Impact of Neurotoxicants on the Physical Development of Children

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Abstract

The present study was undertaken to assess the impact of neurotoxicants on the physical development of children aged 6-7 years in two eco-settings of Ludhiana city of the Punjab State in India. The locality around industrial area was termed as neurotoxicant polluted setting (NPS) and the locality away from the industrial area was termed as neurotoxicant free setting (NFS). The sample comprised of 240 children aged 6-7 years (belonging to low socio-economic status, born and brought up in the specified setting and not of migrant family), randomly taken from the Government schools located in the two settings. Out of these 240 children, 120 each were drawn purposively from the two eco-settings. Standard instruments (anthropometer, weighing balance and fiberglass measuring tape) and procedures were used for anthropometric assessment. The results showed children of NPS to be better in weight whereas those of NFS were better on height. They were almost similar with respect to their head circumference.

Key Words: Environmental Pollution, Lead Poisoning, Health, Anthropometry, Weight, Height

Introduction

Children are partly the products of the environment both material and non-material, consequently any change in it is likely to affect them. 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 land they live on. 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. Even a low-to-moderate level of lead poisoning results in neuromotor problems (Sciarillo & Alexender, 1991). In the presence of underlying iron deficiency, the absorption of lead from various sources increases (Wasserman et al., 1994), thus aggravating the toxic effects of lead. Besides lead, other neurotoxicants like copper, selenium, arsenic etc. also affect the development of children. In addition, if the child is from low socio-economic status, neurotoxicants have an even more damaging effect on him/her as his immediate home environment is already deficit because of poverty.

Ludhiana, the industrial capital of Punjab in India, has a large share of industrial pollutants and effluents from industries such as hosiery, textile, spare parts, cycles, dyeing, sewing machines, auto parts, chemicals and vegetable hydrogenated fats. The impact of neurotoxicants on the physical development of children of the city was studied.

Materials and Methods

The study is based on a sample of 240 children aged 6-7 years, randomly drawn from the Government schools located in the two eco-settings of the Ludhiana district of the Punjab State in India. Neurotoxicant polluted eco-setting (NPS) was adjoining the industrial area and neurotoxicant free eco-setting (NFS) was about 15-20 km from the industrial area. The drinking water samples' qualitative

analysis in terms of heavy metal content is presented in Table 1 for the two settings.

Out of 240 children, 120 children each were drawn purposively from these two eco-settings. Further, the children included in the sample from the two eco-settings satisfied the following criteria for their inclusion: -

(a) belonging to the low socio-economic status
(b) should be attending the school
(c) should not be of a migrant family
should have minimum six years residence in the specified area.

Table 1. Toxicants found in the drinking water samples

Toxicants (mg/litre)	Maximum permissible limits	Neurotoxicant Polluted Setting (NPS)	Neurotoxicant Free Setting (NFS)
Lead	0.1	0.38	0.1
Arsenic	0.05	0.37	0.1
Cadmium	0.005	0.85	0.0008
Copper	0.05	0.26	0.009

Standard instruments (anthropometer, weighing machine and fiber glass measuring tape) and procedures were used to measure the height (cm.), weight (kg.) and head circumference (cm.) of the subjects (Weiner & Laurie, 1969). The anthropometric measurements were taken in the morning and individuals wore minimal clothing during examination. Height was appreciated to the nearest 0.1 cm. with the subject in standing position, bare foot and with the head held in the Frankfurt-Horizontal plane. A gentle pressure was applied on the mastoid process during measurement. On the other hand, weight was measured to the nearest 0.1 kg. Similarly, head circumference was measured from glabella to glabella through occiput to the nearest 0.1 cm. Sahil's haemoglobinometer was used to test their haemoglobin level.

Data was analyzed by applying appropriate statistical methods such as arithmetic mean, standard deviation, percentages, co-efficient of variation and Z-test. Both 0.05 and 0.01 level of 'p' were considered for ascertaining the significance of results.

Results

Table 2. Haemoglobin (Hb) level of respondents of two Eco-settings

Eco-settings/Sex	Range	Various grades of Hb-level (NIN- 1986)			
		Non Anaemic (>12g/dl.)	Mild anaemic (> 10-11.9 g/ dl.)	Moderate anaemic (> 8-9.9g/dl.)	Severe anaemic (> 7.9g/ dl.)
Neurotoxicant Free					
Boys					
Girls	9-12	7(11.67)	35(58.33)	18(30)	-
Combined	8-12	3(5)	33(55)	19(31.67)	5(8.33)
		10(8.33)	68(56.67)	37(30.83)	5(4.17)
Neurotoxicant Polluted					
Boys					
Girls	7-12	3(5)	24(40)	27(45)	6(10)
Combined	6-12	2(3.33)	28(46.67)	21(35)	9(15)
		5(4.17)	52(43.33)	48(40)	15(2.5)

The respondents in both the eco-settings were almost similar in their socio-personal aspects. The respondents in both the eco-settings were also similar in that both these eco-settings housed respondents who were in majority anaemic, though the

respondents of NPS were more anaemic than the respondents of NFS (Table-2), indicating their greater susceptibility to neurotoxicants. Respondents of NPS also showed greater variability in their Hb level to the extent that if some children had Hb level of 10 or 11g/100 ml., then there were also children who had Hb level as low as 6 or 7g/ml.

It is evident from Table 3 that the boys of NFS of both the age groups i.e. 6 and 7 years were comparatively taller (mean height 110.8 cm. at 6 and 115.7 cms. at 7 years) than their counterparts in NPS with mean height of 108.0 cm. at 6 years and 112.4 cm. at 7 years. Also the difference in height at the age group of 7 years was found to be significant ($p<0.05$); the difference was in favour of the NFS group (Table 4). However, the head circumference of the boys of both the eco-settings were almost similar. Further it was observed that the boys of NPS at both the age-levels weighed more than their counterparts in NFS; differences favouring NPS and being significant both at 6 years ($p<0.05$) and at 7 years ($p<0.01$) (Table 4).

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Table 3. Physical development of respondents from two Eco-settings

Age (years)	BOYS							
	Neurotoxicant Free				Neurotoxicant Polluted			
	N	Height (cm) Mean (SD)	Weight (kg) Mean (SD)	Head circm. (cm) Mean (SD)	N	Height (cm) Mean (SD)	Weight (kg) Mean (SD)	Head circm. (cm) Mean (SD)
6	25	110.8 (6.8)	14.3 (3.0)	50.8 (1.7)	22	108.0 (4.6)	16.1 (1.7)	50.6 (1.4)
7	35	115.7 (7.4)	15.5 (3.0)	50.9 (1.2)	38	112.4 (5.4)	18.5 (1.9)	50.9 (1.0)
	GIRLS							
6	16	106.4 (6.3)	12.4 (2.2)	49.6 (1.1)	25	106.3 (4.0)	15.2 (2.3)	49.4 (1.1)
7	44	114.7 (6.0)	15.1 (2.3)	49.9 (1.4)	35	110.8 (6.0)	17.4 (2.0)	49.7 (1.3)

Table 4. Comparison in physical development from two Eco-settings.

Age (years)	Variable	Boys (Z-value)	Girls (Z-value)
6	Height	1.67	0.06
	Weight	2.57**	3.9*
	Head Circum.	0.87	0.57
7	Height	2.16**	2.89*
	Weight	5.0*	4.79*
	Head Circum.	0	0.67

* Critical value of Z at 1% level of significance = 2.58
** Critical value of Z at 5% level of significance = 1.96

Almost similar results were observed in case of girls. Girls of NFS were taller at both age levels of 6 and 7 years (differences significant at 0.01 level) as compared to those in the NPS (Table 4).

Similarly, girls of both the eco-settings had almost similar head circumferences. As with boys, the mean weight of girls of NPS was more as compared to those in the NFS.

Discussion

The results indicate that respondents (both boys and girls) of NFS were better than their counterparts in NPS

with respect to height. On the other hand, those in NPS were heavier than those in NFS, when their mean weights were compared. They were similar with respect to their head circumferences.

The adverse effect of overt plumbism on physical growth has long been recognized (Nye, 1929, Johnson & Tenuta, 1979). The effect of low lead (Pb) level exposure on physical growth was first explored by Schwartz et al (1986), using data from the National Health and Nutrition Examination Survey (NHANES) II of 1976-1980. From the NHANES II data of 2695, seven years old children predicted that blood lead level (BPb) range of 4 to 35 (µg/dl) was a statistically significant predictor of children's height and weight, 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 & Kunzelman, 1989). Two longitudinal studies did not find any significant association between BPb and physical growth (Sachs & Moel, 1989; Greene & Ernhart, 1991). Covariate adjusted heights at 15 and 33 months of age were negatively associated with postnatal BPb concentration (Shukla et al., 1989 and Shukla et al., 1991). Frisancho and Ryan (1991) reported an inverse relationship between BPb concentration in the range of 0.14-1.92 µm0l/l with stature of 1454 Mexican American children aged 5-12 years i.e. growth retardation was associated even with moderate concentration of BPb. The children with BPb concentration above the median for age and sex were approximately 1.2 cm. shorter than their counterparts with BPb concentration below the median. Ballew et al (1999) found significant negative association between BPb concentration and stature and head circumference among children through 1 to 7 years. Stanek et al (1998) reported negative relationship between head circumference and BPb level of 21 children aged 18 to 36 months residing in Omaha, Nebraska. The children (boys and girls) of present study from Neurotoxicants Polluted Setting (NPS) were shorter in stature by 2-3 cm. when compared with their counterparts from Neurotoxicants Free Setting (NFS) and the differences were statistically significant.

The deleterious effects of Pb have also been reported on animals. Hamilton and Oflaherty (1994) in an experimental model exposed rats to lead acetate and found that tail length was shorter in Pb exposed rats. The reduction in tail growth suggested a Pb-induced inhibition of the development of the distal vertebrae. Given the importance of vertebral development on an individual's height, this finding may explain reports of shorter height in children who live in industrial areas that are highly contaminated with Pb as in the present study. Anderson and Danylchuk (1977) in an experimental study in dogs, found that chronic exposure to Pb decreased bone formation. Hass et al (1967) reported similar findings in an experimental study in rabbits. Similarly, Miyahara et al (1995) observed that Pb increased bone resorption in bone cultures, except when the culture had been treated previously with calcitonin, a hormone that has a strong inhibitory effect on osteoclastic activity (Chambers et al., 1986), the principal cell involved in bone resorption. Escribano et al (1997) in a

study on 35, 50 day-old female Wistar rats reported that Pb did not affect the longitudinal growth of peripheral long bones, but did affect the development of the axial skeleton. Lead exposure in rats also produced important changes in bone remodeling that caused reduction in bone mass as measured by histomorphonietry produced mainly by enhanced resorption and an increase in bone mass, as measured by densitometry, produced by Pb accumulation in bone.

Even previous investigations of human populations with moderate to low Pb exposure have demonstrated a strong positive and relatively linear relationship of both tibia and patella bone and Pb with age (Somerville et al., 1985; Hu et al., 1990; Kosnett et al., 1994 & Watanabe et al., 1994). In the study by Hernandez-Avita et al (1996) as well, tibia and patella Pb levels were positively related with age.

Contrasting results have been found regarding effect of neurotoxicants on weight on animals. Levis et al (1988) found no effect on weight, head circumference, crown-rump or crown-heel length in rhesus monkey observed over the first six months of life and dosed from birth having BPb levels of 25-45 µg/dl. Bushnell (1978) found no effect on body weight in rhesus monkeys observed over the first year of life, dosed from birth and divided into three cohorts totaling 38 monkeys. Similarly Rice (1996) found no evidence of reduced weight as a result of Pb exposure in 34 female and 18 male monkeys in which body weight was modeled from infancy through adulthood and in which BPb concentration were always above 40 g/dl.

The findings of the present investigation do point to the negative effect of Pb on stature. The exact mechanism whereby Pb may retard growth is not known. Here, a possible explanation for the observed association may be that nutritional deficits that retard growth also increase Pb absorption e.g. iron deficiency, as found in number of children who are anaemic. In fact iron deficiency does increase Pb absorption as reported by Danford (1982) and Lin-Fu (1982). Pounds et al (1982, 1983) and Rosen (1983) have also attributed the negative affect on weight and height in human children to the lead's effect on heme-dependent enzymes or calcium messengers.

Reduced pituitary responsiveness to hypothalamic stimuli in terms of growth hormone releasing factor or thyrotropin releasing hormone has been postulated as a pathophysiologic mechanism for lead's effect on physical growth (Huseman et al., 1992). They showed that peak human growth hormone and insulin like growth factor I responses to the L-dopa insulin test were low in Pb-poisoned children. They thus, concluded that Pb-induced reduction in stature may be due to diminished human growth hormone secretion which in turn results in reduced insulin like growth factor I secretion, or that Pb may directly inhibit insulin like growth factor I formation. Blunted response of thyroid-stimulating hormone and growth hormone to stimulatory challenge have been observed in Pb-poisoned children (Huseman et al., 1987) as well as in rats exposed to low-level Pb (Camoratto et al., 1993).

There are few studies reporting effect of pollutants on weight of children. The present study, has observed significantly higher body weight of children on NPS. Similar findings have been reported by Kim et al (1995) who in a cross-sectional and longitudinal investigation assessing the relationship between chronic exposure to Pb and

physical growth among a cohort of children re-assessed 13 years after initial examination found that dentin Pb level was positively associated with body mass index suggesting that chronic Pb exposure in childhood may result in obesity that persists into adulthood.

Conclusion

Decrease in height and head circumference and increase in weight of the respondents could be attributed to the excess consumption of neurotoxicants via food chain and water in NPS. A critical analysis of the whole scenario further reveals that weight is the only physical variable that significantly differentiates between the respondents of the two eco-settings at both the age groups and in both the sexes. The effect of toxicity on height becomes more pronounced with increase in age.

References

Anderson, C., and Danylchuk, K. D. 1977. The effect of chronic low-level lead intoxication on the haversian remodeling system in dogs. *Laboratory Investigations*., **37**:466-69.

Angle, C. R., and Kunzelman, D. R. 1989. Increased erythrocyte protoporphyrins and blood lead – a pilot study of childhood growth patterns. *Journal of Toxicology and Environmental Health* **26**:149-56.

Ballew, C., Khan, L. K, and Kautmann, R. 1999. Blood lead concentration and children's anthropometric dimensions in the Third National Health and Nutrition Examination Survey (NHANES III) 1988-1994. *Journal of Pediatrics*., **134**:623-30.

Bushnell, P. J. 1978. *Behavioral toxicology of lead in the infant rhesus monkeys*. Doctoral Thesis, University of Wisconsin, Madison.

Camoratto, A. M., White, L. M, and Lau, Y. S. 1993. Effect of exposure to low level lead on growth and growth hormone release in rats. *Toxicology*., **83**:101-04.

Chambers, T. J., Chambers, J. C., and Symonds, J. 1986. The effect of human calcitonin on the cytoplasmic spreading of rat osteoclast. *Journal of Clinical Endocrinological Metabolism*., **63**:1080-85.

Danford, D. E. 1982. Pica and nutrition. *Annual Review Nutrition.*, **2**:303-22.

Escribano, A., Revilla, M., and Hernandez, E. R. 1997. Effect of lead on bone development and bone mass: A morphometric, densitometric and histomorphometric study in growing rats. *Calcif Tissue Int*., **60**:200-03.

Frisancho, A.R., and Ryan, A. S. 1991. Decreased stature associated with moderate blood lead concentrations in Mexican-American children. *American Journal of Clinical Nutrition.,* **54**:516-19.

Greene, T., and Ernhart, C. B. 1991. Prenatal and preschool age lead exposure: Relationship with size. *Neurotoxicology and Teratology*.,**13**:417-27.

Hamilton, J. D., and Oflaherty, E. J. 1994. Effects of lead exposure on skeletal development in rats. Fundamental Applied Toxicology., **22**:594-604.

Harnandez-Avila, M., Gonzalez-Cossio, T., and Palazuelos, E. 1996. Dietary and environmental determinants of blood and bone lead levels in lactating postpartum women living in Mexico City. *Environmental Health Perspectives.*, **104**:1076-82.

Hass, G. M., Landerholm, W., and Hemmens, A. 1967. Inhibition of intercellular matrix synthesis during ingestion of inorganic lead. *American Journal of Pathology*.,**50**:815-45.

Hu, H., Milder, F., and Burger, D. E. 1990. X-ray fluorescence measurements of lead burden in subjects with low-level community lead exposures. *Archives of Environmental Health*., **45**:335-41.

Huseman, C. A., Varma, M. M., and Angle, C. R. 1992. Neuroendocrine effects of toxic and low blood lead levels in children. *Pediatrics.*,**90**:186-89.

Huseman, C. A., Moriarty, C. M., and Angle, C. R. 1987. Childhood lead toxicity and impaired release of thyrotropin stimulating hormone. *Environmental Research.,* **42**:524-33.

Johnson, N. E., and Tenuta, K. 1979. Diets and lead blood levels of children who practice pica. *Environmental Research*.,**18**:369-76.

Kim, R., Hu, H., and Rotnitzky, A. 1995. A longitudinal study of chronic lead exposure and physical growth in Boston children. *Environmental Health Perspectives*., **103**:952-57.

Kosnett, M. J., Becker, C. E., and Osterloh, J. D. 1994. Factors influencing blood lead concentration in a suburban community assessed by non-invasive K X-ray fluorescence. *Journal of American Medical Association.*, **271**:197-203.

Levis, E. D., Schneider, M. L., and Ferguson, S. A. 1988. Behavioral effects of developmental lead exposure in rhesus monkeys. *Developmental Psychobiology.*, **2**:371-82.

Lin-Fu, J. S. 1982. The evolution of childhood lead poisoning as a public health problem. In: *Lead absorption in children. Ed:* Chrisholm, J. J., O'Hara, D. M. Baltimore: Urban & Schwarzenberg. 1-10.

Miyahara, T., Komiyama H., and Miyanishi, A. 1995. Stimulative effects of lead on bone resorption in organ culture. *Toxicology*., **97**:191-97.

Nye, L.J.J. 1929. An investigation of the extraordinary incidence of chronic nephritis in young people in Queensland. *Medical Journal of Aris.,* **2**:145-59.

Pounds, J. G., and Mittelstaedt, R. A. 1983. Mobilization of cellular Ca-45 and lead-210: Effect of physiologic Stimuli. *Science*., **220**:308-10.

Pounds, J. G., Wright, R., and Kodell, R. L. 1982. Cellular metabolism of lead: a kinetic analysis in the isolated rat hepatocyte. *Toxicology & Applied Pharmacology*., **66**:88-101.

Rice, D. C. 1996. Effect of long term lead exposure on hematology, blood biochemistry and growth curves in monkeys. *Neurotoxicology*., **18**:221-36.

Rosen, J, F. 1983. The metabolism of lead in isolated bone cell populations: Interactions between lead and calcium. *Toxicology & Applied Pharmacology*., **71**:101-12.

Sachs, H. K, and Moel, D. I. 1989. Height and weight following lead poisoning in childhood. *American Journal of Diseases of Childhood*.,**143**:820-22.

Schwartz, J., Angle, C., and Pitcher, H. 1986. Relationship between childhood blood lead levels and stature. *Pediatrics.*,**77**:281-88.

Sciarillo, W.G., and Alexander, G. 1991. Lead exposure and child behaviour. *American Journal of Public Health.,* **82**:1356-60.

Shukla, R., Bomschein, R. L., and Dietrich, K. N. 1989. Foetal and infant lead exposure: effects on growth in stature. *Pediatrics*., **84**:604-12.

Shukla, R., Dietrich, K. N., Bomschein, R. L., and Buncher, C. R. 1991. Lead exposure and growth in the early preschool childhood. *Pediatrics.,* **88**:886-92.

Somervaille, L. J., Chettle, D. R., and Scot, M. C. 1985. In vivo measurement of lead in bone using X-ray fluorescence. *Physical Medicine & Biology*., **30**:929-43.

Stanek, K., Manton, R. D. W., and Angle, C. 1998. Lead consumption of 18 to 36 months old children as determined from duplicate diet collections: Nutrient intakes, blood lead levels and effect on growth. *Journal of American Diet Association.*, **98**:155-58.

Wasserman, G. A., Graziano, J. H., and Factor-Litvak, P. 1994. Consequences of lead exposure and iron supplementation on childhood development at age 4 years. *Neurotoxicol Teratology*., **16**:233-40.

Watanabe, H., Hu, H, and Rotnitzky, A. 1994. Correlates of bone and blood lead levels in carpenters. *American Journal of Medicine*., **26**:255-64.

Weiner, J. S., and Luurie, J.A. 1969. Human biology: a guide to field methods In: *Growth and physique*. Ed: Tanner, J. M., Heirnaux, J., and Jarman, S. Oxford: Blackwell Scientific Publications.