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Prevalence of elevated blood lead levels in children with chronic neurologic disorders in Benin City, Nigeria

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Abstract*: Background:* Excess blood levels of lead in humans are injurious to tissues and organs chief among which is the central nervous system. Children with chronic neurologic disorders (CNDs) with already compromised brain function are unduly predisposed to lead poisoning because of some aberrant conducts and behaviors inherent in CNDs. Such children are therefore at greater risk of further deterioration of brain functions should they have elevated blood lead.
Objectives: To determine blood lead levels in children with CNDs as well as in apparently healthy controls
Methods: The case control, prospective and cross sectional study involving 139 children (89 or 64% males and 50 or 36.0% females) aged 1-15 years (mean ± SD: 6.54±4.04 years) and recruited from the Neurology Clinic (COPD) of the University of Benin Teaching Hospital (UBTH) Benin City was carried out between January and March of 2013. Same number of apparently children matched for age and gender served as controls. A semi structured, pre tested and researcher administered questionnaire was used in obtaining relevant information from the parents or guardians of study subjects. Atomic absorption spectrophotometer was used in determining the blood lead levels while the Vineland Social Maturity Scale was used to assess personal and social sufficiency among subjects while socioeconomic classification of the subjects was in accordance with the recommendations of Ayodeji.
Results: Subjects consisted of 80 (57.5%) with epilepsy, 24(17.3%) with mixed forms, 23(16.5%) with cerebral palsy, 8(5.8%) with attention deficit and hyperactivity disorder and 4 (2.9%) mental retardation. Mean BLL in subjects with CNDs of 25.05±16.65 µg/dl (range: 8-72 µg/dl) was significantly higher than the value in controls (19.24±10.17 µg/dl; range:8-53 µg/dl)(t=3.51, p=0.00). One hundred and one (72.7%) subjects compared to 111 (79.9%) controls had elevated blood lead levels eBLLs. Prevalence of eBLLs was independent of gender, age, family SES and nutritional status. Between subjects and Control show ever, gender (24.92±15.97µg/dl vs 19.35±10.63 µg/dl; t=2.74 = p=0.01/25.28±17.96µg/dl vs 19.50±09.55µg/dl; t=2.01; p=0.04), age (11-15 years only) (36.36±18.56µg/dlvs 19.45 ± 10.76 µg/dl; t= 4.01; p=0.00) and family SES (24.67±17.12 µg/dl vs 18.12±09.17 µg/dl; t=3.42;p=0.00) significantly influenced the mean blood lead level (mBLL). mBLL was significantly higher in children with epilepsy and lowest in those with mental retardation (F=2.75; p=0.03). The only risk factor that predicted eBLLs was finger sucking (OR=2.56; p=0.04 95% CI=1.03-6.40).
Conclusion/Recommendations: eBLLs is rampant in the study locale in both subjects and controls with the latter at heightened risk. Hand sucking is a proven risk factor for eBLLs. Such children and others predisposed to eBLLs should be protected through improved supervision, routine screening and intervention where necessary.

Key words: Blood Lead, Chronic Neurologic Disorders, Children, Benin.

Introduction

Lead is ubiquitous and poisonous heavy metal that is widely distributed in the environment.[1] In 1991, CDC in Atlanta defined elevated blood levels in children as whole blood lead levels (BLLs) ≥10µg/dl. There is however, no known safe blood lead level.[2] Sources of lead poisoning include lead-based paint, lead-contaminated water, food, motor batteries with its waste and inhalation of dust from aerosolized leaded gasoline. Human activities fostering enhanced lead exposure include: use of herbal remedies, ceramic glazes and toys and indiscriminate disposal of leaded materials.[3]

Malnourished children and those of workers in lead smelting factories are particularly predisposed.[1] Other persons unduly predisposed to heightened lead levels/ poisoning are children with chronic neurologic disorders (CNDs), on account of increased "hand to mouth" activity (mouthing and chewing of objects), associated behavioral disorders, attendant malnutrition, poor supervision and neglect.[4]
Unsafe blood lead levels particularly if sustained cause toxicity to organs and tissues as it interferes with extensive body processes and functions.[1] The nervous system in the growing child is worse hit because of its potential to disrupt development of the system.[5]

With already compromised brain functions children with CNDs are at increased risk of further brain damage as elevated BLLs have been reported to disrupt brain function causing disorders of cognition, intelligence, nonverbal reasoning, short term memory and learning.[5,6] Worldwide, the magnitude and burden of mental, neurological, and behavioral disorders are already huge[7] with CNDs being major contributors.[1,8] Determining the BLLs in children with CNDs would be initial steps in identifying those at risk with a view to taking measures aimed at forestalling further deterioration of the already precarious health status of the child with CNDs which equates to preventing a second lifelong tragedy. It is therefore worthwhile evaluating the BLLs in such children.

Subjects and methods

The descriptive cross-sectional study was carried out at the Child Neurology Clinic (CNC) and the General Out-Patient Clinic (GOPC) of the University of Benin Teaching Hospital (UBTH), Benin City. The Hospital is an 860 bed tertiary health care facility providing all levels of care for inhabitants of Edo, Delta and neighboring states.
The CNC holds on Wednesdays and an average of 20 patients are seen during each clinic day. Of the 20 cases eight new ones are seen per week. The clinic is covered by 2 child neurologists, 3 residents and 2 nurses. The GPOC holds daily and an average of 20 children are seen daily in the clinic. In the clinic register are over 500 cases with epilepsy, 105 cases with cerebral palsy, a sizable number of whom have epilepsy as co morbidity. About 100 cases have mental retardation while about a third of this have ADHD.
A semi- structured, pre-tested and researcher administered questionnaire was used to obtain relevant information. (biodata, parent's occupation and level of education, associated risk factors and possible sources of lead exposure. Family socio-economic classification was carried out using the method described by Oyedeji[9]
Permission for the study was obtained from the Ethics and Research Committee of UBTH. Included as subjects were children aged 1-15 years seen in neurology clinic with CND while controls were age and sex matched apparently healthy children with non-neurological conditions seen for minor ailments. Children with suspected epilepsy mimics were however excluded. Only study participants whose parents/guardians gave written informed consent took part in the study. Older children ($\geq$ 10 years) whose mental state permitted (in cases of subjects) also assented to participating in the study.
Vineland Social Maturity Scale (VSMS) was used to assess personal and social sufficiency of the subjects and controls in the areas of communication, daily living skills, socialization, motor skills and maladaptive behavior.[10]

Weight and length measurements and BMI calculation were carried using standard methods to determine their nutritional status. Length of children < 2 years was measured with a length board. The height of children $\geq$2 years was determined using the stadiometer. Average of 3 readings to the nearest 0.1cm was recorded in each case. Heights of those with CP were estimated as recommended by Stevenson.[11] The BMI of subjects were plotted on appropriate BMI percentile charts which enabled their nutritional status to be determined.
Four milliliters of venous blood was obtained from a suitable superficial vein of each child using aseptic techniques and transferred into a metal free, pre- labeled EDTA plastic bottle. The whole blood samples from the participants were stored within 6 hours of collection at -20^{o}C. They were transferred fortnightly in ice packs from Department of Child Health laboratory to the Nigerian Institute for Oil Palm Research (NIFOR), which is a 15minutes drive from UBTH. Determination of blood lead was done using Atomic Absorption Spectrophotometer (AAS, Varian Spectra AA10 model). Average of three readings of the absorbance were taken for each specimen and concentration recorded in parts per million (ppm) and converted to µg/dl by multiplying by a factor of 100.[12]

The test results were made available to the study participants and the managing physician alerted where values were elevated.
SPSS version 20.0 Statistical software was used to analyze the cleaned data. Mean ($\pm$SD) were calculated for continuous data while categorical data were presented as proportions. Test of significance was done using Pearson's Chi-square and Fisher's Exact test where applicable were used to test strength of association between proportions. Comparison of mean values was done using

independent t-test while the multiple logistic regression models were used to test for predictors of elevated BLLs while level of significance was set at $p < 0.05$ at 95% confident interval.

Results

One hundred and thirty nine children were recruited for the study. The 139 subjects with complete data were matched for age group and gender.
There were 89 (64.0%) male and 50 (36.0%) female subjects and same numbers of matched controls. The mean ± SD age of subjects of 6.54 ± 4.04 years was comparable to that of controls (6.45 ± 3.95 years). Among the 139 subjects, 96 (69.1%) were drawn from upper social class while 43 (30.9%) were from lower social class. The proportions of children with CNDs were: epilepsy, 80(57.5%); mixed (children with more than one form of CNDs), 24(17.3%); cerebral palsy, 23 (16.5%); attention deficit hyperactivity disorder, 8 (5.8%) and mental retardation (MR) 4(2.9%). More children in the age bracket 1-10 years compared to older children had the various forms of CNDs (c^2 =19.12, p=0.01) (table 1).

Table 1: Socio-demographic characteristics of subjects according to the different forms of CNDs

Socio-demographic Features	Epilepsy n=80(%)	Mixed n=24(%)	CP n=23(%)	ADHD n=8 (%)	MR n=4(%)	c^2	*p*- value
Age group (years)							
1-5	30(37.5)	11(45.8)	18(78.3)	5 (62.5)	1(25.0)		
6-10	28(35.0)	9(37.5)	5(21.7)	3 (37.5)	1(25.0)	19.12*	0.01
11-15	22(27.5)	4(16.7)	0(0.0)	0(0)	2(50.0)		
Gender							
Male	58(72.5)	12(50.0)	11(47.8)	6(75.0)	2(50.0)	8.0*	0.08
Female	22(27.5)	12(50.0)	12(52.2)	2(25.0)	2(50.0)		
SE status							
Upper	55(68.5)	16(66.7)	16(69.6)	6(75.0)	3(75.0)	0.37*	1.0
Lower	25(31.2)	8(33.3)	7(30.4)	2(25.0)	1(25.0)		

*Fisher's exact test; CP: Cerebral Palsy; ADHD: Attention Deficit Hyperactivity Disorder; MR: Mental Retardation; Mixed: More than one form of the disorders

Influence of gender age and family socioeconomic status on elevated blood lead levels

Among subjects eBLLs was independent of gender, age and family socio- economic status (Table II). Similarly age, gender and family socio- economic status did not influence the occurrence of elevated BLLs in controls.
Mean blood lead levels in subjects and controls.
The mean ± SD BLL in the subjects with CNDs of 25.05 ± 16.65 mg/dl (range; 8 – 72mg/dl) was significantly higher than the value in controls (19.24 ± 10.17 mg/dl, range 8 – 53mg/dl) (t = 3.51, p = 0.00).

Table 2: Influence of gender, age group and socio economic status on elevated blood lead levels in subjects and controls

Socio demographic features	Elevated BLLs Subjects n=101(%)	Controls n=111(%)	c^2	*p*-value
Gender				
Male (n=89)	[A]63(70.8)	71(79.8)	1.93	0.17
Female (n=50)	[B]38(76.0)	40(80.0)	0.23	0.63
Age (years)				
1-5 (n=65)	[C]45(69.2)	51(78.5)	1.43	0.23
6-10 (n=46)	[D]32(69.6)	38(82.6)	2.15	0.14
11-15 (n=28)	[E]24(85.7)	22(78.6)	0.49	0.49
SE status				
Upper (n=96)	[F]66/96(68.8)	*83(79.0)	2.26	0.13
Lower (n=43)	[G]35/43(81.4)	*28(82.4)	0.01	0.91

*For controls total number of children in upper and lower SE statuses respectively were 105 and 34.
Significant: Nil Not Significant
A vs B, c^2 =0.215, p=0.643; C vs D, c^2 = 0.001,p=0.970 ; C vs E, c^2 = 0.190, p=0.124 ; D vs E, c^2 = 0.214, p=0.164; F vs G, c^2=0.029, p=0.864.

Mean blood lead levels in subjects according to age, gender and family socio-economic status.

The mean (±SD) BLL was significantly higher in male subjects (24.92 ± 15.97 µg/dl) compared to male controls (19.35 + 10.63 µg/dl)(t= 2.74, p= 0.01). Similar trend was observed with female subjects compared to controls [(25.28+17.96 µg/dl vs 19.50+9.55 µg/dl, (t=2.01, p=0.04)]. Mean BLL in children aged 11-15 years was significantly higher than the mean value in controls of comparable ages. Among the subjects higher mean BLL was recorded in older children compared to younger ones *(C vs E, t=3.669, p=0.004 (95% CI: 6.801 -21.179; D vs E, t=3.879, p=0.0002 (95% CI: 7.171-22.329)*. Mean BLL in subjects drawn from upper SEC was significantly higher than the value in controls (24.67 + 17.12 µg/dl vs 18.12 + 9.17 µg/dl, t = 3.42, p = 0.00) (Table 3).

Prevalence of elevated blood lead levels in children with chronic neurologic disorders

The prevalence of elevated blood lead levels (eBLLs) (≥10 µg/dl) according to the different forms of CNDs is as shown in Table III. Though fewer (101/139 or 72.0%) subjects compared to controls (111/139 or 80.0%) had eBLLs there were however, more subjects with very high values (> 20 µg/dl) (71/101 or 70.3%) compared to controls (61/111 or 55.0%). Prevalence of eBLLs was highest in children with epilepsy compared with other forms of CNDs (c^2 =8.32, p= 0.06). Furthermore, more

children with eBLLs had values in the third category of elevated BLLs (20-44 µg/dl).

Mean BLL was significantly associated with the form of CNDs (F =2.75 p = 0.03) as children with mixed disorders (more than one form of CNDs) and epilepsy had significantly higher mean BLLs compared to other forms of CNDs. (Table 4). Some intra group differences also existed in the mean BLLs (table 4).

Table 3: Mean blood lead levels in subjects and controls according to gender, age group and socio economic status

Socio-demographic Features	Subject µg/dl	Control µg/dl	t	*P value*
Gender				
Male	[A]24.92 ± 15.97	19.35 ± 10.63	2.74	0.01
Female	[B]25.28 ± 17.96	19.50 ± 9.55	2.01	0.04
Age Group (years)				
1-5	[C]22.62 ± 15.64	19.57 ± 10.05	1.32	0.19
6-10	[D]21.61 ± 13.98	18.80 ± 10.25	1.10	0.28
11-15	[E]36.36 ± 18.56	19.45 ± 10.76	4.01	0.00
SE status				
Upper	[F]24.67±17.12	18.12 ± 9.17	3.42	0.00
Lower	[G]25.91±15.71	23.35±12.35	0.78	0.44

Significant:
C vs E, t=3.669, p=0.004 (95% CI: 6.801-21.179)
D vs E, t=3.879, p=0.0002 (95% CI: 7.171-22.329)
Not Significant: A vs B, t=0.122, p=0.903; C vs D, t = 0.350, p=0.727; F vs G, t=0.405, p=0.686.

Table 4: Mean Blood lead level in subjects according to form of CNDs

Chronic neurologic disorder (CND)	n (%)	Mean blood Lead Level (µg/dl)	F	*p value*
Epilepsy	80(57.5)	[A]27.21 ± 17.00		
Mixed*	24(17.3)	[B]28.54 ± 17.61		
Cerebral palsy	23(16.5)	[C]17.91 ± 13.81	2.75	0.03
ADHD	8(5.8)	[D]21.13 ± 13.09		
Mental retardation	4(2.9)	[E]9.75 ± 2.87		

*Mixed group consist of more than one category of CND
Significant:
A vs C, t=2.403, p=0.018, 95%CI: -16.998 -1.622
A vs E, t=2.041, p=0.044, 95%CI:-34.476-0.442
B vs C, t=2.296, p=0.026, 95%CI:-19.95-1.310
B vs E, t=2.097,p=0.040, 95%CI: -37.21-0.32
Not Significant:
A vs B, t=0.334, p=0.740; A vs D, t=0.981, p=0.330; B vs D, t=1.089, p=0.385; C vs D, t=0.575, p=0.570; C vs E, t=1.159, p=0.257; D vs E, t=1.680, p=0.124.

Risk factors for elevated blood lead levels in children with CNDs

Of the factors evaluated, only finger sucking was predictive of eBLLs. A child engaged in finger sucking was two and half times more likely to have eBLLs compared to those who do not. (OR = 2.56, p = 0.04, C.I 1.03 – 6.40) (table 5).

Subjects' adaptive levels and blood lead levels.

Utilizing the Vineland Adaptive Behavior Scale (range of low, moderately low, adequate, moderately high and high) subjects with "adequate'' and "moderately high" adaptive levels had mean BLLs of 26.05±15.51µg/dl and 29.60 ±20.23 µg/dl respectively. Though these were higher than values in subjects with "moderately low" adaptive level (20.00±12.66 µg/dl) there was however no correlation between adaptive level and BLLs (r=0.04; p=0.59).

Table 5: Risk factors predicting elevated blood lead levels in subjects

	n (%)	*P*	OR	95% C.I.for OR
Habit				
Finger sucking	10(9.9)	0.04	2.56	1.03 – 6.40
Pica	10(9.9)	0.25	0.41	0.09 – 1.89
Environmental				
Water storage	100(99.1)	0.90	0.97	0.57 – 1.64
House type	101(72.7)	0.51	1.27	0.62 – 2.57
Painted house	11(10)	0.56	1.63	0.32 – 8.36
Flaking paint	36(35.5)	0.50	0.75	0.33 – 1.72
Use of generator	76(75.5)	0.13	2.64	0.76 – 9.19
Flat level	20(19.9)	0.87	1.07	0.46 – 2.48
House location	40(40.0)	0.74	0.97	0.79 – 1.18
Others				
Use of eye cosmetics	6(5.9)	0.83	0.78	0.08 – 7.92
Wash toys	30(27.0)	0.21	1.35	0.84 – 2.16
Adaptive levels	101(72.7)	0.17	0.75	0.49 – 1.14

Nutritional status of study participants and mean blood lead levels.

The mean BLL of subjects with adequate nutrition was significantly higher than mean BLL of controls with similar nutritional status (t=3.12, $p = 0.00$). No significant intra group differences in mean BLLs existed among the subjects (table 6). Mean BLL was therefore independent of nutritional status of subjects. (table 6)

Table 6: Mean blood lead level of subjects and controls according to nutritional status

Nutritional Status	Subjects n (%)	Controls n (%)	Subjects Mean BLL (µg/dl)	Control Mean BLL (µg/dl)	*t*	*p-value*
Underweight	17 (12.23)	-	[A]22.76±15.91	-		
Normal	105 (75.53)	123 (88.49)	[B]25.17±16.43	19.50± 10.43	3.12	0.00
Overweight	10 (7.19)	12(8.63)	[C]29.10±20.77	17.00 ± 7.83	1.87	0.08
Obese	07 (5.03)	04(2.88)	[D]23.00± 18.08	23.75± 11.44	-0.07	0.94

Not Significant:
A vs B, t=0.563, p=0.574; A vs C, t=0.893, p=0.380; A vs D, t=0.032, p=0.975; B vs C,t=0.706, p= 0.484; B vs D, t=0.336, p=0.737; C vs D, t=0.627, p=0.540

Discussion

In the study, high prevalence of eBLLs was observed in subjects and in controls suggesting high environmental lead contamination in the study locale. This may be related to increased exposure to lead by the general population as found with the increasing use of lead based paints in homes, indiscriminate importation and disposal of lead containing gadgets (car batteries and electronic waste).[3]

Nigeria has a history of environmental lead contamination from past use of leaded petrol and use of lead based paint in homes.[13] The high prevalence of eBLL of 72.2% noted in the study is higher than the 60.0% reported by Kumar *et al* in children with CNDs in India.[4] Lewendon *et al* in 2001 in South West England[14] carried out a similar study among children with developmental and behavioral problems and reported a low prevalence of 12%. The differences in prevalence between the studies may be a reflection of the levels of environmental lead pollution, characteristics of the study subjects and effectiveness or otherwise of policies instituted by the concerned nations to regulate lead levels in the environment.

Mean BLL in subjects with CNDs was significantly higher than values in age and sex matched controls perhaps affirming the proposition that children with CNDs have higher incidences of social and behavioral problems that predispose them to lead ingestion/inhalation, hence elevated levels of blood lead compared to their apparently healthy counterparts. More male subjects compared to male controls and more female subjects compared to female controls had higher means of BLLs. A less important role for gender as a modifier of BLL is thus suggested though males compared to females, across most age brackets, are more physically active and more likely to interact with an environment that is already polluted with lead.

As earlier noted by Okoronkwo and Dadin Kowa in 2003 in Jos, BLLs were higher in adolescents compared to other age groups.[15] This may reflect the increased levels of socioeconomic and physical activities in such older children. It may also imply the tendency for lead to accumulate over time following consistent exposure.
Subjects from upper SEC had significantly higher mean BLL than controls. In contradistinction, Khan *et al*[16] in 1995 in Pacific North West Washington and Lewendon *et al* [14] in 2001 in South West England found no significant association between socio -economic status and mean blood lead levels in children with CND. Our observation may have to do with the nature of pollutants the subjects are exposed to. Lead contamination from car batteries, electronics and other lead laden gadgets are more likely to be experienced by children with CND drawn from the upper SEC. The regional differences may also be explained by the extent and robustness of the control measures instituted by the various countries.[14,16]

Children with CND suffer from poor care and supervision from care givers. They are also more likely to have behavioural aberrations that can predispose them to increased lead exposure. These may explain the higher mean blood lead levels in the study subjects compared to controls. A similar observation had been made by Kumar *et al* in 1998 among children with neurological disorders in Varanasi India.[4]
About 75% of children with ADHD had eBLLs with a mean value of 21.13µg/dl. Wang *et al* in 2008 in a study involving Chinese children aged 4-12 years with ADHD noted a lower prevalence of 24.0% and mean BLL of 8.77µg/dl. They had suggested that lead exposure early in childhood may have contributed to the occurrence of ADHD in their children.[17] More plausible is the fact that ADHD may be associated with habits that unduly predispose to lead poisoning. The higher mean BLLs and prevalence of eBLLs in individuals with ADHD in the current study may have to do with the higher level of lead pollution in the study locale.

Children with epilepsy and mixed forms of CNDs had higher mean BLLs compared to others suggesting that perhaps there are peculiar social conditions inherent in epilepsy that unduly predispose sufferers to lead poisoning. In between seizures some of such children are known to be hyperactive physically, interacting more with the environment. Later in life some children with epilepsy do also develop psychiatric complications with attendant deviant behaviors that could lead to undue ingestions of materials containing lead.

Children with mental retardation had low mean BLL. In contradistinction Okoronkwo and Dadin Kowa in Jos in 2003 recorded a much higher value of 25.3µg/dl among 100 mentally retarded children aged 6 to 14 years.[15] The lower mean BLL found in Benin compared to what obtained in Jos may have to do with the fact that unlike Jos mining activities in Benin is virtually non-existent. Mentally retarded children compared to other forms of CNDs also tend to get a lot of attention that tend to be protective from care givers and such may reduce their contact with potential sources of lead. Children with mixed disorders/epilepsy are more likely to be disadvantaged neurologically and therefore more prone to neglect, poor supervision and more aberrant behaviors. This may explain why children with mixed disorders and epilepsy had significantly higher mean BLLs compared to children with other forms of CNDs.

A probable implication of high mean BLLs found in children with CND in this study is that it could cause further deterioration of their existing precarious neurological disorders such as worsening cognitive impairment, reduced intelligence, seizure disorders, loss of fine motor skills and more behavioral abnormalities.[6]
Subjects engaged in finger sucking were two and half times more likely to have elevated BLLs. This is an acknowledged risk factor in lead exposure.[4] Children engaged in the act usually have their fingers soiled with materials laden with contaminants including lead prior to sucking. “Hand to mouth” activities such as

mouthing" of objects and finger sucking do coexist. Children with CND are noted to carry out more of these behaviors alongside other aberrant conducts such as pica which do predispose to increased lead levels in such children.[18] This is corroborated by findings in the study. Though not significant the risk of having eBLL was two and half times higher in children with CND who lived in homes that used generators. There is paucity of studies that evaluated the use of generators as a risk factor for eBLLs. It may be inferred from this that the use of generators in homes expose children to lead contaminated fumes. The use of generators in homes in Nigeria to provide power supply is rampant. Suspicion is rife that the state may not have fully implemented the policy to remove lead content in gasoline as leaded gasoline still tops the list of sources of environmental lead exposure via the inhalational route in Nigeria.[19] Adulterated gasoline frequently sold by marketers in Nigeria could be a veritable source of pollution.

Against expectations lead levels in children with CNDs had no correlation with their degree of adaptation. Theoretically, persons with low adaptive level should have high BLLs and reverse could also be true. However CNDs are complex morbidities with diverse management options. The environment including quality of care plays key roles in determining outcome. This may explain why the adaptation at the individual level would not play decisive role(s) in the level of blood lead in such children.

The significantly higher mean BLLs in children with CND who had normal nutritional status compared to their control counterpart also goes to embellish even further perhaps the unique predisposition of the former to lead poisoning.

This study revealed high prevalence of eBLLs in children with CNDs which perhaps may be indicative of high environmental lead pollution in the study locale. Epileptic children and those with mixed CNDs are unduly predisposed to higher levels of BLLs. Patients with CNDs that engage in hand sucking are at accentuated risk of having high BLLs. Children at risk of excessive levels of lead should be protected. Measures to reduce environmental lead pollution as may be instituted by the government may also be helpful.

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