

## Ecotoxicity and Associated Threat of Polycyclic Aromatic Hydrocarbons (PAHs) to Biodiversity: A Review

I.U. ZUNGUM<sup>1\*</sup>, T.S. IMAM<sup>2</sup>

<sup>\*1</sup>Department of Biological Sciences, Federal University Gashua, P.M.B 1005, Yobe State- Nigeria

<sup>2</sup>Department of Biological Sciences, Bayero University Kano, P.M.B 1515, Kano State – Nigeria

Corresponding email: [idezungum@gmail.com](mailto:idezungum@gmail.com)

### Abstract

There is a sustained rise in incidence of cancer and toxicity related to chemicals exerting enormous burden to public health and biodiversity. Polycyclic Aromatic Hydrocarbons (PAHs) are among such contaminants, precisely the sixteen-priority characterized by United States Environmental Protection Agency (USEPA). Therefore, this review is aimed at further elaboration about the 16 USEPA characterized PAHs and threat portend to public health and biodiversity. PAHs are a class of very stable organic pollutants produced most commonly, by incomplete combustion of fossil fuel and are formed when complex organic substances are exposed to heat. PAHs in great amount due to build up over time by bioaccumulation can be perilous: to human beings of all age and levels, aquatic organisms, amphibians and reptiles.

The soil like the aquatic environment contains substantial quantity of PAHs since, atmospheric PAHs sediments on the soil due to dry and wet deposition, terrestrial organism are impacted if the soil is saturated with PAHs. Therefore, PAHs are a great source of trepidation for food safety, public health and biodiversity sustenance. Hence, tackling the spade of the menacing ubiquity of PAHs becomes necessary from its sources by encouragement of alternatives to petroleum fuels for machines and vehicles.

**Keywords:** Polycyclic Aromatic Hydrocarbons (PAHs), Ecotoxicity, Biodiversity, Public Health, Environmental safety.

### Introduction

There is a sustained rise in incidence of cancer and toxicity related to chemicals exerting enormous burden to public health and biodiversity (Zungum *et al.*, 2019; Yaya *et al.*, 2019; Sarki and Roni, 2019). Therefore, this review is aimed at further elaboration about the 16 United States Environmental Protection Agency (USEPA) characterized PAHs and their threat to public health and biodiversity. Polycyclic aromatic hydrocarbons (PAHs) are a class of very stable organic pollutants produced most commonly, by incomplete combustion of fossil fuel and are formed when complex organic substances are exposed to heat plus generally, they do not occur as single compounds but as complex mixtures, (Mojiri, *et al.*, 2019). PAHs represent a group of organic compounds which are highly soluble in organic solvents (lipophilic) but have relatively low solubility in water (hydrophobic) (ATSDR, 2009). They are pollutants that can be generated during processing food and are ubiquitous (Li *et al.*, 2020). PAHs in the atmosphere can react with other pollutants such as sulphur dioxide, nitrogen oxides and ozone forming dinitro-PAHs, nitro-, diones, and sulfonic acids (Tran-Lam *et al.*, 2018). PAHs may also be degraded by some microorganisms in the soil (Salman and Kadhim, 2018). PAHs also manifest various functions such as; conductivity, emission ability, light sensitivity, heat resistance, physiological actions, and corrosion resistance, they also possess very characteristic

UV absorbance spectra, where each ring structure has a unique UV spectrum, thus each isomer has a different UV absorbance spectrum (Shimizu *et al.*, 2020).

PAHs do not occur as single compounds but as complex mixtures in the environmental matrixes because of their related physicochemical properties during the pyrolytic process (Wolf, 2019). They get formed primarily during the incomplete heat induced decomposition of organic materials (Haritash & Kaushik, 2009). The three environment sources of PAHs are: the petrogenic, the pyrogenic and biogenic. Pyrogenic PAHs are products of elevated temperature (>690°C) and rapid oxygen depletion during an incomplete combustion of fossil fuels and organic substances (Zhao *et al.*, 2017; Hailwood *et al.*, 2001). Pyrogenic PAHs are creation resulting from the breakdown of organic matter to low molecular weight radicals during pyrolysis, which is subsequently accompanied by fast recombination into PAHs formation (Patel *et al.*, 2020). Greater concentrations of Pyrogenic PAHs are normally found within the urban areas because their major sources are the incomplete combustion of petrol, gasoline and diesel in automobile, cum heat and power generation, production and use of coal tar and asphalt, and discharges from aluminium smelters and gas plants (Yang *et al.*, 2021).

Biogenic PAHs are produced from biogenic precursors by plants, algae/phytoplankton, and microorganisms (Stogiannidis & Laane, 2015). For example, concentrations of perylene, naphthalene, and phenanthrene concentrations have been found in hydromorphic soils, Magnolia flowers, and *Coptotermes formosanus* termite nests (Wolf, 2019). While, biogenic PAHs are often found at background levels in recent sediments, they are frequently the primary PAHs in older sediments deposited before increased industrialization (Lawal & Fantke, 2017). Whereas, Petrogenic PAHs originates from biogenic processes at fairly low temperatures of about 100-300°C gradually over a long time, resulting in the materialization of petroleum and other fossil fuels comprising of Petrogenic PAHs (Lawal & Fantke, 2017). These PAHs are ushered into the environment accidentally due to oil spills, discharge from tanker operations, and leakage of underground and aboveground storage tanks (Zakaria *et al.*, 2002).

Evidently, ubiquity of PAHs makes it one of the most prevalent persistent organic pollutant in the environment such as water bodies, soil and in some organisms, but the ultimate reservoir is the soil (Huanling *et al.*, 2019). Studies in the United Kingdom affirmed that more than 90% of the PAHs resides in the surface of the soil (Wolf, 2019).

## Discussion

### PAHs of Grave Concern (Sixteen USEPA Characterized)

United States Environmental Protection Agency has classified sixteen PAHs as priority pollutants for human and animal toxicity as listed below: Acenaphthene, Acenaphthylene, Anthracene, Benzo(a)pyrene, Benzo(a)anthracene, Benzo(b)fluoranthene, Benzo(k)fluoranthene, Benzo(g,h,i)perylene, Chrysene, Dibenzo(a,h)anthracene, Fluoranthene, Fluorene, Naphthalene, Indeno(1,2,3-cd)pyrene, Phenanthrene and Pyrene (Wolf, 2019; Domingo, 2017; Hiba, 2015; Keith, 2015).

Acenaphthene appears as white needles: hydrophobic, lipophilic and produces pungent irritating smoke when exposed to heat. It is a natural constituent of coal tar and used in manufacture of pharmaceuticals, dyes, plastics, fungicides and insecticides (NCBI, 2019).

Acenaphthene can induce Cytochrome P450s and undergo oxidation to form toxic PAHs metabolite such as 1-Acenaphthenol which could be of toxic significance to both human and animals (Shimada *et al.*, 2015). It irritates skin and mucous membranes in human (Kamata *et al.*, 2018). Placed by IARC grouping in group 3, it was not classified as to its carcinogenicity

potential in human (Kamata *et al.*, 2018). Therefore, this PAH has not demonstrated any evidence to be considered carcinogenic to both human and animals (Smith & Perfetti, 2019).

Acenaphthylene is a colourless crystalline solid present in petroleum products, coal and also found in automobile exhausts, cigarette, coal and wood emissions. It is used to make products for electronic engineering and in synthesis of fungicides, insecticides, dye and plastics stuffs (NCBI, 2019).

Acenaphthylene can induce Cytochrome P450s and undergo oxidation to form toxic PAHs metabolite such as 1-1, 2-Epoxyacenaphthene which could be of toxic significance to both human and animals (Shimada *et al.*, 2015). It may affect autonomic nervous system, lungs, thorax, or respiration: respiratory depression; blood: haemorrhage and may cause damage and irritation to both eye and skin (NCBI, 2019).

Anthracene is yellowish white particle with mild aromatic odour and are commonly found in petroleum products, cigarette and coal smokes, automobile exhaust, and generated during other combustion and disposal of fossil fuels (Tay *et al.*, 2017). Primarily, used as production of dyes, scintillation counter crystals, and smoke screens (NCBI, 2019). Despite, the substantial existent literature PAHs toxicity, there are very limited data on anthracene however, it is considered a mild irritant of the skin, nose, throat, and eyes (NIOSH, 2019). Though still considered an unverified carcinogen to human (IARC, 2018). But its commonly determined metabolite is trans-1, 2-dihydroxy-1, 2-dihydroanthracene (NCBI, 2019).

Benz[a]anthracene is a crystalline and appears as colourless powder which gives greenish-yellow fluorescence, generated during and incomplete combustion of organic matter (NCBI, 2019). Primarily, it's found in automobile exhaust, coal tar, tobacco, petroleum and coal combustion emissions, cum open heat-treated foods (NCBI, 2019). Meanwhile, its Common metabolites are 3-hydroxybenzo [a] anthracene and 3,4-dihydroxy-1,2-epoxy-1,2,3,4-tetrahydrobenz [a] anthracene (Guntupalli *et al.*, 2019; Schmoldt *et al.*, 1981). There is paucity in literature regarding other health effect in both human and animals. However, evidently its carcinogenic to both human and animals as it was shown to have caused cancer in lung and liver of animals (Siemiatycki & Xu, 2019).

Benzo[a]pyrene appears in liquid state which is formed during a pyrolytic process of an organic substance and primarily, it's found in automobile exhaust, coal tar, tobacco, petroleum and coal combustion emissions, cum during pyrolysis of foods (NCBI, 2019). Its popularly for various irritation and other mild health challenge to human and animals (SEPA, 2019). And this substance has been designated as a biomarker for the monitoring PAHs movement and toxicity to both human and animals due its toxicity potential and prevalence (Ince & Ince, 2019). 3, 4-Benzpyrene is one of its major metabolites produced when cytochrome P450 is induced and it is carcinogenic to both human and animals (IARC, 2019).

Benzo[b]fluoranthene appears as needles or yellow fluffy powder, formed during a pyrolytic process of an organic substance and primarily, it's found in automobile exhaust, coal tar, tobacco, petroleum and coal combustion emissions, cum during pyrolysis of foods pyrolysis products (NCBI, 2019). On a direct contact or smokes causes irritation of the skin, throat and bronchial tubes (Pohanish, 2012). Benzo(b)fluoranthene is a human and animal carcinogen (IARC, 2019).

Benzo[ghi]perylene is a colourless crystalline solid, generated in the incomplete pyrolysis of organic substances and fossil materials. Benzo[ghi]perylene 3,4-oxide and Benzo[ghi]perylene 3,4,11,12-bisoxide are the major metabolites of benzo[ghi]perylene which targets, lungs, skin, eyes and sperm and affects their integrity (NCBI, 2019; Jeng *et al.*, 2013). In flames, it produces

acid, corrosive, irritating and toxic fumes while, in liquid state emits suffocating and dizziness causing vapour (ERG, 2016). Benzo[g,h,i]perylene is a human carcinogen (IARC, 2019).

Benzo(k)fluoranthene appears as pale-yellow needles or yellow crystalline solid and is generated during organic matter combustion. Benzo(k)fluoranthene is primarily located in automobile exhaust, cigarette, coal, coal tar and oil combustion discharges (NCBI, 2019). The compound 8,9dihydroxybenzo(k)fluoranthene is the major metabolite when induced by CP450 enzymes (LaVoie *et al.*, 1980). Meanwhile, the major toxicological effect of concern is its possible carcinogenicity to humans (NCBI, 2019).

Chrysene is a white crystalline substance and among the PAH4 biomarkers of monitoring of toxicity and movement of PAHs and the major metabolites that induces Cytochrome 450 1A enzyme are chrysene-3,6-diphenol, 1-hydroxy chrysene and 6-hydroxychrysene (Lou *et al.*, 2019; NCBI, 2019). It is associated with cancer in both human and animals (IARC, 2019).

Dibenzo[a,h]anthracene is a crystalline, generated from pyrolytic process of organic matter and primarily located automobile exhaust, heat treated foods and fossil deposits (NCBI, 2019). The major is for purpose of research to induce tumour in animals. These are the major metabolites are 1,2dihydro-1,2dihydroxydibenzanthracene and 3,4dihydro3,4dihydroxydibenz anthracene. Dibenzo(a,h)anthracene is a mutagenic and carcinogenic to both human and animals (NCBI, 2019).

Fluoranthene appears as light-yellow fine crystals with no evidence of animal or human carcinogenicity (NCBI, 2019). Commonly, Its Metabolites are 9-fluorenone and 9-hydroxyfluorene (Sepic *et al.*, 2003).

Fluorene is a whitish and generated during incomplete combustion of organic substances and its commonest metabolite is 2-OH fluorine (Peiffer *et al.*, 2013). Causes corrosion/irritation and respiratory tract irritation but there is absent of available data about its association with cancer (NCBI, 2019).

#### Indeno[1,2,3-cd]pyrene

Indeno [1,2,3-cd] pyrene is yellowish in colour, generated from burning of fossil fuel and during incomplete combustion and sourced primarily from fossil deposits (NCBI, 2019). The compound 8-Hydroxyindeno [1,2,3-cd]Pyrene is its most common metabolite (Rice *et al.*, 1986). Indeno[1,2,3-cd]pyrene is carcinogenic to human (IARC, 2019).

Naphthalene is white and a volatile, solid which emits strong mothball odour in contact with flames. Sourced from fossil deposit and generated from incomplete combustion, and is used to synthesis phthalic anhydride and also as a repellent of moth (NCBI, 2019). The major Metabolites are 1, 2 dihydronaphthalene- 1,2-diol, 1, 2-dihydro-1-naphthol (Parke, 1968). Health challenges associated to Naphthalene are cataracts, haemolytic-anaemia, retina-haemorrhage, liver and neurological system damage (NCBI, 2019). It is connected to colorectal and laryngeal cancer and it is a human carcinogen (NCBI, 2019).

Phenanthrene appears as colourless crystals with faint odour and with the following metabolites: trans1,2-dihydro1,2-dihydroxyphenanthrene, trans-3,4-dihydro3,4-dihydroxyphenanthrene and 4-hydroxyphenanthrene (Lou *et al.*, 2019; Rossella *et al.*, 2009). On exposure to Phenanthrene the following symptoms will be exhibited: skin irritation, respiratory irritation, dermatitis, cough, bronchitis, respiratory neoplasm, dyspnoea and kidney neoplasm (NTP, 1992). Shows no evidence of associated cancer risk to both human and animals (IARC, 2019).

Pyrene a colourless solid crystal and its major metabolite is 1-hydroxypyrene (Beach *et al.*, 2010). Can cause respiratory, skin and eye irritation, (NCBI, 2019). 1-hydroxypyrene is its metabolites (Rossella *et al.*, 2009). No sufficient data with regards to its carcinogenicity (IARC, 2019).

### Occurrence of PAHs in Food

Food is a necessity for survival and nourishment of all living organism of which without they seize to exist. Protein component of the food like meat are very popular in low-income communities across the world, where it is served during festivities in abundance (Zungum *et al.*, 2020). However, many studies in recent years have shown that the second major PAHs exposure route is through consumption of food (Zhu *et al.*, 2021; Babić *et al.*, 2018; Duan *et al.*, 2016; Xia *et al.*, 2010). Although, contamination of food by these pollutants is chiefly constraints on: type of fuel applied such as fuel wood, scrape tyre, liquefied petroleum gas, and kerosene. Also, processing procedures adopted: cooking, smoking, grilling, frying, roasting and singeing. Equally, it is dependent on the distance from source of PAHs contamination such as coal mine or volcanic eruption sites, oil spill locations, chemical industrial waste channels or dump sites (Eze *et al.*, 2019). And, even nearness to traffic junctions with lots of automobile exhaust emissions (Tella *et al.*, 2017). According to Abu (2013) there are other mechanisms that result in the generation of PAHs such as pyrolysis of animal fats. Eze *et al.* (2019) in agreement, reported that PAHs are formed when juice and fat of grilled meat and fish placed over naked flames which generates PAHs that adheres to the surface of the meat, fish and other food substances processed.

Over the past 20 years, there are thousands of studies on PAHs in food and persistently raising germane alarms about the toxic potential, transport and fate of metabolites of PAHs within the environment and bodies of organisms (Sun *et al.*, 2019). Meanwhile, several of such studies confirmed the presence of PAHs in food products processed by various methods aforementioned as related by Zachara *et al.* (2017) that PAHs were determine in commercially smoked fish and meat smoked meat and fish products from polish markets. PAHs were also determined in grilled meat by Kamankesh *et al.* (2015). Also, determined in smoked fish by Ghasemzadeh-mohmmadi *et al.* (2012). Equally, was observed in smoked products by Ledesma *et al.* (2015). And in meat smoked with deferent kinds of smoking spices and wood by Hitzel *et al.* (2013). In Similar vein, it was noticed in Swedish smoked fish and meat (Wretling *et al.*, 2010). PAHs in smoked ham at households in Serbia as reported by Kartalovic *et al.*, (2015). Also, PAHs content were seen in smoked common carp meat in Serbia (Babić *et al.*, 2017). PAHs were noticed in smoked duck in china (Chen & Lin, 1997). Studies in Doha, Qatar showed after analysing smoked meat samples, there were none that exceeded European Commission's maximum limits, EC 835/2011 (Hussain *et al.*, 2018). However, similar research undertaken by Tran-Lam *et al.* (2018) in Vietnam revealed thus:

the ranges of average PAHs levels were 9.3–9.6 µg/kg (for instant noodles), 0.22–2.48 µg/kg (for cakes) 0.91–4.83 µg/kg (dried vegetables), 5.14–23.32 µg/kg (teas), 4.82–24.35 µg/kg (coffees), and 1.43–25.2 µg/kg (grilled meats). The results indicated that the total concentrations of residual PAHs and benzo(a)pyrene in the instant noodles and grilled meat samples surpassed the maximum limits tolerated by the European Commission (35 µg/kg and 5 µg/kg, respectively) in many investigated samples.

PAHs in substantial quantity was seen in smoked fish and meat across Finland according to Mirja *et al.*, (2018). In similar cases, PAHs occurrence was established in chocolate candies, tea, milk, toasted bread and flour (Hiba, 2018). In consistence with other studies across the world, PAHs manifestation was confirmed in arrays of consumables across Africa such as; smoked Bush meats in Kumasi, Ghana and was revealed to have contained PAHs below the



EU peg limits for smoked meats, this is according to Abu, (2013). In a related work the 16 priority PAHs were detected in smoked Tilapia Fish and Catfish, revealed through a research conducted by Nnaji & Ekwe, (2018). They were equally, report in smoked cephalopods, crustaceans, bivalve molluscs, and baby foods (Ijeoma *et al.*, 2015a). The presence of PAHs was also established in roasted and smoked fishes, meats, corn, yam and ripe plantain in Eastern Nigeria (Eze *et al.*, 2019). PAHs were seen in grilled fish and meat, smoked fish and roasted yam in Lagos and Ogun states of Nigeria (Oranusi *et al.*, 2018). Rubber tyres singed cattle hides indicate dangerous deposition of high levels of PAHs in Abia State, Nigeria as disclosed in a research undertaken by Nnaji *et al.* (2017). Ijeoma *et al.* (2015b) confirmed the presence of PAHs in barbecued charcoal grilled meat and singed cowhide in Umuahia, Abia state, Nigeria.

### **Ecotoxicity Effects of PAHs**

The toxicity credited to PAHs is rampant and can be observed across all ecological systems including: aquatic and terrestrial realms. Sequel to the far-reaching voluminous research in area of PAHs toxicity, Agency for Toxic Substances and Diseases Registry (ATSDR) ranked them 9<sup>th</sup> on their Substance Priority List (SPL) based on their potential for combine toxicity, frequency and exposure to human (ATSDR, 2017). Correspondingly, The International Agency for research on Cancer (IARC) has classified PAHs into categories based on impending capacity for carcinogenicity: group 1, 2A, 2B, 3... As known, probable, possible, and non-classified human carcinogen. Of these, the 16 USEPA prioritized PAHs were all fitted in to the categories: group 1, Benzo(a)pyrene; group 2A, Dibenzo(a,h)anthracene; group 2B, Chrysene, Benzo(a)anthracene, Benzo(b)fluoranthene, Benzo(k)fluoranthene, Naphthalene, Indeno(1,2,3-cd)pyrene; and group 3, Acenaphthene, Acenaphthylene, Anthracene, Benzo(g,h,i)pyrene, Fluoranthene, Fluorene, Phenanthrene and pyrene (IARC, 2019; 2010).

### **PAHs Toxicity in Human**

PAHs in great amount due to build up over time by bioaccumulation can be dangerous to human beings capable of damaging body cells. They are associated with carcinogenicity, mutagenicity, teratogenicity, genotoxicity, and other related toxin induced complications in human (NCBI, 2019). Historically, PAHs toxicity was first reported by Sir Percival Pott in the year 1775, initially noticed an associated progression of cases of scrotal cancer among workers consistently exposed to chimney sweeps and soot (Pott, 1775). Subsequently, to date tonnes of PAHs examination has reported exposure and cancer incidence to both human and animals (ARTDR, 2015, Kim *et al.*, 2013; Silverman *et al.*, 2012; IARC, 2010; Zhang *et al.*, 2009; Bosetti *et al.*, 2007; ATSDR, 1995). However, there are only limited epidemiological reports of incidence of lone PAHs exposure to human but of their activity in groups (Sasikumar *et al.*, 2018). Conversely, numerous researches from experimental animals were subjected to tests with Benzo(a)pyrene: ingested via gastro intestinal track, inhaled via respiratory track and exposed to dermal contact, all repeatedly kept leading to tumour, cancer and mutation (IARC, 2019; 2012). Of recent, food and occupational exposure to materials contaminated with PAHs have become leading sources of concern associated for cancer (IARC, 2019; 2010).

PAHs can extensively spread all through human body parts, as investigation has confirmed the distribution in several studies and especially lodged in Adipose tissues due to being lipophilic in nature (Abdel-shafy & Mansour, 2016). Once been in the body, they induce cytochrome 450 1a enzyme triggering the Cytochrome450-PAHs-oxidase-pathway (Fakolade *et al.*, 2017). This very metabolic pathway changes the PAHs to intermediate of polar epoxide and further transformed to Phenol and derivatives of dihydrodiol and lastly converted to sulphate and glucuronide conjugates and then excreted into urine and bile (Campo *et al.*, 2010).

However, the conversional metabolism could lead to bio-activation of PAHs toxicity potency resulting in the materialization of electrophiles or metabolites which causes human health challenges (Gelboin, 1980). Evidently, for this reason PAHs are known as pro-carcinogen since they are harmless in their pristine state but require a metabolite to activate the toxicity potential to cause: carcinogenicity, DNA damage, mutagenic effect or genotoxicity (Moorthy *et al.*, 2015). Furthermore, there exist three pathways for the activation of PAHs carcinogenic potency: radical cations pathway, o-quinone pathway and bay-region dihydrodiol epoxide pathway. This pathway leads to synthesis of radical cations, redox-active-o-quinone, electrophiles and diol epoxides which reacts with DNA to form DNA-adducts (Zhang *et al.*, 2012a).

Impact of PAHs on the immune system has been studied extensively and reported its severity on human and animals (Ulieme *et al.* 2019). The immune suppression as the major and most disturbing effect of PAHs was confirmed in series of investigations over the years, and revealed thus, the route of exposure is immaterial as the end effect exerted is systemic disruption (IPCS, 2010). Hence, PAHs induced immune suppression always associated to increase in susceptibility of an individual organism to infectious diseases, cancer of various kind and mortality (Abdel-shafy & Mansour, 2016). More elaborately, immune system potentiation is associated to increased immune cell synthesis of cytokines with a resultant inflammation. Consequently, facilitating expression of hypersensitivity, allergic responses, auto-immunity and tumour development (Rodelli *et al.*, 2016).

The literature bank concerning PAHs and immune system relation claim that the most prevalent and far-reaching effect of PAHs to human and animal systems is immune suppression (Parvez *et al.*, 2019). Additionally, data from the literature confirmed that frequent reported route of exposure *in vitro* as atmospheric and dietary (Burchiel & Luster, 2001). Whereas, route of exposure *in vivo* as inhalation and injection into sub-cutaneous and intra-peritoneal (Pessa *et al.*, 2001). In addition, studies have reported immune toxicity resulting from oral consumption of food (Fakolade *et al.*, 2017). Furthermore, the severing of the immune system due to build-up of PAHs caused DNA-Adducts in several organs of the body and resulted in cancer (Ulieme *et al.*, 2019).

### **Teratogenicity**

Effects due to embryotoxicity of PAHs have been confirmed from a number of experiments on exposure to PAHs mixture (Perera *et al.*, 2005; Wassenberg & Di-Giulio, 2004). Investigation showed that ingestion during pregnancy of substantial quantity of PAHs in food resulted in low baby weight and birth defects (Fakolade *et al.*, 2017). Similarly, it was confirmed to that exposure to PAHs through several route during pregnancy result in birth complications such as: premature delivery, low birth weight and cardiac abnormalities (Edwards *et al.*, 2010). Further, children given birth to in towns contaminated with PAHs like Teplice were reported to have developed multiple health challenges including: growth retardation, respiratory malfunctions and stunted intra-uterine growth (Pratt *et al.*, 2011). Research further demonstrated that PAHs exposure led to the liver inflammation that resulted in cirrhosis (Ulieme *et al.*, 2019). In addition, PAHs was established in prenatal exposure causing behavioural problems, lowering in IQ, Asthma and DNA adduct that resulted in cancer (Drwal *et al.*, 2019).

### **PAHs Acute Health Effects**

The consequence of PAHs contamination in human could be based on the route of contact, quantity and toxicity potency of the PAHs in exposed to (ACGIH, 2005). Many other factors could be responsible for health induced impact due to PAHs including: pre-existing health status and age (Abdel-shafy & Mansour, 2016). The presence of PAHs was confirmed from

different biological samples of human such as: blood, urine, serum, plasma, saliva, breath exhaled, breast milk, placenta and cerebrospinal fluid (Santos *et al.*, 2019). The tendency of PAHs to induced short time health complications in human has been reported resulting in symptoms such as: skin and eye irritation, nausea, vomiting, headache, diarrhoea, allergic reactions, confusion and diaphoresis (ATSDR, 2013; IPCS; Unwin *et al.*, 2006). Though, the priority PAHs mixtures most times, act in unison that its unlikely to identify which one acted or not (Hiba).

### **PAHs Chronic Health Effects**

There is paucity of literature regarding human chronic effects due to PAHs toxicity. However, long term exposure that resulted in health complication as earlier captured leads to immune suppression, eyes defect endocrine disruption, kidney, lung and liver damage (Kuppusamy *et al.*, 2020; IARC, 2019). Equally, sustained skin contact may induce inflammations and withstood ingestion and inhalation can permit breakdown of red blood cells (NCBI, 2019). Essentially, route of exposure, quantity and prolonged exposure increases the susceptibility to chronic health complications that eventually ends in fatality (Diggs *et al.*, 2011). Meanwhile, bioaccumulation is observed due to protracted PAHs interaction to human and animal body systems and consequentially, deepening the impairment and enhance the susceptible to cancer especially to urban dwelling population by (10.7%) ten percent in a long run (Zhu *et al.*, 2019). Plus, copious investigations implicated the sixteen priority PAHs of being the culprit behind the formation of DNA-adducts due to unrelenting exposure in several human body tissues such as: cervix, vulva, oesophagus, bone marrow, prostate and placenta (Rondelli *et al.*, 2016). Further, degenerating to more complications including: teratogenicity, genotoxicity, mutagenicity and carcinogenicity in human of all age brackets (ATSDR, 2013).

### **PAHs in Aquatic Organisms**

#### **Pisces**

Aquatic organisms were reported to be impacted by the toxicity of Poly aromatic hydrocarbon at the detriment of various units of the ecosystem. Aquatic environment was believed to be most susceptible to different sources of PAHs pollutants in contrast to other media owing to been an important basin for most noxious chemicals (Copat *et al.*, 2018; Ferrante *et al.*, 2018). PAHs was found in several species of fishes within the Persian Gulf contaminated due to oil spill (Akhbarizader *et al.*, 2019). Correspondingly, another research on histopathological examination of Tilapia fish was made for liver, gills, kidney, male gonad and female gonad. Liver showed congestion of blood vessels and hepatic sinusoids with focal necrotic changes of hepatocytes, Gills showed congestion of blood vessels, hyperplasia and adhesion of secondary gill lamellae, Kidney showed congestion of blood vessels and degeneration of renal tubules, Male gonad showed degeneration and necrosis of some seminiferous tubules, oedema and severe reduction in all spermatogenic stages and degeneration and Female gonad showed severe necrosis of developmental stages of oocytes along with degenerated mature ripped oocytes in crude oil contaminated water, treated fish (Dighiesh *et al.*, 2019). In a similar work, Vignet *et al.* (2016) reported inhibition of spermatogenesis, germ cell degeneration, seminiferous tubular epithelial degeneration and necrosis as observed in *Oreochromis niloticus*, *Synodonis schall* and *Tilapia zillii* after taken from a PAHs contaminated water of El Salam Canal. Cardiotoxicity is one of the most common effects observed in many teleost species after a developmental exposure to PAHs (Madison *et al.*, 2015). PAHs Exposure has been well established to disrupt or impair the natural function of endocrine systems in teleost (Dey *et al.*, 2019; Alsaadi *et al.*, 2018). Similarly, PAHs were discovered to activate Cytochrome P450-1A, a gene up-regulator of toxins and by induction of the aryl hydrocarbon receptor and were established to be responsible for teratogenic effects and embryonic cardiac deformities on exposed killifish embryos (Volkoff *et al.*, 2019). In addition, Grung *et al.*, 2016



documented PAHs exposure to Eurasian minnow (*Phoxinus phoxinus*) where activation of cytochrome 450 A1 was observed, DNA adduct and PAHs metabolites formation seen. The genotoxic potency of PAHs was confirmed in an *in vitro* study where Zebrafish embryos were exposed PAHs (Mccarrick *et al.*, 2019). A study adduced that bioaccumulation was the primary process that controls pollutants aggregation in aquatic organisms (Akhbarizadeh *et al.*, 2019). In conformity with the findings Finch & Stubblefield, (2019) relates that PAHs being sufficiently lipophilic bioaccumulates into tissues and interferes with normal system functions of these organisms.

### PAHs in Other Aquatic Organisms

Toxicity reports from many previous researches also reveal the noxiousness related to Poly aromatic hydrocarbon in a test where amphipods taken from southern European waters and subjected to series of toxicity assessments in the laboratory: the amphipods die due to multiple complication resulting from PAHs mixture toxicity (Sanz-Lázaro, 2008). In another research by Zychowski & Godard-Codding (2017) who, narrated that European pond turtle and Caspian turtles were picked from industrial waste water plants and oil spilled contaminated waters in Azerbaijan and reported to have recorded high presence of PAHs which subsequently lead to varieties of adverse effects, skin sloughing, hyperkeratosis, necrosis, acanthosis, mutagenicity, DNA and chromosomal damage, and genotoxicity. Other studies observed mutagenicity and damage to DNA in Dragon fly Nymph (*Odonata anisoptera*) due to PAHs in artificial and natural aquatic environments (Meland *et al.*, 2019). Studies also identify presence and toxicity resulting from PAHs in mollusc and eel (Sun *et al.*, 2018). PAHs was detected in numerous tissue and organs of prawns and crabs isolated from oil waste waters of Persian Gulf (Akhbarizadeh *et al.*, 2019). Equally, PAHs was seen to have caused toxicity to crabs (*Callinectes pallidus*) isolated from Niger Delta creek, Nigeria (Erema & Adaobi, 2013). Similar, an investigation undertaken in Persian Gulf waste confirmed that among benthic species studied, including; *Epinephelus coioides*, *Penaeus indicus*, *Liza klunzingeri*, *Panaeus semisulcatus*, and *Portunus armatus* and those that are less motile were more vulnerable to PAHs contaminants accumulation and better candidate species for monitoring aquatic environments (Akhbarizadeh *et al.*, 2019).

### PAHs in Amphibians

All known amphibians have highly permeable membranes to facilitate the exchange of nutrients and gases with the water column. Membrane permeability increases the occurrence of aquatic xenobiotic ingestion and thus toxicity. Most Amphibians are omnivores, consuming algae, macrophytes, detritus and small invertebrates, which may also act as a dietary source of aquatic contaminants resulting in bioaccumulation (Patterson, 2019). Tadpoles and frogs are common Amphibians and are sources of prey in most aquatic ecosystems, fish often target adult frogs as a favoured food source and this interaction may introduce contaminants to trophic webs and eventually human consumers (Patterson, 2019). Amphibian species experience cardiotoxicity on developmental exposures, but predictions of severity and sensitivity are complicated due to large inter-species differences among amphibians and a lack of precedent setting work conducted (Alderman *et al.*, 2017). Exposure to PAHs have been well demonstrated to disrupt or impair the normal function of endocrine systems in amphibians; thyroid hormone receptor beta (TRB) expression was highly affected by PAHs exposure in amphibians (Alderman *et al.*, 2018; Alsaadi *et al.*, 2018). Alterations in metamorphosis timelines and developmental outcomes occur in *L. sylvaticus* raised in reclaimed wetlands contaminated with PAHs (Products *et al.*, 2011; Whyte *et al.*, 2000). The normal activity of the hypothalamus-pituitary-thyroid (HPT) axis is

essential for reproduction, development and growth of amphibians, but is disrupted by exposure to PAHs (Alderman *et al.*, 2018). Exposure to certain PAHs including naphthalene can disrupt the normal gas exchange mechanism in amphibian integument, leading to decreased CO<sub>2</sub> excretion rates, and potentially to asphyxiation (Wallace *et al.*, 2018). Disruptions in gas exchange can impair fitness or survival if respiration is sufficiently impaired. PAHs exposure resulted in the disruption of normal steroid biosynthesis pathways and metabolic disorder related pathways in amphibians (Lara-jacobo *et al.*, 2019). Steroidal pathways are critical for the formation of hormones that regulate the bodies' endocrine systems and homeostasis. Even low concentrations of PAHs can cause teratogenicity to amphibian embryos, and tadpoles that survived the PAHs exposures displayed behavioural and morphological abnormalities such as twisted spines and irregular swimming patterns indicating chronic, sub-lethal effects from embryonic PAHs exposures (Campbell *et al.*, 2019). The jelly coating around amphibians' eggs, known as the extra-cellular matrix confers protection against the embryonic exposure of PAHs, acting as a sequestering agent to shield the embryos (Parrott *et al.*, 2018). The targeted organ accumulation and toxicity is typically localized to the kidneys and liver, and compounds such as B(a)P cause metabolic and transcriptomic toxicity and carcinogenicity in amphibian hepatocytes (Lara-jacobo *et al.*, 2019; Harner *et al.*, 2018). This data provides evidence that PAHs and other similar AhR antagonists in crude oil may lead to adverse biological responses in developing amphibians either through mechanisms of increased genotoxicity, narcosis or disrupted endocrine signalling (Campbell *et al.*, 2019). In addition, high mortality of Amphibians living in PAHs contaminated sediment ponds was documented (Johansen, 2013).

### **PAHs in Terrestrial Organism**

The soil contains substantial quantity of PAHs since, atmospheric PAHs sediments on the soil due to dry and wet deposition, terrestrial organism are impacted if the soil is saturated with PAHs which could cause mild to adverse effects and in some cases tumour and cancers (Beyer, 2010). Mammal have been reported to ingest PAHs in food or inhaled from polluted air (Dong *et al.*, 2012). Plants also, uptake PAHs from soil through their roots and translocate them to other parts of the plants to be stored, factors governing the uptake are physicochemical potential of the plant, water solubility, concentration and hormone (Abdel-shafy and Mansour, 2018). There limited literature on PAHs induced phytotoxicity (Beyer *et al.*, 2010). Some plants provide resistance and protection against PAHs whereas, other plants synthesis PAHs to serve as growth hormone (Abdel-shafy & Mansour, 2018).

### **PAHs Toxicity in Reptiles**

Polycyclic aromatic hydrocarbons and other pollutants have been identified as major contributors to receding factor for reptilian population and as contributors to toxicity reported from number of studies (Zychowski & Godard-Codding, 2017; Sparling *et al.*, 2010; Gibbons *et al.*, 2000). These toxicity effects are many including: hatching and other reproductive difficulties, feeding and digestive abnormalities, endocrine disruption, immune system suppression, mutagenicity and cancers (Sparling *et al.*, 2010; Van Meter *et al.*, 2006; Aguirre and Lutz, 2004; Romero and Wikelski, 2002; Wikelski *et al.*, 2001; Gibbons *et al.*, 2000; Gibbons *et al.*, 2000; Crain & Guillette, 1998; Vonier *et al.*, 1996; Hutchinson & Simmonds, 1992). Despite the relatively scarce literature regarding ecotoxicological effects due to PAHs, it has been found to interfere with feeding habits, sex determination, egg size, embryonic development and increased burden of accumulation within body parts, and are associated to global reptilian decline (Zychowski & Godard-Codding, 2017; Smith *et al.*, 2007; Hopkins, 2000). Studies on desert lizard (*Acanthodactylus scutellatus*) taken contaminated oil spill sites further revealed that PAHs were found to have exerted ecotoxicological effects in all aspects of the organism's normal physiological functions including cases of cancers (Al-Hashem *et al.*, 2007). Similarly, the lizard, *Pardarcis sicula* was found with extremely high PAHs

concentration has been enzymically distorted and with severed physiological functions (Zychowski & Godard-Codding, 2017).

### Crocodile

There only few studies that documented findings of PAHs ecotoxicological significance, of these all concentrated on the captive crocodile *Crocodylus niloticus* (Arukwe *et al.*, 2016). The study investigated oxidative stress responses finding that severity of PAHs contamination is sex dependent, and PAHs is carcinogenic to the crocodiles (Arukwe *et al.*, 2015). And that is immune suppressant, endocrine disrupting and instrumental to reproductive abnormalities and infantile related deformities (Arukwe *et al.*, 2016).

### Snake

Like other reptiles, there are few researches that attempted to address PAHs exposure in snakes and the earliest investigations focused on control of exposure then subsequently, enzymatic activities, particularly in *Thamnophis species* and *Elaphe guttata* (Bani *et al.*, 1998; Scwen & Mannering, 1982). The species were particularly important for biomonitoring of toxicity in snakes (Zychowski & Godard-Codding, 2017). In a separate investigation, PAHs was seen in the shed skin of *Elaphe guttata* when he had eaten PAHs contaminated mice and further reports biomagnification of PAHs along the food chain (Jones *et al.*, 2009). PAHs were discovered in several body parts of Aquatic snakes: *Lapemis curtus* and *Hydrophis cyanocinctus* taken from crude oil contaminated area along the Persian Gulf (Soltani *et al.*, 2019). Petrogenic sources of the PAHs contributes significantly to the exposure of snakes to the contaminant (Sereshk & Bakhtiari, 2014). Also, studies explored the exposure and toxicity of PAHs from aquatic snakes in Goa, India such that it was found in Kidney, liver and guts (Mote *et al.*, 2015).

### PAHs Toxicity in Rodents

Experimental data available *in vivo* on the prenatal developmental toxicity potency of toxic substances showed that some PAHs were confirmed to induce Prenatal developmental toxicity in experimental animals such as mice (Kamelia *et al.*, 2019a; Feuston, 1989;1996; 1997; Hoberman, 1995). The data generated from *in vivo* investigations are in consistent to that of *in vivo* recent findings in rodents (Kamelia *et al.*, 2019a). Where PAHs content, induced Prenatal developmental toxicity, as quantified in the embryonic stem cells, this further inferring potency for genotoxicity (Kamelia *et al.*, 2017). Further studies, confirmed activation of the aryl hydrocarbon receptor (AhR) from *in vitro* exposure of mouse embryonic stem cell to PAHs (Kamelia *et al.*, 2019b).

PAHs was equally associated to mutagenicity as was confirmed in *in vitro* research where they were found to induce DNA methylation, and reproductive toxicity in rats with consequential infertility, immune suppression, neurotic and pancreatic secretion abnormalities (Zhang *et al.*, 2018). PAHs also damage selectively endocrine glands and targets hormones in female rodents (Kamelia *et al.*, 2018). PAHs compounds depress synthesis of DNA in ileum and spleen and severely injure ovary and elicit ovarian tumours, leukaemia and causes adrenal apoplexy (Uematsu & Huggins, 1968). It also elicits DNA adduct formation and apoptosis further resulting in tumour and carcinogenicity of the rat liver (Trilecová *et al.*, 2011). Recent research also affirmed that PAHs exposure leads to hepatic inflammation and apoptosis, and increase in susceptibility to diabetes type 2 and other related abnormalities (Ulieme *et al.*, 2019).

### Conclusion

The sustained exposure to PAHs could lead to extinction of some organisms dwelling in contaminated environment. And, if unchecked, the extant open-fire-food processing method could seamlessly increase the prevailing food safety challenges and general public health

concerns. Therefore, the 16 USEPA characterized PAHs are a great threat to food safety, public health and biodiversity.

### Recommendations

1. The use of solar and other clean energy sources should be encouraged to cut down emission of PAHs from petroleum driven vehicles.
2. To avoid biodiversity lost; oil spills need to be tackled expediently without any unnecessary delays and infected organisms due PAHs contamination should be isolated to avoid breeding mutants.
3. To cut-down health costs; the authorities in developing countries ought to be religiously serious in enforcing the band in use of scrap tyre and petroleum derived fuels to process foods in an open-fire.

### Conflicts of Interest

The authors declare no conflict of interest.

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