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Concentrations and Health Risk Assessment of Potentially Toxic Elements in Ambient Air around some OGONI Communities in Rivers State Nigeria

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Abstract
The concentrations of particulate matter and potentially toxic elements (PTEs) in ambient air around some Ogoni communities in Rivers State were assessed. PTEs were collected with Kanomax 3900 Portable High Volume sampler and analysed using Atomic Absorption Spectrophotometer. The health risk of the elements were estimated using United States Environmental Protection Agency (USEPA) standards; non-carcinogenic risk was assessed by estimating the hazard quotient (HQ) and hazard index (HI) while carcinogenic risk was evaluated by target cancer risk (TLCR) in the three main pathways of ingestion, inhalation and dermal contact. The mean concentrations of the PTEs were As 0.113±0.061mg/kg, Cd 0.004±0.002 mg/kg, Cr 0.003±0.002 mg/kg, Cu 0.001±0.00 mg/kg, Ni 0.029±0.027 mg/kg and Pb 0.003±0.002 mg/kg. Concentrations of As, Cd, Cu, Ni and Pb at the stations were below WHO and NAAQS recommended limits. The results of non-carcinogenic risk assessment of PTEs evaluated in the three main pathways of ingestion, inhalation and dermal contact showed that the HQ and HI values were <1 and therefore there are no non-carcinogenic risk of the PTEs in both children and adults at the study area. Cancer risk assessment for carcinogenic PTEs (As, Cd, Cr, and Pb) were all below acceptable minimum range of 1.0×10^{-6} to 1.0×10^{-4}. In addition to automobile activities, other anthropogenic activities such as illegal artisanal refining of crude oil and other domestic activities contributed to the concentrations of PTEs at the study areas. The study therefore recommended that more studies should be carriedout to monitor the concentrations and sources of pollutants in the areas.

Keywords: Health Risk, Toxic Elements, Ambient Air, Ogoni, Nigeria

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I. INTRODUCTION

There is increasing recognition of the need for concerted and effective action to improve the quality of air in our environments. Air pollution is becoming a major factor in the quality of life of urban and rural dwellers, posing a risk both to human health and to the environment. In order to develop appropriate air quality management plans, however, it is necessary first to have reliable information about the state of pollution (Ideriah and Stanley, 2008a).

Man in his quest to live a comfortable life on earth, has introduce some dangerous gases into the atmosphere thereby changing the composition of air environment from its original status (Odoemelam and Jonah, 2020). Air pollution may be indoor or outdoor (ambient). Both outdoor and indoor air quality data represent the true exposure for human beings (Ideriah, 2019; 2022).

Ambient air, also known as clean or unpolluted air, is a major determinant of environmental stability, sustainably good for human health (WHO, 2016). Greater emphasis has been laid on air quality globally because of its direct impact on man and other environmental components. Air is one of the basic necessities required for human existence (Akinfolarin *et al*., 2017). However, this environmental compartment has been severely impaired with inimical substances such as particulate matter (PM), Sulphur oxides, Nitrogen oxides, Carbon (II) oxide. This interference with the purity of air in its natural state has become a growing environmental issue both in developed and developing nations of the world (Munir *et al*., 2017). This is occasioned by industrialization and urbanization which has brought about higher concentration of human population in cities with viable economic opportunities such ascommercial cities (Koop and Van Leewen, 2017).

Air pollution can be defined as the presence of pollutants, such as sulphur dioxide (SO_2), particulate matter (PM), nitrogen oxides (NOx), hydrocarbons and ozone (O_3) in the air that we inhale at levels which can create some negative effects on the environment and human health (Olayinka and Abdullahi, 2008). It can be classified into natural air pollution which includes wind-blown dust, volcanic ash, and gases, smoke and trace

gases from forest fires, and anthropogenic air pollution which includes products of combustion such as nitrogen oxides (NOx), carbon oxides (COx), sulphur dioxide (SO_2) (Akinsanmi *et al*., 2019). Pollutants that are pumped into the atmosphere and directly pollute the air are called primary pollutants while those that are formed in the air when primary pollutants react orinteract are known as secondary pollutants (Agbaire, 2009).

Commercial cities are characterized with diverse commercial or business activities that range from small scale industries such as ceramics, glass and textile industries, to sales of electronics and household items, abattoir services, auto-repairs, hotel and restaurants, shopping malls, from small unit of cigarettes and freelance street side trading (Ezejiofor *et al*., 2013). In Nigeria, commercial cities such as Kano, Zaria, Aba and Port Harcourt serve as backbone of national development (Satope and Akanbi, 2014). Rapid growth in motor vehicles, roasting of animals with tyres and burning of large municipal waste generated in these cities has led to presence of particulate matter in ambient air (Njoku *et al*., 2016).

Animals are exposed to air pollutants via three pathways: Inhalation of gases or small particles, ingestion of particles suspended in food or water, or Absorption of gases through the skin. An individual's response to a pollutant varies greatly and depends on the type of pollutant involved, the duration of exposure and the amount is taken up by the animal. Factors such as the individual's age, sex, health and reproductive condition also play a role in its response. In addition to affecting individual animals or populations directly, air pollutants also affect wildlife indirectly by causing changes in the ecosystem (Omoko *et al*., 2021; Duce and Hoffman, 1976).

Irrespective of air pollution sources and classification, their impact on man and the environment is a major issue of concern. These impacts are pronounced in their dispersion, travel distance, particle size, transformations and final effect. According to the WHO, air pollution constitutes the largest health risk among all environmental risks and 92% of the world's population breathes substandard air as they live in places where air pollution exceeds safe limits. WHO attributed annual deaths around the world resulting from poor air quality inside and outside as about 6.5 million, making air pollution the world's fourth-largest threat to human health, behind high blood pressure, dietary risks and smoking. It has also been showed that air pollutant with small particle size (decreased diameter), are able to infiltrate finer lung structures and cause severe health effects such as asthma (WHO, 2006), chronic obstructive pulmonary disease (COPD) or increased cardiovascular risks (Gauderman *et al*., 2007). Other air pollution effects include the development of upper airways diseases such as sinusitis, mild otitis, olfactory impairment, rhinitis and sinonasal cancer (Shusterman *et al*., 2011). Kalagbor *et al*. (2019) investigated the presence and levels of heavy metals in soot along with a cancer risk assessment of heavy metals exposure in Port Harcourt, Nigeria and found significant correlation among the metals. The results of their study also showed that the carcinogenic health risks of the heavy metals were within the acceptable limits for cancer risks. However, the cancer health risks for Cd and Pb for children were found to be 3 times higher than those for adults.

Potentially toxic elements (PTEs) in particulate matter from ambient air are mostly generated from vehicle fumes and industrialization (Qadeer *et al*., 2019). In previous studies, PTEs were found to have accumulated in soil, water and the atmosphere, and exceeded the standard for environmental quality (Fry *et al*., 2021; Soltani *et al*., 2021). The presence of PTEs can be estimated from the combination of geogenic and anthropogenic effects (Jeong *et al*., 2021a). A high level of PTES was observed in the contributions of Zn, As, Cd, Pb and Hg that could transport to the environments via runoff and resuspension for 39.6% (Zn), 57.9% (As), 63.8% (Cd), 52.3% (Pb) and 51.3% (Hg) of the total dust in Onsan industrial complex in Korea (Krupnova *et al*., 2020). The essential metals, in certain amounts, may lead to acute exposure and non-carcinogenic effects from dermal absorption or oral inhalation (Soltani *et al*., 2021).

Long-term exposure to PTEs in the air is harmful to human health as they contain toxic particulates in the form of heavy metals (Jaishankar *et al*., 2014). For example, exposure to chromium (Cr) can cause lung tumors in mice through the inhalation pathway, while arsenic (As) may be associated with low birth weight and infant mortality. As is found in industrial dust and enters the human body via inhalation, dermal exposure and orally, and affects the metabolic processes. Pigmentation and keratosis are specific skin lesions that exhibit chronic As toxicity. Low levels of As can reduce the production of erythrocytes and leukocytes, abnormal heart beat, motor dysfunction, and pricking sensation in hands and legs (Jaishankar *et al*., 2014). In children, the toxicological effects of Cr and Pb pose the risk of neurological diseases and typical symptoms on the skin in the form of redness, burns and itching (Koszewicz *et al*., 2021). Repeated exposure may possibly cause atopic dermatitis and skincancer.

USEPA (2002) considered human health risk assessment as the characterization of the potential adverse health effects of humans as a result of exposures to environmental hazards. According to Lushenko (2010) potential health risk is a numerical value calculated using information from an identified and measured hazard and the possible route of exposure. Thus, a human health risk assessment involves hazard identification, dose-response assessment, exposure assessment, and risk characterization. Health risk assessment classifies elements as, carcinogenic or non-carcinogenic. Based on the classification, the procedure to be followed when potential

risks are calculated is determined. For non-carcinogenic chemicals a threshold is assumed. The threshold is considered as a dose below which no adverse health effects will be observed and an essential part of the dose-response portion of a risk assessment includes the use of a reference dose (RfD). For carcinogens, they are assumed to have no effective threshold. This assumption implies that there is a risk of cancer developing with exposures at low doses and, therefore, there is no safe threshold for exposure to carcinogenic chemicals. Carcinogens are expressed by their Cancer Potency Factor (Lushenko, 2010).

The levels of PTEs in the air around residential areas should be of major concern as the health of the human population can be greatly affected. Residents living nearby industrial areas are vulnerable populations due to the higher concentrations of PTEs, and they experience more frequent exposure than those in non-industrial areas (Fry *et al.*, 2021).

Ogoni communities in Rivers State is a typical example of a place that is seriously affected as a result of air pollution from mobile, stationary and indoor sources. Both outdoor and indoor air quality data represent the true exposure for human beings (Ideriah *et al*., 2008b). Air pollution could be serious. This is more so around Ogoni communities as a result of major air pollutants, particulates, dust and sooth constantly emitted from the Port Harcourt Refinery, Eleme Petrochemical Company limited, the National Fertilizer Company (NAFCON) but now called Notore Chemical Industries, incomplete combustion of fossil fuel and waste incineration, oil in homes, industries, power plants, burning of tires and the illegal artisanal refinery operations (Ideriah *et al*., 2008b).

II. MATERIALS AND METHODS

Description of Study Area

Bori is a city in Khana Local Government Area, Rivers State, Southern Nigeria. Bori city is located approximately on Latitude 4.67482° or 4° 40' 29" N Longitude 7.36561° or 7° 21' 56" E with a population of about 200,000 people and an elevation of 18 metres (59 feet). Bori is the traditional headquarters of the Ogoni people. Bori serves as a commercial centre for the Ogoni, Andoni, Opobo Annang and other ethnic nationalities of the Niger Delta. Bori is the second largest city in Rivers state after Port Harcourt and the commercial center of the Rivers southeast senatorial district in Rivers state.

Bodo City is located within Latitude 4° 37′ North of the equator and longitude 7° 16′ east of the meridian. Bodo is an Ogoni community located in Gokana Local Government Area of Rivers State. It has an estimated population of 69,000 people, which easily makes it the largest indigenous community in the state. 'Bodo City' as it is fondly called, plays host to the Anglo-Dutch super oil and gas major, Shell's 24- and 28-inch Trans-Niger pipeline (NDDC, 2004).

The meteorological conditions of the study areas display climatic characteristics that could be classified as semi-hot equatorial zone. The equatorial maritime air mass characterizes the climate with high humidity and heavy rainfalls (annual mean ranges between 72% -81% and 3,000mm-4,000mm). Specifically, these climatic characteristics range from the hot equatorial forest type in the southern lowlands to the humid tropical in the northern highlands. The wet season is relatively long, lasting between seven and eight months of the year, from the months of March to October (considered as rainy season). There is usually a short break around August, otherwise termed the "August break". The dry season begins in late November and extends to February or early March, a period of approximately three months although; the atmosphere sustains adequate moisture throughout the year (Gobo and Abam, 1991). The climatic characteristics are governed by the general circulatory patterns of two air masses: the dry dusty North-East Trade wind (Tropical continental air masses) from the Sahara Desert which come in the dry season (October - March), bringing in the harmattan from December – January and the moisture laden southwest wind (tropical maritime air masses) which bring rain during the wet season (April - October). The meteorological analysis of the prevailing wind patterns in the study area revealed that the wind direction persists from the southwest for most of the year (Ojo, 1977). Temperatures are generally high in the region and fairly constant throughout the year. Average monthly maximum and minimum temperatures vary from 28^oC to 33^oC and 21^oC to 23^oC, respectively, increasing northward and westward with the warmest months being February, March and early April. The coolest months are June through to September during the peak of the wet season. The map of the study area is shown in Figure 1.

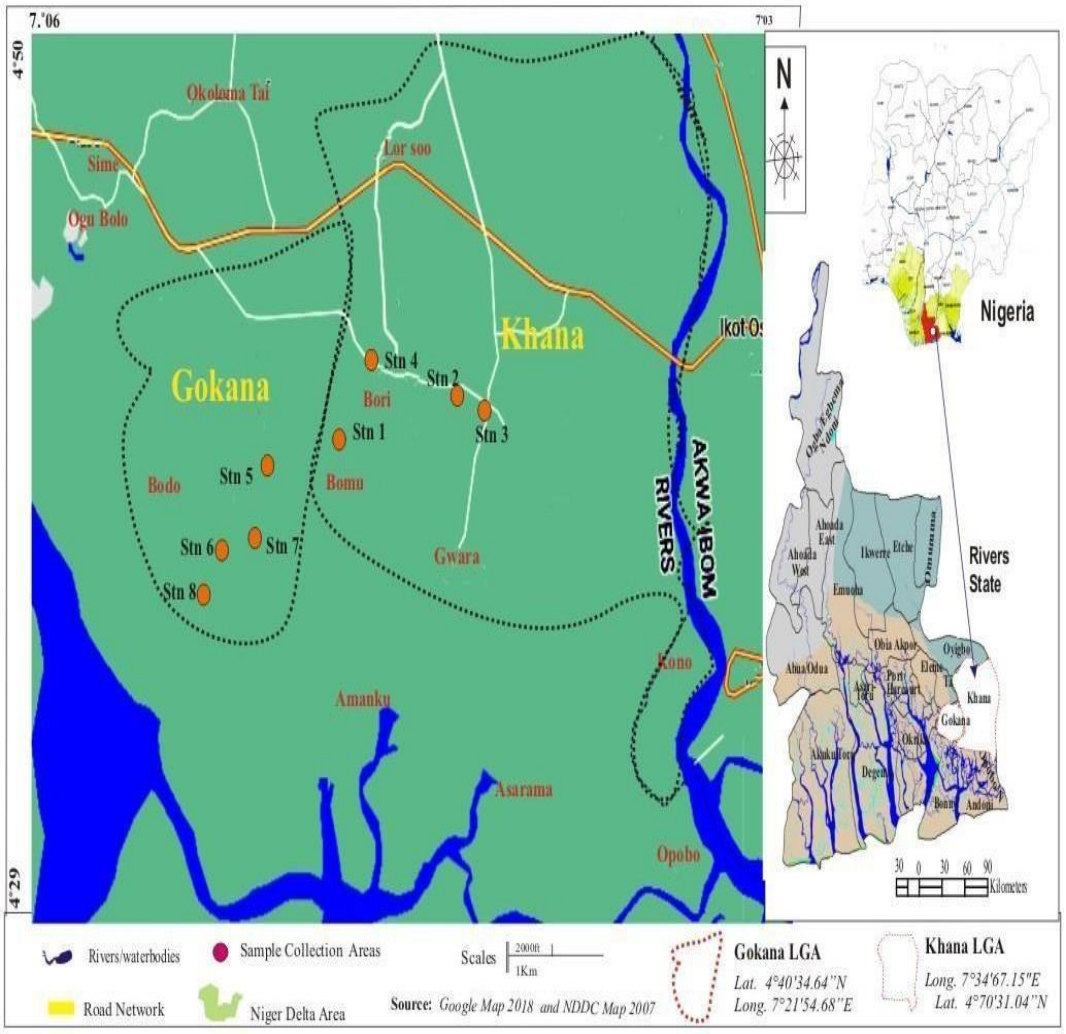

Fig. 1: Map of the Study Area Showing Sampling StationsSampling Stations

Samples were collected at 8 stations identified as (1) Okwale road (2) General hospital gate Bori (3) Ken Saro Wiwa Polytechnic campus main gate Bori (4) Bori Main Market (5) Bera road (6) Station junction Bodo, (7) Bodo-Bonny road (8) Bodo city market.

Potentially Toxic Elements (Cd, Ni, Cr, As, Cu and Pb)

The method recommended by ASTM D1971/4691 was employed for the analysis of the elements. The levels of the elements in the ambient air were determined from the collected particulates from the Kanomax 3900 Portable Counter High Volume Sampler sampler (Andover, USA). The fibre filters holding the fine particulates were digested as per standard procedure and thereafter analyzed with an Atomic Absorption Spectrophotometer (Shimadzu AA-6650).

Human Health Risk Exposure Assessment

The daily environmental exposures to metals in air were assessed for carcinogenic and non- carcinogenic elements. Three main pathways of exposure were assessed which are: ingestion of dust particle through the mouth, inhalation through the nose and mouth, and dermal contact through the skin (Ferreira- Baptista and De-Miguel, 2005; Zheng *et al.,* 2010). This study estimates the potential health risk assessment of children and adults exposed to heavy metals and other gaseous pollutant using USEPA standards (The United States Environmental Protection Agency (USEPA), 2002).

The estimated daily dose exposure was calculated using equations (3.2), (3.3) and (3.4).

$$DD_{ing} = \frac{C \times IngR \times CF \times EF \times F \times ED}{BW \times AT} \quad (3.2)$$

$$DD_{inh} = \frac{C \times InhR \times EF \times F \times ED}{BW \times AT} \quad (3.3)$$

$$DD_{der} = \frac{C \times SA \times CF \times SAF \times EF \times DAF \times F \times ED}{BW \times AT} \quad (3.4)$$

Where DD_{ing} DD_{inh}, DD_{der} in mg/kg/day are the adsorbed daily dose of exposure to heavy metals through ingestion, inhalation and dermal contact respectively.
The C is the concentration of heavy metals in air in mg/kg
IngR and InhR are ingestion and inhalation rate respectively. In this study, IngR was taken as 200mg/day for children and 100mg/day for adults (USEPA, 2002) while InhR was taken as 7.6m^3/day for children and 20m^3/day for adults.
F is a fraction of time spent at station in a day and value of carcinogenic and non-carcinogenic effectsis 0.0694.
EF is the exposure frequency in day/year. The value used in this study was 350 day/year (USEPA, 2002).
ED is the exposure duration in year. The value used in this study was 6 years for children and 24 years for adults (USEPA, 2002) while for carcinogenic and non-carcinogenic effects are 50 and 40 years respectively (Zheng *et al.*, 2010).
CF is the conversion factor and the value of carcinogenic and non-carcinogenic effects is 0.000001kg/mg (USEPA, 2002).
BW is the average body weight. The average body weight in this study was taken as 15kg for children and 70kg for adults (USEPA, 2002).
AT is the averaging time in days and values for carcinogenic and non-carcinogenic effects is 70×365 and ED×365 respectively (USEPA, 2002; Zheng *et al.*, 2010).

PEF is the particle emission factor in m^3/kg and values for carcinogenic and non-carcinogenic effects for both populations is 1,360,000,000 m^3/kg (USEPA, 2002). SA is the exposed skin surface area in cm^2 and is assumed to be 2800cm^2 for children and 5700cm^2 for adults (USEPA, 2002).
SAF is the dermal adherence factor in mg/cm^2/day and the assumed values for carcinogenic and non-carcinogenic effects used in this study is 0.2 mg/cm^2/day for children and 0.07 mg/cm^2/day.
DAF is the dermal absorption factor and the value for carcinogenic and non-carcinogenic effects used in this study is 0.001 except for arsenic which value is 0.03 (Zheng *et al.*, 2010).

Risk Assessment
Assessment of non-carcinogenic risks was achieved by estimating the hazard quotient (HQ). HQ was calculated as the quotient between the environmental exposure and the reference dose (RfD). The formula is given in equation (3.5).

$$HQ = \frac{ADD}{RfD} \quad (3.5)$$

Where ADD in mg/kg/day are the adsorbed daily dose of exposure to heavy metals through ingestion, inhalation and dermal contact and RfD is the Reference Dose (an estimated maximum permissible risk posed to human through daily exposure.

HQ values were obtained for each element and exposure pathway.

Hazard Index (HI) refers to the total risk through health exposure pathway. This was obtained by summing the HQ of each element using equation (3.6) (USEPA, 2011).

$$HI = HQ_{ing} + HQ_{inh} + HQ_{der} \quad (3.6)$$

Total HI is calculated by summing the HI through all exposure pathway) (USEPA, 2000). Values of HI under unity are considered as safe i.e if HI<1, it implies that there is no remarkable risk of non- carcinogenic effects, but if HI>1 then there is a possibility that non-carcinogenic effect may occur and this possibility increases as HI increases (USEPA, 2002).

Carcinogenic risk was evaluated by target cancer risk (TR). The method for estimating TR was provided in USEPA Region III Risk-Based Concentration equation (3.7) (USEPA, 2002).

$$TR = \frac{(C \times IR \times 10-3 \times CPS \times EF \times ED)}{BW \times AT} \qquad (3.7)$$

Where TR is the target cancer risk; C is the concentration of the element in air (μg/m^3); IR is the ingestion, inhalation or dermal contact rate; CPS is the carcinogenic potency slope, (mg/kg bw day^{-1}); BW is the average body weight and AT is the averaging time, carcinogens (days year^{-1}).

Another way to estimate Carcinogenic risks is by calculating the increase possibility of an individual to develop cancer as a result of exposure to the potential carcinogen over a lifetime. The estimated daily intake of toxin is converted by slope factor which is averaged by direct exposure over a lifetime to the increased chances of an individual to develop cancer using equation (3.8) (USEPA, 2000).

$$LCR = ADD \times SF \qquad (3.8)$$

Where LCR = Lifetime Carcinogenic Risk and it is unit less. ADD = Absorbed daily dose in mg/kg/day of exposure to the elements through ingestion, inhalation and dermal contact. SF = carcinogenicity slope factor (per mg/kg/day). Risk is therefore a unit less chances of an individual developing cancer when exposed over a lifetime.

The Total Lifetime Carcinogenic Risk (TLCR) was calculated by summing all the LCRs calculated for ingestion, inhalation and dermal contact equation (3.9).

$$TLCR = LCR_{ing} \times LCR_{inh} \times LCR_{der} \qquad (3.9)$$

For regulatory purposes, risks values exceeding 1×10^{-4} are regarded as intolerable, risks less than 1×10^{-6} are not regarded to cause significant health effects, and risks lying between 1×10^{-4} and 1×10^{-6} are regarded generally as satisfactory range, but circumstances and condition of exposure determine the range of the value of the circumstance (Hu *et al*., 2012).

III. RESULTS

The results of PTEs are presented in Table 1 and Figs. 2 & 3. The results of Human Health Risk Assessment of the PTEs are presented in Tables 2 - 10.

Potentially Toxic Elements

Arsenic concentrations at the stations ranged from 0.002mg/kg to 0.469mg/kg with a mean value of 0.113±0.061mk/kg. The concentrations of cadmium ranged from 0.001mg/kg to 0.008mg/kg with a mean value of 0.004±0.002mg/kg in the stations. Chromium concentrations in the stations ranged from 0.001mg/kg to 0.008mg/kg with mean value of 0.003± 0.002mg/kg. The concentrations of copper has a value of 0.001mg/kg in all stations and mean value of 0.001±0.000mg/kg. Nickel concentrations in the stations ranged from 0.005mg/kg to 0.099 mg/kg with mean value of 0.029±0.027mg/kg. The concentrations of lead ranged from 0.001mg/kg to 0.005mg/kg with mean value 0.003± 0.002mg/kg.

Table 1: Mean Levels of Potentially Toxic Elements at the Study Area

Parameters(mg/kg)	Min	Max	Mean
As	0.002	0.469	0.113±0.061
Cd	0.001	0.008	0.004±0.002
Cr	0.001	0.008	0.003±0.002
Cu	0.001	0.001	0.001±0.000
Ni	0.005	0.099	0.029±0.027
Pb	0.001	0.005	0.003±0.002

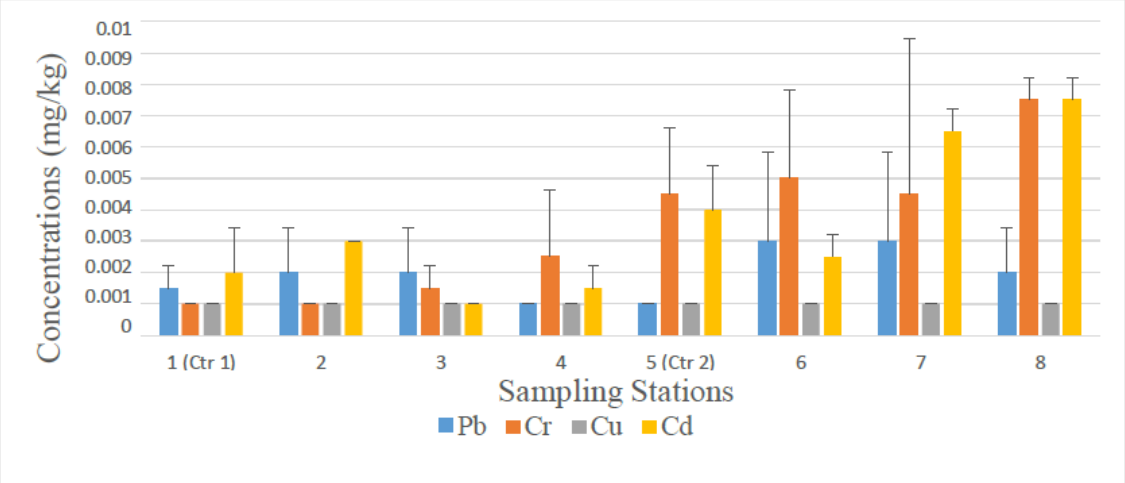

Fig. 2: Mean Concentrations of some Potentially Toxic Elements at the Study Area

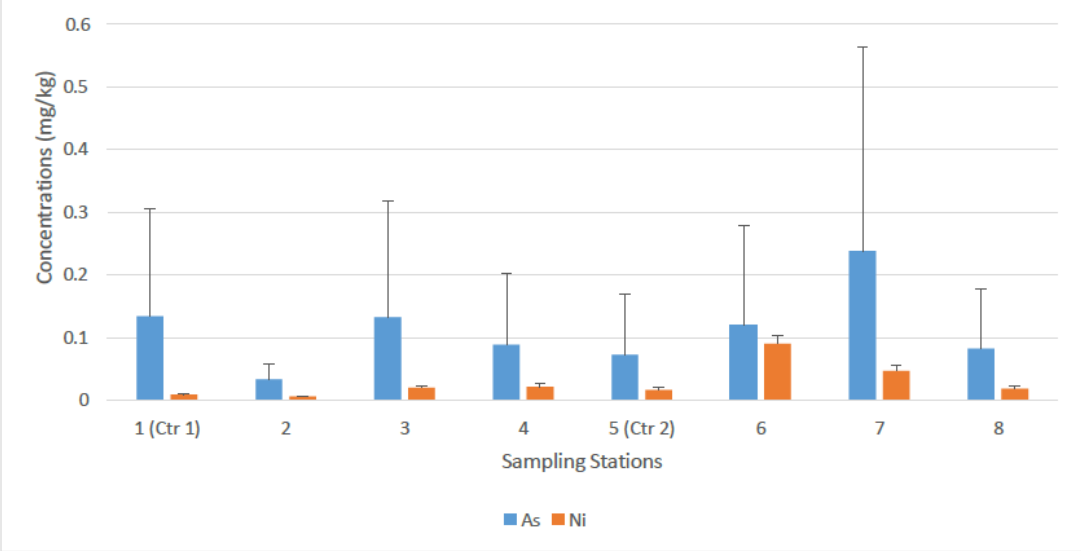

Fig 3: Mean Concentrations of some Potentially Toxic Elements at the Study Area Human HealthRisk Assessment

The results of the human health risk assessment of the PTEs concentrations in air at the study area for both children and adults as evaluated in the three main pathways Ingestion (DDing), Inhalation (DDinh) and Dermal contact (DDder) are presented in Tables 2 - 10.

Daily Dose of Exposure

The DDing values for Arsenic in the children ranged from 2.19×10^{-9} to 5.14×10^{-7} while the values for adults ranged from 9.39×10^{-10} to 2.20×10^{-7}. The values for cadmium in the children ranged from 1.10×10^{-9} to 8.77×10^{-9} while the values for adult ranged from 4.70×10^{-10} to 3.76×10^{-9}. The DDing values for chromium in the children ranged from 1.10×10^{-9} to 8.77×10^{-9} while the value for adults ranged from 4.70×10^{-10} to 3.76×10^{-9}. The DDing values for copper in the children from is 1.10×10^{-9} while the values in adults is 4.70×10^{-10}. The DDing values for Nickel in children ranged from 1.10×10^{-8} to 1.08×10^{-7} while the values for adults ranged from 4.70×10^{-9} to 4.65×10^{-8}. The DDing values for lead in children ranged from 1.10×10^{-9} to 3.29×10^{-9} while the values for adults ranged from 4.70×10^{-10} to 1.41×10^{-9}.

The DDinh value for arsenic in children ranged from 6.12×10^{-4} to 1.44×10^{-11}while the value for adults ranged from 1.38×10^{-13} to 3.24×10^{-11}. The value for cadmium in children ranged from 3.06×10^{-14} to 2.45×10^{-13} while the value for adults ranged from 6.91×10^{-14} to 5.53×10^{-13}. The DDinh value for chromium in children ranged from 3.06×10^{-14} to 2.45×10^{-13} while the value for adults ranged from 6.91×10^{-14} to 5.53×10^{-13}. The value for copper in children is 3.06×10^{-14} while the value for adults is 6.91×10^{-14}. The DDinh value for nickel in children

ranged from 3.06×10^{-13} to 3.03×10^{-12} while the value for adults ranged from 6.91×10^{-13} to 6.84×10^{-12}. The DD_{inh} value for lead in children ranged from 3.06×10^{-14} to 9.19×10^{-14} while the value for adults ranged from 6.91×10^{-14} to 2.07×10^{-13}.

The DD_{der} value for arsenic in children ranged from 1.84×10^{-10} to 4.32×10^{-8} while the value for adults ranged from 1.12×10^{-10} to 2.64×10^{-8}. The values for cadmium in children ranged from 3.07×10^{-12} to 2.45×10^{-11} while the value for adults ranged from 1.87×10^{-12} to 1.50×10^{-11}. The DD_{der} value for chromium in children ranged from 3.07×10^{-12} to 2.45×10^{-11} while the value for adults ranged from 1.87×10^{-12} to 1.50×10^{-11}. The DD_{der} value for copper in children is 3.07×10^{-12} while the value for adult is 1.87×10^{-12}. The value for nickel in children ranged from 3.07×10^{-11} to 3.04×10^{-10} while the value for adult ranged from 1.87×10^{-11} to 1.86×10^{-10}. The DD_{der} value for lead in children ranged from 3.07×10^{-12} to 9.21×10^{-12} while the value for adults ranged from 1.87×10^{-12} to 5.62×10^{-12}.

Table 2: Minimum Daily Intake of Potentially Toxic Elements in Adults and Children

	Routes	As	Cd	Cr	Cu	Ni	Pb
	DD_{ing}	9.39×10^{-10}	4.70×10^{-10}	4.70×10^{-10}	4.70×10^{-10}	4.70×10^{-9}	4.70×10^{-10}
Adult	DD_{inh}	1.38×10^{-13}	6.91×10^{-14}	6.91×10^{-14}	6.91×10^{-14}	6.91×10^{-13}	6.91×10^{-14}
	DD_{der}	1.12×10^{-10}	1.87×10^{-12}	1.87×10^{-12}	1.87×10^{-12}	1.87×10^{-11}	1.87×10^{-12}
	DD_{ing}	2.19×10^{-9}	1.10×10^{-9}	1.10×10^{-9}	1.10×10^{-9}	1.10×10^{-8}	1.10×10^{-9}
Children	DD_{inh}	6.12×10^{-14}	3.06×10^{-14}	3.06×10^{-14}	3.06×10^{-14}	3.06×10^{-13}	3.06×10^{-14}
	DD_{der}	1.84×10^{-10}	3.07×10^{-12}	3.07×10^{-12}	3.07×10^{-12}	3.07×10^{-11}	3.07×10^{-12}

Table 3: Daily Intake of Potentially Toxic Elements in Adults and Children

	Routes	As	Cd	Cr	Cu	Ni	Pb
	DD_{ing}	5.31×10^{-8}	1.88×10^{-9}	1.41×10^{-9}	4.70×10^{-10}	1.36×10^{-8}	9.39×10^{-10}
Adult	DD_{inh}	7.80×10^{-12}	2.76×10^{-13}	2.07×10^{-13}	6.91×10^{-14}	2.0×10^{-12}	1.38×10^{-13}
	DD_{der}	6.35×10^{-9}	7.50×10^{-12}	5.62×10^{-12}	1.87×10^{-12}	5.43×10^{-11}	3.75×10^{-12}
	DD_{ing}	1.24×10^{-7}	4.38×10^{-9}	3.29×10^{-9}	1.10×10^{-9}	3.18×10^{-8}	2.19×10^{-9}
Children	DD_{inh}	3.46×10^{-12}	1.22×10^{-13}	9.19×10^{-14}	3.06×10^{-14}	8.88×10^{-13}	6.12×10^{-14}
	DD_{der}	1.04×10^{-8}	1.23×10^{-11}	9.21×10^{-12}	3.07×10^{-12}	8.90×10^{-11}	6.14×10^{-12}

Table 4: Maximum Daily Intake of Potentially Toxic Elements in Adults and Children

	Routes	As	Cd	Cr	Cu	Ni	Pb
	DD_{ing}	2.20×10^{-7}	3.76×10^{-9}	3.76×10^{-9}	4.70×10^{-10}	4.65×10^{-8}	1.41×10^{-9}
Adult	DD_{inh}	3.24×10^{-11}	5.53×10^{-13}	5.53×10^{-13}	6.91×10^{-14}	6.84×10^{-12}	2.07×10^{-13}
	DD_{der}	2.64×10^{-8}	1.50×10^{-11}	1.50×10^{-11}	1.87×10^{-12}	1.86×10^{-10}	5.62×10^{-12}
	DD_{ing}	5.14×10^{-7}	8.77×10^{-9}	8.77×10^{-9}	1.10×10^{-9}	1.08×10^{-7}	3.29×10^{-9}
Children	DD_{inh}	1.44×10^{-11}	2.45×10^{-13}	2.45×10^{-13}	3.06×10^{-14}	3.03×10^{-12}	9.19×10^{-14}
	DD_{der}	4.32×10^{-8}	2.45×10^{-11}	2.45×10^{-11}	3.07×10^{-12}	3.04×10^{-10}	9.21×10^{-12}

Non-Carcinogenic Risk Assessment

The non-carcinogenic risk for children and adults associated with the daily dose intake of the PTEs were assessed for each exposure pathway as their Hazard Quotients (HQs) and Hazard Indexes. Tables 5 – 7 shows the HQs and the HIs.

The maximum HQ for arsenic in children was HQ_{ing} (1.71×10^{-3}), the minimum HQ was HQ_{inh} (2.04×10^{-10})

while the maximum value for adults was HQder (8.80×10^{-4}), and the minimum value was HQinh (4.60×10^{-10}). The maximum HQ for cadmium in children was HQing (8.77×10^{-6}), the minimum value was HQinh (5.37×10^{-10}) while the maximum value for adults was HQing (3.76×10^{-6}), and the minimum value was HQder (1.87×10^{-9}). The maximum HQ for chromium in children was HQing (2.92×10^{-6}), the minimum was HQder (1.02×10^{-9}) while the maximum value for adults was HQing (1.25×10^{-6}), and the minimum value was HQder (6.25×10^{-10}). The maximum HQ for copper in children was HQing (2.75×10^{-8}), the minimum value was HQinh (6.80×10^{-13}) while the maximum value for adults was HQing (1.18×10^{-8}), and the minimum value was HQinh (1.54×10^{-12}). The maximum HQ for nickel in children was HQing (5.40×10^{-6}), the minimum value was HQinh (1.49×10^{-11}) while the maximum value for adults was HQing (2.33×10^{-6}), and the minimum value was HQinh (3.35×10^{-11}). The maximum HQ for lead in children was HQing (9.40×10^{-7}), the minimum value was HQinh (8.69×10^{-12}) while the maximum value for adults was HQing (4.03×10^{7}), and the minimum value was HQinh (3.92×10^{-11}). The maximumHI for children was 2.04×10^{-3} while the value for adults was 1.62×10^{-3}.

Table 5: Minimum Hazard Quotient (HQ) and Hazard Index (HI) Values from Ingestion, Inhalation and Dermal Route

	Routes	As	Cd	Cr	Cu	Ni	Pb	HI
	HQ_{ing}	3.13×10^{-6}	4.70×10^{-7}	1.57×10^{-7}	1.18×10^{-8}	2.35×10^{-7}	1.34×10^{-7}	
Adult	HQ_{inh}	4.60×10^{-10}	1.21×10^{-9}	2.30×10^{-9}	1.54×10^{-12}	3.35×10^{-11}	1.96×10^{-11}	4.52×10^{-6}
	HQ_{der}	3.73×10^{-7}	1.87×10^{-9}	6.23×10^{-10}	1.56×10^{-10}	3.46×10^{-9}	3.53×10^{-9}	
	HQ_{ing}	7.30×10^{-6}	1.10×10^{-6}	3.67×10^{-7}	2.75×10^{-8}	5.50×10^{-7}	3.14×10^{-7}	
Children	HQ_{inh}	2.04×10^{-10}	5.37×10^{-10}	1.02×10^{-9}	6.80×10^{-13}	1.49×10^{-11}	8.69×10^{-12}	9.39×10^{-6}
	HQ_{der}	6.13×10^{-7}	3.07×10^{-9}	1.02×10^{-9}	2.56×10^{-10}	5.69×10^{-9}	5.79×10^{-9}	

Table 6: Average Hazard Quotient (HQ) and Hazard Index (HI) Values from Ingestion,Inhalation and Dermal Route

	Routes	As	Cd	Cr	Cu	Ni	Pb	HI
	HQ_{ing}	1.77×10^{-4}	1.88×10^{-6}	4.70×10^{-7}	1.18×10^{-8}	6.80×10^{-7}	2.68×10^{-7}	
Adult	HQ_{inh}	2.60×10^{-8}	4.84×10^{-9}	6.90×10^{-9}	1.54×10^{-12}	9.71×10^{-11}	3.92×10^{-11}	1.80×10^{-4}
	HQ_{der}	5.16×10^{-5}	7.50×10^{-9}	1.87×10^{-9}	1.56×10^{-10}	1.01×10^{-8}	7.08×10^{-9}	
	HQ_{ing}	4.13×10^{-4}	4.38×10^{-6}	1.10×10^{-6}	2.75×10^{-8}	1.59×10^{-6}	6.26×10^{-7}	
Children	HQ_{inh}	1.15×10^{-8}	2.14×10^{-9}	3.06×10^{-9}	6.80×10^{-13}	4.31×10^{-11}	1.74×10^{-11}	5.05×10^{-4}
	HQ_{der}	8.46×10^{-5}	1.23×10^{-8}	3.07×10^{-9}	2.56×10^{-10}	1.65×10^{-8}	1.16×10^{-8}	

Table 7: Maximum Hazard Quotient (HQ) and Hazard Index (HI) Values from Ingestion, Inhalation and Dermal Route

	Routes	As	Cd	Cr	Cu	Ni	Pb	HI
	HQ_{ing}	7.33×10^{-4}	3.76×10^{-6}	1.25×10^{-6}	1.18×10^{-8}	2.33×10^{-6}	4.03×10^{-7}	
Adult	*HQinh*	1.08×10^{-7}	9.70×10^{-9}	1.84×10^{-8}	1.54×10^{-12}	3.32×10^{-10}	5.88×10^{-11}	1.62×10^{-3}
	HQder	8.80×10^{-4}	1.50×10^{-8}	5.00×10^{-9}	1.56×10^{-10}	3.44×10^{-8}	1.06×10^{-8}	
	HQing	1.71×10^{-3}	8.77×10^{-6}	2.92×10^{-6}	2.75×10^{-8}	5.40×10^{-6}	9.40×10^{-7}	
Children	HQinh	4.80×10^{-8}	4.30×10^{-9}	8.17×10^{-9}	6.80×10^{-13}	1.47×10^{-10}	2.61×10^{-11}	2.04×10^{-3}
	HQder	1.44×10^{-4}	2.45×10^{-8}	8.17×10^{-9}	2.56×10^{-10}	5.63×10^{-8}	1.74×10^{-8}	

Carcinogenic Risk Assessment

The Lifetime Cancer Risk (LCR) and Total Lifetime Cancer Risk (TLCR) in the study was assessed foe arsenic, cadmium, chromium and lead which have been identified as carcinogens (USEPA, 2011). The cancer slope factor for nickel and copper are zero (0.00). Tables 8 – 10 shows the LCR and TLCR for children and adults with respect to their daily intake doses of each metal.

The maximum LCR value for arsenic in children was LCR_{ing} (7.71×10^{-10}) while in adults was LCR_{ing} (3.30×10^{-10}). The maximum value for cadmium in children was LCR_{ing} (4.39×10^{-9}) while in adults was LCR_{ing} (1.88×10^{-9}). The maximum value for chromium in children was LCR_{ing} (4.39×10^{-9}) while in adults was LCR_{ing} (1.88×10^{-9}). The maximum value for lead in children was LCR_{ing} (2.79×10^{-11}) while in adults was (1.19×10^{-9}). The TLCR for children was 9.68×10^{-9} while in adults was 4.18×10^{-9}.

Table 8: Minimum Carcinogenic Risk (LCR) and Total Carcinogenic Risk (TLCR) for Adults andChildren

	Routes	As	Cd	Cr	Pb	TLCR
Adult	LCR_{ing}	1.41×10^{-12}	2.35×10^{-10}	2.35×10^{-10}	3.99×10^{-12}	4.81×10^{-10}
	LCR_{inh}	2.07×10^{-16}	4.35×10^{-13}	2.83×10^{-12}	2.90×10^{-15}	
	LCR_{der}	1.68×10^{-13}	7.11×10^{-13}	7.85×10^{-13}	7.85×10^{-13}	
Children	LCR_{ing}	3.29×10^{-12}	5.50×10^{-10}	5.50×10^{-10}	9.35×10^{-12}	1.12×10^{-9}
	LCR_{inh}	9.18×10^{-17}	1.93×10^{-13}	1.25×10^{-12}	1.29×10^{-15}	
	LCR_{der}	2.76×10^{-13}	1.17×10^{-12}	1.29×10^{-12}	1.29×10^{-12}	

Table 9: Carcinogenic Risk (LCR) and Total Carcinogenic Risk (TLCR) for Adults and Children

	Routes	As	Cd	Cr	Pb	TLCR
Adult	LCR_{ing}	7.97×10^{-11}	9.40×10^{-10}	7.05×10^{-10}	7.98×10^{-12}	1.76×10^{-9}
	LCR_{inh}	1.17×10^{-14}	1.74×10^{-12}	8.49×10^{-13}	5.80×10^{-15}	
	LCR_{der}	9.53×10^{-12}	2.85×10^{-12}	2.36×10^{-12}	1.58×10^{-12}	
Children	LCR_{ing}	1.86×10^{-10}	2.19×10^{-9}	1.65×10^{-9}	1.86×10^{-11}	4.08×10^{-9}
	LCR_{inh}	5.19×10^{-15}	7.69×10^{-13}	3.77×10^{-14}	2.57×10^{-15}	
	LCR_{der}	1.56×10^{-11}	4.67×10^{-12}	3.87×10^{-12}	2.58×10^{-12}	

Table 10: Maximum Carcinogenic Risk (LCR) and Total Carcinogenic Risk (TLCR) for Adults and Children

	Routes	As	Cd	Cr	Pb	TLCR
Adult	LCR_{ing}	3.30×10^{-10}	1.88×10^{-9}	1.88×10^{-9}	1.19×10^{-11}	4.18×10^{-9}
	LCR_{inh}	4.86×10^{-14}	3.48×10^{-12}	2.27×10^{-11}	8.69×10^{-15}	
	LCR_{der}	3.96×10^{-11}	5.70×10^{-12}	6.30×10^{-12}	2.36×10^{-12}	
Children	LCR_{ing}	7.71×10^{-10}	4.39×10^{-9}	4.39×10^{-9}	2.79×10^{-11}	9.68×10^{-9}
	LCR_{inh}	2.16×10^{-14}	1.54×10^{-12}	1.00×10^{-11}	3.86×10^{-15}	
	LCR_{der}	6.48×10^{-11}	9.31×10^{-12}	1.03×10^{-11}	3.87×10^{-12}	

IV. Discussion

Potentially Toxic Elements

Arsenic (As) concentrations ranged from 0.002mg/kg to 0.469mg/kg with a mean value of 0.113± 0.061mg/kg. Station 7 has the highest concentration in compared to other stations. The mean concentration of arsenic in station 1 (control 1) was higher than station 2, 3, 4, 5, 6 and 8. The concentration of arsenic in station 5 (control 2) was higher than station 2. The results in the concentrations of As from this study showed lower values as compared to Rauf *et al.* (2021) who reported a concentration of 2.08±0.24 mg/kg.

Cadmium (Cd) concentrations ranged from 0.001mg/kg to 0.008mg/kg with mean value of 0.004±0.002mg/kg. The concentration of cadmium in station 8 was higher than other stations. Station 1 (control 1) was higher than station 3 and 4. Station 5 (control 2) was higher than station 1, 2, 3, 4 and

6. These results were similar to the results of Oweisana *et al*. (2021a) and Kalagbor *et al*. (2019) which reported low concentrations of 0.006±0.001mg/kg and 0.018±0.005mg/kg of Cd respectively. Chromium (Cr)

concentrations ranged from 0.001mg/kg to 0.008mg/kg with mean value of 0.003±0.002mg/kg. The concentration of chromium in station 8 was higher than other stations. Station 5 (control 2) showed higher concentration than station 1, 2, 3 and 4. Station 5 had same concentration with station 7. Station 1 (control 1) had the same concentration with station 2. These results were similar to the result reported by Kalagbor *et al*. (2019) which had a low concentration of 0.001±0.00mg/kg. Copper concentration did not vary in all stations during the period of study. The concentration was observed to be 0.001mg/kg. The result from the study was low when compared to the concentration of4.68±1.64mg/kg reported by Rauf *et al*. (2021).

Nickel concentrations in the stations ranged from 0.005mg/kg to 0.099mg/kg with a mean value of 0.029±0.027mg/kg. The concentration of nickel in station 6 showed a higher value compared to other stations. The results of Ni from this study was higher when compared to the result reported by Kalagbor *et al.* (2019) who had a low value of 0.0086±0.01mg/kg.

Lead concentrations in the stations ranged from 0.001mg/kg to 0.005mg/kg with a mean value of 0.003±0.002mg/kg. The concentrations of station 6 and 7 was higher than other station while station 4 and 5 has the same concentration of 0.001mg/kg. The findings from this study showed a lower value when compared to the results of Kalagbor *et al.* (2019) who had a value of 0.034±0.04mg/kg and Oweisana *et al.* (2021a) who reported a value of 0.010±0.00mg/kg.

Continuous exposure to PTEs in residential areas can cause adverse health effects in humans, including cardiovascular diseases, genetic issues and internal organ damage (Raul *et al.,* 2021). In this study, toxic metals such as Pb and Cr were present in the SPM. These elements are classified as carcinogenic pollutants and harmful to humans (Raul *et al.,* 2021). Pb exposure in males causes a decrease in sperm quality and a possible alteration of serum levels. Young children and infants absorb more Pb and are particularly susceptible to neurological effects (Assi *et al.,* 2016). The inhalation and oral intake of Cr causes Cr poisoning, pneumonia, kidney disease, gingivitis and lung cancer (Raul *et al.,* 2021). With dermal absorption Cr exposures causes damage to the epidermis cells leading to dermatitis, which causes redness and burning in the skin. Furthermore, the toxicity of As is responsible for epigenetic alterations, DNA damage and heart problems (Oweisana *et al.,* 2021a). Toxicity from cadmium results in disease and abnormalities of the kidney, liver skeleton and reproductive functions. The other elements, such as Cu and Ni are some of the most abundant elements in the earth's crust. These types of element are not dangerous as toxic trace metals, but in certain amounts have acute effects. Dizziness and irritation of nose, mouth and eyes can result from long-term exposure to Cu dust. Ni in high concentrations can lead to genetic determinants for nickel induced lung toxicity and dermatitis (Jeong and Ra, 2021b).

Human Health Risk Assessment

Daily Dose and Non-Carcinogenic Risk

According to the DD calculation the major intake of PTEs in adults and children took place via ingestion followed by dermal and inhalation route. This is a reminder that the role of ingestion route from PTEs exposure cannot be ignored. The DD values followed the order of As > Ni > Cd > Cr > Pb
> Cu for ingestion, inhalation and dermal route for adults and children.

The HQ and HI values for PTEs from multiple pathways (ingestion, inhalation and dermal) are presented in Table 4.8 – Table 4.10. Based on the HQ value, adult showed the highest potential for non-carcinogenic effects for HQ_{der} of As (8.80×10^{-4}) followed by HQ_{ing} of As (7.33×10^{-4}). In children the major results were HQ_{ing} of As (1.71×10^{-3}) followed by HQ_{der} of As (1.44×10^{-4}). This results did not exceed the threshold value of 1. However, the sum of HQ was expressed as HI value and was used to assess the overall estimation for non-carcinogenic risk poses by more than one chemical. The maximum HI values for adults and children were 1.62×10^{-3} and 2.04×10^{-3} respectively. This results indicate that children have a higher possibility of adverse health effects from PTEs than adults. However, the non- carcinogenic risk from PTE exposure in adults and children are negligible. The low concentrations of metals influence the daily dose and hazard index values in human. Children and infants are vulnerable population and have a greater risk of experiencing health problems due to PTE exposure from the atmosphere than adults. This is because the amount of air inhalation and other intake by children is twice that of adults (considering the intake per weight unit) and the fact that their lung function and immune systems are not yet fully developed (Mathiarasan and Huls, 2021). The findings from this study was similar to Kalagbor *et al*. (2019) who reported that the cumulative non-carcinogenic health risk indices were less than 1 (HRI < 1), which implies that there was no risk associated with these heavy metals during the study period. However, these values were opposed by Rauf *et al.* (2021) who reported that the HI values for adults and children were greater than 1 indicating a non-carcinogenic adverse health effects from PTE exposure.

Carcinogenic Risk

The carcinogenic health risk to adults and children were determined based on the exposure to As, Cd, Cr and Pb, which are the carcinogenic pollutants. The LCR values for ingestion, inhalation and dermal exposure are listed in Table 4.11 – Table 4.13. The LCR value decreased in the order ingestion > dermal > inhalation, with ingestion posing the most significant risk. The carcinogenic risk of As, Cd, Cr and Pb through combined pathways were less than the acceptable level range (1.0×10^{-4} to 1.0×10^{-6}). However, exposure to As even in small amounts is very dangerous in the long term because of its strong toxicity. The TLCR was calculated as the sum of all LCRs for all combine routes. The TLCR values for adults (4.18×10^{-9}) was lower than children (9.68×10^{-9}). These values were negligible as they were less than the maximum acceptable risk. However, lifetime cancer risk was 5 times higher in children than in adults in the communities. These findings were similar to the results of Oweisana *et al.* (2021a) which reported no carcinogenic health risks both in adults and children. However, Life timecancer risk was 10 times higher in children than in adults in the communities.

V. Conclusion

The control stations showed significant higher levels of particulate matter and PTEs concentrations than some of the study stations. Thus, in addition to automobile activities, other anthropogenic activities such as illegal artisanal refinery of crude and other domestic activities may have contributed to the concentrations of particulate matter and PTEs at the study areas.

The results also indicated spatial variations of the PTEs; although they did not constitute any health risk both in adults and children. However, life time cancer risk was 5 times higher in children than in adults in the communities.

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