

Toxicological Profiling of Dusts from Selected Public Schools in Rivers State, Nigeria

Olua, Victor^{1*}, Patrick-Iwuanyanwu, Kingsley Chukwuemekam^{2,3}, Nwaichi, Eucharia Oluchi², Ajie Bliss Isioma Adaego⁴, Chigozirim, S. Amadi⁴, Ukaigwe, Agbonma Barbara², Adele, Habiganuchi²

1. Energy Technology Institute, University of Port Harcourt,
2. Department of Biochemistry, Faculty of Science, University of Port Harcourt P.M.B. 5323, Choba, Rivers State.
3. Africa Centre of Excellence for Public Health and Toxicological Research (ACEPUTOR)
4. Department of Biochemistry, Faculty of Science, Rivers State University

**E-mail of the Corresponding Author: vyckol@yahoo.com*

Abstract

The potential toxicity effects and health risk of exposure to heavy metals in dust samples from selected primary schools' windows in Rivers State was studied. Twenty-seven samples from nine Public Primary Schools were collected from selected Local Government Areas based on factors such as proximity to urban industrialization, and high traffic ways. Dusts from window corners were analysed for Pb, Cd, Cr and As using Atomic Absorption Spectrophotometer (AAS, GF, Flame HVG, model: S4=71096). Results showed mean maximum levels for Pb ($21.26 \pm 0.12 \text{ mgkg}^{-1}$) and Cd ($2.36 \pm 0.00 \text{ mgkg}^{-1}$) in Schools B₂ and A₁ while mean maximum levels for Cr ($42.46 \pm 0.12 \text{ mgkg}^{-1}$) and As ($0.94 \pm 0.1 \text{ mgkg}^{-1}$) was seen in school C₂. Heavy metal pollution profiling revealed high Cd load in all samples except in B₃. Multiple Pollution Index (MPI) decreased in the order A₁ > A₂ > B₂ > B₁ > A₃ > C₂ > C₃ > C₁ > B₃. Chronic daily intake dose (CDI_{ingestion, dermal and inhalation}) were below the recommended reference doses with ingestion contributing more to exposure than dermal or inhalation. Target Hazard Quotients (THQ) and Hazard Index (HI) were below 1. Furthermore, Life cancer Risks (TLCR) indicated that children population in the study areas may be more vulnerable to carcinogenic risk and may pose a public health concern.

Key words: Heavy Metals, Dusts, Public Primary School, Health Risk Assessment, Carcinogenic Risks.

DOI: 10.7176/JEES/15-4-05

Publication date: September 30th 2025

1. Introduction

Anthropogenic activities around the Niger Delta region of Nigeria has largely contributed to environmental pollution. These intensive oil and gas exploration and exploitation activities are majorly responsible for the release of contaminants such as heavy metals, organic and inorganic substances into the environment. Consequently, dusts from urban and industrial wastes in this region are usually made up of a complex mixture of particles. Heavy metals in dust may arise from various sources such as vehicular emission, industrial wastes, road surface wear, atmospheric deposition and particulate emission (López et al., 2005; Al-Khashman & Shawabkeh, 2006; Popoola et al., 2012; and Olua, Patrick-Iwuanyanwu, & Nwaichi, 2018). Human beings are generally exposed to lots of these contaminants, environmental hazards and potential toxicant. Studies have shown that these contaminated dusts are found around our homes, offices, classrooms (Popoola et al., 2012 and Olua et al., 2018) etc.

The Niger Delta region of Nigeria and in particular Rivers State has witnessed a long term exposure to illegal crude refining, open air burning (Babayemi et al. 2009; Ighariemu et al. 2023), high traffic congestion, gas flare etc all of which contribute to air pollution. This no doubt settles together with suspended dust particles both indoors and outdoors and are thereby been exposed to humans.

These enter into the human system either by oral ingestion, dermal contact, and inhalation or through these three (3) exposure means (Tong, and Lam, 1998; Latif et al. 2014; Olujimi et. al. 2015 & Olua et. al., 2018). Several studies have revealed presence of contaminants such as heavy metals in dusts seen in offices and class rooms (Popoola et al., 2012; Addo, 2012, and Olua et. al., 2018). Heavy metals can cause adverse health effects on humans (Sanborn et al. 2002; Faiz et al. 2009; Sarda et al. 2013; Ighariemu et al. 2023; Olua et al. 2021; Nyimone et al. 2024; Olua et al. 2024). Lead (Pb) on entering the body system could limit water imbalance, enzyme activities and change in hormonal status. It could also alter the permeability of membranes; high concentrations could restrain cell activities leading to cell death (Seregin, and Ivaniov, 2001; Nyimone et al. 2024). Cadmium causes bone disorders on chronic exposure (Asia et al. 2008). It could have toxic effect even on low absorption via ingestion. No known beneficial effect of Cd is known yet (Gough, et al. 1979 and Aydinalp, and Marinova, 2009). Chromium produces pulmonary defects or symptoms on exposure to humans, on inhalation it could cause ulceration of the

nasal mucosa (Dayan, and Paine, 2001; Lindberg, E. and Vesterberg, 1983). Arsenic has the ability of disrupting ATP production, it as well inhibits lipoic acid in tricarboxylic acid cycle (Klaassen, C. and Watkins, 2003). As arsenate it can uncouple oxidative phosphorylation, thus stalling reduction of NAD^+ . Some of these metabolic meddling could however lead to death (Sabina, 2005). This study however examined the presence of heavy metals in dusts obtained around classroom window corners in selected public schools in Rivers State.

Materials and Methods

Materials

Solar thermo elemental Atomic Absorption Spectrophotometer (Flame AAS) model: S4=71096, Analytical balance: Capable of weighing 0.1 mg. Stock metal solution, Aluminum nitrate solution, Hydrogen tetraoxosulphate (vi) acid (H_2SO_4), Trioxonitrate (v) acid (HNO_3), Perchloric acid (HClO_4).

Study Area

The study area covers selected public primary schools within Rivers State.

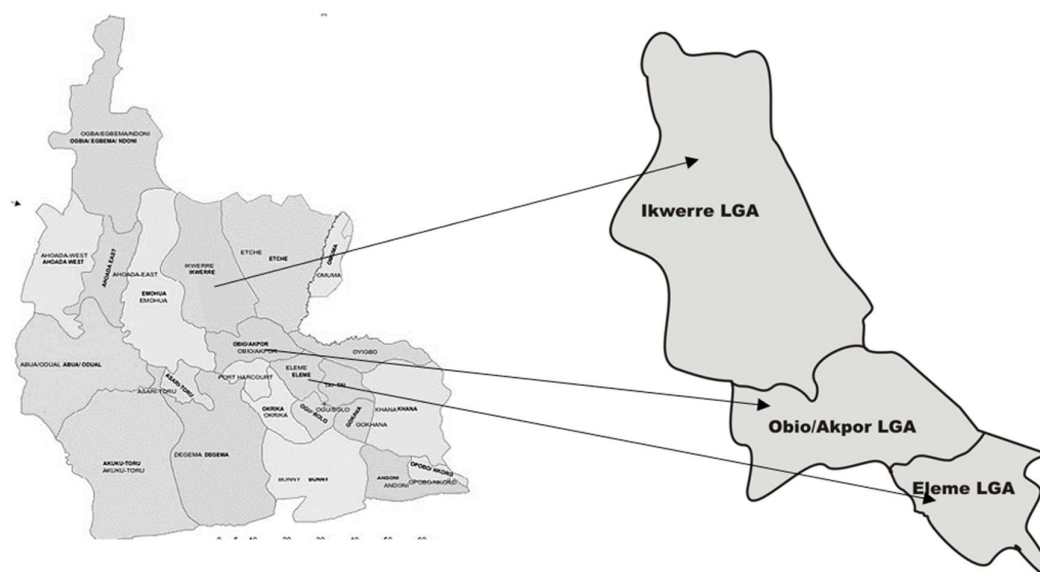


Fig. 1 Map of Rivers State showing sampled LGAs

Sample Collection Preparation and Analysis

Dust samples were collected from the sample sites using hard brush and foil:

At each sampling point, 50g of dust samples were collected from classroom window corners. The collected samples were put into sterile bottles and labelled appropriately before taking them to the laboratory for digestion (Acid digestion) and analysis.

Each sample was analysed for heavy metals (Pb, As, Cd, Cr and Ni) using Atomic Absorption Spectrophotometry using the method described by Olua *et al.* (2018)

Quality assurance and control (QA/QC)

Trace elements in indoor dust, from NIST, USA was used as the certified reference elements (CRM) NIST® 2584 and was evaluated using same experimental procedure in order to authenticate integrity/accuracy of procedure and results obtained. About 95% accuracy was recorded. Cross contamination and interferences were put on check using spiked Standard sample and procedural blank. All readings were taken in triplicate ($n=3$). Blank values for sampled metals were below the limit of detection (LOD- this measures concentration of analyte that gives 3 times the baseline's noise level i.e signal to noise ratio). Obtained LOD was 0.079mgkg^{-1} for Pb, Cr and As while that of Cd was 0.01mgkg^{-1} . Limit of quantification (LOQ), (given as the concentration which gives 10 times the baseline's noise level) was 0.25mgkg^{-1} for Pb, Cr and As while that of Cd was 0.03mgkg^{-1} .

Contamination/pollution index (C/PI): The C/PI was calculated using (Lacatusu, 1998)

$$C/PI = \frac{\text{Concentration of metal in dust}}{\text{Reference value}} \quad (1)$$

The reference value was taken from the crusta abundance value of respective sampled metals (Turekian & Wedepohl, 1961). A C/PI value >1 shows pollution range while values <1 shows contamination range. Multiple pollution index (MPI) was calculated from the sum of respective metal from sampled dust's C/PI values greater than 1.

Index of geo-accumulation (I_{geo}): The I_{geo} values as proposed by Muller (1969) was used to evaluate degree of metal contamination in the sampled dusts.

This was calculated using;

$$I_{geo} = \log_2 \frac{C_n}{1.5B_n} \quad (2)$$

where C_n denotes concentration of individual heavy metal in collected dust sample while B_n is background level of same metal as obtained from their crustal abundance value respectively (Turekian & Wedepohl, 1961). The factor 1.5 normalizes the likely lithological differences which exists between test and background samples (Rogan, et al. 2010).

Table 1. Significance of indices for contamination/pollution, and Index of geo-accumulation.

C/PI	Significance	I_{geo}	Class	Significance
<0.1	Very slight contamination	<0	Class 1	Practically unpolluted
0.10–0.25	Slight contamination	0-1	Class 2	Unpolluted to moderately Polluted
0.26–0.5	Moderate contamination	1-2	Class 3	Moderately polluted
0.51–0.75	Severe contamination	2-3	Class 4	Moderately to strongly polluted
0.76–1.0	Very severe contamination	3-4	Class 5	Strongly polluted
1.1–2.0	Slight pollution	4-5	Class 6	Strongly polluted to very polluted
2.1–4.0	Moderate pollution	>5	Class 7	Extremely polluted
4.1–8.0	Severe pollution			
8.1–16.0	Very severe pollution			
>16.0	Excessive pollution			

(Lacatusu, 1998)

Health Risk Assessment

Health risk assessment is a risk characterization of the potential adverse health effects that could occur on human exposure to contaminants (Luo et al. 2012). The International Agency for Research on Cancer has classified As, Cd, Cr and Pb as potential non-carcinogenic and carcinogenic elements. According to the Exposure Factors Handbook (USEPA. 2002), the Chronic daily intake, CDI $\text{mgkg}^{-1}\text{day}^{-1}$ of heavy metals via ingestion, dermal contact and inhalation was estimated using the following equations, respectively:

$$CDI_{\text{ingest}} = C \times \text{IngR} \times \text{EF} \times \text{ED} \times \text{CF} / \text{BW} \times \text{AT} \quad (3)$$

$$CDI_{\text{dermal}} = C \times \text{SA} \times \text{AF} \times \text{FE} \times \text{ABS} \times \text{EF} \times \text{ED} \times \text{CF} / \text{BW} \times \text{AT}, \quad (4)$$

$$CDI_{\text{inhale}} = C \times \text{InhR} \times \text{EF} \times \text{ED} / \text{PEF} \times \text{BW} \times \text{AT}, \quad (5)$$

where C is the concentration of heavy metals (mg/kg); IngR = the ingestion rate (mgday^{-1}); SA = the surface area of the skin exposed to heavy metal (cm^2); AF = the skin adherence factor ($\text{mg/cm}^2\text{day}^{-1}$); ABS = the dermal absorption factor (mg/cm^2); InhR = the inhalation rate ($\text{m}^3\text{day}^{-1}$); PEF, the particle emission factor (m^3/kg); EF, the exposure frequency (days/year); ED=the exposure duration (year); BW= the body weight (kg); AT= the averaging time (days); FE=Dermal exposure ratio and CF= the conversion factor.

The parameters of the CDI, reference dose (RfD) and cancer slope factor (CSF), which were obtained from the Exposure Factors Handbook(USEPA. 2002; USDOE. 2011), Integrated Risk Information System (USEPA. 2007, 2011) are shown in Tables 2a and b. Furthermore, these risks can be grouped into non-carcinogenic and carcinogenic risks. Both risks exposure for children and adult population were estimated using Hazard Quotient (HQ) and Life Cancer Risk (LCR), respectively.

For non-carcinogenic risk, the HQ for children and adult population during a lifetime was estimated by dividing the CDI from each exposure pathway by a specific RfD as shown, whereas CDI is the chronic daily intake and RfD is the estimated maximum permissible risk posed to humans through daily exposure. Afterward, the estimated

HQ from the three (3) exposure pathways (dermal contact, ingestion and inhalation) was summed to obtain the Hazard Index (HI). If it is less than 1 ($HI \leq 1$), then adverse health effects would be unlikely to occur. Nevertheless, potential non-carcinogenic effects would occur when $HI > 1$ as this indicates significant non-carcinogenic risk posed to human health.

The formula used for calculation of HQ

$$HQ = ADD/RfD, \quad (6)$$

$$HI = HQ_{\text{ingest}} + HQ_{\text{dermal}} + HQ_{\text{inhale}}. \quad (7)$$

For carcinogenic risk, the LCR of children caused by potential carcinogen exposure over a lifetime can be calculated as shown below, for CDI and CSF is the slope factor for cancer. Total Life Cancer Risk (TLCR) adds up all LCRs calculated for ingestion, dermal contact and inhalation. The acceptable range of TLCR for carcinogenic risk is in the range of $1 \times 10^{-6} - 1 \times 10^{-4}$. If the risk exceeds the range, this implies that carcinogenic risks exist, and the potential carcinogenic effect would likely occur.

$$LCR = CDI \times CSF, \quad (8)$$

$$TLCR = LCR_{\text{ingest}} + LCR_{\text{dermal}} + LCR_{\text{inhale}}. \quad (9)$$

Table 2a. Recommended Standard Values for Dusts Health Risk Assessment

Parameters	Pb	Cd	Cr	As
RFD _{ing}	0.0035	0.001	0.003	3.00E-04
RFD _{inh}	3.50E-02	0.001	0.0001	1.00E-03
RFD _{dermal}	5.25E-04	0.00001	0.00006	1.00E-05
CSF _{ing}	0.0085	6.3	0.5	1.5
CSF _{inh}	4.20E-02	6.3	4.10E-01	1.50E+01
CSF _{dermal}	-	-	2.0E+1	1.5

(USEPA. 1989, 2002, 2007, 2011; USDOE. 2011 and MEP. 2014)

Table 2b Recommended Standard Values for Dusts Health Risk Assessment Contd

Parameter	Symbol	Value	
		ADULT	CHILD
Ingestion rate	IngR	100 mg	200 mg
Exposure duration	ED	24 years	6 years
Exposure frequency	EF	350 days	350 days
Average body weight	BW	70	15 kg
Averaging time (AT)	ATnon-carcinogenic	$ED \times 365$ days	$ED \times 365$ days
	ATcarcinogenic	70×365 days	70×365 days
Conversion factor	CF	1×10^{-6} kg/mg	1×10^{-6} kg/mg
Surface area of skin	SA children	5800 cm ²	2800 cm ²
Skin adherence factor	AF _{dust}	0.07 mg/cm ² /day	0.2 mg/cm ² /day
Dermal absorption factor	ABS non-carcinogenic	0.001 mg/cm ²	0.001 mg/cm ²
	ABS carcinogenic	0.03 mg/cm ²	0.03 mg/cm ²
Inhalation rate	InhR	20	10 m ³ /day
Particle emission factor	PEF	1.36×10^9 m ³ /kg	1.36×10^9 m ³ /kg
Dermal exposure ratio	FE	0.61	0.61

(USEPA. 1989, 2002, 2011; Rogan. et al. 2010 20USDOE. 2011)

Statistical Analysis

The significant difference of heavy metal mean values obtained were subjected to Analysis of variance (ANOVA) using SPSS version 20.

Results and Discussion

Profiling of Heavy metals contamination and pollution

Heavy Metal contamination and pollution profiling of sampled dusts from window corners of selected Public Primary Schools in Obio/Akpor, Eeleme and Ikwerre Local Government Areas (LGAs) of Rivers State was evaluated using individual metals C/PI, MPI and I_{geo} . The respective values are shown in Tables 3 and 4. C/PI values were shown to vary from one metal to the other and one location to the other with highest level 17.295 seen in Cd in school A₁, this shows that the window corner dust at A₁ is heavily polluted with Cd. Cadmium pollution (1.0125 – 17.2893) was notable in all sampled schools except at B₃ (0.586) which however shows severe contamination. Most analysed heavy metals were within the range of slight contamination to slight pollution; however, Arsenic shows no contamination in all sampled school in Ikwerre LGA, B_{1&3} in Eleme LGA and A₁ in Obio/Akpor LGA. The MPI showed that schools B₃ and C₁ were devoid of pollution (on multiple pollution index). However, other sampled schools showed multiple pollution index from slight pollution to heavily polluted in the order A₁>A₂>B₂>B₁>C₂>C₃.

The degree of metal contamination as measured with I_{geo} revealed A₁ to be strongly polluted with Cd (3.53) while other schools showed degree of pollution from practically unpolluted to moderately polluted with various sampled heavy metals.

Table 3: Contamination/Pollution Index (C/PI) of Heavy Metals from sampled Public Primary Schools Window Corner Dusts

SAMPLE	C/PI				MPI
	Pb (mg/kg)	Cd(mg/kg)	Cr(mg/kg)	As(mg/kg)	
A ₁	0.664045	17.28938	0.197779	0.019022	17.28938
A ₂	1.019663	10.18315	0.151442	0.19837	11.20281
A ₃	0.953933	5.934066	0.148985	0.105978	5.934066
B ₁	0.889888	6.007326	0.106776	0.07337	6.007326
B ₂	1.194382	6.886447	0.204609	0.220109	8.080829
B ₃	0.471348	0.586081	0.080094	0.005435	0
C ₁	0.122941	0.9875	0.2851	0.001724	0
C ₂	0.236471	1.425	0.4246	0.032414	1.425
C ₃	0.186353	1.0125	0.2173	0.00931	1.0125

Table 4: I_{geo} of Heavy Metals from sampled Public Primary Schools Window Corner Dusts

I_{geo}				
SAMPLE	Pb (mg/kg)	Cd(mg/kg)	Cr(mg/kg)	As(mg/kg)
A ₁	-1.17561	3.526852	-2.923	-6.30117
A ₂	-0.55687	2.76315	-3.30812	-2.9187
A ₃	-0.653	1.984058	-3.33172	-3.82312
B ₁	-0.75327	2.00176	-3.8123	-4.35364
B ₂	-0.3287	2.198797	-2.87402	-2.76867
B ₃	-1.6701	-1.35579	-4.22712	-8.10852
C ₁	-1.35334	1.947989	-3.42052	-6.7866
C ₂	-0.40964	2.477098	-2.84589	-2.55394

Heavy Metal Contents

Table 5 shows heavy metal levels as seen from collected dust samples from classroom window corners of nine (9) public primary schools. The study showed in $mgKg^{-1}$ Pb mean level of 21.26 ± 0.12 and Cd 2.36 ± 0.00 , from School B₂ and A₁ respectively, these schools are located within Eleme and Obio/Akpor LGAs respectively of Rivers State Nigeria. These LGAs are within the urban and industrialised region of Rivers State. This could however account for the high level of the heavy metals seen. Cr and As maximum mean levels were seen as 42.46 ± 0.12 and 0.94 ± 0.1 ,

as seen from School C₂ located at a major junction in Ikwerre Local government within Port Harcourt metropolis Rivers State. However, it is notable that the level of these metals on estimation of their chronic daily intake were below the respective reference doses for oral, inhalation and dermal contact in both adults and children populations (Tables 6 to 8). The non-carcinogenic risk assessment using hazard quotient and hazard index as the toxicological indices gave values below the bench mark of 1, which implies that these heavy metals at the level found in the sampled sites were potentially free of non-carcinogenic risks.

Popoola *et al.*, 2012; Addo *et al.*, 2012 and Olua *et al.*, 2018, have previously reported the presence of Pb with values within the range as seen in this study. Pb is known for its ability to restrain water imbalance, alter enzyme activities and hormonal status with lots more potential adverse effects to human (Sanborn *et al.* 2002, Seregin & Ivanjoy, 2001; Asia *et al.* 2008; Uaboi – Egbenni *et al.* 2010 & Ademoroti, 1996).

It has been reported that Cd is toxic even at low absorption by ingestion and chronic exposure to high levels of cadmium in food could lead to osteoporosis, osteomalacia etc (Asia *et al.* 2008). However no known beneficial effect due to Cd exposure have been reported (Gough *et al.* 1979; Aydinalp, and Marinova, 2009).

Exposure to Cr(III) has been associated with respiratory diseases and substantiated with coughing, wheezing, tracheobronchitis, chronic rhinitis, asthma polyps of the upper respiratory tract, etc (Dayan, and Paine, 2001; Lindberg, E. and Vesterberg, 1983; Novey, HS., and Habib, 1983). Lewis, and several other authors (Lewis, 2004, Bruynzeel, and Hennipman, 1988; Polak, 1983) also reported that dermal exposure to chromium elicits irritation and allergic responses, localized erythematous or vesicular lesions at points of contact or generalized eczematous dermatitis.

Several adverse health effects could be associated with long-term ingestion of inorganic arsenic some of which are developmental effects, neurotoxicity, diabetes, pulmonary disease and cardiovascular disease(WHO. 2016). Excessive mortality has been shown in previous studies (Rose *et al.* 2007).

Table 5: Heavy Metal Content of Window Corner Dusts in Public Primary Schools

SAMPLE SITE	Pb (mg/kg)	Cd(mg/kg)	Cr(mg/kg)	As(mg/kg)
A ₁	11.82±0.01 ^a	2.36±0.00 ^a	40.25±0.12 ^a	0.07±0.01 ^a
A ₂	18.15±0.03 ^a	1.39±0.17 ^a	30.82±0.01 ^a	0.73±0.00 ^b
A ₃	16.98±0.00 ^a	0.81±0.01 ^a	30.32±0.01 ^a	0.39±0.12 ^a
B ₁	15.84±0.02 ^a	0.82±0.06 ^a	21.73±0.03 ^a	0.27±0.12 ^a
B ₂	21.26±0.12 ^a	0.94±0.02 ^a	41.64±0.01 ^a	0.81±0.01 ^c
B ₃	8.39±0.17 ^a	0.08±0.03 ^a	16.30±0.17 ^a	0.02±0.01 ^a
C ₁	10.45±0.01 ^a	0.79±0.06 ^a	28.51±0.01 ^a	0.05±0.03 ^a
C ₂	20.10±0.03 ^a	1.14±0.07 ^a	42.46±0.12 ^a	0.94±0.1 ^a
C ₃	15.84±0.02 ^a	0.81±0.01 ^a	21.73±0.11 ^a	0.27±0.06 ^a
Ketu Ghana	22.89	-	744.02	-
Lagos Nigeria	23.33	0.09	8.35	-

Values are Mean±SEM Means in the same column with same superscript alphabet are significantly different at $p \leq 0.05$. A_{1,2,3} = Sampled public primary schools in Obio/Akpor, B_{1,2,3} =sampled public primary schools in Eleme, C_{1,2,3} = sampled public primary schools in Ikwerre. (Addo *et al.* 2012; (López *et al.* 2005).

Table 6 Chronic Daily Intake Dose (CDI_{ingest})

SAMPLE	Pb (mgkg ⁻¹ day ⁻¹)		Cd(mgkg ⁻¹ day ⁻¹)		Cr(mgkg ⁻¹ day ⁻¹)		As(mgkg ⁻¹ day ⁻¹)	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	1.69E-05	1.58E-04	3.37E-06	3.15E-05	3.45E-06	5.37E-04	1.00E-07	9.33E-07
A ₂	2.59E-05	2.42E-04	1.99E-06	1.85E-05	2.64E-06	4.11E-04	1.04E-06	9.73E-06
A ₃	2.43E-05	2.26E-04	1.16E-06	1.08E-05	2.60E-06	4.04E-04	5.57E-07	5.20E-06
B ₁	2.26E-05	2.11E-04	1.16E-06	1.08E-05	1.86E-06	2.90E-04	3.86E-07	3.60E-06
B ₂	3.04E-05	2.83E-04	1.34E-06	1.25E-05	3.57E-06	5.55E-04	1.16E-06	1.08E-05
B ₃	1.20E-05	1.12E-04	1.14E-07	1.07E-06	1.40E-06	2.17E-04	2.86E-08	2.67E-07
C ₁	1.49E-05	1.39E-04	1.13E-06	1.05E-05	2.44E-06	3.80E-04	7.14E-08	6.67E-07
C ₂	2.87E-05	2.68E-04	1.63E-06	1.52E-05	3.64E-06	5.66E-04	1.34E-06	1.25E-05
C ₃	1.90E-05	1.77E-04	1.87E-06	1.75E-05	2.24E-06	3.48E-04	1.86E-07	1.73E-06

E=exponential, A_{1,2,3} = Sampled public primary schools in Obio/Akpor, B_{1,2,3} =sampled public primary schools in Eleme, C_{1,2,3}= sampled public primary schools in Ikwerre.

Table 7 Chronic Daily Intake Dose ($CDI_{inhalation}$)

SAMPLE	Pb (mgkg ⁻¹ day ⁻¹)		Cd(mgkg ⁻¹ day ⁻¹)		Cr(mgkg ⁻¹ day ⁻¹)		As(mgkg ⁻¹ day ⁻¹)	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	2.48E-09	5.79E-09	4.96E-10	1.16E-09	2.54E-09	1.97E-08	1.47E-11	3.43E-11
A ₂	3.81E-09	8.90E-09	2.92E-10	6.81E-10	1.94E-09	1.51E-08	1.53E-10	3.58E-10
A ₃	3.57E-09	8.32E-09	1.70E-10	3.97E-10	1.91E-09	1.49E-08	8.19E-11	1.91E-10
B ₁	3.33E-09	7.76E-09	1.70E-10	3.97E-10	1.37E-09	1.07E-08	5.67E-11	1.32E-10
B ₂	4.47E-09	1.04E-08	1.97E-10	4.61E-10	2.62E-09	2.04E-08	1.70E-10	3.97E-10
B ₃	1.76E-09	4.11E-09	1.68E-11	3.92E-11	1.03E-09	7.99E-09	4.20E-12	9.80E-12
C ₁	2.20E-09	5.12E-09	1.66E-10	3.87E-10	1.80E-09	1.40E-08	1.05E-11	2.45E-11
C ₂	4.22E-09	9.85E-09	2.39E-10	5.59E-10	2.68E-09	2.08E-08	1.97E-10	4.61E-10
C ₃	2.79E-09	6.50E-09	2.75E-10	6.42E-10	1.65E-09	1.28E-08	2.73E-11	6.37E-11

E = exponential

Table 8 Chronic Daily Intake Dose (CDI_{dermal})

SAMPLE	Pb (mgkg ⁻¹ day ⁻¹)		Cd(mgkg ⁻¹ day ⁻¹)		Cr(mgkg ⁻¹ day ⁻¹)		As(mgkg ⁻¹ day ⁻¹)	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	4.18E-08	2.69E-07	8.35E-09	5.37E-08	1.42E-07	9.17E-07	2.48E-10	1.59E-09
A ₂	6.42E-08	4.13E-07	4.92E-09	3.17E-08	1.09E-07	7.02E-07	2.58E-09	1.66E-08
A ₃	6.01E-08	3.87E-07	2.87E-09	1.84E-08	1.07E-07	6.90E-07	1.38E-09	8.88E-09
B ₁	5.60E-08	3.61E-07	2.87E-09	1.84E-08	7.69E-08	4.95E-07	9.55E-10	6.15E-09
B ₂	7.52E-08	4.84E-07	3.33E-09	2.14E-08	1.47E-07	9.48E-07	2.87E-09	1.84E-08
B ₃	2.97E-08	1.91E-07	2.83E-10	1.82E-09	5.77E-08	3.71E-07	7.08E-11	4.55E-10
C ₁	3.70E-08	2.38E-07	2.80E-09	1.80E-08	1.01E-07	6.49E-07	1.77E-10	1.14E-09
C ₂	7.11E-08	4.58E-07	4.03E-09	2.60E-08	1.50E-07	9.67E-07	3.33E-09	2.14E-08
C ₃	4.69E-08	3.02E-07	4.63E-09	2.98E-08	9.24E-08	5.95E-07	4.60E-10	2.96E-09

E = exponential

Table 9 Hazard Quotient (HQ_{ingest})

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	0.005	0.045	0.003	3.15E-02	1.15E-03	1.79E-01	3.33E-04	3.11E-03
A ₂	0.007	0.069	0.002	1.85E-02	8.81E-04	1.37E-01	3.48E-03	3.24E-02
A ₃	0.007	0.065	0.001	1.08E-02	8.66E-04	1.35E-01	1.86E-03	1.73E-02
B ₁	0.006	0.060	0.001	1.08E-02	6.21E-04	9.66E-02	1.29E-03	1.20E-02
B ₂	0.009	0.081	0.001	1.25E-02	1.19E-03	1.85E-01	3.86E-03	3.60E-02
B ₃	0.003	0.031	0.0001	1.07E-03	4.66E-04	7.24E-02	9.52E-05	8.89E-04
C ₁	0.004	0.040	0.0011	1.05E-02	8.15E-04	1.27E-01	2.38E-04	2.22E-03
C ₂	0.008	0.077	0.0016	1.52E-02	1.21E-03	1.89E-01	4.48E-03	4.18E-02
C ₃	0.005	0.051	0.0019	1.75E-02	7.46E-04	1.16E-01	6.19E-04	5.78E-03

Table 10 Hazard Quotient (HQ_{inhalation})

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	7.09E-07	1.66E-06	4.96E-07	1.16E-06	2.54E-05	1.97E-04	4.90E-08	1.14E-07
A ₂	1.09E-06	2.54E-06	2.92E-07	6.81E-07	1.94E-05	1.51E-04	5.11E-07	1.19E-06
A ₃	1.02E-06	2.38E-06	1.7E-07	3.97E-07	1.91E-05	1.49E-04	2.73E-07	6.37E-07
B ₁	9.51E-07	2.22E-06	1.7E-07	3.97E-07	1.37E-05	1.07E-04	1.89E-07	4.41E-07
B ₂	1.28E-06	2.98E-06	1.97E-07	4.61E-07	2.62E-05	2.04E-04	5.67E-07	1.32E-06
B ₃	5.04E-07	1.18E-06	1.68E-08	3.92E-08	1.03E-05	7.99E-05	1.40E-08	3.27E-08
C ₁	6.27E-07	1.46E-06	1.66E-07	3.87E-07	1.80E-05	1.40E-04	3.50E-08	8.17E-08
C ₂	1.21E-06	2.82E-06	2.39E-07	5.59E-07	2.68E-05	2.08E-04	6.58E-07	1.54E-06
C ₃	7.97E-07	1.86E-06	2.75E-07	6.42E-07	1.65E-05	1.28E-04	9.10E-08	2.12E-07

Table 11 Hazard Quotient (HQ_{dermal})

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	7.97E-05	5.13E-04	8.35 E-04	5.37E-03	2.37E-03	1.53E-02	2.48E-05	1.59E-04
A ₂	1.22E-04	7.87E-04	4.92 E-04	3.17E-03	1.82E-03	1.17E-02	2.58E-04	1.66E-03
A ₃	1.14E-04	7.37E-04	2.87 E-04	1.84E-03	1.79E-03	1.15E-02	1.38E-04	8.88E-04
B ₁	1.07E-04	6.87E-04	2.87 E-04	1.84E-03	1.28E-03	8.25E-03	9.55E-05	6.15E-04
B ₂	1.43E-04	9.22E-04	3.33 E-04	2.14E-03	2.46E-03	1.58E-02	2.87E-04	1.84E-03
B ₃	5.65E-05	3.64E-04	2.83E-05	1.82E-04	9.61E-04	6.19E-03	7.08E-06	4.55E-05
C ₁	7.04E-05	4.53E-04	2.8 E-04	1.80E-03	1.68E-03	1.08E-02	1.77E-05	1.14E-04
C ₂	1.35E-04	8.72E-04	4.03 E-04	2.60E-03	2.50E-03	1.61E-02	3.33E-04	2.14E-03
C ₃	8.94E-05	5.76E-04	4.63 E-04	2.98E-03	1.54E-03	9.91E-03	4.60E-05	2.96E-04

Table 12 Hazard Index (HI)

SAMPLE	Pb		Cd		Cr		As		Σ HI	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	4.90E-03	4.55E-02	4.21E-03	3.69E-02	3.55E-03	1.94E-01	3.58E-04	3.27E-03	1.30E-02	2.80E-01
A ₂	7.53E-03	6.99E-02	2.48E-03	2.17E-02	2.72E-03	1.49E-01	3.74E-03	3.41E-02	1.65E-02	2.75E-01
A ₃	7.05E-03	6.54E-02	1.44E-03	1.26E-02	2.68E-03	1.47E-01	2.00E-03	1.82E-02	1.32E-02	2.43E-01
B ₁	6.57E-03	6.10E-02	1.44E-03	1.26E-02	1.91E-03	1.05E-01	1.39E-03	1.26E-02	1.13E-02	1.91E-01
B ₂	8.82E-03	8.19E-02	1.68E-03	1.46E-02	3.68E-03	2.01E-01	4.15E-03	3.78E-02	1.83E-02	3.35E-01
B ₃	3.48E-03	3.23E-02	1.42E-04	1.25E-03	1.44E-03	7.87E-02	1.02E-04	9.35E-04	5.16E-03	1.13E-01
C ₁	4.34E-03	4.03E-02	1.41E-03	1.23E-02	2.51E-03	1.38E-01	2.56E-04	2.33E-03	8.51E-03	1.93E-01
C ₂	8.34E-03	7.74E-02	2.03E-03	1.78E-02	3.74E-03	2.05E-01	4.81E-03	4.39E-02	1.89E-02	3.44E-01
C ₃	5.51E-03	5.11E-02	2.33E-03	2.05E-02	2.30E-03	1.26E-01	6.65E-04	6.08E-03	1.08E-02	2.04E-01

CARCINOGENIC RISKS

The carcinogenic risk factor was estimated using Life cancer risk (LCR) and Total life cancer risk (TLCR). Results from this study revealed that the exposure population of pupil and adults are to a higher extent prone to cancer risks. The values (2.08E-06 to 2.45E-05) obtained for adult population are within the upper tolerable limit 10^{-6} to 10^{-4} within which it is seen as been potentially risk free. While the pupils showed value (8.49E-05 to 2.54E-04) which is higher than the upper tolerable unit except at school B₃ which showed values below 10^{-4} .

Studies by International Agency for Research on Cancer (IARC) have shown Pb, Cd, Cr, and As, as potential carcinogens (IARC, 1987), this is also in agreement with the recognition of these heavy metals as carcinogenic agents by World Health Organization (WHO) and Agency for Toxic Substances and Disease Registry (ATSDR), (WHO, 2016, ATSDR, 2015). This study has revealed that exposure to these metals by inhalation could be associated with increased risk of cancer of the respiratory system (ATSDR, 2000), lung cancer risk, lung cancer death (Katz, and Salem, 1993).

Table 13 LIFE CANCER RISK (LCR_{ingest})

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	1.44E-07	3.35E-07	2.12E-05	4.96E-05	1.73E-06	6.71E-05	1.50E-07	3.50E-07
A ₂	2.20E-07	5.14E-07	1.25E-05	2.92E-05	1.32E-06	5.14E-05	1.56E-06	3.65E-06
A ₃	2.06E-07	4.81E-07	7.29E-06	1.70E-05	1.30E-06	5.05E-05	8.36E-07	1.95E-06
B ₁	1.92E-07	4.49E-07	7.29E-06	1.70E-05	9.31E-07	3.62E-05	5.79E-07	1.35E-06
B ₂	2.58E-07	6.02E-07	8.46E-06	1.97E-05	1.78E-06	6.94E-05	1.74E-06	4.05E-06
B ₃	1.02E-07	2.38E-07	7.20E-07	1.68E-06	6.99E-07	2.72E-05	4.29E-08	1.00E-07
C ₁	1.27E-07	2.96E-07	7.11E-06	1.66E-05	1.22E-06	4.75E-05	1.07E-07	2.50E-07
C ₂	2.44E-07	5.70E-07	1.03E-05	2.39E-05	1.82E-06	7.08E-05	2.01E-06	4.70E-06
C ₃	1.61E-07	3.76E-07	1.18E-05	2.75E-05	1.12E-06	4.35E-05	2.79E-07	6.50E-07

Table 14 Life Cancer Risk ($LCR_{inhalation}$)

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	1.04E-10	6.08E-11	3.12E-09	1.82E-09	1.04E-09	2.02E-09	2.21E-10	1.29E-10
A ₂	1.60E-10	9.34E-11	1.84E-09	1.07E-09	7.96E-10	1.55E-09	2.30E-09	1.34E-09
A ₃	1.50E-10	8.74E-11	1.07E-09	6.25E-10	7.83E-10	1.52E-09	1.23E-09	7.17E-10
B ₁	1.40E-10	8.15E-11	1.07E-09	6.25E-10	5.62E-10	1.09E-09	8.51E-10	4.96E-10
B ₂	1.88E-10	1.09E-10	1.24E-09	7.26E-10	1.08E-09	2.09E-09	2.55E-09	1.49E-09
B ₃	7.40E-11	4.32E-11	1.06E-10	6.18E-11	4.21E-10	8.19E-10	6.30E-11	3.68E-11
C ₁	9.22E-11	5.38E-11	1.05E-09	6.10E-10	7.37E-10	1.43E-09	1.58E-10	9.19E-11
C ₂	1.77E-10	1.03E-10	1.51E-09	8.80E-10	1.10E-09	2.13E-09	2.96E-09	1.73E-09
C ₃	1.17E-10	6.83E-11	1.73E-09	1.01E-09	6.75E-10	1.31E-09	4.10E-10	2.39E-10

Table 15 Life Cancer Risk (LCR_{dermal})

SAMPLE	Pb		Cd		Cr		As	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	-	-	-	-	1.26E-06	1.37E-04	1.11E-08	1.79E-08
A ₂	-	-	-	-	9.67E-07	1.05E-04	1.16E-07	1.87E-07
A ₃	-	-	-	-	9.51E-07	1.04E-04	6.21E-08	9.99E-08
B ₁	-	-	-	-	6.82E-07	7.42E-05	4.30E-08	6.92E-08
B ₂	-	-	-	-	1.31E-06	1.42E-04	1.29E-07	2.08E-07
B ₃	-	-	-	-	5.11E-07	5.57E-05	3.18E-09	5.12E-09
C ₁	-	-	-	-	8.94E-07	9.74E-05	7.96E-09	1.28E-08
C ₂	-	-	-	-	1.33E-06	1.45E-04	1.50E-07	2.41E-07
C ₃	-	-	-	-	8.19E-07	8.92E-05	2.07E-08	3.33E-08

Table 16 Total Life Cancer Risk (TLCR)

SAMPLE	Pb		Cd		Cr		As		Σ TLCR	
	Adult	Child	Adult	Child	Adult	Child	Adult	Child	Adult	Child
A ₁	1.44E-07	3.35E-07	2.12E-05	4.96E-05	2.99E-06	2.04E-04	1.61E-07	3.68E-07	2.45E-05	2.54E-04
A ₂	2.20E-07	5.14E-07	1.25E-05	2.92E-05	2.29E-06	1.56E-04	1.68E-06	3.84E-06	1.67E-05	1.90E-04
A ₃	2.06E-07	4.81E-07	7.29E-06	1.70E-05	2.25E-06	1.55E-04	8.99E-07	2.05E-06	1.06E-05	1.74E-04
B ₁	1.92E-07	4.49E-07	7.29E-06	1.70E-05	1.61E-06	1.10E-04	6.23E-07	1.42E-06	9.72E-06	1.29E-04
B ₂	2.58E-07	6.02E-07	8.46E-06	1.97E-05	3.09E-06	2.11E-04	1.87E-06	4.26E-06	1.37E-05	2.36E-04
B ₃	1.02E-07	2.38E-07	7.20E-07	1.68E-06	1.21E-06	8.29E-05	4.61E-08	1.05E-07	2.08E-06	8.49E-05
C ₁	1.27E-07	2.96E-07	7.11E-06	1.66E-05	2.11E-06	1.45E-04	1.15E-07	2.63E-07	9.47E-06	1.62E-04
C ₂	2.44E-07	5.70E-07	1.03E-05	2.39E-05	3.15E-06	2.16E-04	2.16E-06	4.94E-06	1.59E-05	2.45E-04

Conclusion

This study investigated the concentration of heavy metals in dust samples from classroom windows of selected public primary schools in Rivers State. Data from the study showed that the concentration of Pb detected in classroom dust was lower while the Cd load was higher than those previously reported in Ketu (Ghana) and Lagos (Nigeria). Health risk assessment revealed that children population may be more susceptible to both non-carcinogenic and carcinogenic risks. Generally, results from the study suggests that exposure to dusts contaminated by heavy metals may pose serious health concerns considering the hand to mouth attitude of children leading to high heavy metals exposure and ingestion rate.

References

- Addo, M. A., Darko, E. O., Gordon, C., Nyarko, B. J. B., & Gbadago, J. K. (2012). Heavy Metal Concentrations in Road Deposited Dust at Ketu-South District, Ghana. *International Journal of Science Technology*, 2(1): 28-29.
- Ademoroti, C. M. A. (1996). *Environmental chemistry and toxicology*. Ibadan, Nigeria: Foludex Press Ltd.
- Agency for Toxic Substances and Disease Registry (ATSDR). (2000). *Toxicological profile for chromium*. Atlanta, GA: ATSDR.
- Agency for Toxic Substances and Disease Registry (ATSDR). (2015). *Public health statement for chlorfenvinphos*. Atlanta, GA: ATSDR.
- Al-Khashman, O., & Shawabkeh, R. (2006). Metal distribution in soils around the cement factory in southern Jordan. *Environmental Pollution*, 140: 387-394.
- Asia, I. O., Ekpo, K. E., Amayo, K. O., & Jegede, D. A. (2008). Determination of lead, cadmium and mercury in surrounding water and organs of some species of fish from Ikpoba River in Benin City, Nigeria. *International Journal of Physical Sciences*, 3(11), 289–292.
- Aydinalp, C., & Marinova, S. (2009). The effects of heavy metals on seed germination and plant growth on alfalfa plant (*Medicago sativa*). *Bulgarian Journal of Agricultural Science*, 15(4), 347–350.
- Babayemi, O. J., Khadijah, T. D., Abideen, A. A. K., & Davies, O. (2009). Determination of potash alkali and metal Contents of ashes obtained from peels of some varieties of Nigeria grown Musa species. *Bioresources*, 5, 1384-1392
- Bruynzeel, D. P., & Hennipman, G. (1988). Irritant contact dermatitis and chrome-passivated metal. *Contact Dermatitis*, 19(3), 175–179.
- Dayan, A. D., & Paine, A. J. (2001). Mechanisms of chromium toxicity, carcinogenicity and allergenicity: Review of the literature from 1985 to 2000. *Human & Experimental Toxicology*, 20(9), 439–451.
- Faiz, Y., Tufail, M., Javed, M. T., Chaudhry, M. M., & Siddique, N. (2009). Road dust pollution of Cd, Cu, Ni, Pb and Zn along Islamabad Expressway, Pakistan. *Microchemical Journal*, 92, 186–192.
- Gough, L. P., Shacklette, H. T., & Case, A. A. (1979). *Element concentrations toxic to plants, animals and man*. Washington, DC: U.S. Geological Survey.
- Ighariemu, I., Wegwu M.O., Chuku, L.C., Olua, V. and Obadesagbo, O. (2023). Toxicological assessment of marine sediment in oil spilled impacted area of Nembe, Niger Delta, Nigeria. *International Journal of Scholarly Research and Reviews*, 02(01), 011–024. DOI: <https://doi.org/10.56781/ijssr.2023.2.1.0015>
- International Agency for Research on Cancer. (1987). *IARC monographs on the evaluation of carcinogenic risk of chemicals to humans. Supplement No. 7: Overall evaluations of carcinogenicity: An updating of IARC monographs volumes 1 to 42*. Lyon: International Agency for Research on Cancer.
- Katz, S. A., & Salem, H. (1993). The toxicology of chromium with respect to its chemical speciation: A review. *Journal of Applied Toxicology*, 13(3), 217–224.
- Klaassen, C., & Watkins, J. (2003). *Casarett and Doull's essentials of toxicology*. New York, NY: The McGraw-Hill Companies Inc.
- Lacatusu, R. (1998). Appraising levels of soil contamination and pollution with heavy metals. In H. J. Heineke, W. Eckelmann, A. J. Thomasson, R. J. A. Jones, L. Montanarella, & B. Buckley (Eds.), *Land information systems: Developments for planning the sustainable use of land resources* (pp. 393–402). Luxembourg: Office for Official Publications of the European Communities.

- Latif, M. T., Saw, M. Y., Saad, A., Mohamad, N., & Baharuddin, N. H. (2014). Composition of heavy metals in indoor dust and their possible exposure: a case study of preschool children in Malaysia. *Air Quality and Atmospheric Health*, 7:181–193.
- Lewis, R. (2004). Occupational exposures: Metals. In J. LaDou (Ed.), *Current occupational & environmental medicine* (3rd ed., pp. 439–441). New York, NY: Lange Medical Books/McGraw-Hill Companies, Inc.
- Lindberg, E., & Vesterberg, O. (1983). Urinary excretion of proteins in chromeplaters, exchromeplaters and referents. *Scandinavian Journal of Work, Environment & Health*, 9(6), 505–510.
- López, J. M., Callén, M. S., Murillo, R. T., García, R. T., Navarro, M. V., de la Cruz, M. T., & Mastral, A. M. (2005). Levels of selected metals in ambient air PM10 in an urban site of Zaragoza (Spain). *Environmental Research*, 99: 58–67.
- Luo, X. S., Ding, J., Xu, B., Wang, Y. J., & Li, H. B. (2012). Incorporating bioaccessibility into human health risk assessments of heavy metals in urban park soils. *Science of the Total Environment*, 424, 88–96.
- Ministry of Environmental Protection of the People's Republic of China (MEP). (2014). *Technical guidelines for risk assessment of contaminated sites*. Beijing, China: China Environmental Science Press. (In Chinese)
- Muller, G. (1969). Index of geoaccumulation in sediments of the Rhine River. *Geological Journal*, 2, 108–118.
- Novey, H. S., & Habib, M. (1983). Asthma and IgE antibodies induced by chromium and nickel salts. *Journal of Allergy & Clinical Immunology*, 72(4), 407–412.
- Nyimone, D. P., Patrick-Iwuanyanwu, K. C., Anacleto, F. N., Nwauche, K., Olua, V., Habiganuchi, A., Amadi, C. S., (2024). Toxicological Evaluation of Selected Seafoods and Water from Akpajo, Rivers State. *Journal of Environment and Earth Science*. 14, No.1, 2024
- Olua, V. Wegwu, M.O., Nwaichi, E.O., Riyadh S. Almalki and Samar F. M. (2024). Nephrotoxicity and oxidative stress response of Wistar rats fed with plant extracts from bioremediated crude oil impacted soil. *European Chemical Bulletin*. 13(4), 42 – 50. DOI: 10.53555/ecb/2024.13.04.07
- Olua, V., Patrick-Iwuanyanwu, K. C., & Nwaichi, E. O. (2018), “Heavy Metals Contents and Health Risk Assessment of Classroom Corner Dusts in Selected Public Primary Schools in Rivers State, Nigeria.” *Journal of Environment Pollution and Human Health*, 6,(4): 138-147.
- Olua, V., Wegwu, M. O and Nwaichi, E. O. (2021). Hepatotoxicity Of Farm Produce From Recently Remediated Crude Oil Polluted Sites Amended Using Formulated Agrowastes. *International Journal of Innovative Science and Research Technology*. 6(10) 353-358.
- Olujimi, O., Steiner, O., & Goessler, W. (2015). Pollution indexing and health risk assessments of trace elements in indoor dusts from classrooms, living rooms and offices in Ogun State, Nigeria. *Journal of African Earth Science*, 101:396–404.
- Polak, L. (1983). Immunology of chromium. In D. Burrows (Ed.), *Chromium: Metabolism and toxicity* (pp. 51–135). Boca Raton, FL: CRC Press.
- Popoola, O. E., Bamgbose, O., Okonkwo, O. J., Arowolo, T. A., & Popoola, A. O. (2012) Heavy metals content in classroom dust of some public primary schools in metropolitan Lagos, Nigeria. *Research Journal of Environment and Earth Science*, 4:460–465.
- Rogan, N., Dolenc, T., Serafimovski, T., Tasev, G., & Dolenc, M. (2010). Distribution and mobility of heavy metals in paddy soils of the Kočani Field in Macedonia. *Environmental Earth Sciences*, 61, 899–907.
- Rose, M., Lewis, J., Langford, N., Baxter, M., Origi, S., & Barber, M. (2007). Arsenic in seaweed—Forms, concentration and dietary exposure. *Food and Chemical Toxicology*, 45(7), 1263–1267.

Sabina, C., Grund, K., Hanusch, H., & Uwe, W. (2005). Arsenic and arsenic compounds. In *Ullmann's Encyclopedia of Industrial Chemistry*. Weinheim: Wiley-VCH.

Sanborn, M. D., Abelsohn, A., Campbell, M., & Weir, E. (2002). Identifying and managing adverse environmental health effects: 3. Lead exposure. *Canadian Medical Association Journal*, 2002;166:1287–92.

Sardar, K., Ali, S., Hameed, S., Afzal, S., & Fatima, S. (2013). Heavy metals contamination and what are the impacts on living organisms. *Global Journal of Environmental and Management Policy Studies*, 2, 172–179.

Seregin, I. V., & Ivaniov, V. B. (2001). Physiological aspects of cadmium and lead toxic effects on higher plants. *Russian Journal of Plant Physiology*, 48, 606–630.

Tong, T. Y., & Lam, K. C. (1998). Are nursery schools and kindergartens safe for our kids? The Hong Kong study. *Science of the Total Environment*; 216:217–225

Turekian, K. K., & Wedepohl, K. H. (1961). Distribution of the elements in some major units of the earth's crust. *Bulletin of the Geological Society of America*, 72, 175–192.

Uaboi-Egbenni, P. O., Okolie, P. N., Martins, O., & Teniola, O. (2010). Studies on the occurrence and distribution of heavy metals in sediments in Lagos Lagoon and their effects on benthic microbial population. *African Journal of Environmental Science and Technology*, 4(6), 343–351.

United States Department of Energy (USDOE). (2011). *The risk assessment information system (RAIS)*. Oak Ridge, TN: U.S. Department of Energy's Oak Ridge Operations Office.

United States Environmental Protection Agency (USEPA). (2002). *Supplemental guidance for developing soil screening levels for superfund sites* (OSWER 9355/4–24). Washington, DC: Office of Emergency and Remedial Response.

United States Environmental Protection Agency (USEPA). (2011). *Integrated risk information system*. Washington, DC: US EPA.

United States Environmental Protection Agency. (1989). *Risk assessment guidance for Superfund Volume 1: Human health evaluation manual (Part A)*. Washington, DC: Office of Emergency and Remedial Response.

United States Environmental Protection Agency. (2007). *Framework for determining a mutagenic mode of action for carcinogenicity: Review draft*. Washington, DC: US EPA.

World Health Organization (WHO). (2016). *Arsenic*. Geneva: WHO.