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Lead Exposure in Children through Water and Soil

Abstract:

Lead is a metal which has the ability to spread in the earth's crust and has corrosive property. It is a naturally occurring metal which is soft in nature. Lead exposure in children is through various pathways and the major concentrated sources are the soil and drinking water. Children are most susceptible to lead exposure is due to their growing/developing bodies which are very sensitive to lead. Lead poisoning in children is a preventable environmental disease affecting many children around the world. This paper discusses how soil and water plays a major role in lead exposure to children's routine life. The adverse effects of lead poisoning range widely from delayed to severe health outcomes. According to the Center for Disease Control and Prevention (CDC) the blood lead levels should be no higher than 5µg/dl, which is considered as the new reference value. The epidemiological studies in the article details some statistical evidence that how children are affected by lead exposure through soil and water. Animal behavioral studies are mentioned in order to compare the toxic levels to humans. Even though there has been decline in the blood lead levels in children from 1970's, very small amounts of blood lead can also result in various health outcomes. These blood lead levels in children are due to the lead based paints in old houses and the service lines made by lead and lead solder. Due to some regulatory interventions by the state and federal agencies the developed countries have reduced the lead exposure to an extent, but the developing countries are still at greatest concern in childhood lead poisoning.

1 Introduction:

Lead is a metal which has been used by humans over 5000 years and has properties of corrosion-resistant, ductile, dense and malleable. Lead production has increased with increase in population and economic growth. For Native Americans the estimated blood lead levels were calculated as $0.016 \mu g/dl$ before European settlement in the America when usage of lead was less, whereas the estimated average blood levels in 1999-2004 was 1.9 $\mu g/dl$. These variations in the above mentioned estimates are due to the usage of lead during 1900-1975, and also are the indicators that the sources of lead exposure exist in the environment (Mary Jean Brown & Margolis, 2012). Blood lead levels are minimal in the absence of industrial activities. In the United States the two major sources identified historically are the airborne lead form the combustion of gasoline and the leaded chips and dust mostly from deteriorating lead paint. These two sources results in contamination of the soil. Water in lead is an essential source of lead contamination especially in children. However, a gradual decrease in airborne exposure has occurred since 1980 and also is no longer a community source of exposure (Village, 2005).

Lead is one of the toxic elements which occur naturally in the earth's crust. Lead has some beneficial uses and also harmful effects on both humans and animals. The beneficial uses of lead are it is used in automobile industries in the form of batteries, used as coloring pigment, as soldering material. The main exposure route of lead is from human activities such as usage of fossil fuels, industrial facilities, and past usage of lead based paints (EPA, 2015). After many studies and efforts done by federal agencies, the lead exposures have been declined to some extent in children and adults. However, chronic lead toxicity is still alarming as a major public health issue in the United States (National Academy of Sciences, 1993). Over the past years children in the United States have shown declined levels of blood levels from 890,000 in 1991-1994 to 310,000 in 1999-2002 (Mary Jean Brown& Jacobs, 2006). This makeable decline is due to the efforts made by federal, state and local efforts to develop a pool of lead- safe housing in the communities who are more prone to lead toxicity (Mary Jean Brown& Jacobs, 2006). According to the Centers for Disease Control and Prevention (CDC) approximately 2 million preschool children were estimated to have blood levels more than the federal guidelines of 10µg/dl and 200,000 had levels above 25µg/dl (Todd et al., 1996).

Lead toxicity is a preventable health problem. Efforts should be made to reduce or avoid the usage of lead consumption by using it in certain products like batteries, solder, pipes, ammunition, roofing and x-ray shielding (Todd et al., 1996). This lead exposure will continue to be a threat for years. "The American epidemic of lead toxicity is not yet over." (Wedeen, Ty, Favata, & Jones, 1995). In children, age groups between 1 to 5 years are at more risk for lead poisoning. The signs and symptoms of lead poisoning are very much similar to other diseases in childhood like vomiting, abdominal pain, constipation and often meningitis is confused with encephalitis and in late 90's meningitis was diagnosed as intra cranial tumors (Rabin, 1989). Recent research illustartes negative health effects like learning disabilities and behavioral disorders in children having lead levels lower than $2\mu g/dl$. However in contrast to these studies, the CDC has specified that there is no safe blood lead level in children (Miranda, Anthopolos, & Hastings, 2011).

2 <u>Common Sources of Lead</u>:

Lead pathways are through the air, soil, water and including our homes. Children's environment is almost surrounded by lead, such as solder and consumer products. The other sources through which children are exposed through are artificial turf, candy, food additives, toy jewelry, toys and water, which are used in their daily routine life (CDC, 2015). The other sources are preventable easily by avoiding the usage of these toys which does not play a major role and can be replaced with other alternatives, for example lead free plastics to prepare toys and jewelry.

2.1 The Air:

Lead smelters contain the highest concentrations of lead. Various sources of lead in the air are ore, metals processing, piston-engine aircraft operating on leaded aviation fuel, waste incinerators, and lead-acid battery manufactures (EPA, 2014). In U.S. "The Doe Run Company" was one of the largest sources for lead emissions, which released large amounts of lead in air. Also there are many other facilities (battery recyclers, smelters, metal foundries power plants) in U.S which were estimated up to 200 facilities emitting between one-half and 1 ton of the metal per year. In 2008, EPA reduced the lead National Ambient Air Quality Standard (NAAQS) from 1.5μ g/m³ to 0.15μ g/m³ (Schmidt, 2010). According to EPA studies in 1970's and 1980's automobiles produced huge amounts of lead into the environment than industrial sources. However, over time there was a decline in the lead emissions into the atmosphere through the Clean Air Act (Callender & Van Metre, 2011).

2.2 The Soil:

The major source in the soil is from the lead-based paint from our homes. It is also contaminated from gasoline cars and industrial sources (EPA, 2015). Soil is the most common source through which children are exposed to lead (Muir & Campbell, 1995). Due to the lead based paints at home, the exterior paint fades and gets deposited in soil. The most common method due to which children get exposed to lead is through hand to mouth contact (Xintaras, 1992; Jacobs et al., 2002). Children of approximately 5.9 to 11.7 million are at risk in being exposed to lead through contaminated soil/dust (Xintaras, 1992). Lead's duration in the soil is of longer period time. The mobility of lead in soil is due to its pH, humid acids and organic matter. The acidic conditions in the soil play a major role in solubility of lead (Hansen *et al.*, 2004). The lead adsorbed in soil is to organic matter and might enter the surface waters which further results in erosion of lead- containing soil particles (U.S. ATSDR, 2005).

2.3 The Water:

The major source of lead in water is through the corrosion of plumbing materials, homes built before 1986 have lead pipes, fixtures and solder. It is mostly found in the hot water due to the brass faucets and fixtures with lead solder (EPA, 2015). The only way to avoid the lead exposure is to removal of old pipes, which is an expensive way but one of the effective way to reduce lead exposure from water (WHO, 2015). According to researchers, studies done on contaminated water inferred that lead contribution in drinking water results in blood lead. Seven percent of blood lead is due to 10µg/dl of lead in drinking water; these have been estimated for adults in general population (Bois, Tozer, Zeise, & Benet, 1989). The pollution that comes from industries is one of the sources to contaminate water by lead. The quantity of lead that gets dissolved in water depends on factors like pH, temperature, water hardness and standing time of the water (WHO, 2015). Chloramine causes unintended release of lead from plumbing materials in to the drinking water (Edwards, 2014). Erosion of natural deposits and corrosion of plumbing systems in homes are the important sources of lead in drinking water. A chemical reaction between water and plumbing occurs when lead leaches into water through corrosion from pipes, solder and fixtures (EPA, 2015b). Schools and child care facilities have irregular water use patterns, which may have elevated lead levels in drinking water. Even though water is a source of no lead or very low levels of lead, children are affected by the chronic levels even today and are suffering from neurological disorders (EPA, 2015a). Other important source through which the infant gets exposed to lead is the "Transplacental Exposure and Lead in Human Milk".

2.4 Prenatal Exposure:

Lead is a metal which can cross the placenta. The source of lead in infant's blood is the mixture of dietary and skeletal lead. The dietary lead is about two-thirds and the skeletal lead is of one-third. The concentrations of lead levels in blood are same in both the infants and mother. (Village, 2005). Lead levels in human milk are comparatively low than the plasma lead levels, therefore the mobility of lead through milk is in very small amounts (Village, 2005). Maternal blood can have higher proportion of lead and its exposure from water when compared to lead paint in the age group of 1.5 to 6 years (Edwards, 2014).

3 Significance of topic:

The exposure of lead is through various sources and has major impact on children like behavioral problems, learning disabilities and other health effects. Lead toxicity in children is preventable. In 1991, CDC established an action for defining risk for having major issues in IO and cognitive development. The established lead blood level in children was 10 µg/dl for children aged less than or equal to 6 years (Jones et al., 2009). Approximately 24,000 children age group of six years or less had blood lead levels of greater than 10 µg/dl and 243,000 children of the same age group had blood lead levels greater than 5 µg/dl. These readings were obtained in the year of 2010, in a total of 34 states in the United States and also the District of Columbia (Raymond, Wheeler & Brown, 2014). The CDC's new reference value 5µg/dl for lead has been based on 97.5 percentile of the National Health and Nutrition Examination Survey (NHANES)'s blood distribution in children (Roberts, Allen, Ligon, & Reigart, 2013). The two common sources through which children get exposed are the soil and water. The most concentrated source of lead exposure in the soil is due to the deteriorating lead paint at home and dust. Another major source of lead exposure is water. The higher the concentration of lead in drinking water, higher is the amount of lead contaminated water consumed. In children the blood lead levels associated with lead in drinking water is greater than or equal to 10 µg/dl (Mary Jean Brown & Margolis, 2012).

4 **<u>Clinical effects</u>**:

Lead toxicity can result in various health effects and affect every organ in our body. Children have a greater risk to lead toxicity due to their growing bodies and sensitive nervous systems. The exposure effects are seen over long period of time and are irreversible and untreatable (Needleman, Schell, Bellinger, Leviton, & Allred, 1990). In children, the primary routes of lead absorption are through ingestion and respiration. The gastrointestinal tract of children can absorb ingested lead in higher rates than that of adults (Lidsky & Schneider, 2003). The ingestion of lead can occur through playing in the soil and drinking water which is contaminated by lead and also the paint dust at homes can result in inhaling the lead particles. In the body absorbed lead is carried by erythrocytes up to 95% and the remaining 5% is through the plasma to the tissues. The half- life of lead in blood is 35 days which is equivalent to an erythrocyte, in brain it is nearly 2 years and in bone it can be up to longer periods (Lidsky & Schneider, 2003).

These toxic effects vary from acute, clinical, symptomatic poisoning and subclinical which can be at lower levels of lead exposure. The major organs affected are central and peripheral nervous system, cardiovascular, gastrointestinal, renal, endocrine, immune and haematological systems (World Health Organization, 2010).

4.1 Acute Clinical Toxicity:

In acute toxicity, children are exposed to a very high levels of lead, initially this exposure would lead to symptoms like lethargy, abdominal cramps, anorexia and irritability (Lidsky & Schneider, 2003). In younger children below two years show symptomatic poisoning over a period of time. The symptomatic poisoning can be characterized by colic, constipation, fatigue,

anemia and neurological features. Fatal acute encephalopathy with ataxia, convulsions and coma can occur in most severe cases (World Health Organization, 2010). Children who can tolerate high exposure of lead are either cognitively compromised or mentally retarded. These children are characterized with a blood lead level of 70 μ g/dl and in few children it may be at 50 μ g/dl (Adams, Victor, & Ropper, 1997).

4.2 Subclinical Toxicity:

It can be termed as low-dose exposure of lead for longer period of time. During this exposure the clinical symptoms might be not obvious but the toxic effects are very harmful. This type of toxicity is based on dose-related range of toxic effects (Landrigan, 1989; Bellinger & Bellinger, 2006)

Blood lead levels	Health Effects
150µg/dl	Death
Below 100 µg/dl	Nephropathy Encephalopathy
	Frank anemia Colic
$40 \ \mu g/dl - 50 \ \mu g/dl$	Decreased hemoglobin synthesis
30 µg/dl	Increases vitamin D metabolism
20 μg/dl to 10 μg/dl	Increased nerve conduction velocity Increased level of erythrocyte protoporphyrin Decreased Vitamin D Metabolism Decreased calcium homeostasis
Below 10 µg/dl	Decreased IQ level Decreased Hearing Decreased growth Impaired peripheral nerve function Trancplacental transfer

Table: Health effects associated with ranges of blood lead levels in children

4.3 Haematological Effects:

Lead toxicity in erythrocytes is a usual clinical indicator of anemia. It is commonly seen in young children with iron deficiency. The anemia caused by lead is due to the impairment of heme biosynthesis or due to the increased rate of erythrocyte destruction (Schwartz, Landrigan, Baker, Orenstein, & Von Lindern, 1990). A study done in preschool children from Montevideo, Urugay, there was an association between anemia and blood lead concentrations in the children. In this study low hemoglobin was an important predictor of blood lead concentration of 10 μ g/dl, especially among children less than 18 months of age (Queirolo, Ettinger, Stoltzfus, & Kordas, 2010).

4.4 Neurological Effects:

The Neurological effects act both on peripheral nervous system and also central nervous system. Motor axons are affected in the peripheral nervous system during long term exposure to lead, which results in segmental demyelination and axonal degeneration. The symptoms would be ankle drop or extensor muscle palsy with wrist (World Health Organization, 2010). Lead in central nervous system results in asymptomatic impairment of neurobehavioral function. Many studies were conducted to rule out the association between lead and intelligence quotient (IQ) (Needleman et al., 1979). Recent studies have estimated that for children ranging 10-20 μ g/dl of blood lead levels lost a quarter to half of an IQ point for each 1 μ g/dl increase in the blood lead levels (Schwartz, 1994).

4.5 Effects of prenatal exposure:

Lead as a neurotoxin can pass through the placenta to the fetus, when the mother is having high blood lead levels. The maternal blood lead level and umbilical cord levels are found to nearly same. This lead is accumulated in the bone for long period of time in the mother. This lead is the major source for the blood lead during pregnancy and post-partum period. This lead may result in cognitive development of children, with a range of 10 μ g/dl placental blood lead levels. This is due to the penetration of lead in to the immature blood- brain barrier to pass in the developing brain (Lidsky & Schneider, 2003). The lead which occurs in the breast feeding is much lesser in concentration when compared with the blood lead levels and also the amount of lead transferred is very minimal (World Health Organization, 2010).

5 Epidemiological studies on lead

5.1 Studies of Lead in Water:

The health consequences of contaminated water with lead in the United States were identified in early 1845. The survey conducted in 1990 nationwide, around 3.3 million lead water service lines were used and according to this estimates, nearly 61,000 lead service lines have been removed in the previous 10 years (Brown & Margolis, 2012). A study conducted in Edinburgh, Scotland during 1972, collected 949 first-flush water samples (stagnant water samples in plumbing pipes for 6 years) were equivalent with 949 blood lead levels and also running water samples of 205 were equivalent to 205 blood lead levels. These results in the study have challenged the necessity to lower the water lead concentration to less than 100 ppb. According to WHO, acceptable lead concentration was 100 ppb in 1972. (Brown & Margolis, 2012).

Another study was to determine whether residential water sampling supports the belief of the formation of stable lead oxide coating on lead service lines (LSLs) provides an effective lead release control strategy. The samples were from the eight home kitchen taps in three U.S. cities with noticed lead oxide coated LSLs. These LSLs released lower or equal lead levels after different water standing times (1 to 18 μ g/L) when compared to the lead levels from the kitchen pipes, which was 1 to 130 μ g/L. The maximum contaminant level is 15 μ g/L and "zero" as maximum contaminant level goal (ATSDR, 2007). Due to the prolonged stagnation (10-101h) at Cincinnati sites, the results were varying from minimal 0-4 μ g/L increase in one site and 3 fold

increase in another site. These results concluded that the lead release was lower than the previous lead (II) coated LSL case studies, suggesting that natural formation of lead oxide in LSLs is an actual lead "corrosion" control strategy (Triantafyllidou, Schock, DeSantis, & White, 2015).

An ecological study was conducted to note the fetal death rates during the Washington DC drinking water "lead crisis" during 2000-2004. These elevated lead levels in drinking water due to the change in the water disinfectant from chlorine to chloramine. These Water lead levels were controlled after 2006 by treating with orthophosphate, which is a corrosion inhibitor. The study states that the water lead levels in DC were highest during 2001 and decreased during 2004 due to the public health interventions programs to protect pregnant women. The fetal death rates and water lead levels were compared in between two cities in U.S. due to the similarities in risk factors for miscarriage. In DC, 2007 and 2009 the fetal death rates increased due to the removal of public health intervention programs and had the consistent high water lead levels. Whereas, in Baltimore City the water lead levels, childhood lead poisoning and fetal death rates declined from 2000 to 2009 (Edwards, 2014).

Another study was conducted in Uganda in 2005, to rule out the possible risk factors for the exposure of lead. The study was conducted among the school children of Kampala, collected blood samples, questionnaire data, and soil samples and other possible risk factors like water in the communities were collected from the schools and homes of 163 children aged 4 to 8 years. The results of this study included mean blood lead levels which were 7.15 μ g/dl and 20.5% of the children were having elevated blood lead levels. The study concluded that the possible risk factors played a major role such as canned food, and the primary source was the community water supply for elevated blood levels. Whereas the soil/dust lead was not significantly predictive of elevated blood levels (Graber et al., 2010).

The study is to assess the blood lead levels and risk factors in young children in France in 2008-2009. It was a cross-sectional study, which included 3831 children who were hospitalized. Blood samples, socio-demographic characters and environmental data which include soil and water samples were collected. The blood lead levels were at high levels and the environmental factors associated with blood lead levels were the tap water consumption at homes with lead service connections, peeling of old paints and hand to mouth behavior (Etchevers et al., 2014).

5.2 Studies of lead in Soil:

To rule out children's death due to lead poisoning in villages of Northwestern Nigeria, Dooyema et al., conducted a study, in which they collected blood from 2-59 months of age children and obtained soil samples from family compounds. In the survey of 119 family compounds, 25 % of children had died and 59% were lead poisoned and 97% children had blood lead levels greater than 45 μ g/dl. At this blood lead level decreased hemoglobin synthesis occurs. The results also included two-thirds of the family compounds contained processed gold-ore. The gold ore mining was the major source for contaminating soil due to lead weathering which occurred naturally from the gold ores. The possible risk factors concluded in this study were the lead poisoning associated with the age of child, mother's work at ore- processing activities, community well as primary water source, and the soil concentrations in the compounds (Dooyema et al., 2012). The study was conducted on the lead contamination due to lead smelting and resulted in epidemic of lead poisoning in children. The soil samples from 79 locations were collected and bio-accessible lead concentration were evaluated before and after remediation with 60 mmol/ kg Ethylenediaminetetraacetic acid EDTA. This extraction had shown positive results in reducing the soil lead concentration in the towns. The locations were interpreted, where the blood lead levels were higher than the expected (10 μ g/dl) would reduce to much higher extent after remediation of the soil. The results inferred that the soil washing with EDTA is an efficient way to reduce the lead contamination and also soil capping in Slovenia towns in Europe (Jez & Lestan, 2015).

This study evaluated lead concentrations in farmland soil, hair and blood of residents in Henan Province surrounded by a lead smelter. The main objective of this study was to disclose the contamination of soil by lead smeltery. The lead concentrations in the 66.7% of topsoils (0-20cm) were crossing the grade II (350 mg/kg, pH >7.5) of the standards of National Soil Environmental Quality (GB 15618-1995 standards of China) and reaching the maximum level up to 1687 mg/kg. The standard level for soil contamination according to EPA is 400 mg x kg¹ in play areas and 1200 mg x kg-1 in non-play areas and the pH should be maintained at 6.5 or above. This high level of soil contamination is the crucial source for the children's health problems near the smeltery, as children tend to play in the soil and have hand to mouth contact and have dermal exposure to the contaminated soil (Zhou et al., 2013).

A study was done on children in three states of Russian cities. The kindergarten school children were participants, who were close to the industrial areas and major traffic corridors. The capillary blood samples were collected and also tap water, indoor dust and soil samples in the play areas and also paint on the walls. Overall 23% of these children had elevated blood levels, 2% were anemic and the city Krasnouralsk had detectable levels of lead in all the soil samples and also 4% children were anemic and 60% children had elevated levels of blood. This study concluded that the elevated levels are associated with soil and dust contaminated by lead and also the source of contamination is through the lead-related industrial emissions (Rubin et al., 2002).

6 Toxicological Studies on Lead:

Animal behavioral studies are more plausible for comparing the lead toxicity in humans. The reasoning that low-level exposure to lead results in human behavioral illness becomes more accurate when these behavioral changes can be observed after lead is administered to animals in experimental settings. The similarities between the general outline of behavioral abnormalities in lead exposed animals and in any range lead exposed children provides support, although indirect, for the reading that the relationships observed in humans are contributory. But, sometimes these results of animal studies do not reflect contributory influence of humans. The Animal studies on lead toxicity are very useful in having a clear understanding on childhood lead toxicity with the only reason is, it allows deep level of analysis which allow in the effort to identify the behavioral mechanism of functional defect (Bellinger, 2004).

6.1 Study on behavioral deficits:

The behavioral deficits observed in children due to lead exposure have been observed in studies on animals, especially monkeys. For an example, in an animal study, Rhesus monkeys were treated orally from birth at different lead doses ranging from 4 μ g/dl, 32 μ g/dl and 65 μ g/dl. The results observed were the treated monkeys attended to irrelevant cues in a systematic way. Therefore, the study suggests that the constant lead exposure at different developmental period can affect the behavior (Rice, 1990).

6.2 Study on hearing deficits:

The analysis done in Second National Health and Nutrition Examination Survey data set had results with uncertain lead levels in blood due to community exposures (Ex: drinking water) were associated with increased hearing threshold in children. But in an animal study done on 31 rhesus monkeys, in the first 2 postnatal years with 35 to 40 μ g/dl suggested a doubt on the conclusions of the low lead exposures can result in hearing defects in children.(Lasky, Luck, Torre, & Laughlin, 2001).

6.3 Study on developmental anomalies:

Hamsters, chicks and rats when subjected to prenatal lead exposure have resulted in a characteristic pattern of malformations. Prominent malformations seen in the animals were urorectocaudal malformation, vertebral, lower extremity and hydrocephalus. Genital anomalies and hydrocephalus was observed in animal models of lead embryopathy and these malformations were associated with the Vertebral, Anal atresia, Cardiac, Tracheo-Esophageal, Renal, Limb anomalies (VACTERL) in patients (McClain & Becker, 1975).

6.4 Study to reduce lead levels from the body:

Many studies were conducted to reduce the lead exposure and blood lead levels. An animal study has been done on succimer chelation to reduce the level of lead from the body. In the primate study the succimer treatment was effective at increasing the elimination of lead from the body. The results also showed that the brain lead levels were reduced with succimer chelation for 21-day treatment regimen in rodents. But the blood levels were poor surrogate and the reduction of lead levels were poor predictor of the chelation treatment (Smith & Strupp, 2013).

6.5 Study on visual defects:

The studies done in rats was to assess the relation between the visual problems in infants with elevated blood lead levels due to breast feeding. The study was in agreement with the findings in humans. The rats exposed to lead through mother's breast feeding had 19 μ g/dl lead blood levels, resulted in retinal sensitivity caused due to selective alterations of the rods (Fox, Campbell, & Blocker, 1997).

7 Methods and Interventions to Reduce Lead Exposure:

In children with blood lead concentrations greater than 45 μ g/dl are treated with chelating agents, these agents can lower the mortality of acute lead encephalopathy but the majority of lead

is not removed from the bone and does not reverse the neuropsychological effects (Dietrich et al., 2004). The primary goal for the management of lead as public health issue is to prevent the lead induced effects by controlling lead hazards in the environment (Chisolm, 2001). The prevention of lead exposure in children can be mainly by removing the environmental source in community as well as individual level. The next level of prevention would be identifying children at risk from their environment and reduce further exposure (Campbell & Osterhoudt, 2000). The three main interventions which can play a major role in prevention techniques are:

- Environmental interventions: These interventions primarily focuses on improvement in risk-assessment, improving the standards of housing for lead-based paint effects, reduce the contamination of lead in soil and water. This can be done by replacing the lead pipes of water and removal or covering of the contaminated soil. These are to be done in safe and cost-effective manner (Campbell & Osterhoudt, 2000). According to CDC some of the interventions are to visit houses or buildings built before 1950 and also remodeled houses. Check siblings who had lead poisoning. The blood lead levels of children must be tested at 1 to 2 years, and 3-6 years if they haven't been tested for lead.
- Educational Interventions: These interventions are based on bringing awareness among parents and children. The awareness programs are primarily on prenatal awareness of lead exposure pathways, home cleaning, hygiene and control measures to prevent ingestion of lead through soil and water (Campbell & Osterhoudt, 2000). CDC implemented public assistance programs for the poor such as Medicaid or the Supplemental Food Program for women, infants and children (Public Health Statement for Lead, 2007).
- **Regulatory Interventions:** These are the regulations made by government around the world to protect the people against the risks of lead- contamination in soil, water, air and other consumer products. The acute and chronic exposure of lead can be prevented by the regulations and bans of products on lead based paint, air, drinking water, work place environments and other consumer products (Pfadenhauer, Burns, Rohwer, & Rehfuess, 2014). EPA suggests the concentration of lead in air to be no higher than 1.5 micrograms per cubic meter averaged for 3 months. The EPA regulations have banned lead in gasoline and also the sale of leaded gasoline in 1995. EPA suggests testing of public water systems, and if 10% the samples contain lead levels over 0.015 milligrams per liter, then measures should be taken to reduce these levels. Tests on drinking water at schools and daycare centers should be done if they are regulated under public water system (Public Health Statement for Lead, 2007).

8 Conclusion:

The wide distribution and mobilization of lead in the environment has resulted in increased uptake and exposure to lead. This exposure to lead had concluded several complicated health hazards among the socio-economically poor children. They tend to live in old houses where lead concentrations are high in the paint, and are nutritionally poor. Over the past 20 years the blood lead levels have been decreased in the developed countries due to bans on lead releasing products and other consumer products. In developing countries due to the growth in industrialization, the persistence of lead in the environment can remain as a public health issue for several years. But the major concern is regarding the chronic level exposure due to its adverse effects especially on children. The effects of lead in children are mostly irreversible. In

spite of reducing or eliminating lead levels in the body, the neurophysiological disturbances cannot be improved. Childhood lead poisoning prevention programs should be implemented in schools and communities. Children are exposed to many sources of lead in spite of having elevated blood lead levels. Hence, all sources at homes should be assessed.

References:

- Adams, R. D., Victor, M., & Ropper, A. H. (1997). *Principles of neurology*. New York: McGraw-Hill, Health Professions Division.
- ATSDR Environmental Medicine & Environmental Health Education CSEM. (2007, August 20). Lead (Pb) Toxicity: What Are the U.S. Standards for Lead Levels? |. Retrieved from http://www.atsdr.cdc.gov/csem/csem.asp?csem=7&po=8
- Bellinger, D. C. (2004). Lead. PEDIATRICS, 113(4 Suppl), 1016–1022.
- Bellinger, D. C., & Bellinger, A. M. (2006). Childhood lead poisoning: the torturous path from science to policy. *The Journal of Clinical Investigation*, 116(4), 853–7. http://doi.org/10.1172/JCI28232
- Bois, F. Y., Tozer, T. N., Zeise, L., & Benet, L. Z. (1989). Application of clearance concepts to the assessment of exposure to lead in drinking water. *American Journal of Public Health*, 79(7), 827–31.
- Brown, M. J., & Jacobs, D. E. (2006). Sources of blood lead in children. *Environmental Health Perspectives*, 114(1), A18–A19. http://doi.org/10.1289/ehp.7713
- Brown, M. J., & Margolis, S. (2012). Lead in drinking water and human blood lead levels in the United States. *Morbidity and Mortality Weekly Report. Surveillance Summaries (Washington, D.C. : 2002)*, 61 Suppl, 1–9.
- Campbell, C., & Osterhoudt, K. C. (2000). Prevention of childhood lead poisoning. *Current Opinion in Pediatrics*, *12*(5), 428–37. http://doi.org/10.1097/00008480-200010000-00002
- Chisolm, J. J. (2001). The road to primary prevention of lead toxicity in children. *Pediatrics*, 107, 581–583. http://doi.org/10.1542/peds.107.3.581
- Dietrich, K. N., Ware, J. H., Salganik, M., Radcliffe, J., Rogan, W. J., Rhoads, G. G., ... Jones, R. L. (2004). Effect of chelation therapy on the neuropsychological and behavioral development of lead-exposed children after school entry. *Pediatrics*, 114(1), 19–26. http://doi.org/10.1542/peds.114.1.19
- Dooyema, C. A., Neri, A., Lo, Y.-C., Durant, J., Dargan, P. I., Swarthout, T., ... Brown, M. J. (2012). Outbreak of fatal childhood lead poisoning related to artisanal gold mining in northwestern Nigeria, 2010. *Environmental Health Perspectives*, 120(4), 601–7. http://doi.org/10.1289/ehp.1103965
- Edwards, M. (2014). Fetal death and reduced birth rates associated with exposure to leadcontaminated drinking water. *Environmental Science & Technology*, 48(1), 739–46. http://doi.org/10.1021/es4034952
- Etchevers, A., Bretin, P., Lecoffre, C., Bidondo, M.-L., Le Strat, Y., Glorennec, P., & Le Tertre, A. (2014). Blood lead levels and risk factors in young children in France, 2008-2009. *International Journal of Hygiene and Environmental Health*, 217(4-5), 528–37. http://doi.org/10.1016/j.ijheh.2013.10.002
- Fox, D. A., Campbell, M. L., & Blocker, Y. S. (1997). Functional alterations and apoptotic cell death in the retina following developmental or adult lead exposure. *Neurotoxicology*, 18(3),

645–664.

- Graber, L. K., Asher, D., Anandaraja, N., Bopp, R. F., Merrill, K., Cullen, M. R., ... Trasande, L. (2010). Childhood lead exposure after the phaseout of leaded gasoline: an ecological study of school-age children in Kampala, Uganda. *Environmental Health Perspectives*, 118(6), 884–9. http://doi.org/10.1289/ehp.0901768
- Hansen, E., Lassen, C. and Elbaek-Jørgensen, A. (2004a): Advantages and drawbacks of restricting the marketing and use of lead in ammunition, fishing sinkers and candle wicks. European Commission, Directorate General Enterprise, Brussels.
- Jacobs, D.E., Clickner, R.P., Zhou, J.Y., Viet, S.M., Marker, D.A., Rogers, J.W., Zeldin, D.C., Broene, P., & Friedman, W. (2002). The prevalence of lead-based paint hazards in U.S. housing. Environmental Health Perspectives, 120, A599-A606.
- Jez, E., & Lestan, D. (2015). Prediction of blood lead levels in children before and after remediation of soil samples in the upper Meza Valley, Slovenia. *Journal of Hazardous Materials*. http://doi.org/10.1016/j.jhazmat.2015.04.049
- Jones, R. L., Homa, D. M., Meyer, P. a, Brody, D. J., Caldwell, K. L., Pirkle, J. L., & 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), e376–85. http://doi.org/10.1542/peds.2007-3608
- Lasky, R. E., Luck, M. L., Torre, P., & Laughlin, N. (2001). The effects of early lead exposure on auditory function in rhesus monkeys. *Neurotoxicology and Teratology*, 23(6), 639–49. http://doi.org/10.1016/S0892-0362(01)00175-1
- Landrigan PJ (1989). Toxicity of lead at low dose. *British Journal of Industrial Medicine*, 46(9):593–596.
- Lidsky, T. I., & Schneider, J. S. (2003). Lead neurotoxicity in children: Basic mechanisms and clinical correlates. *Brain*, *126*(1), 5–19. http://doi.org/10.1093/brain/awg014
- McClain, R. M., & Becker, B. A. (1975). Teratogenicity, fetal toxicity, and placental transfer of lead nitrate in rats. *Toxicology and Applied Pharmacology*, 31(1), 72–82. http://doi.org/10.1016/0041-008X(75)90053-8
- Miranda, M. L., Anthopolos, R., & Hastings, D. (2011). A geospatial analysis of the effects of aviation gasoline on childhood blood lead levels. *Environmental Health Perspectives*, 119(10), 1513–1516. http://doi.org/10.1289/ehp.1003231
- Muir, M., & Campbell, M (1995). Why are barns red: The health risks from lead and their prevention: A resource manual to promote public awareness, [manual], Toronto, Canada.
- National Academy of Sciences. Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations Washington: National Academy Press, 1993.
- Needleman, H. L., Gunnoe, C., Leviton, a, Reed, R., Peresie, H., Maher, C., & Barrett, P. (1979). Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *The New England Journal of Medicine*. http://doi.org/10.1056/NEJM197903293001301

Needleman, H. L., Schell, A., Bellinger, D., Leviton, A., & Allred, E. N. (1990). The long-term

effects of exposure to low doses of lead in childhood. An 11-year follow-up report. *The New England Journal of Medicine*, *322*(2), 83–8. http://doi.org/10.1056/NEJM199001113220203

- Pfadenhauer, L. M., Burns, J., Rohwer, A., & Rehfuess, E. A. (2014). A protocol for a systematic review of the effectiveness of interventions to reduce exposure to lead through consumer products and drinking water. *Systematic Reviews*, *3*, 36. http://doi.org/10.1186/2046-4053-3-36
- Protect Your Family | Lead | US EPA. (2015, October 21). Retrieved from http://www2.epa.gov/lead/protect-your-family#soil
- Public Health Statement for Lead. (2007, August). Retrieved from http://www.atsdr.cdc.gov/ToxProfiles/tp13-c1-b.pdf
- Queirolo, E. I., Ettinger, A. S., Stoltzfus, R. J., & Kordas, K. (2010). Association of anemia, child and family characteristics with elevated blood lead concentrations in preschool children from Montevideo, Uruguay. Archives Of Environmental & Occupational Health, 65, 94– 100. http://doi.org/10.1080/19338240903390313
- Rabin, R. (1989). Warnings unheeded: a history of child lead poisoning. *Public Health Then and Now*, *79*(12), 1668–1674.
- Rice, D. C. (1990). Lead-induced behavioral impairment on a spatial discrimination reversal task in monkeys exposed during different periods of development. *Toxicology and Applied Pharmacology*, 106(2), 327–33.
- Roberts, J. R., Allen, C. L., Ligon, C., & Reigart, J. R. (2013). Are children still at risk for lead poisoning? *Clinical Pediatrics*, *52*(2), 125–30. http://doi.org/10.1177/0009922812464549
- Rubin, C. H., Esteban, E., Reissman, D. B., Daley, W. R., Noonan, G. P., Karpati, A., ... Zlepko, A. (2002). Lead poisoning among young children in Russia: concurrent evaluation of childhood lead exposure in Ekaterinburg, Krasnouralsk, and Volgograd. *Environmental Health Perspectives*, 110(6), 559–562
- Schmidt, C. W. (2010). Lead in Air: Adjusting to a New Standard.*Environmental Health Perspectives*, *118*(2), A76–A79.
- Schwartz, J. (1994). Low-level lead exposure and children's IQ: a meta-analysis and search for a threshold. *Environmental Research*, 65(1), 42–55. http://doi.org/10.1006/enrs.1994.1020
- Schwartz, J., Landrigan, P. J., Baker, E. L., Orenstein, W. a., & Von Lindern, I. H. (1990). Leadinduced anemia: Dose-response relationships and evidence for a threshold. *American Journal of Public Health*, 80(2), 165–168. http://doi.org/10.2105/AJPH.80.2.165
- Smith, D., & Strupp, B. J. (2013). The Scientific Basis for Chelation: Animal Studies and Lead Chelation. *Journal of Medical Toxicology*, 9(4), 326–338. http://doi.org/10.1007/s13181-013-0339-2
- Sources of Lead. (2015, May 29). Retrieved September 28, 2015, from http://www.cdc.gov/nceh/lead/tips/sources.htm.

- Todd, A. C., Wetmur, J. G., Moline, J. M., Godbold, J. H., Levin, S. M., & Landrigan, P. J. (1996). Unraveling the chronic toxicity of lead: An essential priority for environmental health. *Environmental Health Perspectives*. http://doi.org/10.1289/ehp.96104s1141
- Triantafyllidou, S., Schock, M. R., DeSantis, M. K., & White, C. (2015). Low Contribution of PbO2-Coated Lead Service Lines to Water Lead Contamination at the Tap. *Environmental Science & Technology*, 49(6), 3746–54. http://doi.org/10.1021/es505886h
- U.S. ATSDR (2005): Toxicological profile for lead. (Draft for Public Comment). U.S. Department of Health and Human Services. Public Health Service. Agency for Toxic Substances and Disease Registry, Atlanta, U.S.A.
- US EPA. (2014, December 31). Lead in Outdoor Air | Lead |. Retrieved from http://www2.epa.gov/lead/lead-outdoor-air
- US EPA (a). (2015, October 15). Learn about Lead | Lead |. Retrieved from http://www2.epa.gov/lead/learn-about-lead#lead
- US EPA (b). (2015, August 20). Lead in Drinking Water | Lead |. Retrieved from http://water.epa.gov/drink/info/lead/index.cfm
- Village, E. G. (2005). Lead exposure in children: prevention, detection, and management. *Pediatrics*, *116*(4), 1036–46. http://doi.org/10.1542/peds.2005-1947
- Wedeen, R. P., Ty, A., Favata, E. A., & Jones, K. W. (1995). Clinical application of in vivo tibial K-XRF for monitoring lead stores. *Archives of Environmental Health*, 50(5), 355–373. http://doi.org/10.1080/00039896.1995.9935967
- World Health Organization. (2010). Childhood lead poisoning. Retrieved from http://www.who.int/ceh/publications/leadguidance.pdf.
- Xintaras, C. (1992). Analysis Paper: Impact of Lead-Contaminated Soil on Public Health. U. S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry - Impact of Lead-Contaminated Soil on Public Health.
 - Zhou, X., Lei, M., Yang, J., Zhou, G., Guo, G., Chen, T., ... Qiao, P. (2013). [Effect of lead on soil quality and human health around a lead smeltery]. Huan Jing Ke Xue= Huanjing Kexue / [Bian Ji, Zhongguo Ke Xue Yuan Huan Jing Ke Xue Wei Yuan Hui "Huan Jing Ke Xue" Bian Ji Wei Yuan Hui.], 34, 3675–3678.