ORIGINAL ARTICLE

Occupational and Environmental Lead Exposure in Port Harcourt, Nigeria: Analysis of its association with renal function indices

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Abstract

Background: *In spite of the high risk of lead exposure in Nigeria, there is a paucity of data on the occupational and environmental burden of lead exposure and its impact on human health especially its nephrotoxic effects. This study aims to assess the degree of occupational and environmental lead exposure in Port Harcourt Nigeria and the relationship between lead exposure and indices of renal function.*

Methods: *A cross sectional comparative study of 190 adult subjects with occupational lead exposure and 80 matched controls. Blood lead was used as the biomarker of lead exposure. Serum urea, creatinine, uric acid, urine albumin and glomerular filtration rate were the renal function indices measured.*

Results: *Occupationally lead exposed subjects had higher mean blood lead 50.37±24.58 ug/dl, than controls 41.40±26.85 ug/dl (p= 0.008). The mean values of serum urea, creatinine and uric acid were significantly higher in study subjects compared to controls 3.06±0.81 mmol/L vs. 2.7±0.84 mmol/L (p = 0.002), 87.2±14.30 umol/L vs. 80.68±14.70 umol/L (p = 0.001) and 271.93±71.18 umol/L vs. 231.1±62.70 umol/L (p = 0.000) respectively. Creatinine clearance was significantly lower in subjects compared to controls 98.86±21.26 ml/min/1.72m^2 vs.108.18±25.16 ml/min/1.72m^2 (p = 0.002). Blood lead correlated positively only with blood urea [r = .031, r2 = .017, p = .031] and negatively [r = -.144, r2 = .021, p = .018] with serum phosphate.*

Conclusion: *The level of environmental and occupational lead exposure in Port Harcourt, Nigeria is high, with occupational lead exposure increasing the risk of lead toxicity and renal function impairment.*

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Introduction

Lead has been a known toxicant for thousands of years, and it remains a persistent environmental and occupational health threat [1-3]. A range of adverse health outcomes which may manifest at low concentrations of blood lead (BPb) in the range of 5 10 micrograms (ug)/dl[2,4], have been attributed to lead nephropathy. These toxic effects include lead induced nephropathy[2]. The Center for disease control Atlanta (CDC) has defined elevated blood lead level (BLL) as BPb = 10 ug/dl [5]. This limit is justified by evidence which demonstrates subtle adverse health effects at lower levels[1,2].

Environmental [6-9] and occupational lead exposure [10-12] has been shown to result in renal function impairment and chronic kidney disease (CKD)[13] in addition to accelerating the progression to end stage renal disease (ESRD) in patients with CKD from other causes[1,13] and without diabetes[14].

In spite of the known hazards of lead exposure and its role as a causative factor and promoter of kidney disease, the level of environmental and occupational lead exposure still remains high in many developing countries[1,3], due poor prevention and control[3].

The significantly higher degree of environmental and occupational lead exposure in developing countries like Nigeria is largely attributed to the use of high percentage leaded gasoline[3,4] and poor regulation and monitoring of occupational exposure in contrast to the status in most highly industrialized countries[4,15].

The higher level of occupational and environmental lead exposure in Nigeria is thus expected to result in higher body lead burden as demonstrated by reports of earlier lead exposure surveys from Nigeria[4,16].

In spite of the high risk of lead toxicity in Nigeria, there is a paucity of data, on the environmental and occupational burden of lead exposure and its impact on kidney function. The objective of this cross sectional study was to evaluate environmental and occupational lead exposure and the association between lead exposure measures and indices of renal function among adults in Port Harcourt, Nigeria.

Materials and Methods

Study setting: The study was conducted in Port Harcourt, Nigeria, a city with significant volume of industrial activity, high traffic density and petrol refining

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industries. Study population and study design: The study population were adults between the ages of 18 to 60 years divided into two groups. Study subjects with occupational risk for lead exposure, who had been engaged in such occupations for over one year and age matched controls with limited occupational risk for lead exposure. The subjects were selected by stratified sampling on an occupational basis, from the following occupational groups: Welding/Metal works, Paint/Pigment workers, Radiator repairers, Battery workers and Petrol workers. Subjects who had prior history and treatment for Hypertension and Diabetes Mellitus, a previous history of renal disease, chronic use of mercury and hydroquinone containing cosmetics and a cumulative lifetime analgesic use exceeding 20 pills were excluded[17]. A clinical evaluation including socio-demographic assessment, clinical history and blood pressure (BP) measurements was done. Blood lead (BPb) was measured in subjects as a marker of lead exposure and analyzed by atomic absorption spectrometry (AAS) at the Fugro industrial and environmental laboratory in Port Harcourt observing all qualitative precautions. Renal function was assessed using serum urea, creatinine and serum uric acid (SUA). Creatinine clearance was calculated using the Cockcroft and Gault formula [Creatinine clearance (ml/min) = (140-Age) x body weight (kg)/plasma creatinine (mg/dl) x72], multiplied by 0.85 for females [18]. The estimation of urine albumin was done using the urine albumin creatinine ratio (ACR) after the collection of spot urine samples. In addition haemoglobin, serum calcium, serum phosphate and serum albumin were also measured. The laboratory assessment of renal function and other parameters were done at the University of Port Harcourt Teaching Hospital clinical chemistry and haematology laboratories. Ethical consideration: The approval of the Ethics and Research Committee of the University of Port Harcourt Teaching hospital was obtained before proceeding with the study. Informed consent was also obtained from each participant in the study. Data analysis model: The analysis of data in this study is based on a model for the quantitative assessment of the health impacts of lead in population groups which involves the assessment of lead exposure based on the blood lead levels in the general and specific population group; the classification of blood lead levels (BLL) into the following blood lead intervals, level 1 to 7: with the corresponding blood lead values. Level 1 (510 µg/dl), Level 2 (1015 µg/dl), Level 3 (1520 µg/dl), Level 4 (> 20 µg/dl), Level 5 (= 60 µg/dl), Level 6 (= 70 µg/dl) and Level 7 (= 80 µg/dl) and the correlation of lead exposure with various health effects for the study population such as impairment in renal function[4]. Tests of significance were calculated using the student t-test for independent samples and the F-test using analysis of variance (ANOVA) for continuous variables. Qualitative and categorical data were compared using the chi-square tests and relative risk (RR) measure. Pearson and Spearman bivariate correlation analyses and linear regression was used to determine the relationship between blood lead levels, renal function indices and other study variables with p values of < 0.05 considered significant as appropriate.

Results

Demographic characteristics of study population: There was no significant difference in the mean age of lead exposed subjects 34.78(10.05) years compared to controls, 35.95(10.09) years, p (0.385). There was a higher proportion of males participants, but no significant difference was observed in the proportion of males and females in both groups, p = 0.21.

(Table I and II). ***Duration of occupation:*** The mean duration of occupation in the study subjects was 11.91 (9.2) years with a range of (2 48) years (Table I), while the value in controls was 8.03 (7.32) range of (2 30) years (Table II). A significant difference was observed between the means, with a p = 0.001 (Table III)

Blood pressure in study population: The mean systolic blood pressure (SBP) in study subjects was 118.49 (14.67) mmHg compared to 113.62 (11.31) mmHg in controls with a significant difference observed, expressed by p = 0.008. (Table III). The mean diastolic blood pressure (DBP) in study subjects was 74.64 (10.98) mmHg compared to 73.10 (7.47) mmHg in controls. No significant difference was observed with a p = 0.285. (Table III). A significantly higher proportion of subjects 9.47% had SBP = 140mmHg compared with 1.25 % in controls, p = 0.016 with a RR = 1.38<1.21<RR<1.58>. A significantly higher proportion of subjects 9.5% had DBP = 90mmHg compared to 2.5% of controls, p = 0.028 and a RR =1.33<1.13<RR<1.55>. BLL in study population: There was a significant difference in the mean BPb in the study subjects 50.37(24.58) ug/dl compared to 41.40(26.85)ug/dl in controls. (p = 0.008), (Table III). The distribution of BLL by blood lead groups in both subjects and controls shows that 92.1% of subjects and 72.6% of controls had BPb above level 2 >20ug/dl, with a significant difference in the proportions, expressed by p = 0.000 and RR=1.85<1.25<RR<2.76>. Renal function indices in study population: The mean values of renal function indices of the subjects and controls are

shown in (table III). The mean serum urea in the study subjects was 3.06(0.81) mmol/L compared with a mean of 2.7 (0.84) mmol/L in the controls with p = 0.002. The mean serum creatinine in the study subjects was 87.2 (14.30) umol/L while the mean in controls was 80.68 (14.70) umol/L with p = 0.001. The mean SUA in the study subjects was 271.93 (71.18) umol/L while the mean in controls was 231.1 (62.70) umol/L with p = 0.000. The mean calculated creatinine clearance was significantly lower in study subjects compared to controls 98.86 (21.26) ml/min/1.72m^2 BSA vs. 108.18 (25.16) ml/min/1.72m^2 BSA (p = 0.002) respectively. The comparison of urine albumin excretion using the urine ACR did not show any significant difference, though study controls had a higher mean 32.71 (11.72) mg/g creatinine compared to subjects, 30.99 (13.30) mg/g creatinine (p = 0.316). Haemoglobin, calcium and phosphate in study population: The mean haemoglobin level in subjects was 12.5(1.45) g/dl compared with controls 12.6(1.16) g/dl, (p = 0.518), (Table 3). The mean serum calcium in the study subjects was 2.13(0.156) mmol/L compared with 2.10(0.101) mmol/L in the controls with (p = 0.083), (Table 3). The mean serum phosphate level of the study subjects was 1.4 (0.161) mmol/L while the mean in controls was 1.37(0.097) mmol/L. With (p = 0.139), (table 3).

Bivariate Correlation Analysis: The pattern of correlation between blood lead and the study groups (subjects and controls) showed a trend of higher blood lead in the subjects compared to controls [r = -0.180, p = .003]. There was a significant positive correlation between blood lead and blood urea [r = 0.131, p = 0.031] and a significant negative correlation between blood lead and serum phosphate [r = -0.144, p = 0.018].

Simple and multiple Linear Regression analysis: With simple linear regression analysis blood lead level was significantly and positively associated with blood urea [r = .031, r2 = .017, p = .031] (Figure 1) and significantly negatively associated with serum phosphate [r = -.144, r2 = .021, p = .018] (Figure 2). With multiple linear regression analysis blood lead was also independently significantly and positively correlated with serum urea [r = .178, r2 = .032, p = .047] and negatively with serum phosphate [r = -.187, r2= .035, p = .036]. Serum phosphate was the only predictor of lead induced serum urea change.

Table I. Demographic characteristics of subjects

Parameters	Frequency distribution of variables	N (%)	Range	Mean (SD)
Age (Years)	18-30	78 (41.6%)	18 58	34.78(10.05)
	31-40	49 (25.8%)		
	41-50	42 (22.1%)		
	51-60	21 (11.1%)		
Sex	Male	151(79.5%)		
	Female	39 (20.5%)		
Marital status	Married	109(57.4%)		
	Single	81 (42.6%)		
Occupation	Welding and metal	42 (22.1%)		
	Paint and Pigment	38 (20.0 %)		
	Radiator repairer	37 (19.5%)		
	Battery workers	37 (19.5%)		
	Petrol	36 (18.5%)		
Duration of occup ation (Years)			2 48	11.91 (9.2)
Cigarette smoking	Smokers	9 (4.7%)	0 15	0.41 (2.4)
(Pack years)	Non Smokers	181 (95.3%)		
Alcohol consumption	Take alcohol	53 (27.9%)	0 -216	19.32 (4.10)
(grams/week)	Do not take alcohol	137 (72.1%)		
BMI(Kg/m ^{2})	< 18.5	4 (2.1%)	16.94 -	24.91(3.638)
	18.5 24.9	97 (51.1%)	40.65	
	25 29.9	74 (38.9%)		
	30 34.9	13 (6.8%)		
	35 39.9	1 (0.5%)		
	> 40	1(0.5%)		
Fasting blood sugar (mmol/L)			3.3 5.7	4.21 (0.59)

Table II. Demographic characteristics of controls

Parameters	Frequenc y distribution of variables	N (%)	Range	Mean(SD)
Age (Years)	18-30	26 (32.5%)	19 - 59	35.95(10.09)
	31-40	24 (30.0%)		
	41-50	19 (23.8%)		
	51-60	11 (13.7%)		
Sex	Male	58 (72.5%)		
	Female	22 (27.5%)		
Marital status	Married	25 (31.2%)		
	Single	55 (68.8%)		
Occupati on	Hospital workers	80 (100%)		
Duration of occupation (Years)			2 - 30	8.03 (7.32)
Cigarette smoking (Pack years)	Smokers	3 (3.7 %)	0 - 10	0.30 (1.62)
	Non Smokers	77 (96.3 %)		
Alcohol consumption (grams/week)	Take Alcohol	20 (20 %)	0 - 126	12.15 (26.7)
	Do not take alcohol	60 (75 %)		
BMI (Kg/m ^{2})	< 18.5	1(1.3%)	18.30 - 47.96	26.13(5.107)
	18.5 - 24.9	39 (48.8%)		
	25 - 29.9	24 (30.0%)		
	30 - 34.9	13 (16.3%)		
	35 - 39.9	2 (2.5%)		
	> 40	1(1.3%)		
Fasting Blood Sugar (mmol/L)			3.4 - 5.3	4.12 (0.532)

Table III. Comparison of study variables between subjects and controls

	Subjects (N=190)	Controls (N=80)	
Variables	Mean (SD)	Mean (SD)	P. value
Age (Years)	34.78(10.04)	35.95 (10.09)	0.385
Duration of occupation (years)	11.91(9.26)	8.03(7.32)	0.001
Alcohol us e (g/week)	19.32(41.10)	12.15(26.75)	0.152
Cigarettes (pack years)	0.47(2.40)	0.30(1.62)	0.555
BMI (Kg/m2)	24.91(3.63)	26.13(5.10)	0.028
Systolic BP (mmHg)	118.48(14.67)	113.62(11.31)	0.008
Diastolic BP (mmHg)	74.67(10.98)	73.23(7.31)	0.285
Blood Lea d (ug/dl)	50.37(24.58)	41.40(26.85)	0.008
Urea (mmol/L)	3.06(0.81)	2.72(0.84)	0.002
Creatinine (umol/L)	87.21(14.30)	80.68(14.70)	0.001
Uric acid (umol/L)	271.93(71.18)	231.18(62.70)	0.000
Creatinine clearance (ml/min/ 1.73m2 BSA)	98.86(21.26)	108.18(25.16)	0.002
Urine Alb (mg/g cr)	30.99(13.30)	32.71(11.72)	0.316
Haemoglobin (g/dl)	12.66(1.16)	12.56(1.45)	0.518
Serum Ca $^{+}$ (mmol/L)	2.10(0.10)	2.13(0.15)	0.083
Serum PO ^{4} (mmol/L)	1.37(0.16)	1.40(0.09)	0.139

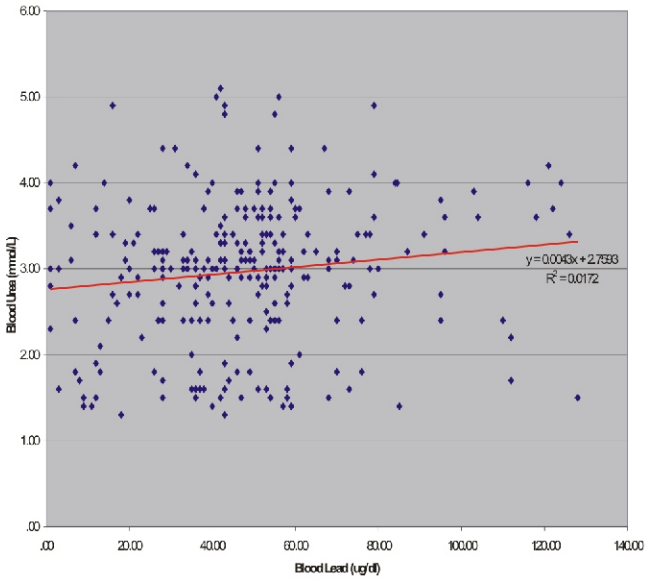

Figure 1. Simple linear regression plot of Blood lead against serum urea

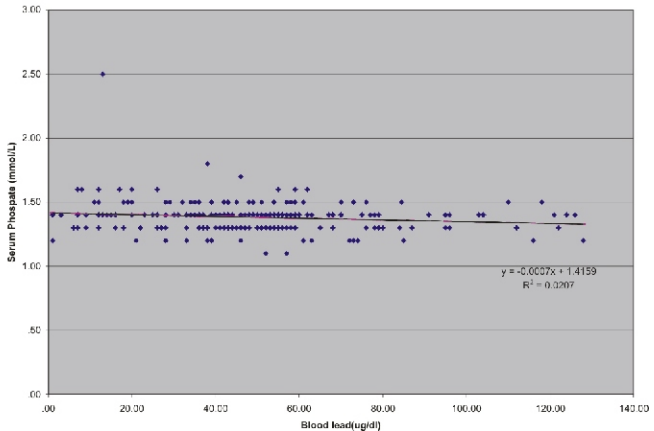

Figure 2. Simple linear regression plot of Blood lead against serum phosphate

Discussion

Lead exposure in Port Harcourt: The classification of study participants by their BLL showed that 92.1% of subjects and 72.6% of controls had BPb above level 2 >20ug/dl. The figures reported are higher when compared to that of other previous reports in occupationally exposed workers[19], from a study in Boston who found a prevalence of 39% for lead workers with blood lead > 40ug/dl. Another study [20] in Alexandria, Egypt among adolescents in lead exposed occupations showed a prevalence of 96.1% of subjects with BPb > 10ug/dl and 20.1% = 25ug/dl. Correspondingly the figures in environmentally exposed controls also followed the same trend, when compared to previous reports in Ibadan, Nigeria[16] that reported 72% of the study population n with BPb >10ug/dl, while data from the WHO[4] indicate that 14.3% of adults in Nigeria have BPb > 20ug/dl. The results of this study therefore indicate a high risk of lead toxicity in Port Harcourt as well as a higher frequency for the risk of lead toxicity in occupationally exposed persons compared to controls.

Occupational Lead exposure in Port Harcourt: The results of this study established significantly higher mean BPb in study subjects compared to controls 50.3(24.5) ug/dl vs. 41.40(26.85). This finding is consistent with reports from another Nigerian study [21] that reported a mean BPb of 56.3 (0.95) ug/dl in occupationally exposed subjects compared to 30.47(1.4) ug/dl in controls and studies in other countries. A study of Korean lead workers reported a mean BPb of 32.00(15.00) ug/dl compared with 5.8(1.8) ug/dl in controls [12]. Similarly Jung et al [22] reported the following mean BLL of 74.6 (7.8) µg/dl, 46.5 (5.9) µg/dl and 24.3 (2.7) µg/dl respectively in the highly exposed, moderately exposed and slightly exposed lead workers and 7.9 (1.4) ug/dl in the control group. Pergande and co-workers in a German study[23] also reported higher mean BPb in lead workers 40.6ug/dl with a range of (20.2 70.6)ug/dl against 6.8ug/dl with a range of (4.8 10.6) ug/dl in controls, while Medhi et al [24] in Iraq also reported higher mean BPb in three groups of lead exposed workers 71.70ug/dl, 58.00ug/dl and 36.35ug/dl compared with 14.63ug/dl in controls. In a Taiwanese study[25] the mean BPb of 15.8 µg/dl in male and 11.6 µg/dl in female lead exposed workers was significantly higher than the 8.6 µg/dl and 6.7 µg/dl for male and female controls respectively.

Ogunshola et al[26] reported that the mean blood lead level of 18.1 (6.4) µg/dl in traffic wardens in Lagos was

significantly higher than 12.9 (7.0) ug/dl in controls in Lagos. in the same way another study[10] among South African lead workers also reported high mean BPb of 53.5(ug/dl) with a range of (23 110) ug/dl. Cardenas et al[27] in France, Goldman et al[19] in Boston and Omae et al[28] in Tokyo reported mean BPb of 48.0ug/dl, 37.1ug/dl and 36.5ug/dl respectively among lead exposed workers, while Nomiyama et al[29] reported a mean BPb of 55.42 (13.52) ug/dl in Korean female lead workers. The results from this study reveal a higher degree of lead exposure in occupational exposed persons in Port Harcourt in comparison with controls. Furthermore the similarity in the BLL of 50.6ug/dl reported by Anetor[21] in Ibadan, South West Nigeria may reflect uniformity in the National occupational lead exposure risk, especially with the similarities in the occupational groups studied. The difference in the occupational groups studied may explain the significant variance in the BPb reported by Ogunshola et al[26], in a study of traffic wardens in Lagos. It is important to note that the BLL reported in this study subjects approximates with that of other studies in South Africa[10], Korea[12,22] and Iraq[24] but is lower than the levels in studies from America[19] and Europe[23]. These findings seem to corroborate earlier data which indicate higher level of occupational lead exposure in developing countries. Several factors may be responsible for the high level of occupational lead exposure found in this study. These include the poor regulation of occupational lead exposure among risk groups, resulting from the lack of occupational lead exposure monitoring and reduction activities and the low level of awareness of lead toxicity among people engaged in occupations which put them at risk.

Environmental Lead exposure in Port Harcourt: The mean BPb of 41.40 ug/dl reported for the controls in this study is worrying when compared with data from other studies in urban areas in Nigeria. Anetor [21] reported a mean BPb of 30.47 ug/dl while Omokhodion[16] found the average blood lead in an adult population of Ibadan, South-West Nigeria to be 11.4ug/dl for females and 12.3 ug/dl for males. Ogunshola et al[26] in a study of non-occupationally exposed adults in Lagos, Nigeria found the mean blood lead to be 13.0 ug/dl. The value reported in this study is also higher than the regional mean blood lead level in adults in urban areas in Africa which is estimated[4] to be 11.6 ug/dl and 10.4 ug/dl. Further comparison of the mean blood lead level in the study controls with the levels reported in civil servants in a London study [30], who had a mean blood lead of 11.6 ug/dl in men and 9.2 ug/dl in women and that of a Korean study [22] of 7.9 ug/dl, indicate higher levels of environmental lead exposure in Port Harcourt. The high level of environmental lead exposure found in this study may result from a high level of lead in air, arising from various emission sources. These sources include high traffic density, with resultant emissions from second hand vehicles run on leaded gasoline and the use of gasoline power generators in homes and home based work places. The open burning of solid wastes is a major method of waste management in Port Harcourt; this process has also been found to contribute significantly to the air lead level [31]. In addition there is a significant level of unregulated cottage and other industrial processes such as printing, battery repair, welding, electronic repair, automobile repair and the fabrication of plastics which are usually carried out in crowded residential areas, typically in stores or work shades adjoining houses. These sources are further compounded by poor urban planning and development which result in narrow, dusty and overcrowded streets as well as the predominant use of untreated ground water and a high level of petrochemical activities which accelerates the process of environmental exposure.

Lead exposure and renal function indices: The Comparison of renal function indices showed significantly higher mean values of serum urea levels in study subjects compared with controls. This is consistent with the findings of Jung et al[22] and Endo et al[32]. In addition serum urea was the only index of renal function with significant positive correlation with blood lead. This correlation between blood lead and urea in this study may indicate that urea is a sensitive indicator of lead nephropathy as urea is a more acute marker of renal disease compared to creatinine. The mean serum creatinine was significantly higher in subjects compared to controls, a similar observation was found in other studies in South Africa[10] and brazil[33]. In spite of the higher mean level of creatinine in the study subjects, there was no significant correlation between creatinine and BPb in this study consistent with the findings of some other studies [22,25,34]. The higher creatinine levels in study subjects may indicate a higher probability of renal impairment. SUA was significantly higher in the study subjects than controls as also reported by other studies [33,35]. No significant correlation was observed between SUA and BPb in this study, which is similar to previous observations Jung et al[22] and Omae et al[28]. The significant differences in SUA may suggest a higher possibility of lead induced renal impairment of in study subjects, as hyperuricaemia is a common feature of lead nephropathy.

Creatinine clearance was significantly lower in the subjects compared to controls as reported in a study[23] of

German battery workers. In this study there was no significant correlation between creatinine clearance and BPb, a similar finding was reported in a cohort of Japanese lead exposed workers [28]. The comparison of urine albumin excretion using the urine ACR did not show any significant difference in albuminuria between subjects and controls. Jung et al[22] also did not observe a significant difference in urine albumin between subjects occupationally exposed to lead and controls. There was no significant correlation between blood lead and urine albumin in this study. Reports from other studies [10,22] also did not show a significant correlation. Albuminuria results both primarily from glomerular damage and tubular dysfunction, however in lead nephropathy which is an interstitial renal disease with principal tubular involvement glomerular albumin excretion is minimal especially in the early stages. Therefore results from this study seem to support earlier assertions [22,36] that urine albumin is not a helpful indicator of renal dysfunction in lead nephropathy. In summary significant differences were observed in serum urea, SUA, serum creatinine and creatinine clearance in study subjects compared to controls with a positive correlation between BPb and urea level. These findings may indicate a higher risk of renal function impairment in the occupationally exposed subjects as the differences observed between study subjects and controls could be related to lead exposure, though the findings do not imply causality. In addition, the hypothesis that the initial effects of lead on renal function could result in hyper filtration [37], may also make identification of early lead induced renal impairment with creatinine clearance difficult.

Blood pressure and lead exposure: The association between BPb and BP, with or without renal dysfunction, has been the subject of various epidemiological investigations. While several studies[38-42] have established a significant positive correlation between increased BLL and Hypertension there are other studies[43-45] which contradict this assertion. In this study the mean SBP was significantly higher in the study subjects compared to controls while there was no significant difference in the DBP of both groups. In addition a significantly higher frequency of SBP and DBP elevation was observed in the study subjects compared to controls. In spite of the poor correlation of blood pressure with blood lead in this study the significantly higher systolic blood pressure observed in the subjects and the significant higher rates of SBP and DBP elevations in the study subjects does suggests that occupational lead exposure may predispose to higher level of SBP and rates of hypertension.

Haemoglobin and blood lead: There was no significant correlation between haemoglobin and blood lead in this study. Therefore, this study did not establish significant association between occupational and environmental lead exposure and haemoglobin levels, even with mean blood lead level in the study subjects above 50ug/dl, a level at which the haematological effects of lead exposure occur[46].

Serum Calcium, Serum Phosphate and blood lead**:** The absorption and distribution of lead is influenced by the dietary intake and blood levels of calcium and phosphate [47,48]. The mechanisms explaining the effect of calcium and phosphorus on lead toxicity are related to the absorption of lead from the gastrointestinal tract and renal tubule and to the function of the parathyroid glands[47,48]. The effects of vitamin D, calcium and phosphorus on lead absorption are thus complex and interrelated. These effects are dependent on the duration of lead exposure, the magnitude of body lead stores and dietary content of calcium and phosphate. It is has thus been established that low levels of calcium and phosphate are associated with higher lead levels [47,48]. The significant negative correlation between serum phosphate and BPb observed in this study is consistent with the findings of a study which evaluated the relationship between lead and cadmium with renal function, calcium and phosphorus [49]. The result of this study does imply that low serum phosphate levels are associated with increasing BLL and that serum phosphate is a significant predictor of lead induced increase in serum urea levels.

In conclusion the outcome of this study indicates a significant level of environmental and occupational lead exposure in Port Harcourt. This is supported by the frequency of risk for lead toxicity from occupational and environmental lead exposure in the study subjects based on the proportion of subjects and controls with BPb > 20ug/dl which was 92.1% and 72.6% respectively. This proportion is higher than 14.3% with BPb > 20ug/dl for adult Nigerians in urban areas reported by the WHO[4]. The mean BPb of the study subjects and controls was 50.37(24.58) ug/dl and 41.40 (26.85) ug/dl respectively. This value far exceeds the 10ug/dl set by CDC as the limit of acceptable BLL. The findings also demonstrate a higher risk of lead exposure in occupational exposed subjects compared to environmentally exposed controls, with occupational lead exposure increasing the likelihood of renal function impairment. On the other hand, it should be noted that this report is a relatively small scale study thus the generalization of the findings may be limited.

Resultantly further larger scale studies on environmental and occupational lead exposure and lead health effects assessment in both rural and urban areas of Nigeria will be required in addition to public health programmes aimed at promoting public understanding and awareness; concerning the effect on human health associated with exposure to lead and lead exposure pathways.

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