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Lead: The inevitable slow poison eroding our society

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Abstract
Child development and growth patterns are indicators of environmental health, whose degradation leads to diseases of various kinds in them. This is so because children are uniquely vulnerable to environmental toxicants due to their greater relative exposure, less developed metabolism and higher rates of cell production, growth and change. These environmental insults of childhood may manifest themselves over a life time of growth to adulthood and senescence, thus producing defective future population of a nation which would instead of being assets would be liabilities for the nation.
In addition to these physiologic vulnerabilities, majority of children in our society are also exposed to a number of social vulnerabilities as well such as poverty, malnutrition and environmental injustice. Moreover, since the society is not willing to do away with a number of these toxicants due to their role in the industrial progress of the society, the only alternative, that remains in these conditions is searching their alternatives.

Keywords: Lead, children, development, environment and toxicants**.**

Introduction
"Development of human resources is as important as the development of material resources. The best way to develop national human resources is to take care of children as they constitute the nation's principle human assets"

Radhakrishnan

Children are partly the products of the environment both material and non- material, consequently any changes in it are likely to affect them as well. Child development and growth patterns are an indicator of the environmental health. For the same reason increasing concern is being expressed to the increase in environmental pollution that is releasing potentially dangerous chemicals or toxicants in the air the children breathe, water they drink and the land they live on.
It has been widely recognized that developing individuals (embryos, foetuses, newborns, infants and children) are a uniquely susceptible population to insults from environmental hazards (Guzelian *et al.* 1992 and Bearer 1995) [19, 4]. Their increased susceptibility can arise from increased exposure to environmental toxins (pound for pound of body weight children drink more water, eat more food and breathe more air than adults), increased exposure of individual organ systems from differences in distribution of toxins, immaturity of excretory pathways, alterations in target organ susceptibility, and a longer life span in which to express illness. Children are indeed different from adults, both in patterns of exposure to environmental risk and in their responses to environmental hazards. There are several examples in the literature demonstrating that exposure to a chemical during a critical period of development will produce toxicity, whereas exposure to the same chemical during adulthood will have little or no effect. The major determinants of these differences are however related to the rapid growth and development of children.
Of the various toxicants, childhood lead poisoning is now recognized as the number one preventable global environmental disease of children. Lead poisoning affects children's health and development, especially in densely populated urban and industrial cities. Plumbism or Pb poisoning has been recognized for many centuries and is one of the oldest ailments afflicting humans.

Nikander (200 B.C.) was the first to describe the symptoms of Pb poisoning, which included colic and pallor (Wedeen 1982) [51]. The English aristocracy of the 17th and 18th centuries suffered from widespread Pb poisoning from consumption of Portuguese wine, transported with submerged Pb bars to enhance flavour and to prevent spoilage (Batuman 1993) [3]. After this many studies have been done on Pb.

The sources of exposure to this metal, which have been identified, include the use of lead glazed pottery for cooking and storing food (Rothenberg *et al.* 1990, Hernandez-Avila *et al.* 1991, Jimene'z *et al.* 1993 and Munoz *et al.* 1993) [38, 20, 22, 30], residing in areas with heavy vehicular traffic, consumption of canned foods (Rothenberg *et al.* 1993) [37] and the presence of Pb in indoor and outdoor dust (Romieu *et al.* 1995) [39]. Specifically Pb exposure in urban areas comes from Pb in soil (usually due to emissions of automobiles run on leaded fuel from the 1920's to the 1970's), lead in paint and Pb in plumbing (Bellinger and Matthews 1998 and Bruening *et al.* 1999) [10, 11]. The Center for Disease Control and Prevention (CDC 1997) [14] estimates that 83% of houses built before 1978 contain dangerous level of Pb in paint with housing built prior to 1950 posing an even higher risk. Pb exposure in rural areas comes from a variety of sources including Pb in point (in older barns as well as homes), soil, plumbing and contaminated waste dumps. However, the mechanisms producing children's elevated Pb levels include inhalation of dust containing Pb as well as ingestion of dust through normal hand-to-mouth and toy mouthing behaviour among very young children (e.g. Bruening *et al.* 1999) [11].

The importance of Pb in soil in predicting children's blood Pb levels was emphasized in a recent study of children living in one urban and one rural parish in Louisiana. Soil Pb was more closely related to children's blood Pb (BPb) level than was the age of the housing in which the children were residing (Mielke *et al.* 1997) [27]. In contrast, studies conducted by researchers at many urban universities continue to report significant associations between older housing and BPb level in children (e.g., Lanphear *et al.* 1998) [25]. Also it has been reported that bone serves as a long-term repository of 75% and 90-95% of Pb in children and adults, respectively. Many studies, have demonstrated that bone Pb levels remain elevated despite declines in BPb. In addition to bone and blood, the level of Pb in body is also assessed through the hair and urine.

Pb, infact, is a highly toxic metal, as even a low-to-moderate level of Pb poisoning results in irreversible loss of intelligence, behaviour and neuromotor problems and poor school performance (Sciarillo and Alexander 1991) [43]. High doses are on the other hand reported to cause anemia, frank encephalo pathology with lethargy, seizures, cerebral oedema and even death (Hugelmeyer *et al.* 1988) [21].

Early work in developmental neuro behaviour suggested that Pb was related to a variety of children's behaviours that indexed problems in both their cognitive and their social functioning. In a 1979 study that addressed both cognitive and social functioning, Needleman and his colleagues at Harvard found that; compared to children with low dentin Pb levels, children with high dentin Pb levels scored lower on the Wechsler Intelligence Scale for children revised on three measures of auditory or speech processing and on a measure of attention. These children were also rated by their teachers as scoring higher on such classroom behaviour problem as distractibility, decreased persistence, impulsivity, day dreaming and dependency (Needleman *et al.* 1979) [32]. More recent studies replicate the finding that Pb is related to children's non-adaptive social functioning. Among 212, 11-14 year old boys in the Pittsburgh metropolitan area, those with higher levels of bone lead were rated by their teachers and parents as higher on both internalizing and externalizing behaviour problems than those with lower levels of bone Pb (Needleman *et al.* 1996, 2011) [33, 34]. Among more than 1000 Boston school-age children, dentin Pb level were significantly positively related to teacher's ratings of internalizing and externalizing behaviour problems (Bellinger *et al.* 1994b) [8]. Similar results were reported by Burns *et al.* (1999) [13] but he assessed BPb level instead of dentin Pb.

Even more widely investigated than the relationship of Pb to children's behaviour problems and social functioning is the relationship between Pb and children's cognitive functioning. In the late 1970's and early 1980's researchers initiated a series of international prospective studies of the impact of Pb on children's development. Study sites were Boston, Cincinnati, Cleveland, Kosovo, Port Pirie and Sydney. Sampling strategies varied across sites. In the Boston and Kosovo sites, children were enrolled in the study as a function of prenatal Pb exposure as measured by umbilical cord Pb levels. In the Boston study, children with both high and low prenatal Pb exposures were over sampled. In the sites other than Boston and Kosovo, enrollment was by consecutive births or consecutive registration for prenatal care or by other criteria, all of which resulted in lower rates of sampling of children with higher rates of exposure to Pb (Bellinger 1995) [5]. In the Boston study, Pb explained significant variance in children's impaired cognitive functioning on a variety of measure and over time periods as long as 12 years. For example, BPb level at age 2 was inversely related to cognitive score on the Mc Carthy Scale of Children's Abilities at age 5 (Bellinger *et al.* 1991) [6] and to IQ and achievement scores at age 10 (Bellinger *et al.* 1992) [7].

Among the other international prospective studies, results tended to be consistent with the Boston results although not always as dramatic. The Cincinnati group reported a significant inverse relationship between postnatal BPb level and performance IQ on the WISC-R at age 5-6 (Dietrich *et al.* 1993) [15]. The Port Pirie group reported a significant inverse relationship between postnatal BPb level and IQ at age 7. The Kosovo group reported a significant inverse relationship between prenatal and postnatal Pb exposure and children's cognitive score at age 4 (Wasserman *et al.* 1994) [48].

Consistent with these findings is Needleman *et al.* (1990) [35] report of an 11 year follow up of 132 children who were evaluated in Needleman *et al.* (1979) [32] report. Results revealed that dentin Pb level was inversely related to a number of measures of educational attainment-highest grade achieved, reading grade equivalent, class standing, grammatical reasoning and vocabulary and positively related to school absences. Bellinger *et al.* (1994a) [8] reported on the relationship between dentin Pb and attention in a sub sample of these same individuals who returned for additional follow up one year later. Two of the four measures of attention were significantly related to dentin Pb. It should be noted that all analyses were adjusted for potential confounding variables, which included family socio-economic status.

Dudek and Merecz (1997) [17] also found a significant impact of Pb upon intelligence and attention concentration. They also revealed that the short term memory deteriorates with growing level of exposure of Pb.

Citing the WHO'S 1995 International Program on Chemical Safety, Bellinger and Matthews (1998) [10] point out that the average impact of Pb exposure on children's IQ ranges from 0 to 5 points for each 10 µg of Pb per dl of blood. Schmidt (1999) [42] cites a range of 1 to 3 points of IQ decreased as associated with each 10 µg 's of Pb per dl of blood.

Although the adverse effect of overt plumbism on physical growth has long been recognized (Nye 1929 and Johnson and Tenuta 1979) [31, 23], the effect of low level Pb exposure on physical growth was first explored by Schwartz *et al.* (1986) [40], using data from the National Health and Nutrition Examination Survey (NHANES) II of 1976-1980 (Schwartz *et al.* 1986) [40]. The NHANES II data for 2695 7 years old children indicated that BPb level (range=4-35 µg/dl) was a statistically significant predictor of children's height, weight and chest circumference, with control for age, race, sex and nutritional covariates.

The results of subsequent studies have been inconsistent. A retrospective study of the growth of 54 children from birth to 48 months of age suggested a negative correlation between weight gain and higher BPb between 15 and 24 months of age (Angle and Kunzelmar 1989) [1]. Also in another longitudinal study, covariate-adjusted heights at 15 and 33 months of age were negatively associated with postnatal BPb concentrations (Shukla *et al.* 1991) [44]. Frisancho and Ryan (1991) [18] also reported an inverse relationship between BPb concentration in the range of 0.14 - 1.92 nmol/l with stature of children aged 5 – 12 years. The children with BPb concentrations above the median for age and sex were approximately 1.2 cm shorter than their counterparts with BPb concentration below the median. It was also reported that increase in stature with age also decreased in children with BPb concentration above average. Similarly Shenoi *et al.* (1991) [45] reported that slum school children of Bombay from low socio-economic class were less than 5th percentile of weight for age with BPb levels greater than 70 µg/dl. Ballew *et al.* (1999) [2] also concluded that there are significant negative associations between BPb concentration and stature and head circumference among children through 1 to 7 years.

Regarding the effect of Pb on psychomotor and visuomotor performance majority of researchers after doing research in different parts of the world viz. India, Beijing and Yugoslavia have come to the conclusion that Pb level, be that in hair, umbilical cord or blood is inversely related to psychomotor and visuomotor performance (Wasserman *et al.* 1997, Zang *et al.* 1997 and Sinha and Sharma 1998) [49, 52].

On the research pertaining to susceptibility to Pb and its effects; several factors have been reported to increase the absorption of Pb. According to Doss *et al.* (1984) [16] a genetic deficiency in delta–aminolevulinic acid dehydratase is associated with increased susceptibility to clinical Pb poisoning. Deficiencies of protein and minerals, particularly iron and calcium have also been found to significantly influence the gastrointestinal absorption of Pb (Kaul 1999) [24]. Generalized fasting also has been found to increase gastrointestinal absorption of Pb (Rabinowitz *et al.* 1998) [36]. Further, inspite of the conclusion by Tong *et al.* (1998) [47]. that the cognitive deficits associated with exposure to environmental Pb in early childhood appear to be only partially reversed by a subsequent decline in BPb level, several researchers have reported that diet can modify Pb Kinetics. Mahaffey *et al.* (1996) [26] reported that milk intake and calcium supplement use were significant negative predictors of BPb level. Similar results were reported by Johnson and Tenuta (1979) [23], Muldoon *et al.* (1994). West *et al.* (1994) on a study conducted among African American women during pregnancy reported a positive association between BPb and serum calcium (r=0.44) and significantly higher BPb level among women who did not consume vitamin–mineral supplements. However Morris *et al.* (1990) [28] reported no effect of calcium supplements on BPb levels in a sample of 142 subjects who received lg calcium supplements for 12 weeks. Severe Pb poisoning is however treated with hospitalization and chelation therapy (CDC 1997) [14].

Early subclinical exposure to lead thus appears to result in lifelong disability. Multiplied by the tens of thousands of children at risk, the societal and fiscal impact of this disability is enormous (Schwartz 1994) [41]. From a public health perspective it is thus highly desirable and prudent to reduce the dispersive uses of lead. Further since lead is ubiquitous and persistent in the environment and has a subtle and persistent effect on the overall development, how to set the environmental standard for lead exposure is an important scientific issue still open for debate.

In addition, if the child is living in poverty or is from low socio-economic status, toxicants have an even more damaging effect on him as his immediate home-environment is already deficit. It is beyond doubt that too often in our society the children most heavily exposed to environmental toxicants are poor children in underprivileged communities i.e. there is a pattern of disproportionate exposure most commonly termed as environmental injustice (Bullard and Wright 1993) [12].

This potential for environmental contaminants to produce neurological, cognitive, motor or other behaviour deficits as a result of developmental exposure is receiving increasing attention. The focus has shifted from description of frank toxicity observed in a relatively few individuals to more subtle impairment in a much greater number of children. With this shift has come the recognition that subtle deficits such as a small decrease in IQ can have important societal impact when large numbers of children are affected. For example, the result of a 1 microgram/dl decrease in blood lead concentration in children in the United States with blood lead concentration between 10 and 20 micrograms/ dl would translate into a savings of 5-7.5 billion U.S. dollars a year in increased earning power alone. In addition, behavioural problems such as increased aggression and poor social adjustment identified early in childhood may escalate to serious antisocial behaviour such as delinquency as the child approaches puberty.

Conclusion

Exposure to neurotoxic agents during development or over a significant portion of the lifespan may result in acceleration of age-related neurodegenerative diseases. Such changes in the functional abilities of a significant proportion of a population have potentially serious consequences (health, economic etc) for society as well as for affected individuals. It has been rightly said that:-

"Children are our Future, and our Future lies before us like a Path of Driven Snow. Carefully we have to tread it, for every mark will show."

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