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STATUS OF LEAD LEVELS IN DRINKING WATER AND BLOOD OF CHILDREN IN KASHMIR, INDIA

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ABSTRACT

Lead is one of the most abundant heavy metals on earth considered as number one environmental persistent toxin and health hazard affecting millions of people round the globe. The diagnosis of lead toxicity has traditionally been based on significantly elevated blood lead levels. Therefore, the most commonly accepted and verifiable biomarker for lead exposure is measuring blood lead level. Present study was conducted in SKUAT-K, Srinagar. Data was collected from families who visited the G.B Panth Hospital, Srinagar; Department of Psychiatry, Govt. Medical College Srinagar for normal health examination. The study was approved by the Ethical Committee of Govt. Medical College, Srinagar. In order to be included in the final data analysis, participants had to fulfill the following criteria: (i) Parental consent to participate in the study, (ii) Children's agreement to participate in the study, (iii) Participants were the residents of Kashmir, and (iv) Children of age group ≤12 years. Exclusion criteria were: (i) Parental dissent to participate in the study, (ii) Children's objection, (iii) Children reported any occupational/accidental exposure to lead, and (iv) Children under any medication. For final analysis 71 subjects (42 boys and 29 girls, 2-12 years) were included in the study. Drinking water samples were also collected to check the status of lead level in drinking water. Water and blood samples were collected by standard methods and processed and analyzed for lead level estimation by Atomic Absorption Spectrophotometer.

Key words: BLL, Children, Drinking Water lead levels

I. Introduction

The elemental symbol of lead is Pb, taken from the Latin word *plumbum*. Its atomic number is 82 and, it is dense, malleable, readily fusible, and has a low melting point. It is a relatively soft metal. Because of these characteristics, lead has been one of the most widely used metals in the history of mankind. The first reported uses of lead dated back to 4000 BC, and toxicological effects have been linked to lead since antiquity. Lead is known to bio-accumulate in most organisms, whereas it is generally not biomagnified up the food web (UNEP, 2010).

Lead is one of the most abundant heavy metals on earth considered as number one environmental persistent toxin and health hazard affecting millions of people round the globe. Environmental lead exposure is still a worldwide problem and has been associated with renal and cardiovascular disease, hematologic toxicity, and irreversible neurologic damage (ATSDR, 2000). As lead poisoning can effect at any age but children are more because of their behavioural patterns that place them at higher risk for exposure to lead, and the body of children retains more percentage of lead they ingest therefore, they exhibit lead toxicity at lower levels of exposure than adults (ATSDR, 2000).

Lead toxicity has also been found to cause degradation of structurally and functionally important phospholipids in cell membranes, with the subsequent of oxidative damage of the biological system (Shafiq-ur-Rehman, 1984; Shafiq-ur-Rehman *et al*., 1995). In most recent investigations in human blood, Shafiq-ur-Rehman (2013) have shown that lead toxicity tarnishes phospholipids classes, which fabric membranes, and damages erythrocytes function with the induction of oxidative stress.

II. MATERIALS AND METHODS

The clinical symptoms in children were assessed for mild ataxia, bilateral wrist drop and a peculiar blue line at the base of gums.

Collection of blood samples

Blood was then drawn from each child from the antecubital vein after carefully washing the puncture site with isopropanol to minimize the potential for external contamination. The blood samples were collected into lead-free heparinised tubes for analysis of Lead.

Digestion of blood samples

The method described by Memon *et al.* (2007) was employed for the digestion of blood samples using Microwave Oven (LG, model: MG-396 WA). A volume of 0.5 ml of whole blood samples was taken into Pyrex conical flask. A volume of 3 ml of freshly prepared mixture of concentrated nitric acid and hydrogen peroxide (HNO_3–H_2O_2; 2:1, v/v) were added into the sample and kept for 10 minutes at room temperature. Thereafter, the samples were transferred in microwave oven with heating at 800W for 3 minutes. The digestion flasks were cooled at room temperature. The samples were separately diluted with 0.1M nitric acid up to the mark in 25 volumetric flasks..

Collection and digestion of drinking water samples

Water samples were collected from North, Central and South Kashmir regions where the subjects belong. The sampling sites were Lal Chowk, Batmaloo and Shalimar (Srinagar) Main Chowk, Tral and Chandgam (Pulwama) Wavoora, Sogam and Lasipora (Kupwara), and Bijbehara, Lal chowk and Yasu (Anantnag). Water samples were collected in polyethylene bottles, and were centrifuged at 6000 rpm to make the suspended particles settle and to obtain clear water sample. A portion of 50 ml of water sample was subjected to acid digestion. For digestion, 5 ml of 6N HNO_3 was added to 50 ml of the sample followed by heating at 70 °C for 30 min and cooled at room temperature.

III. Results and Discussion

The results obtained for lead in drinking water and in blood of the children. The data are given in Mean ± SE.

Mean Lead level (µg/dl) in drinking water of different districts

The table 1 shows lead level (mg/l) of water samples collected from different districts. Lead level in drinking water of district Srinagar was found 0.097 ± 0.015 followed by district Kupwara (0.093 ± 0.015), district Pulwama (0.077 ± 0.014) and district Anantnag (0.070 ± 0.014).

Table – 1 Mean Lead level (µg/dl) in drinking water samples of four districts of Kashmir valley

S. No.	Water Location	Population of children (%)	Lead Level (mg/L) Mean ± SE	WHO Standard (mg/L)
1.	Kupwara	19	0.093 ± 0.005	0.1
2.	Srinagar	20	0.097 ± 0.015	0.1
3.	Pulwama	32	0.077 ± 0.014	0.1
4.	Anantnag	29	0.070 ± 0.014	0.1

Among total number of subjects (71, 2- 12 years age) 39.44% of children had greater than 5 blood lead level while 60.56% possess less than 5 blood lead level (Table 2). In detail, while considering finding percentage of subjects in each age group, It was observed that in age group of 2-4, a sum of 27.78% children were having blood lead level greater than 5 while 72.22% children had less than 5 blood lead level (Table 2). In age group 4-8, 45.16% of children were having blood lead level greater than 5 while 54.84% children had less than 5 blood lead level (Table 2). In age group 8-12, 40.90% of children were having blood lead level greater than 5 while 59.10% children had less than 5 blood lead level (Table 2).

In detail, while considering finding percentage of subjects in gender, It was observed that in gender, 45.23% of males had blood lead level above 5 and 54.76% of males had blood lead level below 5 (Table 2). In female children 31.04% had blood lead level above 5 and 68.96% had blood lead level below 5 (Table 2).

In detail while considering results on percentage of subjects in different father's education groups, It was observed that in children of illiterate fathers 70% of children had blood lead level above 5 while 30% had blood lead level below 5 (Table 2). In children of primary educated fathers, 77.78% of children had blood lead level above 5 while 22.22% had blood lead level below 5 (Table 2). In children of middle pass fathers, 25% of children had blood lead level above 5 while 75% had blood lead level below 5 (Table 2). In children of metric pass fathers, however, 40% of children had blood lead level above 5 while 60% had blood lead level below 5 (Table 2). In children of 10+2 fathers, 50% of children had blood lead level above 5 while 50% had blood lead level below 5 (Table 2). In children of graduate fathers, 36.84% of children had blood lead level above 5 while 63.16% had blood lead level below 5 (Table 2). In children of above graduate fathers, 57.14% of children had blood lead level above 5 while 42.86% had blood lead level below 5 (Table 2).

In addition, the finding pertaining to percentage of subjects in each mother's education group, It was observed that in children of illiterate mothers, 85% of children had blood lead level above 5 while 15% had blood lead level below 5 (Table 2). In children of primary educated mothers, 70% of children had blood lead level above 5 while 30% had blood lead level below 5 (Table 2). In children of middle pass mothers, 81.82% of children had blood lead level above 5 while 18.18% had blood lead level below 5 (Table 2). In children of metric pass mothers, 33.33% of children had blood lead level above 5 while 66.67% had blood lead level below 5 (Table 2). In children of 10+2 mothers, 20% of children had blood lead level above 5 while 80% had blood lead level below 5 (Table 2). In children of graduate mothers, 80% of children had blood lead level above 5 while 20% had blood lead level below 5 (Table 2).

In detail, while considering finding percentage of subjects in each father's occupation group, It was observed that in children of unemployed or unskilled fathers 52.94% of children had blood lead level above 5 while 47.06% had blood lead level below 5 (Table 2). In children of professional fathers, 37.84% of children had blood lead level above 5 while 62.16% had blood lead level below 5 (Table 2). In children of businessmen fathers, 29.42% of children had blood lead level above 5 while 70.58% had blood lead level below 5 (Table 2).

In detail, while considering finding percentage of subjects in each family income group, It was observed that 43.33% of children having average monthly family income less than Rs 50,000 had blood lead level above 5 while 56.67% of children had blood lead level below 5 (Table 2). A portion of 35.71% children, having average monthly family income of Rs 50,000-1, 00,000, had blood lead level above 5 while 64.29% of children had blood lead level below 5 (Table 2). In 46.15% children having average monthly family income above Rs 1, 00,000 the blood lead level was above 5 while 53.85% of children had blood lead level below 5 (Table 2).

If we consider finding with the percentage of subjects in each distance from home to highway traffic group, it was observed that in children having distance from home to highway traffic less than 1Km, 48.28% of children were having blood lead level above 5 while 51.72% of children had blood lead level below 5 (Table 2). According to the distance from home to highway traffic with less than 1-2 Km, 42.86% of children were found having blood lead level above 5 while 57.14% of children had blood lead level below 5 (Table 2). In children having distance from home to highway traffic with less than 2-5 Km, 25% of children were having blood lead level above 5 while 75% of children had blood lead level below 5 (Table 2).

Presenting these finding with percentage of subjects in each group of proximity to school, It was observed that in the group with less than 1 Km proximity to school, 37.74% of children were having blood lead level above 5 while 62.26% of children had blood lead level below 5 (Table 2). In children falling under the group of proximity to school less than 1- 2.5 Km, 44.44% were having blood lead level above 5 while 55.56% had blood lead level below 5 (Table 2).

In detail of the results with percentage of subjects in each group of highway traffic density is presented herein. It was observed that 25% of children residing in low traffic density area were having blood lead level above 5 while 75% had blood lead level below 5 (Table 2). 66.67% of children residing in medium traffic density area were having blood lead level above 5 while 33.33% had blood lead level below 5 (Table 2). 84.62% of children residing in high traffic density area were having blood lead level above 5 while 15.38% had blood lead level below 5 (Table 2).

Considering finding in detail for the percentage of subjects in each residence condition group, it was observed that in children having serious residence conditions, 60% were having blood lead level above 5 while 40% had blood lead level below 5 (Table 2). In children having poor residence conditions, 70% were having blood lead level above 5 while 30% had blood lead level below 5 (Table 2). In children having moderate residence conditions, 71.43% were having blood lead level above 5 while 28.57% had blood lead level below 5 (Table 2). In children having good residence conditions, 64% were having blood lead level above 5 and 36% subjects had blood lead level below 5 (Table 2). In children having excellent residence conditions, 50% were having blood lead level above 5 while 50% had blood lead level below 5 (Table 2).

Table – 2 Relation of selected baseline socio-demographic factors to varying blood lead levels

Socio-Demographic Factors	Blood Lead Levels			
	< 5 µg/dl N = 43		≥ 5 µg/dl N = 28	
	N	%	N	%
Total number of subjects	43	60.56	28	39.44
Age (years)				
2-4	13	72.22	5	27.78
4-8	17	54.84	14	45.16
8-12	13	59.10	9	40.90
Sex				
Male	23	54.76	19	45.23
Female	20	68.96	9	31.04
Father's Education				
Illiterate	3	30	7	70
Primary	2	22.22	7	77.78
Middle	1	75	3	25
Metric	6	60	4	40
10+2	6	50	6	50
Graduate	12	63.16	7	36.84
Above Graduate	3	42.86	4	57.14
Mother's Education				
Illiterate	3	15	17	85
Primary	3	30	7	70
Middle	2	18.18	9	81.82
Metric	10	66.67	5	33.33

10+2	8	80	2	20
Graduate	1	20	4	80
Father's Occupation				
Unemployed/Unskilled	8	47.06	9	52.94
Businessman	12	70.58	5	29.42
Professional (Govt. employee)	23	62.16	14	37.84
Family Income				
Less than ₹ 50,000	17	56.67	13	43.33
₹ 50,000 - ₹ 1,00,000	18	64.29	10	35.71
Above ₹ 1,00,000	7	53.85	6	46.15
Distance from Highway Traffic				
< 1Km	15	51.72	14	48.28
1-2Km	8	57.14	6	42.86
2-5Km	21	75	7	25
Proximity of School to Highway Traffic Density				
<1Km	33	62.26	20	37.74
1-2.5Km	10	55.56	8	44.44
School Highway Traffic Density				
<500	30	75	10	25
500-1000	6	33.33	12	66.67
>1000	2	15.38	11	84.62
Residence condition				
Serious	2	40	3	60
Poor	3	30	7	70
Moderate	6	28.57	15	71.43
Good	9	36	16	64
Excellent	5	50	5	50

Lead has been seen as nuisance affecting individuals of any age, but children were found to be disproportionately affected at lesser levels of exposure than adults (ATSDR, 2000). The socio-demographic characteristic and the living environment in which children are growing and experiencing their living, have vital role in body burden of lead.

In many countries such as Israel (Tepferberg and Almog, 1999), Germany (Jacob *et al*., 2000), Denmark (Nielsen *et al*., 1998), France (Flurin *et al*., 1998), Greece (Vasilios *et al*., 1997), and Sweden (Bergdahl *et al*., 1997), the mean BLL as low as 3.5 µg/dl in urban children were reported. Moreover, regional mean BLL in urban children is found to be 2.2 µg/dl in Canada and USA (Koren *et al*., 1990; CDCP, 2000). The BLL of 2.7 µg/dl was found in urban children of Australia (AIHW, 1996) was similar as found in Japan (Watanabe *et al*., 1996; Zhang *et al*., 1997), New Zealand (Fawcett *et al*., 996) and Singapore (Chia *et al*., 1996, 1997; Neo *et al*., 2000). This was as the U.S.

Government and some other countries followed implementation of environmental and occupational regulations for reduction of lead pollution that resulted in decline of BLL in children by >80% (CDCP, 2000; Howson *et al.,* 1995). However in India and other developing countries, lead poisoning continues to be a major and serious problem where environmental health concern is still inaccessible (Romieu *et al.*, 1997; Krishnaswamy and Kumar, 1998). Riddell *et al*., (2007) conducted a study in Philippines to assess the prevalence of lead poisoning among children with a sample of 2861 which revealed 601 children had BLL more than 10 µg/dl. Recent studies have reported BLL in children of 6.4 µg/dl from South Africa (Mathee *et al*., 2006), 9.3 µg/dl from China (Wang and Zhang, 2006), 15.6 µg/dl from Pakistan (White *et al*., 2002), and 15.0 µg/dl from Bangladesh (Kaiser *et al*., 2001).

In our study performed in non-industrial Kashmir, the mean BLL of 5.15 µg/dl was determined in children of 2-12 years age. More than 51% of children in Indian metros below the age of 12 years have their BLL above 10 µg/dl (Venkatesh, 2009). Ahmad and co-worker (2010) highlighted 9.3 µg/dl mean BLL among children of Lucknow. They further reported that 37% of the children still had BLL >10 µg/dl. A BLL of 11.4 µg/dl was determined by the same group among the newborns in Lucknow during the year 2000 when leaded-petrol was in practice (Srivastava *et al*., 2001) and 54% newborns exceeded the intervention level. Human age has a link with the risk for environmental pollution impact and diseases. Children as compared to adults remain more at risk for lead poisoning as because their smaller bodies are in a continuous state of growth and development (Landrigan *et al.,* 2002).

Gender or sex may have relationship to environmental lead pollution risks and disease generation. An increased vulnerability in male rats to some effects induced by lead exposure such as increased in corticosterone concentration and decreased fixed-interval response rates have been reported (Cory-Slechta *et al*. 2004). Gender or sex may have relationship to environmental lead pollution risks and disease generation. An increased vulnerability in male rats to some effects induced by lead exposure such as increased in corticosterone concentration and decreased fixed-interval response rates have been reported (Cory-Slechta *et al*. 2004). McBride *et al.,* (1982) have reported that association of BLL with father's occupational type such as professional/technical, managerial/administrative/executive, clerical, sales, transport/communication, tradesman/process worker/laborer, service/sport and recreation, had no relationship. Other studies by Lindbohm *et al*. (1991); Sallmen *et al*., (1992) and Winder (1993) suggested a possible relationship between paternal lead exposure and disorders of spermatogenesis among exposed fathers and spontaneous abortion and birth defects among offspring. Studies have evaluated the relationship between maternal and paternal occupational lead exposures and low birth weight among offspring (Savitz *et al.,* 1989; Kristensen *et al*., 1993). A population-based case-control study conducted by Yuan *et al.,* in 1996 suggested a possible relationship between maternal and paternal occupational exposures to lead and low birth weight. Verrula and Noah (1990) reported that occupational exposure is main cause for lead poisoning and is entirely unregulated in many developing countries but little monitoring is conducted in developed countries.

Socio-demographic factors such as low income was reported to be associated with higher blood lead levels (Pirkle *et al.,* 1994). The present study gives explanation for a possible relationship between BLL and low income group since children of unemployed or unskilled fathers were examined with BLL of >5 µg/dl. Whereas, a reverse association between BLL and high income group was found apparent. Our study supports that poor children in developing countries are at especially high risk for lead poisoning (Meyer, PA. 2003). One of the early studies reported IQ score deficits of four points at BLL of 30-50 µg /dl and one to two points at levels of 15-30 µg /dl among 75 black children of low socioeconomic status (Schroeder and Hawk, 1986). Of North American children, 7% have blood lead levels >10 µg/dl, whereas among Central and South American children, the percentage was evaluated 33 to 34% (Payne, 2008). Furthermore, about one fifth of the world's disease burden from lead poisoning occurs in the Western Pacific, and another fifth is in Southeast Asia (Payne, 2008).

Residence condition is one of the important factors that indicate hygiene scenario of the living situation of a family and exposure guide to environmental toxicity. Underprivileged people living in crowded areas in old styled houses in one or two rooms, having poor sanitation/drainage facility, low air quality and light condition, etc. play vital role for diseases and toxic exposure, especially forming worse state of living to children. In developed countries, non white population living in poorer hygienic areas were most at the risk for elevated lead hazards (Pokras *et al*., 2008). In the US however, the people most at risk for lead exposure are the impoverished, city-dwellers, and immigrants (Cleveland, *et al*. 2008). In the United States, African-American children and those living in old housing with poor sanitation have also been found to be at elevated risk for high BLL (Jones, 2009). Low-income people often live in old houses with leaded paint, which may begin to peel and evaporate, exposing residents to high levels of lead-containing dust. Venkatesh (2009) has reported that lead levels of dust in Delhi homes was 31% greater which was dangerous to children who ingest lead from playing close to the ground and having frequent hand to mouth contact. He also reported that in India lead based paints were found in the homes of three children whose blood lead level were 40 µg/dl. He further studied that more than 51% of children in Indian metros below the age of 12 years have their BLL above 10 µg/dl. Considering our finding It was found that children living in poor or below the poor (serious) residence conditions show BLL higher than 5 µg/dl; 60-70% of children were from serious and poor residence conditions. So, residence conditions with good hygienic environment could be healthy for children in respect to toxic exposure of lead and prevention of toxicity and diseases. For underprivileged public the initiative should come from the government agencies.

IV. Conclusion

Lead poisoning is the fore-most environmental health threat to children in most of the developed countries. No attention on children's risks from environmental lead exposure has been seriously given in poor or under developing countries or in India, while it should have been a priority research. The data base on children affected with lead toxicity and estimates of money expended on prevention and healthcare of children are not available. Results of the present study show that the lead contents in drinking water collected from different study sites were below the drinking water norms (0.01 mg/l, IS 10500 : 2012).

The mean BLL of 5.15 µg/dl was determined in children of 2-12 years age. Furthermore, we found that 39 percent of children in Kashmir were found to having BLLs higher than 5 µg/dl. Our finding on selected age groups (2-4, 4-8 and 8-12 years) further demonstrates that 28 percent children in age group of 2-4, 45 percent of children in age group 4-8, and 40.90 percent of children in age group 8-12 were having BLL higher than 5 µg/dl.

Bibliography

[1] Ahmad, M., Verma, S., Kumar, A. and Siddiqui, M. K. J. 2010. Blood lead levels in children of Lucknow. *Indian Environmental Toxicology* **12:** 48-54.

[2] AIHW, Australian Institute of Health and Welfare, 1996. *A report on Lead in Australian children: summary of the National Survey of Lead*. Environment Protection Agency Canberra, Australia, p.45-61.

[3] ATSDR, 2000. Lead toxicity, Agency for Toxic Substances and Disease Registry, Centers for Disease Control and Prevention. Atlanta. http:// www.atsdr.cdc.gov.

[4] Bergdahl, I.A., Schutz, A., Gerhardsson, L., Jensen, A. and Skerfving, S. 1997. Lead concentrations in human plasma, urine and whole blood. *Scandinavian Journal of Work, Environment and Health* **23**: 359-363.

[5] CDCP, 2000. Blood lead levels in young children-United States and selected states, Morbidity and Mortality Weekly Records **49**: 1133-1137.

[6] Chia, S.E., Chia, H.P., Ong, C.N. and Jeyaratnam, J. 1996. Cumulative concentrations of blood lead and postural stability. *Occupational and Environonmental Medicine* **53**(4): 264-268.

[7] Chia, S.E., Chia, H.P., Ong, C.N. and Jeyaratnam, J. 1997. Cumulative blood lead levels and neurobehavioral test performance. *Neurotoxicology* **18**(3): 793-803.

[8] Cleveland, L.M., Minter, M.L., Cobb, K.A. Scott, A.A. and German, V.F. 2008. Lead hazards for pregnant women and children. *The American Journal of Nursing* **108**(10): 40-49.

[9] Fawcett, J. P., Williams, S. M., Heydon, J. L., Walmsley, T.A. and Menkes, D. B. 1996. Distribution of blood lead levels in a birth cohort of New Zealanders at age 21. *Environmental Health Perspectives* **104**(12): 1332-1335.
[10] Flurin, V., Mauras, Y., Le Bouil, A., Krari, N., Kerjan, A. and Allain, P. 1998. Lead blood levels in children under 6 years of age in the Le Mans region. *Medical Press* **27:** 57-59.
[11] Howson, P.C., Hernandez-Avila, M. and Rall, D.P. 1995. Lead in the Americas. A call for action. Institute of Medicine, USA, p.92.
[12] Jacob, B., Ritz, B., Heinrich, J., Hoelscher, B. and Wichman, H.E. 2000. The effect of low-level blood lead on haematological parameters in children. *Environmental Research* **82:**150-159.
[13] Jones, R.L., Homa, D.M., Meyer, P.A., Brody, D.J., Caldwell, K.L., Pirkle, J.L. and Brown, M.J. 2009. Trends in blood lead levels and blood lead testing among US children aged 1 to 5 years, 1988–2004. *Pediatrics* **123**(3): 376-85.
[14] Kaiser, R., Henderson, A.K., Daley, W.R., Naughton, M., Khan, M.H. and Rahman, M. 2001. Blood lead levels of primary school children in Dhaka, Bangladesh. *Environmental Health Perspectives* **109**: 563-566.
[15] Koren, G., Chang, N., Gonen, R., Klein, J., Weiner, L., Demshar, H., Pizzolato, S., Radde, I. and Shime, J. 1990. Lead exposure among mothers and their newborns in Toronto. *Canadian Medical Association Journal* **142**(11)**:** 1241-1244.
[16] Krishnaswamy, K. and Kumar, B.D. 1998. Lead toxicity. *Indian Pediatrics* **35**: 209-216.
[17] Kristensen, P., Urgens, L.M., Daltveit, A.K. and Andersen, A. 1993. Perinatal outcome among children of men exposed to lead and organic solvents in printing industry. *American Journal of Epidemiology* **137**: 134-144.
[18] Landrigan, P.J., Schechter, C.B., Lipton, J.M., Fahs, M.C. and Schwartz, J. 2002. Environmental pollutants and disease in American children: estimates of morbidity, mortality and costs for lead poisoning, asthma, cancer, and developmental disabilities. *Environmental Health Perspectives* **110**: 721-728.
[19] Lindbohm, M.L., Sallmen, M., Antilla, A., Taskinen, H. and Hemminiki, K.1991. Paternal occupational lead exposure and spontaneous abortion. *Scandinavian Journal of Work, Environment and Health* **17**: 95-103.
[20] Mathee, A., Rollin, H., Von Schirnding,Y., Levin, J. and Naik, I. 2006. Reduction in blood lead levels among school children following the introduction of unleaded petrol in South Africa. *Journal of Environmental Research* **100:** 319-322.
[21] McBride, W.G., Black, B.P. and Brian, J.E. 1982. Blood lead levels and behavior of 400 pre-school children. *The Medical Journal of Australia* **1**: 26-29.
[22] Memon, A.R., Tasneem, G.K Hassan, I.A. and Nasreen, S. 2007. Evaluation of Zinc status in whole blood and scalp hair of female cancer patients *International Journal of Clinical Chemistry and Diagnostic Laboratory Medicine* **10***:*54-64.
[23] Meyer, P.A., Brown, M.J. and Falk, H. 2008. Global approach to reducing lead exposure and poisoning. *Mutation research* **659**(2): 166-75.
[24] Neo, K. S., Goh, K.T. and Sam, C. T. 2000. Blood lead levels of a population group not occupationally exposed to lead in Singapore. *Southeast Asian Journal of Tropical Medicine and Public Health* **31**: 295–300.
[25] Nielsen, J. B., Grandjean, P. and Jorgensen, P. J. 1998. Predictors of blood lead concentrations in the lead-free gasoline era. *Scandinavian Journal of Work, Environment and Health* **24**: 153-156.
[26] Payne, M. 2008. Lead in drinking water. *Canadian Medical Association Journal* **179**(3): 253–4.
[27] Pirkle. J.L., Brody, D.J. and Flegal, K.M. 1994. The decline in blood lead levels in the U.S.: The National Health and Nutrition Examination Surveys (NHANES). *Journal of American Medical Association* **272**: 284-291.
[28] Pokras, M.A. and Kneeland, M.R. 2008. Lead poisoning: using transdisciplinary approaches to solve an ancient problem. *Ecohealth* **5**(3): 379-85.
[29] Riddell, T. J., Solon, O., Quimbo, S.A., Tan, C.M.C., Butrick, E. and Peabody J.W. 2007. Elevated blood-lead level among children living in the rural Philippines. *Bulletin of World Health Organization*. **85**(9): 674-680.
[30] Romieu, I., Lacasann, M. and McConnell, R. 1997. Lead exposure in Latin America and the Caribbean. *Environmental Health Perspectives* **105**: 398-405.
[31] Sallmen, M., Lindbohm, M.L., Antilla, A., Taskinen, H. and Hernminki, K. 1992. Paternal occupational lead exposure and congenital malformations. *Journal of Epidemiology and Community Health* **46**: 519-522.
[32] Savitz, D.A., Whelan, E.A. and Kleckner, R.C. 1989. Effect of parents occupational exposures on risk of stillbirth, preterm delivery, and small for gestational age infants. *American Journal of Epidemiology* **129**: 1201-1218.
[33] Schroeder, S.R., Hawk, B., Otto, D.A., Mushak, P. and Hicks, R.E. 1986. Separating the effects of lead and social factors on IQ. *Environmental Research* **38**:144-54.
[34] Shafiq-ur-Rehman. 1984. Lead-induced regional lipid peroxidation in brain. *Toxicology Letters* **21**: 333-338.
[35] Shafiq-ur-Rehman. 2013. Effect of lead on lipid peroxidation, phospholipids composition, and methylation in erythrocyte of human. *Biological Trace Element Research* **154**: 433-439.
[36] Shafiq-ur-Rehman., Chandra, O. and Abdulla, M. 1995. Evaluation of malondialdehyde as an index of lead damage in rat brain homogenates. *BioMetals* **8:** 275-279.
[37] Srivastava, S., Siddiqui, M. K. J., Mehrotra, P. K., Srivastava, S.P. and Tondon, I. 2001. Blood lead and zinc in pregnant women and their offspring in intrauterine growth retardation case. *Analytical Toxicology* **25**: 461-465.

[38] Tepferberg, M. and Almog, S. 1999. Prenatal lead exposure in Israel: an international comparison. *Israel Medical Association Journal* **1**:250-253.
[39] Vasilios, D., Theodor, S., Konstantinos, S., Evangelos, P. E., Fotini ,K. and Dimitrios, L.1997. Lead concentrations in maternal and umbilical cord blood in areas with high and low air pollution. *Clinical and Experimental Obstetrics and Gynecology* **24**:187–189.
[40] Venkatesh, T. 2009. Global perspective of lead poisoning. *Al-ameen Journal of Medical Science* **2**(2): 1-4
[41] Verrula, G.R. and Noah, P.K. 1990. Clinical manifestations of childhood lead poisoning. *Journal of Tropical Medicine and Hygiene* **93:** 170-177.
[42] Wang, S. and Zhang, J. 2006. Blood lead levels in children, China. *Environmental Research* **101**: 412-418.
[43] Watanabe, T., Nakasuka, H., Shimbo, S., Iwami, O., Imai, Y., Moon, C. S., Zhang, Z. W., Iguchi, H. and Ikeda, M. 1996. Reduced cadmium and lead burden in Japan in the past 10 years. *International Archives of Occupational and Environmental Health* **68**(5): 305-314.
[44] White, F., Rahbar, H. and Agboatwalla, M. 2002. Elevated blood lead in Karanchi children. *Bulletin of World Health Organisation* **79**: 173-175.
[45] Winder, C. 1993. Lead, reproduction and development. *Neurotoxicology* **14**: 303-318.
[46] Zhang, Z. W., Moon, C. S. and Watanabe, T. 1997. Background exposure of urban populations to lead and cadmium: comparison between China and Japan. *International Archives of Occupational and Environmental Health* **69**: 273-281.

