspect.* 2001;109:563–566.
34. Rubin CH, Esteban E, Jones R, et al. Childhood lead poisoning in Russia: a site specific pediatric blood lead evaluation. *Int J Occup Environ Health.* 1997;3:241–248.
35. Yao J, Shen GP. Study on blood lead level among children aged 0–6 years in Huzhou city. *Modern Prev Med.* 2007;34:1716–1718.
36. Cao J, Li M, Wang Y, et al. Environmental lead exposure among preschool children in Shanghai, China: blood lead levels and risk factors. *PLoS One.* 2014;9:e113297.
37. Baghurst PA, McMichael AJ, Wigg NR, et al. Environmental exposure to lead and children's intelligence at the age of seven years: the Port Pirie study. *N Engl J Med.* 1992;327:1279–1284.
38. Kennedy C, Lordo R, Sucosky MS, et al. Primary prevention of lead poisoning in children: a cross-sectional study to evaluate state specific lead-based paint risk reduction laws in preventing lead poisoning in children. *Environ Health.* 2014;13:1–10.
39. Gulson BL, Davis JJ, Bawden-Smith J. Paint as a source of recontamination of houses in urban environments and its role in maintaining elevated blood leads in children. *Sci Total Environ.* 1995;164:221–235.
40. González-Estecha M, Trasobares E, Fuentes M, et al. Blood lead and cadmium levels in a six hospital employee population. PESA study, 2009. *J Trace Elem Med Biol.* 2011;25:S22–S29.
41. Sharaf NE, Abdel-Shakour A, Amer NM, et al. Evaluation of children's blood lead level in Cairo, Egypt. *American-Eurasian J Agric Environ Sci.* 2008;3:414–419.
42. Paoliello MM, De Capitani EM, da Cunha FG, et al. Exposure of children to lead and cadmium from a mining area of Brazil. *Environ Res.* 2002;88:120–128.
43. Schwartz J, Landrigan PJ, Baker EL, et al. Lead-induced anemia: dose-response relationships and evidence for a threshold. *Am J Public Health.* 1990;80:165–168.
44. Froom P, Kristal-Bonch E, Benbassat J. Lead exposure in battery-factors workers is not associated with anemia. *J Occup Environ Med.* 1999;41:120–123.
45. Omar M, Ibrahim M, Hala A, et al. Teeth and blood lead levels in Egyptian schoolchildren: relationship to health effects. *J Appl Toxicol.* 2001;21:349–352.

46. Harari R, Cullen M. Childhood lead intoxication associated with manufacture of roof tiles and ceramics in the Ecuadorian Andes. *Arch Environ Health.* 1995;50:393.
47. Zaki A, el Shazly M, Abdel Fattah M, et al. Lead toxicity among working children and adolescent in Alexandria. *Egypt Eastern Mediterranean Health J.* 1998;4:520–529.
48. Kumar BD, Krishnaswamy K. Detection of occupational lead nephropathy using early renal markers. *J Toxicol Clin Toxicol.* 1995;33:331–335.
49. Grandjean P, Kon SH. Lead exposure of welders and bystanders in a ship repair yard. *Am J Ind Med.* 1981;2:65–70.
50. World Health Organization. Childhood Lead Poisoning (2010) (ISBN 978 92 4 150033 3): http://www.who.int/ceh/publications/childhood-poisoning/en/. Accessed July 9, 2015.
51. Price Maslías J, Castro Nureña C. Serie: Documento de Trabajo, 190. [Regional thematic evaluation: Child labour in the segregation and management of urban solid waste in Latin America and the Caribbean] [In Spanish]. Lima: International Labour Office (ILO), International Programme on the Elimination of Child Labour (IPEC); 2004. Available at: http://www.oit.org.pe/ipec/boletin/documentos/ct190.pdf and http://www.oit.org.pe/ipec/pagina.php?seccion=6&pagina=173 (accessed October 6, 2015).
52. Cuadra S. Child labour and health hazards. Chemical Exposure and Occupational Injuries in Nicaraguan Children Working in a Waste Disposal Site. Lund, Sweden: Faculty of Medicine, Lund University; 2005.
53. Ide LSR, Parker DL. Hazardous child labor: lead and neurocognitive development. *Public Health Rep.* 2005;120:607–612.
54. Nasralla MM. Lead in urban environment. Cairo, pp: 27–29. *Egyptian J Ind Med.* 1984;8:53–63.
55. Wu Y, Huang Q, Zhou X, et al. Study on the effects of lead from small industry of battery recycling on environment and children's health. *Zhonghua Liu Xing Bing Xue Za Zhi.* 2002;23:167–171.
56. Amira T. Child Labor in Egypt (*Doctoral dissertation, Cairo University*) 2011.
57. Tsekrekos SN, Buka I. Lead levels in Canadian children: do we have to review the standard? *Paediatr Child Health.* 2005;10:215.
58. Boreland F, Lyle D. Screening children for elevated blood lead—learnings from the literature. *Sci Total Environ.* 2008;390:13–22.