

Availability of Polycyclic Aromatic Hydrocarbons in Environment and Food Chains: A Review

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Doi: 10.23918/eajse.v6i2p83

Abstract: Polycyclic aromatic hydrocarbons (PAHs) due to their detrimental threat to human life, widespread existence in food chains and incremental lifetime cancer risk are of great concern around the globe. Ensuing their carcinogenic, genotoxic, and mutagenic effects, this paper reviews the status of PAHs pollution in air, sediment, soil and water of different world regions and food chains. Besides this, the review will also summarize the data available on the content of PAHs in various food types and effects of PAHs intake on human health along with some preventive measures. The result showed that the overall concentration of PAHs ranged from 0- 3842 ng.m⁻³ in air, 0- 205398.76 ng. L⁻¹ in water and 0-233320 ng. g⁻¹ in sediment while agriculture soil possesses lower concentration of PAHs than urban soil. Moreover, higher content of PAHs in air, water and sediment have found in Asia, Middle East, and Africa, respectively. At the end, discussion will be extended to describe some recommendations to reduce emission rate of PAHs around the world.

Keywords: PAHs, Ecotoxicology, Carcinogenic, Mutagenic, Genotoxic, Regional Distribution

1. Introduction

Polycyclic aromatic hydrocarbons are the organic pollutants occur as white or pale yellow solid, which consist of two or greater than two integrated aromatic rings. PAHs produced during biological processes and partial ignition of organic matter, which not only includes natural process (Slezakova et al., 2013; Wang et al., 2015) but also anthropogenic activities (Cao et al., 2017; Yang et al., 2013). The general properties of PAHs are notable melting and boiling point, low aqueous solubility (decreases with each additional ring) and low vapour pressure. Due to high lipophilicity, they are soluble to organic solvent. In addition, by increasing molecular weight, the latter two characteristics tend to decrease and their resistance to oxidation and reduction increases.

They are available in all compartments of nature because of their persistent, bio accumulative and long-range transport ability (Ashraf, Sarfraz, Naureen, & Gharibreza, 2015). Atmosphere is the first and foremost mean of PAH dispersal in the environment (Abdel-Shafy & Mansour, 2016; Dat & Chang, 2017) through wet or dry deposition these pollutants are introduced into aquatic system (Alves et al., 2016; Franco et al., 2017), deposited, absorbed and reemit from soil. Moreover, plants can consume and accumulate these pollutants in food chains ultimately leading to human exposure.

Received: November 15, 2020 Accepted: December 25, 2020

Bhutto, A.U., Manzoor, S., Bhutto, M.U., & Abodif, A.M. (2020). Availability of Polycyclic Aromatic Hydrocarbons in Environment and Food Chains: A Review. *Eurasian Journal of Science & Engineering*, 6(2), 83-116.



It has proven that PAHs can cause carcinogenic and mutagenic effects and are potent immune suppressants (Kameda, Shirai, Komai, Nakanishi, & Masunaga, 2005; Lee & Shim, 2007; Yoon, Park, Lee, Yang, & Lee, 2007; Zhang, Tao, Shen, & Ma, 2009). Due to their toxic characteristics, United Nations Environmental protection Agency (USEPA) has listed 16 PAHs as major pollutants, from which of seven were classified as carcinogenic (Cancer, 1983).

These pollutants are introduced into the nature via variety of sources (Sanjrani et al., 2017). Anthropogenic sources are considered as the primary sources of PAHs availability in environment and food chains such as, petroleum refineries, smoke from automobile, combustion of refuse, power plants heating, sewage deposition, tobacco fumes, smog from barbeque, coke production and oil/gasoline spills (Christensen & Bzdusek, 2005; Moon, Kannan, Lee, & Ok, 2006). Furthermore, PAHs transport rely on their number of rings molecular weight and phases.

Recently, organic pollution has attracted great concern by environmental agencies and policy makers due to their diversity of sources and lifetime cancer risk. Since 1990's, several investigations have documented the availability of semi volatile organic contaminants in air, soil, water and sediments. Several studies have been conducted on PAHs including polycyclic aromatic hydrocarbons in the region of Asia (Chang, Fang, Chen, & Wu, 2006), PAH diagnostic ratios (Tobiszewski & Namieśnik, 2012), a review on PAHs (Abdel-Shafy & Mansour, 2016), radiation from energy production (Mastral & Callen, 2000), fate and transport in environment and adverse impacts on human (Kim, Jahan, Kabir, & Brown, 2013) and PAHs pollution in food and their impact on health (Bansal & Kim, 2015). Still there is a gap regarding their concentration around the world in different compartments. Therefore, this review will provide the background information on sources, exposure, emission rates and availability in environment and food chains around the world. This paper will also discuss about the impact of PAHs exposure on human health, preventive measures, and some recommendation regarding control strategies to decrease the quantity of PAHs emission in environment.

2. Genesis of PAHs

Polycyclic aromatic hydrocarbons emitted from both natural sources (volcanic eruption and forest fire) and anthropogenic sources (incomplete combustion of fossil fuel, biomass as well as spilling of petroleum products and industrial processes). Mainly distributed into two groups' i-e pyrogenic and petro genic. Whenever at high temperature organic combustion takes place when there is no oxygen, it forms pyrogenic PAH. These include intentionally produced PAHs as destructive distillation of coal and thermal cracking of petroleum residue also unintentionally produced PAHs such as combustion of fuel in automobiles and combustion of wood in forest fires and fireplaces. Unlike pyrogenic, oil maturation of crude and similar process forms petro genic PAH; It includes oil spills, storage tanks leaks and releases due to transportation of oil and gasoline (Abdel-Shafy & Mansour, 2016). Various factors are responsible for PAH emission such as fuel type, combustion situation such as turbulence, resident time, temperature, kind of combustor, moisture content, genus of air pollution control devices (Dat & Chang, 2017). However, pyrogenic sources produce PAH dominated by four and five ring PAHs (Hu et al., 2017). While petro genic sources mainly produce, two and three ring PAHs (Kwon & Choi, 2014; Zhi, Zhao, & Zhang, 2015).

Origin of PAHs in surrounding samples is usually identified by utilizing diagnostic ratios (Pongpiachan, Tipmanee, Khumsup, Kittikoon, & Hirunyatrakul, 2015; Tobiszewski & Namieśnik, 2012). Composition of PAHs emission rely on combustion source such as particulates release from diesel are dominated by 3-4 rings compound such as fluoranthene, phenanthrene, and pyrene (Rogge,



Hildemann, Mazurek, Cass, & Simoneit, 1993; Westerholm & Li, 1994) while Fluoranthene, benzo[a]fluoranthene, benzo[g,h,i]perylene, indeno[1,2,3-c,d]pyrene, phenanthrene, and chrysene were predominant in municipal waste incinerator (Williams, Meares, Brooks, Watts, & Lemieux, 1994). Petroleum related PAHs are characterised by higher content of alkyl – substituted derivatives. Therefore, diagnostic ratios are based on proportions of alkyl-substituted, parent PAHs and nonsubstituted molecules. Ant/ (Ant + Phe) > 0.1 suggest the dominance of petroleum, while <0.1 indicates the dominance of fuel combustion. BaA/ (BaA + Chr) > 0.5 indicates pyrogenic source, ratio <0.2 shows the dominance of petro genic source while 0.1-0.5 indicate both sources. Fla/ (fla + pyr) < 0.4 shows petro genic source, 0.4 - 0.5 indicates fuel combustion and ratio > 0.5 shows coal, grass or wood burning. IcdP/(IcdP + BghiP) < 0.2 shows petrogenic source, > 0.5 shows coal, grass or wood burning while 0.2 – 0.5 indicates fuel combustion (Tobiszewski, 2014; Zhi et al., 2015). In addition to this, Nap, Phe and Ant account for main part of PAHs in fuel oil product and indicates petroleum as major sources where bicyclic and tricyclic aromatics are dominant. Diagnostic ratios can easily be affected by change in climatic, environmental conditions so these are not much effective as a tool for source identification (Katsoyiannis & Breivik, 2014). Because of limitations, it is not possible to draw any conclusive analysis (Duodu et al., 2017). Therefore, along with diagnostic ratios, multivariate statistical analysis methods including principal components analysis (PCA) and positive matrix factorization (PMF) can also be used for receptor modelling of sources/source identification of PAHs. Different researchers have used some diagnostic ratios for source apportionment in their studies that are mention in the Table 1.

Positive matrix factorization disintegrates a matrix of speciated sample data into two matrices: factor profiles and factor contributions. USEPA in 2014 and other researchers interpret these profiles by using measured source profile as well as discharge information, in order to identify the type of sources (Paatero, 1997; Paatero & Tapper, 1994). Source profiles can be compared with the available literature values and conclude that PAHs originate from different sources. PMF provides physically realistic result by using the component digits while reducing matrix dimensionality simultaneously to maximum viable (Saraga et al., 2010). Number of elements in the PMF solution can be determined by examining the values by adding the square route of the difference between the real dataset and the PMF output (Q) concluded from the values contributing to sample (Reff, Eberly, & Bhave, 2007). PMF model could yield physically realistic results because of pointwise evaluation of uncertainty errors in the dataset and non-negativity constraints (Khairy & Lohmann, 2013).

Another multivariate statistical analysis is also used for source apportionment studies i-e principal component analysis. It extracts definite factors to analyse correlation among observed variables by reducing set of original variables (Jiang et al., 2009; Picasso, 2014). Principle components can be extracted from loadings of different factors by using orthogonal transformation method. After that, each principal component is evaluated and recognized by source markers/profiles. Furthermore, factor profiles are compared with available literature that have same characteristics of source profile. PCA is of great importance because the factor loading scores are higher score values and type of correlation coefficient (Agarwal, Khillare, Shridhar, & Ray, 2009). Commonly, two principal components were used for analysis of PAHs sources in environment. However, multiple approaches can be used in combination for identification of sources such as (Kwon & Choi, 2014) as well as (Zeng, Jeppesen, Gu, Mao, & Chen, 2018) used diagnostic ratios along with PMF receptor model in their studies.

3. Composition of PAHs in Environment

Content of PAHs in environment mostly depend upon the PAH transport modes, level of industrialization and distance from source. However, spatial distribution of PAHs wholly relies on their basic characteristics i-e molecular weight, phases, and number of rings. Polycyclic aromatic hydrocarbons enter the air in gaseous as well as particulate form when combustion of pyrogenic and petrogenic sources occurs. They deposit on soil by wet or dry deposition, where many air soil exchange processes take place according to their physiochemical characteristics and environmental conditions. Moreover, soil work as a basic sink for PAHs, re-volatilization and re-deposition of these contaminants takes place simultaneously in soil during summer and winter seasons (Ren, Wang, Wang, Gong, & Yao, n.d.).

Runoff and infiltration transport these pollutants into deeper soil and water estuaries. In water, suspended particles absorbed these pollutants and entrapping them permanently due to lack of air-soil exchange processes (Bozlaker, Muezzinoglu, & Odabasi, 2008). Sediments act as permanent sink as well as secondary origin of polycyclic aromatic hydrocarbons. In this way, fate, and transport of PAHs in environment and food chains and ultimately to mammalian organisms takes place.

3.1 Concentration in Atmosphere

In previous decade, polycyclic aromatic hydrocarbons due to its characteristics and long-range transport in atmosphere have drawn constant attention around the globe (Albuquerque, Coutinho, & Borrego, 2016; Liu, Xue, Zhu, & Jia, 2017; J. Zhang et al., 2016). PAHs when emitted into the air, went through multiple processes, such as particle size distribution, gas-particulate partitioning, reaction, deposition, and degradation (Cheruiyot et al., 2015). In atmosphere, they mostly found in gas and particulate phase. Due to hydrophobicity, atmospheric particulate-soaked low molecular element more rapidly than high molecular weight element. PAHs concentration in atmosphere can be different because of different vapour pressure of different PAH compounds. Furthermore, it also depends upon the nearby sources, population, wind direction and weather variations. Generally, different studies documented greater composition of gas phase availability in atmosphere than particulate phase (Lai, Tsai, Chen, & Chang-Chien, 2017) (Gregoris et al., 2014) (Wu, Yang, Wang, Yuan, & Hong, 2014). In addition to this, it is reported that PAHs concentration in winter is much higher than that of other seasons (Lv et al., 2016; Tomaz et al., 2016; Wang et al., 2016; Xia et al., 2013). These number of factors are responsible for such as increased heating demand (Birgul & Tasdemir, 2015), lower photothermo and chemical oxidations, and lower combustion of vehicles (Arhami et al., 2010). Furthermore, decrease in PAHs concentration observed with the rise of wind speed and temperature because wind speed could enhance concentration and suspension of air mass (Masih, Singhyi, Taneja, Kumar, & Masih, 2012). Elorduy, Elcoroaristizabal, Durana, García, & Alonso (2016) reported atmospheric pressure, wind speed and temperature as major parameters for PAHs concentration. Hence, its shows that atmosphere is dominated by gas phase particles contain low molecular PAHs and high concentration in winter season than in summer.

Air quality standard has established by many countries to deal with atmospheric PAHs level but guidelines for emission from immobile sources have not been established yet. To set up the guidelines regarding presence of PAH in atmosphere BaP was utilized as a referral (Garrido, Jiménez-Guerrero, & Ratola, 2014). Limited concentration of PAH in atmosphere can be calculated by the addition of the mass concentration of individual congeners multiplied by their TEF values and expressed in Nano gram (ng). Maximum value of 1 ng BaP-TEQ m⁻³ for annual presence of PAHs has come into force in



2013 by European Commission, Air Quality Standard. China and India also established that limit as standard value in 2012 as well as Chinese government also settled 24 hours average exposure limit of 2.5 ng BaP-TEQ m⁻³. In addition, France established limit for annual exposure of 0.9 ng BaP-TEQ m⁻³, applies to 41 PAHs parent along with their derivatives.

3.2 Concentration of PAHs in Soil

Terrestrial soil act as a primary reservoir for PAHs (Wang et al., 2015). They deposited into soil through atmospheric precipitation i-e wet and dry deposition (Demircioglu, Sofuoglu, & Odabasi, 2011; Westgate & Wania, 2013). In soil, PAHs absorption by organic matter and adsorption in black carbon plays important role in the dispersal of polycyclic aromatic hydrocarbons. PAHs in soil went through variety of process based on their characteristics and multiple conditions of environment. Sorption and desorption are the most influential processes for bioavailability and mobility of individual PAH which depend upon the properties of PAH and soil. Due to sorption, PAHs may be detected in the greater depth of the soil. Bioavailability of PAHs depend upon the hydrophilicity and their binding to soil particles. The lower water-soluble a PAHs is, the lower is the uptake by earthworms (Van Brummelen, Verweij, Wedzinga, & Van Gestel, 1996) while uptake by plants is higher when more water-soluble a PAH is. Octanol—water partitioning Kow can determine sorption of PAHs in land because it belongs to the solubility of organic matter in water. The tendency for sorption to a particular soil increases as the Kow increases because of decrease in aqueous solubility.

Urbanization and industrialization are important characteristics for greater number of PAHs in soil. Less crop production and permanent change in soil can be the terrible impact of long-term contamination of soil. PAH level in remote regions is 2-20 fold lower than that of urban areas (Maisto, De Nicola, Iovieno, Prati, & Alfani, 2006; Wang et al., 2015). The span of developed areas and accumulation time are prominent factors for higher concentrations and proportion of high molecular PAHs in soils (Liu, Xia, Yang, Shen, & Liu, 2010). Carrero had documented that the soil near highways has higher concentration of PAHs (Carrero et al., 2013). In 1995 ASTDR has reported about the additional factors that contribute towards PAHs concentration in soil are soil type, altitude, humidity, pattern in soil and gap between the sources and sink. As the distance from point source increases PAH decreases. Order of PAHs concentration in different types of soil are contaminated sites > urban soil > permanent grassland > mineral soil under forest > arable soils. The chemistry between dissolved PAHs and soil, momentum of emission from source materials, and the configuration of the solution phase are responsible for the existence of PAHs in tainted soil. PAHs composition in air can also influence the PAH to soil flux. Once PAHs deposited into soil, they can be transferred to plants, to animals and then to humans by soil ingestion or through plants (Knoche et al., 1995). Volatilization helps to minimize the content of PAHs in soil particularly for low molecular weight compounds. PAHs with heavy molecular mass commonly binds with soil organic matter while low molecular weight compounds tend to evaporate. Air temperature, henry coefficient, wind speed and atmospheric turbulence can directly influence the volatilization rate while soil moisture has indirect influence (Ophoff, Stork, Veerkamp, & Führ, 1996; Sims & Overcash, 1983). In summer, it volatilizes into gaseous phase while in winter it precipitates again into soil. Gas Chromatography – Mass Spectrometry is the most frequently used method for identification of the particular polycyclic aromatic hydrocarbons in loam (Włóka & Smol, 2014). Table 4 demonstrates the worldwide composition of PAHs in various type of soil. In addition, it has been documented that the highest concentration of PAH found in urban areas or in high population regions.

3.3 Accumulation of PAHs in Water

Polycyclic aromatic hydrocarbons enter into water by many routes such as atmospheric deposition, gas-water exchange, run-off (Heintzman, Anderson, Carr, & McIntyre, 2015), oil spillage/ leakage, industrial effluents (Duodu et al., 2017; Kwon & Choi, 2014) and municipal sewage (Tongo, Ezemonye, & Akpeh, 2017). With increasing industrialization and urbanization, number of pollutants discharged into environmental waters that poses threat to ecosystem. PAHs due to their fat soluble and hydrophobic characteristics, it is very likely to dissolve in water, deposited into sediment or adsorbed into suspended solids (Liu et al., 2016). According to Kim, carcinogenicity of polycyclic aromatic hydrocarbons intensifies with the rise of molecular weight. Lightweight PAH are dominant in environmental waters that can pose threat to aquatic organisms (Kim et al., 2013). Composition, agglomeration and grit of suspended sediment, biological cycle, precipitation, salinity, temperature, and water discharge are influential factors for PAHs concentration in water estuaries. The more phytoplankton available in water the less will be the concentration of PAHs. PAHs load in the base is associated with mass of water while in upper layer it is correlated with runoff, deposition, and air water exchange. Seasonal variation is another factor that influences the collection of PAHs in natural waters. World health organisation has established maximum level i-e 50 ng. l⁻¹ for surface water in 1997. In 1994, Environment Canada has also established maximum limit of 10 µg. l⁻¹ for groundwater. These values play their part in detection of these pollutants in different estuaries around the word. Table 4 shows the concentration of PAHs in water estuaries of different countries. However, highest concentration has been found in Colombia whereas the lowest found in India.

3.4 Concentration of PAHs in Sediments

Polycyclic aromatic hydrocarbon enter into sediment by numerous mechanisms such as, pyrogenic origin (fossil fuel combustion), petro genic origin (slow maturation of organic matter) and diagenic origin (biogenic precursors) (Dudhagara et al., 2016; King, Readman, & Zhou, 2004; Sun et al., 2016). Sediment deposition follows the similar processes that govern soil deposition. In water, transport of PAHs in multiple phases vary according to their source and physiochemical properties. Distribution variability of PAHs in sediment can be described by distribution coefficient (Kd) which is the measure of the content fraction of PAH in solid and dissolved phase. Distribution coefficient, Koc is also helpful to measure PAHs availability in sediments because of organic matter presence, which is the ratio of Kd and foc (sediment fraction organic carbon). Furthermore, compounds with high log Kow i-e greater than five most likely detected in sediments while compounds with low log Kow i-e is less than three probably observed in water. Compounds having log Kow between 3 and 5 depend upon contamination degree. There are number of factors that control dispersion of PAHs in sediments including, texture, bio-chemical characteristics of sediments, environmental conditions and geochemical attributes of pollutants (Chakraborty, Ramteke, & Chakraborty, 2015). Eggleton and Thomas (2004), and Tang et al. (2015) have reported that mobilization and remobilization takes place due anthropogenic as well as natural activities (waves and storms), change in sediment chemistry and resuspension that can enhance PAHs solubility in aquatic systems. Transfer, degradation and sequestration are helpful processes to define the providence of PAHs in sediments (Pierzynski, Vance, & Sims, 2005). Generally, PAHs desorb quickly from sediments but with increasing time, rate of desorption decreases. PAHs can be absorbed into organic matter and diffuse into Nano pore (Reid, Jones, & Semple, 2000). Organic matter content and concentration of contaminants can affect the rate of sequestration. Hence, greater the PAHs sequestration bio accessibility/bioavailability of contaminants decreases significantly. Table 5 describes the agglomeration of PAHs in worldwide sediments.

4. Occurrence of PAHs in Food Chains

Food is the first and foremost exposure pathway of PAHs. PAHs occurrence in food chains is due to food production and preparation techniques. Food production in contaminated soil and air / uptake by plants and marine life or fish lives in contaminated waters are the major sources of food chains deterioration. Following PAHs availability in environment, it has been also found in raw food. The phenomenon of PAHs in foodstuff relies on its solubility in organic solvents and water which not only govern the transfer and distribution in environmental compartments but also the uptake and accumulation by living organisms. PAHs accumulate in lipid tissue of plants and animals. PAHs perhaps settled onto or absorbed by plants, might be washed of by precipitation, volatilize or remain deposited into soil due to degradation (Eisler, 1987). Plants uptake PAHs from soil through roots and take them to other parts of plants. Concentration, water solubility and their physicochemical state and soil type govern their uptake by plants. Vegetables and fruits having glutinous surface can adsorbed low molecular PAHs. On the contrary, PAHs are generally lower in tissues than the plant surface (outer leaves, peel), also greater the water content the less will be the accumulation in tissues.

Seafood most likely can be subject to PAH available in water and sediments, while the concentration wholly depends upon the metabolizing ability of marine organism. Mussels and oysters have ability to filter out greater mass of water so they cannot effectively metabolize all PAHs and cumulate PAHs with high molecular mass (Afolabi, Adesulu, & Oke, 1983; Murray, 1998). The maximum quantity of PAHs observed in fish is lower than that of mollusks, fish has oxidation ability also it metabolizes PAHs into water-soluble compounds, excreted by living organism. Ramalhosa has documented that only four low molecular PAHs has been found in several fish species but none had high ring PAH (Ramalhosa et al., 2012). The mussels from urban area showed excessive number of PAHs than rural areas. Furthermore, PAHs concentration in smoked fish depend upon the temperature and exposure. However, direct exposure of fish or meat smoked at high temperature possess high PAHs than indirect smoking methods, but hot smoking also accounted for outrageous PAH levels unlike cold smoking (Roda et al., 1999). It has been delineated that regardless the setting of parameters, all smoking processes led to higher concentration of low ring PAHs.

Moreover, food processing (drying or smoking) or cooking at high temperature (frying, grilling and roasting) can induce PAHs in food (Davidek, 1995; Fretheim, 1983; Mottier, Parisod, & Turesky, 2000). The extent of PAHs in foodstuff can vary depending upon the factors i-e duration, fuel type, loss of fat, range from the cooking source, food process type (grilling, frying, roasting, and smoking) and cooking. Drying of seeds is the major cause of edible oil contamination with PAHs. Majority of PAHs i-e 95% noticed in cooking oil are low molecular PAHs while sunflower and rapeseed oil contain increased amount of high molecular weight PAHs i-e 50% and 30% respectively. Several preservation techniques under inappropriate conditions also engendered the contamination of food. Drying and roasting of tealeaves, coffee beans and cocoa beans may also increase the content of PAHs. It has been documented that food Processing and preservation/ packaging techniques led to even high percentage of PAHs in foodstuffs.

Consumption of foodstuffs plays major in supply of pollutants(Falcó, Bocio, Llobet, & Domingo, 2005). Vegetables, cereals, oils and barbequed meat are the common components of health risk (Grova, Rychen, Monteau, Le Bizec, & Feidt, 2006). PAHs in food chains are utmost components leading to skin and lung cancer (Kameda et al., 2005; Lee & Shim, 2007; Zhang et al., 2009). They enhance urinary elimination rates and causes non-genotoxic diseases i-e cardiovascular disorders and diabetes



mellitus (DM)(Buckley & Lioy, 1992; Burstyn et al., 2005; Grant, 2009; Hu, Kan, Kearney, & Xu, 2015). Xia has documented that PAHs have incremental lifetime cancer risks (Xia et al., 2013). The potential of cancer and health hazard of PAHs usually measured by using toxic equivalency factors (TEFs^{carc}) by calculating the approximation of BAP equivalent doses (Qiao, Wang, Huang, Wang, & Wang, 2006).

Various organizations have estimated the Incidence and noxiousness of PAHs such as the United States Environmental Protection Agency (USEPA), the International Agency for Research on Cancer (IACR), the Scientific Committee on Food (SCF), the Joint FAO/WHO Expert Committee on Food Additives (JECFA), the International Programme on Chemical Safety (IPCS), and the European Food Safety Authority (EFSA). In 2008, the scientific community on foods (SCF) confirmed the mutagenicity / Geno toxicity of 15 PAHs. It includes benz[a]anthracene, benzo[b]fluoranthene, benzo[j]fluoranthene, benzo[k] fluoranthene, benzo[ghi]perylene, benzo[a]pyrene, chrysene, cyclopenta[cd] pyrene, dibenzo[a,h]anthracene, dibenzo[a,e]pyrene, dibenzo[a,h]pyrene, dibenzo[a,i]pyrene, dibenzo[a,l]pyrene, indeno[1,2,3-cd]pyrene, and 5-methylchrysene. Also declared that the BaP was not only the marker for distribution of PAHs in food. Therefore, a combination of PAHs such as PAH2 (BAP+CHY), PAH4 (PAH2+BAA+BBF), and PAH8 (PAH4+BKF, BPY, DBA, and INP) were instigated by European Food Safety Authority in 2008. It also concluded that integration of PAH4 would provide more accurate value than the fusion of PAH8. For the maximal quantity of PAH4 in edibles matrices, European Commission has also established the regulation guidelines. In 2006, maximum level of PAHs in food chain was introduced by EU commission (Commission Regulation (EC) No. 1881/2006) was then updated by commission regulation in 2011 (EU No 835/2011). Table 6 documented different studies, which shows PAHs contamination in different type of foods. The maximum concentration has been found in smoked food products that causes lung cancer in human.

5. Eco Toxicological Effects of PAHs

Most of the PAHs released into environment by anthropogenic activities including, coal processing, partial ignition of organic matter (e.g., wood, and fossil fuels), from cigarettes, motor vehicle exhaust. Forest fires, volcanoes and hydrothermal processes are some natural sources of PAHs. In addition, Vegetation in industrial/urban area is more contaminated than rural areas. Moreover, contamination of food chains depend upon the Physiochemical properties of PAHs including, volatility, chemical reactivity, biological and abiotic degradation and water and fats/oils dissolvability (Edwards, 1983; Nielsen, Jørgensen, Larsen, & Poulsen, 1996). Photo-oxidation and metabolism also affects the toxicity of PAHs. Unlikely, PAHs in soil exert toxic impact on terrestrial invertebrates except when soil is contaminated. To birds and aquatic life, they have average to inflated poisonousness. PAHs exposures can pose adverse impact on organism include reproduction, tumors, development, and immunity. PAH – induced phytotoxic effect are rare. As per ASTDR research in 2010, some plants contain substances from which certain act as a shield and protect against effect, while other synthesize the PAH that play as development hormones. Due to persistent and bioaccumulation, the number of PAHs in environment is much lower than that of fish and shellfish. Moreover, terrestrial invertebrates also showed the bioaccumulation while PAHs metamorphosis is adequate to hinder bio magnification (Borosky, 1999; Inomata et al., 2012).

6. Human Exposure to PAHs

Humans quiet possibly are subjected to PAHs through air, water, soil, sediments, and food chains. In both employment and non-employment settings, the routes of exposure are respiration, ingestion and cutaneous. In occupational settings, workers jeopardize by PAHs by breathing fumes from industrial exhaust, mining area, smelting, and refining. Total uptake dose probably affected by multiple vulnerable route i-e sum of two or more route simultaneously (e.g., skin and inhalation exposures from polluted air)(Wu, Zhu, Luo, & Wang, 2016). In non-occupational setting, food consumption is the important route of PAHs for non-smoking person (Xia et al., 2010). Smoking and burning of coal and wood are other non-workplace source of human exposure.

6.1 Acute/ Short Term Impact of PAHs

The health risk relies on the number of factors such as, route and length of exposure, amount of pollutant one is subjected and toxicity. Several other subjective factors also responsible are age and health status of the person. Although the ability of PAHs to engender acute impact is unforeseeable. Several symptoms such as allergies in eyes, nausea, diarrhoea, and confusion has been resulted by occupational exposure to high quantity of PAHs contamination (Bølling et al., 2009; Collins, Brown, Alexeeff, & Salmon, 1998; Unwin, Cocker, Scobbie, & Chambers, 2006) while the component responsible for effect is not known either it can be PAHs or any other component attached with it. The mixture of PAHs causes' inflammation and skin irritation. Anthracene, Benzo pyrene and naphthalene are known to be skin irritants from which two were documented as skin sensitizer which causes skin allergies in human and animal (Kim et al., 2013), IPCS 2010. Exposure levels can be estimated by measuring minimal Risk Levels or MRLs. It can be determined by having specific data such as source of PAHs exposure, targeted organ, and health effect. To calculate acute, intermediate, and chronic duration exposures for inhalation and oral routes it is possible to drive maximum detection level. Several techniques have attained by EPA in 1990 to measure these levels but uncertainties lie in these techniques. ASTDR documented that uncertainty lies in the procedure to obtain less than lifetime MRLs.

6.2 Chronic/ Long Term Impacts

Long-term health hazards include lever damage like jaundice, respiratory problems such as asthma symptoms and lungs abnormalities, opacity, weaken immune system and skin inflammation. According to Collins, naphthalene can foster red cells breakdown if inhaled or ingested in greater amount. Furthermore, occurrence of harmful effect depend upon the way of exposure (Collins et al., 1998).

6.2.1 Carcinogenicity

Besides the reality that un-metabolized PAHs can have dangerous impacts, a noteworthy concern is the capacity of their responsive metabolites to tie to cell proteins and DNA, for example, dihydrodiols and epoxides. Cell damage and biochemical disruption lead to mutations, tumors, developmental malformations and cancer (Ramesh, Archibong, & Niaz, 2010). Some studies on occupational workers reported that the mixture of PAHs is carcinogenic to human (Grimmer et al., 1988). In addition to this, these studies not only have shown the higher risk of predominantly skin and lung cancer but also bladder and gastrointestinal cancers. Nonetheless, it is not clear whether the PAHs are major cause or workers were exposed to other cancer-causing agent (e.g. aromatic amines) simultaneously (Bach,



Kelley, Tate, & McCrory, 2003; Grimmer et al., 1988). Table 8 shows several PAHs that is has been identified as carcinogenic by different agencies.

According to USEPA research centre investigations in 2008, that animals are exposed to large amount of several PAHs on significant lots causes lung cancer due to respiration, stomach cancer through ingesting of PAHs in sustenance and skin cancer because of dermal contact. BaP is the significantly recognized PAH to cause malignant growth in animals, and remarkable first chemical as cancercausing agent have been found. Furthermore, it has been determined that PAHs can supress immune system reactions in gnawing mammal. Nevertheless, PAHs effect on immune system is not clear but it has been considered that immune suppression mechanism can instigate cancer and other infectious diseases.it has been investigated that Ingestion of food contaminated with PAHs was the prominent factor in Immune toxicology. In addition, it causes DNA adducts in lungs.

6.2.2 Impacts on Development

In laboratory experiment, the embryo toxic impact of PAHs i-e benzo (a) anthracene, benzo (a) pyrene and naphthalene have been observed on mice. Greater amount of benzo (a) pyrene ingestion during pregnancy resulted decrease in the body weight of offspring as well as birth defects while these result in human is not known. Nonetheless, the Centre for children's Environmental Health reports studies evaluated that the consequences of PAHs exposure throughout the pregnancy are premature delivery, heart malformation and low birth weight. Exposure of PAHs before birth can also cause childhood asthma, IQ problems at the age of three and behavioural problems when turn to six and eight years (Perera et al., 2012). Cancer in exposed babies could identified by the DNA damage in cord blood.

6.2.3 Genotoxicity

Genotoxicity plays vital role in both the carcinogenetic process and in certain forms of developmental toxicity. According to ASTDR report in 2010, majority of the PAHs do not consider as genotoxic, but they metabolized to the diol epoxides that react with DNA and instigate genotoxic damage. In laboratory, effects of PAHs on rodents and in visto tests using mammalian cell lines (including human) has been observed which demonstrated that PAHs undergo multiple metabolic transformation which became the origin to produce electrophilic derivatives that react with nucleophilic centre of macromolecules. In addition to this, different scholars have also studied about the mutagenicity mechanism. Resultantly to base pair substitutions, bulky adducts of PAH to DNA bases can prompt frameshift mutations, a variety of chromosomal alterations, deletions, strand breakage and S-phase arrest (Jung et al., 2013), IPCS 1998.

7. Conclusion

This paper systematically summarized the composition of polycyclic aromatic hydrocarbons in different compartments and food chains. It evaluated that higher concentration of PAHs were found in the air of Asian region with average and standard deviation of 1447.018 and 2046.341569. Pollution status follows descending order i-e Asian region> Africa> Middle East> Europe> America. For sediment, higher concentration found in Middle East region with average and SD i-e 116660 and 164982.154. Region wise pollution status in ascending order follow as; Asia< America< Africa< Europe< Middle East. Moreover, for urban soil, highest concentration was found in Poland ranged from 41-77143ng.g⁻¹ while for agricultural soil highest concentration was found at Slovakia varied from 2400- 17000ng.g⁻¹. In addition, overall concentration of PAHs in water estuaries of five regions

ranged from 0- 205398.76 ng. L⁻¹ with average 102699.38 and standard deviation 145238.956, respectively. Risk factor of exposure has also been analysed which results carcinogenic, mutagenic, genotoxic effects on human and animals. Apart from this, long time exposure has reported lifetime cancer risk. In addition to this, PAHs emission sources and behaviour were also traced which suggested that diverse sources were responsible for distribution of PAHs around the globe whereas food processing were major contributor of PAHs formation in food chains. However, as exposure involve compounds of PAHs therefore some gaps are still existing in terms of their availability and toxicity that needs further evaluation. Continuous assessment of environment and exposure on monthly basis should be encouraged. Awareness about PAHs exposure, effects and Proper methods of PAHs reduction should cover and taught to people around the globe. Moreover, Industries should evolve some strategies for minimizing PAHs content in exhaust gases and they should provide effective Personal Protective Equipment's to workers to reduce the amount of PAHs exposure.

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Diagnostic ratio	Pyrogenic	Petrogenic	Reference		
Anth/Anth+Phe	>0.1	<0.1	(Pies et al., 2008)		
BaA/BaA+Chr	0.33 or >0.5	<0.2 (Katsoyiannis, Terz Cai, 2007)			
Fla/Fla+Pyr	>0.4	<0.4	(Yunker et al., 2002)		
IcdP/IcdP + BghiP	>0.2	<0.2	(Tobiszewski, 2014)(Zhi et al., 2015)		
LMW/HMW	<1.0	> 1.0	(W. Zhang et al., 2008)		
Anth/178	≥0.1	<0.1	(Sofowote, Allan, & McCarry, 2010)		
BaA/228	>0.35	<0.2 (Yunker et al., 200			

Table 1: Diagnostic ratio for source apportionment

Low molecular weight LMW, High molecular weight HMW, (Adeniji, Okoh, & Okoh, 2018; Moyo, McCrindle, Mokgalaka, Myburgh, & Mujuru, 2013).

Table 2: Concentration of PAHs in atmosphere around the world

Region	Country	City	Pahs	Concentratio		Avera	Standard	Referenc
				n	n		Deviatio	e
				Max	Min		n	
Asia	China	Hefei	Σ16	4.92	71			(R. Hu et
								al.2017)
		Tumen River	Σ15	3.2 776				(Jin Et
		Area						Al.2012)

	Dalian	Σ6	4.32	112.2		(Y.
		20	4.32	112.2		Wang et
						al. 2019)
	Shanghai	Σ16	36.01	10.85		(X. Y.
	Shanghar	210	30.01	10.05		Wang Et
						Al. 2010)
India	Tamil Nandu	Σ16	5	47.5		(Sampath
		210				et al.
						2015)
	Delhi	Σ16	388	672		(H.
						Sharma,
						Jain, And
						Khan
						2017)
	Urban, Rural	Σ16	9.91	164		(WJ.
	and Background					Hohg et
	Area					al. 2016)
	West Coast	Σ16	0.71	2.99		(Gune Et
						Al. 2019)
Japan	Tokyo	Σ 36	2.37	9.34		(Saha et
						al. 2017)
	Kamihaya	Σ17	0.036	10.16		(Jadoon,
						Kondo,
						And
						Sakugaw
	Halian Daniel	D16	4.1	9.27		a 2015)
	Urban, Rural	Σ16	4.1	8.27		(Y. Hong et al.
	and Background					2015)
Korea	Area	Σ16	38.2	78.8		(YH.
Korea		210	30.2	70.0		Kim And
						Kim
						2015)
	Urban, Rural	Σ16	8.63	51.8		(Y. Hong
	and Background	210	0.05	21.0		et al.
	Area					2015)
Malaysia	Kuala Lumpur	Σ36	3.44	10.35		(Saha et
	r ··					al. 2017)
	Klang Vally		1.35	4.92		(Mohd
						Tahir et
						al. 2014)
	Bangi (Semi		1.66	5.23		
	Urban)	<u> </u>	<u> </u>	<u> </u>	 	
Pakistan	Gujranwala	Σ16	1375	2430		(Kamal
						et al.
						2016)
	Rawalpindi	Σ16	1613	2894	 	
	Lahore	Σ16	2351	2366	 	
	Twin Cities	Σ16	1630	2.893		(Hamid
	(Islamabad And					et al.
1	Rawalpindi)					2018)

	Vietnam	Urban, Rural and Background Area	Σ16	15.2	87			(Y. Hong et al. 2015)
		Hanoi	Σ36	4.06	28.77			(Saha Et Al. 2017)
Sum				0.036	2894	1447. 018	2046.341 569	111. 2011)
Africa	Egypt	Cairo	Σ16	0.03	567.1			(Shakour et al. 2011)
		Alexandria	Σ 44	330	1770			(Khairy And Lohmann 2013)
	Nigeria	Lagos	Σ 20	89.2	96.48			(Salaude en et al. 2017)
		Ogun	$\Sigma 20$	72.52	142.9 1			
	Sierra Leone	Westerin Part	Σ11	20.8	87.6			(E T Taylor et al. 2015)
		Waterloo and Tombo	Σ11	38	335			(Eldred Tunde Taylor And Nakai 2012)
		Free Town	Σ11	42.13	75.95 NG/ M			
	Uganda		Σ30	19.3	311			(Arinaitw e et al. 2012)
Sum				0.03	1770	885.0 15	1251.557 789	
Americ a	Brazil		Σ16	0.7	90			(Meire et al. 2019)
		Campo Grande	Σ15	8.94	62.5			(Re and Santiago- Silva 2005)
		Dourados	Σ16	0.375	8.407			(Re et al. 2015)
	Canada	Toronto	Σ16	0.27	51			(Melymu k et al. 2012)
		Hamilton, Ontraio	Σ16	10.2	83.7			(Anastas opoulos et al. 2012

	Chile	Temuco	Σ15	0	70			(Pozo Et
	Cinic	Temaco	213	U	70			Al. 2015)
		Nortern. Central and Sourhern	$\Sigma 4$	0	1.25			Shunthira singham et al. 2011)
	Mexico	Urban Atmosphere	Σ12	11.04	20.77			Mugica et al. 2010)
		Guadalajara	Σ14	0.65	19.62			(Murillo- Tovar et al. 2018)
Sum				0	90		63.63961 031	
Europe	Different (Σ15	1.772	2.243			(Carrido, Jimenez- Guerrero, And Ratola 2014)
	Germany	Dettenhausen	Σ21	0.4	121.4			Bari et al. 2010)
		Bechtoldsweiler	$\Sigma 21$	0.41	25.8			
			Σ12	1.9	5			(Schauer Niessner, And Poschl 2003)
	Spain		Σ18	2.71	366.9			(Ramirez Et Al. 2011)
		Basque Country	Σ16	0.85	9.86			(Oleagoit ia et al. 2019)
	Italy	Naples	Σ16	2	130			Caricchia , Chiavari ni, And Pezza 1999)
	Kurkey	Four Sites	Σ12	30	198			Birgul And Tasdemir 2015)
		Iskenderun	Σ16	21	314			(Falay et al. 2013)
		Kocaeli	Σ15	4.2	3842			(Cetin et al. 2017)
Sum				0.4	366.9	183.6 5	259.1546 353	
Middle East	Bahrain	33 Islands	Σ32	3.1	9.1			(Madany And

								Raveendr an 1992)
	Iran	Ahvaz	Σ16	0.5	25.5			(Nahmed din And Keshavar zi 2018)
	Kuwait	Kuwait City	Σ16	5	13			(Gevao et al. 2006)
	Qatar	Doha	Σ36	0.134	2.439			(Javed et el. 2019)
	Saudi Arabia	Riyadh	Σ16	0.13	515.7 6			(Bian et el. 2016)
Sum				0.13	515.7 6	257.9 45	364.6054 696	
Total				0	3842	1921	2716.704 2	

Table 3: Contents of PAHs in urban and agricultural soil in different regions

Country	PAHs	Compo	osition (ng. g ⁻¹)	Reference
		Min	Max	
Urban soil				
Bahrain	∑16	0.01	42.58	(Al-Haddad, Madany, & Abdullah, 1993)
Brazil	∑16	132.94	410.5	(Amato-Lourenco, Saiki, Saldiva, & Mauad, 2017)
China	∑16	91.5	46834.5	(S. Wu et al., 2017)
	∑15	42.8	28600	(Y. Wang et al., 2020)
	∑16	366	27,825	(L. Tang, Tang, Zhu, Zheng, & Miao, 2005)
	∑16	65.01	23603.05 μg/kg	(Yu, Li, Liu, & Ma, 2019)
China	∑16	371	2224	(Z. Wang, Liu, & Zhang, 2019)
Europe	∑50	6	88	(Vane et al., 2014)
	∑15	1487	51 822 μg/kg	(Morillo González et al., 2007)
	∑31	2.47	852mgkg	(A. W. Kim et al., 2019)
Florida	∑16	59	58640 μg/kg	(Y. Liu et al., 2019)
	∑16	43	30428µg/kg	
	∑16	797	7909 μg/kg	(Gao et al., 2019)
	∑16	1133	30691µg/kg	
	∑16	950	11451µg/kg	
	∑16	922	17698 μg/kg	
Germany	∑59	60	140	(Hindersmann & Achten, 2018)



Guatemala	∑17	460	3251 μg/kg	(Kasaraneni & Oyanedel- Craver, 2016)
India	Σ16	1.59	22.7 ng/g	(Gune et al., 2019)
Italy	$\frac{2}{\sum 16}$	0	4191 ng/g	(Qu et al., 2019)
Iran	<u>Σ</u> 16	148.4	721	(Khoshand, Tabiatnejad, Siddiqua, Kamalan, & Fathi, 2017)
Iraq	∑13	19	855	(Alawi & Azeez, 2016)
Kyrgyzstan	∑16	52	9439	(Li, Wu, Zhou, Sakiev, & Hofmann, 2020)
Lebanon	∑17	33.5	4062.9	(Soukarieh, El Hawari, El Husseini, Budzinski, & Jaber, 2018)
Malaysia	∑16	12,324	18,384	(Mohd Radzi, Abu Bakar, Emenike, & Abas, 2016)
Nepal	∑15	17.1	6219	(Pokhrel et al., 2018)
Pakistan	∑16	62.79	1080	(Riaz et al., 2019)
Poland	∑17	41	77,143	(Bojakowska, Tomassi- Morawiec, & Markowski, 2018)
Portugal		6	73395 µg/kg	(Reis et al., 2016)
Saudi Arabia	∑6	30.5	1016.6	(EL-Saeid & Sapp, 2016)
Turkey	∑15	835	1141	(Yurdakul, Çelik, Çelen, Öztürk, & Cetin, 2019)
	∑16	89	43481	(Falay et al., 2013)
Agriculture soil				
Brazil	∑21	16	38µg/kg	(Krauss et al., 2005)
China	∑16	12.9	2271.03	(Ding et al., 2018)
	∑16	491.65	1007.73	(Chen, Zhang, Zhang, Liu, & Zhou, 2018)
	∑16	0	27,580 ng/g,	(J. Sun et al., 2018)
Germany	∑29	271	2407	(Bandowe et al., 2019)
Guatemala	∑17	350	2087 μg/kg	(Kasaraneni & Oyanedel- Craver, 2016)
India	∑16	830	3880 μg/kg	(Agarwal et al., 2009)
Japan	∑21	52.9	2180 μg/kg	(Honda, Mizukami, Ueda, Hamada, & Seike, 2007)
Korea	∑15	38.54	4826.63 ng/g	(L. Kim et al., 2019)
Mexico	∑16	11.43	35.77	(Ortiz et al., 2012)
Moscow	∑21	59	1350	(Wilcke, Krauss, Safronov, Fokin, & Kaupenjohann, 2005)
New York	∑16	0.12	149.5	(Marquez-Bravo et al., 2016)
Norway	∑16	<0.01	2.6 mg/kg	(Jensen, Reimann, Finne, Ottesen, & Arnoldussen, 2007)

Poland	∑16	96	4348 μg/kg	(Klimkowicz-Pawlas,
	_			Smreczak, & Ukalska-Jaruga,
				2017)
	$\sum 8$	30	4108 μg/kg	(Maliszewska-Kordybach,
				Smreczak, & Klimkowicz-
				Pawlas, 2009)
	Σ16	80	7264 μg/kg	(Maliszewska-Kordybach,
				Smreczak, Klimkowicz-
				Pawlas, & Terelak, 2008)
Slovakia	∑29	2400	17,000	(Bandowe, Bigalke, Kobza, &
	_			Wilcke, 2018)

Table 4: Concentration of PAHs in Water Estuaries around the World

Region	Water Estuary	PAHs	Concentra	ation (ng. L-1)	Reference
			Min	Max	
Asia	Langkawi Island	Σ18	1300	26000	(Surif 2013)
	Bohai Bay	Σ16	48	607	(Tong et al. 2019)
	Chenab River	Σ17	289	1290	(Farooq et al. 2011)
	Coastal areas	Σ16	679.4	12639.3	(Habibullah-Al-Mamun, Ahmed, and Masunaga 2018)
	Dongjin River	Σ15	0	53.9	(Son et al. 2014)
	Ganges River	Σ16	0.05	65.9	(B. M. Sharma et al. 2018)
	Gomti River	Σ16	60	84,210	(Malik et al. 2011)
	Guanlan River	Σ16	121.8	8371.7	(Liang et al. 2019)
	Huaihe River	Σ15	79.94	421.07	(M. Liu et al. 2016)
	Northeastern	Σ16	122.7	639.8	(Yu Liu et al. 2013)
	Northwestern Japan Sea	Σ13	7.4	10.2	(Chizhova et al. 2013)
	River Brahmaputra and River Ganga	Σ16	0	31	(Paromita Chakraborty et al. 2014)
	Soan River	Σ17	61	207	(Aziz et al. 2014)
	southeastern Japan Sea	Σ16	6.38	13.81	(Hayakawa et al. 2016)
	Three Gorges Reservoir	Σ15	130.8	227.5	(Zhu et al. 2015)
	Tumen river area	Σ16	12.9	383	(Jin et al. 2012)
	Wenzhou	Σ15	910	1520	(Jianwang Li et al. 2010)
	Yellow River estuary	Σ16	11.84	393.12	(Jing Li, Li, and Liu 2017)
	Yeongil bay	Σ16	3	33	(H. B. Moon et al. 2001)
	Yinma River Basin	Σ16	175	325	(C. Sun et al. 2017)
Range			0	84210	
Average			42105		

S. D			59545.4		
			62		
Africa	River nile	Σ16	235.92	10367.6	(Haiba 2019)
	Suez Gulf	Σ16	13.352	205398.76	(Farid, Mahmoud, and Ahmed 2015)
	Alexandria coasts	Σ15	9.89	677.25	(Shreadah et al. 2013)
	Lake Victoria	Σ16	3.32	55.8	(Kwach and Lalah 2009)
	Ngong River	Σ16	2.69	14.22	(Kiage 2015)
	Nairobi River	Σ16	5.19	9.14	\ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \ \
	Atlas Cove	Σ11	46	507	(Olayinka et al. 2018)
	Ogbese river	Σ13	6250	13930	(Ololade et al. 2017)
	Buffalo River Estuary	∑16	14.91	206	(A O Adeniji, Okoh, and Okoh 2019)
	Msunduzi River	∑7	12.5	1799	(Munyengabe, Mambanda, and Moodley 2017)
	Mvudi and Nzhelele Rivers	Σ16	13.174	26.382 mg. L ⁻¹	(Edokpayi et al. 2016)
Range			2.69	205398.76	
Average			102700.		
_			73		
S. D			145236. 954		
America	Japaratuba River	Σ16	4	119	(Santos et al. 2018)
	Doce and Piracicaba Rivers	Σ16	156.2	1478.9	(da Costa Lima et al. 2015)
	Cauca River	Σ16	52.1	4476.5	(Sarria-Villa et al. 2016)
	Almendares River	Σ14	836	15811	(Santana et al. 2015)
	Gulf of Mexico	∑44	1.4	73	(Y. Hong et al. 2015)
	Narragansett Bay	∑22	3.6	340	(Zhao et al. 2018)
	Mississippi River	Σ18	77	430	(S Mitra and Bianchi 2003)
	Estero de Urias		9	347	(Jaward et al. 2012)
Range			1.4	15811	,
Average			7906.2		
S. D			11179.0 754		
Europe	Inland lakes	Σ17	14	360	(Yao et al. 2016)
•	Czech Rivers	Σ16	5.2	173.9	(Blahova et al. 2014)
	Gulf of Finland	Σ16	150	1280	(Petrova et al. 2009)
	Seawater around England and wales	Σ16	1	24821	(Law et al. 1997)
	Somme River	Σ16	513	831	(Net et al. 2014)
	Seine river basin	Σ 14	1.09	1659	(Motelay-Massei et al. 2007)

	Danube River and its tributaries	Σ17	67	96	(Nagy, Szabó, and Vass 2014)
	Raba River	Σ17	41	437	(Nagy, Szabó, and Vass 2013)
			23.9	72	
	Tiber River Sarno River	Σ6 Σ16	23.9		(Patrolecco et al. 2010)
D	Sarno River	210		2,670.40	(Montuori and Triassi 2012)
Range			1	24821	
Average			12411		
S. D			17550.3 903		
Middle East	Kor River	Σ16	5.02	11.88	(Kafilzadeh, Shiva, and Malekpour 2011)
	Tehran drinking water	Σ16	32.45	733.1	(Karyab et al. 2013)
	Al-Kahlaa River	Σ19	0.739	1.974	(Al-Saad n.d.)
		sea water-soluble fractions,Kuwait		2176	(Saeed and Al-Mutairi 2000)
	Chabahr Bay, Oı	nan sea	0.04	59.6	(Agah et al. 2017)
	West-Northern coasts of the Persian Gulf	Σ16	65	1059	(Aagh et al. 2016)
	coastal sea water	Σ16	0.8	18.34 μg. L ⁻¹	(Sinaei and Mashinchian 2014)
	Northern part of Hormuz strait	Σ16	3.12	5.88	(Bastami et al. 2013)
Range			0.04	2176	
Average			1088.02		
Standard 1	Deviation		2176		
Total					
Range			0	205398.76	
Average			102699. 38		
S. D	1		145238. 856		

S. D= Standard Deviation

Table 5: PAHs concentration in sediment of five regions

Region	Country	PAHs	Concentration (ng. g ⁻¹) Min Max		Reference
Asia	Bangladesh	∑19	208.4	4,687.90	(Zuloaga et al. 2013)
	China	∑16	89.1	749	(Yu Liu et al. 2013)
	Gulf of Thailand	∑16	2.6	78.1	(L. Hu et al. 2017)
	India	Σ16	3.3	630	(Soumita Mitra et al. 2019)

	Indonesia	Σ16	3555	9947	(La Nafie et al. 2016)
	Japan	Σ8	21	1447	(Onozato, Nishigaki, and Okoshi 2016)
	Korea	∑16	8.8	18 500	(Yim, Hong, and Shim 2007)
	Malaysia	∑16	918.1	994.02	(Sany et al. 2014)
	Pakistan	∑16	14.54	437.43	(Riaz et al. 2019)
		∑16	120.3	719	(Kahkashan et al. 2019)
	Southern China	∑16	1639	8766	(J. Chang et al. 2018)
	Taiwan	∑16	2.41	218.54	(T. Li et al. 2016)
	Vietnam	∑16	49	505	(Babut et al. 2019)
Range			2.41	9947	
Average			4974.71		
S. D			7031.89		
Africa	Congo	∑16	22.6	1011.9 μg. kg ⁻¹	(Kilunga et al. 2017)
	Durban	∑16	36	6800	(Vogt, Pieters, and Newman 2018)
	Egypt	∑16	0.74	456.91	(Salem et al. 2014)
	Kenya	Σ16	0.04	31.95	(Kwach and Lalah 2009)
	Limpopo	Σ6	111.6	61 764 µg.kg ¹	(Nekhavhambe, Van Ree, and Fatoki 2014)
	Nigeria	∑13	48	117	(Oyo-Ita et al. 2016)
	Republic of Djibouti	∑16	2.65	3760.11	(mahdi ahmed et al. 2017)
	South Africa	∑16	1107	22,310	(A O Adeniji, Okoh, and Okoh 2019)
	Tanzania	∑16	78	25,000	(Gaspare et al. 2009)
	Togo	∑21	4	257	(Gnandi et al. 2011)
Range			0.74	25,000	
Average			12,500		
S. D			17,677		
America	Alaska	Σ16	1700	2,800	(Apeti and Hartwell 2016)
	Argentina	$\frac{-}{\sum 18}$	15	10,260	(Arias et al. 2010)
	Brazil	$\frac{-}{\sum 16}$	39	2350	(Machado et al. 2014)
	Canada	<u>Σ</u> 16	0.037	336.77µg. kg ⁻¹	(Davis et al. 2018)
	California	∑36	622	16,663	(Neira et al. 2017)
	Colombia	∑16	212.3	1582.7	(Sarria-Villa et al. 2016)
	Jamaica	∑8	0.97	358	(Jaffe et al. 2003)
	Maxico	<u>Σ</u> 17	95	1,482	(Ontiveros-Cuadras et al. 2019)
	Northern Gulf of Mexico	<u>∑</u> 16	100	856	(Z. Wang et al. 2014)
	Puerto Rico	Σ16	40.4	1912	(Aldarondo-Torres et al. 2010)

	USA	∑14	213	1291	(Huang, Chernyak, and Batterman 2014)
	Virgin Islans	∑16	2.94	199.08	(Whitall, Pait, and Hartwell 2015)
Range			0.037	1912	,
Average			956.019		
S. D			1351.96		
Europe	East Antarctica	∑16	12.95	30.93	(Xue et al. 2016)
	Arctic	∑12	37.3	1973	(Pouch, Zaborska, and Pazdro 2017)
	France	∑16	0.3	34.11	(Wafo et al. 2017)
	Hungary	∑17	35.2	288.3	(Nagy, Szabó, and Vass 2014)
	Italy	∑6	157.8	271.6	(Patrolecco et al. 2010)
	Poland	∑17	41	33,158	(Bojakowska, Tomassi- Morawiec, and Markowski 2018)
	Romania	∑16	24.8	575.6	(Barhoumi et al. 2019)
	Slovakia	∑15	7910	29,538	(Hiller et al. 2009)
	Spain	∑16	4	4286	(Hijosa-Valsero et al. 2016)
	Warsaw	∑17	< 41	33158 µg.kg ⁻¹	(Bojakowska, Tomassi- Morawiec, and Markowski 2018)
Range			0.3	33158	2010)
Average			16579.2		
S. D			23446		
Middle East	Iran	∑7	2.09	31,930	(Keshavarzi et al. 2015)
	Iraq	∑19	4.906	35.479	(Salih, Abdul-Hussain, and Hamid n.d.)
	Kuwait	∑ 30	13	1290	(Lyons et al. 2015)
	Lebanon	∑16	1.22	731.93 μg. kg ⁻¹	(Manneh et al. 2016)
	Oman	∑16	0	93	(Agah et al. 2017)
	Persian Gulf	∑16	593.74	53393.86	(Bemanikharanagh et al. 2017
	Persian Gulf	∑16	15.28	75	(Aagh et al. 2016)
	Qatar	∑16	2.6	1025	(Soliman, Al-Ansari, and Wad 2014)
	Sharjah	∑13	1156	233,320	(Samara et al. 2016)
	UAE	∑16	0.6	9.4	(Tolosa et al. 2005)
	Yemen	∑16	2.2	604	(Mostafa et al. 2009)
Range			0	233,320	
Average			116660		
S. D			164982		
Total					

Range	0	233,320	
Average	116660		
Standard Deviation	164982		

Table 6: PAHs Contamination in different types of foods

S.no	Food type	Food sub- type	PAHs	Concentration	Reference
1	Cereal grains	Toasted bread	FLT, PYR, NAP, FLR, and PHN	0.71–1.66 μg.kg ⁻	(Al-Rashdan, Helaleh, Nisar, Ibtisam, & Al- Ballam, 2010)
		Pita bread	Σ8 PAHs	0.94μg.kg ⁻¹	(Alomirah et al., 2011)
2	Fruits	Strawberries and Apples	FLR, ANT, FLT, BAP, CHY, BAA, BKF, BBF, DBA, PYR, and INP	1.9–6.0 4.1– 15 μg.kg ⁻¹	(Wennrich, Popp, & Zeibig, 2002)
3	Barbequed meat	Pork (Charcoal grilling)	Σ15 PAHs	72.6 μg.kg ⁻¹	Food and Environmental Hygiene Department Hong Kong
		Duck (Gas grilling)	-	9.5 μg.kg ⁻¹	
		Beef (Electric grilling)	-	71.8 μg.kg ⁻¹	
4	Smoked Fish	Catfish (Malashya type)	Σ16 PAHs	92.6	(Okoronkwo, Eze, & Egedeuzu, 2019)
		Mangala		4.76	
		Arira		18.4	
		Prawn (oporo)		17.2	

5	Oil	Corn	Σ9 PAHs	6.40–76.08 μg. L ⁻¹	(Dost & İdeli, 2012)
		Butter	Σ16PAHs	18.1	(Martí-Cid, Llobet, Castell, & Domingo, 2008)
		Olive	-	32.25	
		Sesame	-	47.81	(Rose et al., 2007)
6	Beverages	Milk/ Dairy products	Σ13PAHs in term of BaP	1.3–8.2 μg. g ⁻¹	(Rawash et al., 2018)
		Coffee	Σ16 PAHs	0.52 and 1.8 μg. L^{-1}	(Orecchio & Papuzza, 2009)
		Alcohol drinks	-	536–2960 ngL ⁻¹	(Kamangar, Schantz, Abnet, Fagundes, & Dawsey, 2008)
7	Vegetables	Potatoes and Carrots Cabbage Celery Cucumber Egg Plant	Σ16 PAHs -	13μg.kg ¹ 23 μg.kg ⁻¹ 29 μg.kg ⁻¹ 31 μg.kg ⁻¹ 12 μg.kg ⁻¹ 9.79 μg.kg ⁻¹	(M. W. Ashraf, Taqvi, Solangi, & Qureshi, 2013) (Zhong & Wang, 2002)

Table 7: PAHs with their Toxic Equivalent Factor (TEFeq) and Genotoxicity

PAHs	No.of rings	TEF	Genotoxicity
Naphthalene (Nap)	2	0.001	-ve
Acenaphthylene (Acy)	3	0.001	-
Acenaphthene (Ace)	3	0.001	-
Fluorine (Flu)	3	0.001	-ve
Phenanthrene (Phe)	3	0.001	-
Anthracene (Ant)	3	0.01	-ve
Fluoranthene (Fla)	4	0.001	+ve
Pyrene (Pyr)	4	0.001	-

Benz[a] anthracene (BaA)	4	0.1	+ve
Chrysene (Chr)	4	0.01	+ve
Benzo[b] fluoranthene (BbF)	5	0.1	+ve
Benzo[k] fluoranthene (BkF)	5	0.1	+ve
Benzo[a]pyrene (BaP)	5	1	+ve
Indeno[1,2,3-cd] pyrene (IcdP)	6	0.1	+ve
Dibenz[a,h] anthracene (DahA)	5	1	+ve
Benzo[ghi]perylene (BghiP)	6	0.01	+ve

+ve= can damage DNA, -ve= no impact on DNA

World Health Organization (WHO) classification, (Nisbet & Lagoy, 1992)

Table 8: List of PAHs classified as carcinogenic to species

Agency	DHHS	IARC	EPA
Species	Animal	Human	Human
	BaA,	BaA	BaA
	BaP	BaP	BaP
	BbF	BbF	BbF
PAHs	BjF	BjF	BkF
	BkF	BkF	Chr
	DahA	IcdP	DahA
	IcdP		IcdP

benz[a] anthracene (BaA), Benzo[b] fluoranthene (BbF), Benzo[k] fluoranthene (BkF), benzo[j] fluoranthene (BjF), Benzo[a]pyrene (BaP), Dibenz[a,h] anthracene (DahA), Indeno[1,2,3-cd]pyrene (IcdP), Chrysene (Chr), Department of Health and Human Services (DHHS), The International Agency for Research on Cancer (IARC), Environmental Protection Agency (EPA).