INTRODUCTION

Industrial activities such as gas works, refining/distillation of crude oil, fuel/oil storage, transportation, use and disposal etc. are associated with polycyclic aromatic hydrocarbons (PAHs) releases to the environment [1]. Pyrogenic, petrogenic and biogenic sources are the principal sources of PAHs in the environment with biogenic sources not as common as pyrogenic and petrogenic sources [2]. The source of the PAHs polluting the environment can be ascertained by knowledge of the PAH source input into the environment [3,4]. The most common source of petroleum hydrocarbon pollution in Nigeria is petrogenic source arising from accidental spillage of crude oil or/and illegal oil bunkering activities which are prevalent in the Niger-delta region of the country. However, the recent surge of activities in the telecom industry has added the telecom industry as a considerable contributor of hydrocarbon spill incidents across the entire country with the associated potential environmental risks.

Nigeria has undergone massive development in the telecommunication sector over the past decade with huge investment both foreign and local. The risk of hydrocarbon pollution of the environment from the telecom industry in Nigeria is entirely due to the use of generators as alternative energy source with diesel as the main fuel used [5]. The contamination in the study area occurred as a result of vandalism of the diesel generator set and the diesel storage tank containing about 3,000 L in October, 2015. The land use of the polluted soil was for agricultural purposes and is within residential areas. This study identified some habitations and the farmland nearest to the base station as the major environmental receptors of the PAHs contaminants.

The routes of exposure to PAHs at any PAH contaminated soil are non-dietary (inadvertent) ingestion and dermal contact with the soil [6].

The pollution of the environment by PAHs poses very serious environmental and health risks as a result of their mutagenicity and carcinogenicity [7]. Sverdrup et al. [8] reported that PAHs can induce immunotoxicity, reproductive toxicity, carcinogenicity and genotoxicity.

Several studies have reported the ecological and human health risks of exposure to PAHs due to hydrocarbon pollution in soils and sediments [2,9-11]. However, it is not yet very clear the level of ecological and human health risks associated with PAH soil pollution as encountered from spill incidents arising from the Telecom industry. It is very important to understand how spill incidents from the Telecom industry affect ecosystems and human health as most of the telecom facilities unlike those of the conventional oil industry are located within residential areas.
This study would therefore seek to achieve the following objectives:

• Determine the concentration of the PAHs in the polluted soil environment and establish extent of diesel contamination.
• Determine soil toxicity arising from the PAH contamination and estimate human health non-carcinogenic and carcinogenic risks due to the PAH contamination.

EXPERIMENTAL

In this study, the site of diesel contamination is located at Ring Road, Jos, Plateau State Nigeria with geographical reference: N09°54'14.92" E008°54'17.37". The study area for sampling was delineated into three sub locations, AA1, AA2 and AA3. AA1 is the sub-location immediately adjoining the telecom base station and measures approximately 220 m², AA2 adjoining AA1 measures 200 m² and AA3 adjoining AA2 measures 180 m². Nine samples were collected from each sub-location at random at a depth of 0-30 cm using auger borer. The samples for each sub-location were composited, dried and divided into three equal parts for triplicate analysis in the laboratory.

High grade dichloromethane (DCM) and acetone employed for extraction were supplied by Sigma Chemicals, Germany. Deuterated compounds and polycyclic aromatic hydrocarbons (PAHs) standard mix containing naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Flo), phenanthrene (Phe), anthracene (Ant), fluoranthene (Fla), pyrene (Pyr), benzo(a)anthracene (BaA), chrysene (Chy), benzo(b)fluoranthene (BbF), benzo(k)fluoranthene (BKF), benzo(a)pyrene (BaP), benzo(ghi)perylene (BghiP), dibenz(a,h)anthracene (DahA) and indeno (1,2,3-cd)pyrene (IcdP) were supplied by AccuStandard, USA.

Modified USEPA method 3570 was employed in the extraction of PAHs in this study. The procedure is as follows:

Dread sieved soil samples (20 g per sample) were mixed with 10 g of sodium sulphate. The resultant mixture was mixed with 100 mL dichloromethane/acetone (1:1, v/v) and the surrogate standards (50 µL at 100 mg/L surrogates). The mixture was agitated in a mechanical stirrer for 2 h and the extract centrifuged with a centrifuge machine (80-2B Uniscope laboratory centrifuge by Surgifriend Medicals) operated at 3500 rpm for 30 min. The extract was concentrated by means of a rotary evaporator (RES2-2, SearchTech Instruments, England).

Quality control measures employed in the course of this study were triplicate sample analysis, procedural blank, spiked blank and surrogate recovery analysis. The surrogate recovery was used to indicate the efficiency of the extraction method.

Recovery (%) = \( \frac{\text{Amount determined by analysis}}{\text{Amount added}} \times 100 \)

The per cent recovery of the surrogate standards is the equivalent percent recovery of the relevant PAHs and hence giving the extraction efficiency. The recovery of the surrogate standards ranged from 65 to 91 %.

The basis for the relative standard deviation as computed in this study was the fact that samples were collected and analyzed in triplicates. The relative standard deviation (RSD) of the measured PAHs of the soil samples was calculated and the values varied between 5 and 12.6 %.

Determination of the PAHs was carried out using gas chromatograph-mass spectrometry. The GC-MS used was Agilent 7890 GC instrument with split/splitless injector (280°), linked to a 5975 MSD mass spectrometer. The GC was equipped with a fused silica capillary column (30 m × 0.25 mm) coated with 5 % phenyl polysiloxane (HP-5) stationary phase. The initial GC oven temperature was 65 °C, held for 1 min, then increased to 140 °C at 25 °C/min (Ramp 1); and then increased to 290 °C at 10 °C/min for 11 min (Ramp 2). The carrier gas used was helium at a flow rate of about 1 mL/min and initial pressure of 9.0855 psi and average velocity of 37.604 cm/s. Selected ion monitoring (SIM) mode was used to monitor ions of the target PAHs. The PAHs were identified by comparing the retention times of the analytes to those of the external standard and matching the mass spectra of the monitored ions of the analytes with the National Institute of Standards and Technology (NIST) library database spectra. The computation of the analyte concentration is performed automatically by the instrument as it compares the peak areas of the analytes with those of the external standards [12].

Soil toxicity of the study area was evaluated using toxic equivalency factors (TEF). In this study, the TEFs provided by Nisbet and Lagoy [13] were employed for evaluating the toxicities of the non-carcinogenic PAHs whereas the ones provided by DoE [14] were used for evaluating the toxicities of the carcinogenic PAHs. In the assessment of risks via TEF methodology, the total toxicity equivalency (TEQ) is computed as follows:

\[
\text{TEQ} = \Sigma C_n \times \text{TEF}_n
\]

where: TEQ = Total toxicity equivalence; \( C_n \) = concentration of individual PAH in the soil; \( \text{TEF}_n \) = Toxic equivalency factor of the individual PAH in the soil.

This study assessed non-carcinogenic risks to human health due to PAHs by means of hazard quotients which would be summed up to generate the hazard index (HI).

Hazard quotient for non-carcinogenic risks (HQ) = \( \frac{D}{RfD} \) (2)

where: \( D \) = exposure dose (mg/Kg.day); \( RfD \) = reference dose (mg/Kg.day).

The exposure dose via soil ingestion and dermal contact was estimated as provided by Health Canada [6].

In this study, the targeted chemical specific approach was employed in the carcinogenic risk assessment of exposure to the PAHs. In this approach, exposure dose, slope factor and potency equivalency factor (PEF) were used in calculating excess lifetime cancer risks associated with the dermal contact and non-dietary ingestion of the PAH contaminated soil by a typical adult of 70 kg body weight according to eqns. 3 and 4.

Exposure via non-dietary (inadvertent) ingestion of PAH contaminated soil:

\[
\text{ELCR} = D_i \times \text{PEF} \times \text{SF}
\]

Exposure via dermal contact of PAH contaminated soil:

\[
\text{ELCR} = D_d \times \text{PEF} \times \text{SF}
\]

where ELCR = excess lifetime cancer risk; \( D_i \) = exposure dose via non-dietary ingestion of PAH-contaminated soil whereas \( D_d \) = exposure dose via dermal contact. \( D_i \) and \( D_d \) were
calculated as provided in Health Canada [6]. PEF = potency equivalency factor. The PEF employed in this study is as provided in OEHHA [15].

This study adopted the cancer slope factor of 2.3 per mg BaP/kg/day [6].

RESULTS AND DISCUSSION

The concentration and exposure dose of the PAHs for sublocations AA1 and AA2 are presented in Table-1.

The PAHs were not detected in sub-location AA3 indicating that the PAH contamination did not extend beyond sub-location AA2. Among the low molecular weight PAHs, only naphthalene occurred at significant level of 280.2 mg/Kg (Table-1). All the eight carcinogenic PAHs namely benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(ghi)perylen, dibenz(a,h)anthracene and indeno(1,2,3-cd)pyrene were detected. Among the carcinogenic PAHs present in the soil, benzo(k)fluoranthene, benzo(ghi)perylen, dibenz(a,h)anthracene and indeno(1,2,3-cd)pyrene occurred at significant concentration in sub-location AA1 (Table-1). The total concentration of all the PAHs present in sub-location AA1 is 477.2 mg/Kg with the carcinogenic PAHs making up 42 % and the rest 58 % made up by only naphthalene. In sub-location AA2, the total concentration of the PAHs is 24.2 mg/kg with naphthalene accounting for 53 % of all the PAHs measured and the carcinogenic PAHs accounting for 47 %.

Volatileization was expected to drastically reduce the concentration of the low molecular weight PAHs, but if naphthalene was quickest to migrate to the sorption sites of the soil, there would be the tendency of sorption inhibiting its volatilization even though it has the highest vapour pressure among the 16 priority PAHs studied. Other studies have also demonstrated the occurrence of naphthalene in hydrocarbon polluted soil at significant levels [10,11]. This in-turn would mean that the un-adsorbed low molecular weight PAHs would have the tendency to volatilize or migrate to the sub-surface. This is suggested to account for their undetected level of concentration in the top soil [16,17]. The carcinogenic PAHs are heavier with relatively lower vapour pressure and therefore lesser tendency to volatilize and might migrate more slowly to the sub-soil [17]. Hence, their detected presence in the top soil in sub-locations AA1 and AA2. The exposure dose of the PAHs for both inadvertent (incidental) soil ingestion and dermal contact are as presented in Table-1.

The PAHs known to be non-carcinogenic such as naphthalene and benzo(a)pyrene were evaluated for their non-carcinogenic health risks by estimating their hazard quotients and general hazard index. It is important to note that benzo(a)pyrene exhibits both carcinogenic and non-carcinogenic properties and so is considered in both classes in risks evaluation [18].

Potential ability of the individual non-carcinogenic PAHs to cause adverse health effects is dependent on their hazard quotient (HQ). Hazard quotient > 1, indicates potential non-carcinogenic health effect of the individual PAHs [19]. However, non-carcinogenic PAHs could have potential adverse effects derived from their interactive and/or additive effects. The potential ability of PAHs to cause adverse health effects as a result of additive and/or interactive effects is measured by hazard index (HI) [20]. HI < 1, indicates insignificant non-carcinogenic adverse health effect arising from additive and/or interactive effects of all the non-carcinogenic compounds and the converse is true if HI > 1. The HQ and HI are presented in Table-2.

The values of HQ for the individual non-carcinogenic PAHs (naphthalene and benzo(a)pyrene) for both sub-locations AA1 and AA2 << 1 (Table-2). This indicates that the potential to cause non-carcinogenic health effects by either naphthalene or benzo(a)pyrene is quite insignificant. The HI of the PAHs is also << 1 indicating that there is no adverse non-carcinogenic health effect arising from additive and/or interactive effects of the PAHs.

<table>
<thead>
<tr>
<th>PAH</th>
<th>Conc. (mg/kg)</th>
<th>Dose (mg/kg/d)</th>
<th>Dose (mg/kg/d)</th>
<th>Conc. (mg/kg)</th>
<th>Dose (mg/kg/d)</th>
<th>Dose (mg/kg/d)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>D_i × 10^7</td>
<td>D_d × 10^7</td>
<td></td>
<td>D_i × 10^7</td>
<td>D_d × 10^7</td>
</tr>
<tr>
<td>Nap</td>
<td>280.2</td>
<td>30.40</td>
<td>10.60</td>
<td>12.8</td>
<td>1.400</td>
<td>0.480</td>
</tr>
<tr>
<td>Acy</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ace</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Flo</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Phe</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Ant</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Fla</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Pyr</td>
<td>ND</td>
<td>–</td>
<td>–</td>
<td>ND</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>BaA</td>
<td>5.1</td>
<td>0.50</td>
<td>0.45</td>
<td>0.2</td>
<td>0.022</td>
<td>0.008</td>
</tr>
<tr>
<td>Chy</td>
<td>12.0</td>
<td>1.30</td>
<td>1.90</td>
<td>2.1</td>
<td>0.065</td>
<td>0.023</td>
</tr>
<tr>
<td>BbF</td>
<td>4.1</td>
<td>0.45</td>
<td>0.15</td>
<td>0</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>BkF</td>
<td>23.2</td>
<td>2.40</td>
<td>0.90</td>
<td>2.8</td>
<td>0.300</td>
<td>0.110</td>
</tr>
<tr>
<td>BaP</td>
<td>13.5</td>
<td>1.50</td>
<td>0.51</td>
<td>0.5</td>
<td>0.054</td>
<td>0.020</td>
</tr>
<tr>
<td>BghiP</td>
<td>48.0</td>
<td>5.20</td>
<td>1.80</td>
<td>1.1</td>
<td>0.120</td>
<td>0.042</td>
</tr>
<tr>
<td>DahA</td>
<td>26.1</td>
<td>2.80</td>
<td>1.00</td>
<td>1.6</td>
<td>0.170</td>
<td>0.060</td>
</tr>
<tr>
<td>IcdP</td>
<td>65.0</td>
<td>7.10</td>
<td>2.50</td>
<td>3.1</td>
<td>0.340</td>
<td>0.120</td>
</tr>
</tbody>
</table>

ND = not detected or < limit of detection. PAHs were not detected in sub-location AA3. D_i and D_d represent exposure dose due to incidental soil ingestion and dermal contact respectively.
The assessed soil toxicity and estimated lifetime cancer risks for sub-locations AA1 and AA2 are as presented in Table-3.

<table>
<thead>
<tr>
<th>Hydrocarbons</th>
<th>Dose (sum) × 10^7 (mg/kg/d) for sub-location AA1</th>
<th>Dose (sum) × 10^7 (mg/kg/d) for sub-location AA2</th>
<th>HQ × 10^-6 for sub-location AA1</th>
<th>HQ × 10^-6 for sub-location AA2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nap</td>
<td>41.0</td>
<td>1.8</td>
<td>0.2</td>
<td>0.009</td>
</tr>
<tr>
<td>BaP</td>
<td>2.01</td>
<td>0.07</td>
<td>0.6</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Dose (sum) = D_1 + D_2 + D_3 + D_4 as defined in Table-2.

The TEQ values of 31.04 and 1.514 mg/kg for sub-locations AA1 and AA2 respectively exceed the Environment Canada and Department of Ecology (DoE) Washington allowable limits of 0.14 mg/Kg and 0.6 mg/kg respectively required for protecting human life and guaranteeing proper functioning of the habitats of the living organisms in soil ecosystem [14,21] (Table-3). The TEQ value of 31.04 mg/kg for sub-location AA1 is significantly higher than the standard allowable limits and that of sub-location AA2 and therefore poses a higher risk to human health and ecosystem functioning.

Application of the targeted chemical-specific approach for risk assessment is based on the benchmark of 1 in a million risk threshold meaning that risks in excess of 1 × 10^-6 is considered significant for carcinogenic effects [22]. The assumption of ‘additivity’ which is a standard practice in cancer risk estimation of exposure to PAHs was the basis for computing total risks as the sum of the incremental contributions from each of the targeted PAHs [6]. The risk index, \( RI = 1.9135 \times 10^6 \) (Table-3) for sub-location AA1 indicates a risk level in excess of 1 × 10^-6 demonstrating that this section of the soil area poses a carcinogenic health risk to humans. The Telecom base station workers, owners of the polluted farm and the public who walk through this polluted area are at risk of adverse health effects from exposure to the PAHs. The following effects could be observed with time: lung cancer, skin, liver and intestinal tumors among others [23]. The risk index, \( RI = 0.1006 \times 10^6 \) (Table-3) for sub-location AA2 indicates a risk level that is less than 1 × 10^-6 indicating that this section of the soil area poses insignificant carcinogenic health risk to humans.

### Conclusion

For the purposes of assessment of human health risk exposure to PAHs of the contaminated soil, the contaminated soil was delineated into sub-locations AA1, AA2 and AA3. The occurrence and distribution of the PAHs were such that the extent of PAHs contamination was within sub-locations AA1 and AA2. In sub-location AA3, PAHs were undetected. Among the PAHs (with a total concentration of 477.2 mg/Kg), the only non-carcinogenic PAH detected was naphthalene constituting about 58 % of the total PAH in the soil while the carcinogenic PAHs constituted 42 % of the total PAHs in the soil. The assessment of health risk exposure to the non-carcinogenic PAHs indicated insignificant risk for both sub-locations AA1 and AA2. However, the assessment of health risks using soil toxicity method indicates that soil locations AA1 and AA2 are both toxic and therefore could have adverse ecological and human health effects. The employment of targeted chemical-specific approach to estimate the excess cancer risk revealed that sub-location AA2 does not pose any significant cancer risk of exposure to the carcinogenic PAHs whereas in sub-location AA1, there is significant cancer risk of exposure to carcinogenic PAHs.

This study has been able to demonstrate the inherent human health risk of exposure to PAHs associated with the telecom industry as it is presently being operated in the developing countries such as Nigeria where there is reliance on diesel as fuel for generator plants used as alternative source of power. However, the quantity of diesel spill may be an important decisive factor of the level of expected risks.

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