International Journal of Chemistry and Chemical Processes E-ISSN 2545-5265 P-ISSN 2695-1916,
Vol 11. No. 2 2025 www.iiardjournals.org online version

Evaluation of the Toxicity Level of Polycyclic Aromatic Hydrocarbons in *Telfairia occidentalis* Planted in Some Ekpeye Communities, Rivers State, Nigeria

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DOI: 10.56201/ijccp.vol.11.no2.2025.pg1.10

Abstract

The bioaccumulation of polycyclic aromatic hydrocarbons in food crops is viewed as one of the most dangerous threats confronting humanity due to their persistence in the environment and their resultant mutagenic, carcinogenic and teratogenic effect on the health of human beings. The concentrations of sixteen priority polycyclic aromatic hydrocarbons (16 PAHs) were determined in Telfairia occidentalis leaves, where samples were collected from Ahoada, Okporowo, Idu-Ekpeye and Ubeta communities. The results of mutagenic equivalent (MEQ) of the study ranged between 5.01 x10^{-4} ppm (mg/kg) and 1.82 x 10^{-3}mg/kg (ppm) while that of toxic equivalent (TEQ) ranged between 1.88 x 10^{-4}ppm (mg/kg) and 3.71 x 10^{-3}mg/kg (ppm). The highest contributions to the MEQ and TEQ and their respective potentials (MP and CP) which is the capacity to cause modifications in human's DNA thus forming PAH-DNA adduct thereby resulting in mutations and cancer were made in the wet season by IcdP contributing 64% MP and 60 % CP and those of dry season were made by DahA contributing 61% MP and 90% CP. These dominant PAH compounds ie IcdP, DahA, BghiP, BbF, BaP, etc with diagnostic ratios indicated artisanal refineries and other minor contributory sources. Due to the effect of soot toxicants, individuals are to limit exposure while Governments need to take strict measures to reduce the activities of artisanal refineries.

1.0 INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a group of pollutants with highly stable chemical structures that can be originated by different sources (Thiombane et al., 2019). Although natural sources such as the petrogenic process can generate PAHs, anthropogenic sources, such as the incomplete combustion of biomass, coal, diesel, and other fossil fuels, the direct release of oil and its products, and their emissions from power generation plants and coke ovens, contribute more to their concentrations in the natural environment (Qi et al., 2020). Once released into the environment, PAHs are transported and distributed within different environmental media, such as soils and sediments, water bodies, and the atmosphere. Surface soils are considered to be large reservoirs and sinks for PAHs, due to the physicochemical properties of soils that enable the adsorption of PAH compounds to soil particles with high organic matter content (Soukarieh et al., 2018).

Various studies, including work done by the International Agency for Research on Cancer (IARC), have shown that PAHs are a group of toxic compounds with eco- toxic, genotoxic, mutagenic, and estrogenic effects (IARC, 2010; Mohit et al., 2019). Additionally, their adverse effects intensify with their accumulation and amplification in biological organisms (Zhang et al., 2022). In order to evaluate the cancer risks posed by the PAHs in soils and other environmental media, a health risk assessment model and an incremental lifetime cancer risk (ILCR) assessment model were proposed by the US Environmental Protection Agency (US EPA), two approaches which have been widely used by various authors (USEPA,1996; Suman et al., 2016). Considering the different carcinogenic potencies of different PAH compounds, the toxic equivalent quantity for Benzo(a) Pyrene (BaP-TEQ) calculated from the PAH concentrations and their toxic equivalent factors (TEFs) were used in the cancer risk assessment (Hussain & Hoque, 2015). However, the contributions of different PAH sources to the BaP-TEQ concentration and their associated cancer risks were rarely reported in the literature.

In order to apportion the BaP-TEQ concentration and cancer risks to different PAH sources, the potential PAH sources should first be identified, and their source contributions subsequently calculated using a different method. Finally, the contributions of different PAH sources to the BaP-TEQ concentration and cancer risks were obtained. In source identification, although diagnostic ratios were used by some authors, it cannot provide accurate information regarding the PAH sources (Shi et al., 2017). The principal component analysis (PCA) model can attribute portions of the total PAH concentration to different sources, but cannot apportion the BaP-TEQ concentration and cancer risks to each source (Suman et al., 2016). Studies suggested that the chemical mass balance (CMB) model and positive matrix factorization (PMF) model could apportion the PAH concentrations in every sampling station to different PAH sources (Buell et al., 2021). In a study by Li et al. (2014) used a CMB model to identify the PAH sources which contributed to the BaP-TEQ concentrations in the Liao River estuary wetland. However, the apportionment of BaP-TEQ concentrations and cancer risks to each source using the PMF model was rarely reported in the literature.

In the past decades, some authors have reported the PAH concentrations in soils in China (Qu et al., 2020) and other parts of the world (Shi et al., 2021), identified their sources using different methods (Qu et al., 2020), and quantitatively reported the health risks associated with exposure to PAHs in surface soils (Liu et al., 2018). However, the contributions of different PAH sources to the BaP-TEQ concentration have not been reported thus far. In this study, we intended to investigate the PAH pollution in different medias, for example the air and surface soil in Beijing and Tianjin. Unfortunately, there is no adequate PAH data in air samples, and the significant seasonal and spatio-temporal variability that controlled by the Carcinogenesis occur when adduct formation affects DNA repair mechanism thus resulting in accumulation of mutations in DNA (Kumar et al., 2014).

On the other hand, Oloyede et al. (2023), holds that with just one mutation in genome, there is the possibility of generating cancerous cell and consequently a threshold value cannot be assigned. The reactive epoxides and dihydrodiols PAH metabolites have the potential to bind to cellular proteins and DNA with toxic effects which result in biochemical disruption and cell damage can lead to mutations, tumours and cancer (Kumar et al., 2014). USEPA (2008), classified seven PAHs which are benz(a)anthracene, benzo(a)pyrene, benzo (b) fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene and indeno(1,2,3-cd) pyrene as potent carcinogens. Mutagenicity and carcinogenicity are clearly correlated according to Oloyede et al. (2023), who also reported that approximately 90% of known carcinogens are also mutagens. Also, there is increasing evidence to suggest that DNA damage (expressed mainly as mutations) is involved in the induction of many cancers (Bartsch & Tomatis, 1983), Benzo (a) pyrene (BaP) is marker PAH for cancer which is believed to be the most toxic PAH has been well characterized toxicologically. Toxic equivalency factors based on BaP are used for assessment of potential risk of many other PAH compounds. On this, mutagenic equivalent factor (MEF) and toxic equivalent factor (TEF) were developed/proposed by Durant et al. (1999) and Nisbet and LaGoy (1992) respectively. Calculation of BaP-MEQ and BaP-TEQ and to estimate the atmospheric air and swimming pool water PAH mutagenic and carcinogenic hazards in people for adverse health outcomes in both dry and wet seasons. This is as Jung (2010), calculated BaP-MEQ and BaP-TEQ to estimate residential indoor and outdoor PAH carcinogenic and mutagenic hazards in young inner city children, known to be at greater risk for adverse health consequences from exposure to air pollution.

Most studies conducted on swimming pools dwell on microbial contamination of the water while few look into physicochemical parameters for water quality studies. In line with this, Belonwu et al (2020), investigated physicochemical and microbial profiles of selected hotel swimming pools in Port Harcourt, Rivers State, Nigeria and found that physicochemical analysis of most of the studied pools and the microbial loads (ie total coliform, faecal coliform and Escherichia coli) of the studied pools were higher than WHO standard showing that most fall short of WHO standard for recreational activity. Attention was not paid to ubiquitous PAHs pollution particularly during this period of widespread of artisanal refineries in our localities coupled to the fact that the neighbourhood where these operations are carried out are generally under direct deposition of air pollutants from the atmosphere day and night. The aim of this research work was therefore to assess the mutagenicity and carcinogenicity of 16 PAHs in *Telfairia occidentalis* in both the wet and dry seasons.

2.0 Materials and Methods

2.1 Sampling

Soil samples were obtained using a stainless-steel auger at two depths: the lower zone (15–30 cm) and the upper surface zone (0–15 cm). Then the samples from the two depths were mixed to avoid the remarkable differences in the concentration of the LMW-PAHs in both depths due to their evaporation from the above (upper) soil parts. Each homogeneous sample was composed of five subsamples (one from the center and four from the corners of the area); it covered a plot of 100m x 100m, and subsamples were thoroughly mixed to form approximately 500–1000g. The soil samples were stored in a sterile dark container at 2^{O}C prior to extraction procedure. As PAHs are sensitive to light and to avoid degradation or contamination, samples of all kinds including soil, plant, and water were extracted within three days.

Furthermore, from each of the five communities, *Telfairia occidentalis* samples were randomly bought from five different markets, bulked, labeled, wrapped with aluminum foil and stored in ice cool boxes and conveyed to the laboratory for analysis.

2.2 Sample Pretreatment

Vegetable samples were rinsed using Milli-Q ultrapure water, and edible peels were removed to assess PAH concentration. Samples were homogenized and prepared using a high-speed blender, and the smooth purée of the vegetable was kept at -20^{o}C until the analysis was performed. The triplicated water samples were mixed, and one liter was filtrated using a vacuum filtration unit supplied with a membrane pare of 35–40 µm pore size. The filtered water sample was transferred to a SPE system obtained from Agilent Varian, Santa Clara (CA, USA) and equipped with an Oasis HLB cartridge and a vacuum pump from Waters Corporation (Milford, MA, USA). Meanwhile, soil samples were homogenized by sieving (<2 mm), and then stored in a solvent-cleaned glass jar at -20^{o}C until the performance of the instrumental analysis (Al-Nasir et al., 2022).

2.3 Extraction of PAHs

Soil and vegetable samples were prepared using a solid liquid extraction technique explained by Al-Nasir et al. (2022). Briefly, 50 g of soil or 100 g of vegetables wet homogenized samples was placed into a 500 mL Erlenmeyer flask. Samples were placed on a horizontal shaker overnight after adding 100 mL of acetone for the vegetables and 100 mL of a 2:1 acetone/water (v/v) mixture for the soil. The liquid–liquid partitioning was done by 1 h of shacking with 15 g NaCl and 100 mL cyclohexane. The organic layer of the analyte was placed into a 250 mL Erlenmeyer flask and dried using anhydrous sodium sulfate (15g). The extract was concentrated to 1 mL using the rotary evaporator and then dried using a gentle stream of N_2 gas. The residue was dissolved in 3 mL hexane and then dried again and redissolved in 5 mL of a mixture of 1:1 (v:v) ethyl acetate and cyclohexane. Finally, the cleanup was performed using a microfiltration syringe made of a polytetrafluoroethylene (PTFE). The filtrate was concentrated to 1 mL and passed through a deactivated alumina column (10 g) loaded with 2 g of anhydrous sodium sulfate to the top of the column. The sample was eluted with 50 mL of n-hexane and re-concentrated to 4 mL with a help of a gentle nitrogen stream. Finally, the eluate was divided into four portions of 1 mL vials and kept at $-20^{o}C$ for further GC/MS analysis.

2.4 Mutagenic and Carcinogenic Potentials of Polycyclic aromatic hydrocarbons (PAHs)

The toxic equivalent (TEQ) of the PAHs and mutagenic equivalent (MEQ) of high molecular weight PAHs were calculated by multiplying their individual toxic equivalent factor (TEF) and mutagenic equivalent factor (MEF) with the mean concentration of each PAH in the samples as shown in Eqs. (1) and (2) (Adeniji et al., 2018).

$$TEQ = \Sigma Cn \times TEFn \qquad (1)$$

$$MEQ = \Sigma Cn \times MEFn \qquad (2)$$

Where:

Cn = concentration of the individual PAH n in the mixture

TEFn = toxic equivalence factor for individual PAH n.

MEFn = mutagenic equivalent factor for individual PAH n.

In accordance with Błaszczyk et al. (2017), carcinogenic potential (CP) was calculated using the formula:

$$CPi = \frac{\left(\frac{PAHi}{BaP}\right) * TEFi}{\sum_{i}^{n} \left(\frac{PAHi}{BaP}\right) * TEFi} * 100\%$$

$$MPi = \frac{\left(\frac{PAHi}{BaP}\right) * MEFi}{\sum_{i}^{n} \left(\frac{PAHi}{BaP}\right) * MEFi} * 100\%$$

3. Results and Discussion

Table 1: Result of BaP-TEQ of eight carcinogenic PAHS in *Telfairia occidentalis* samples

8 PAHS	**TEF**	**Seasonal Bap-TEQ Levels**		**CP**	
		Dry	**Wet**	**Dry**	**Wet**
BaA	0.1	0.0089	0.0143	5.53	5.42
Chry	0.01	0.00048	0.0008	0.30	0.30
B(b)F	0.1	0.0025	0.0041	1.55	1.55
B(k)F	0.1	0.0045	0.0122	2.80	4.62
BaP	1.0	0.101	0.0154	62.73	5.83
DBA	1.0	0.041	0.071	25.47	26.89
IND	0.1	0.0026	0.0073	1.61	2.77
B(ghi)P	0.01	0.00019	0.00049	0.12	0.19
TOTAL		**0.161**	**0.264**		

Table 2: Result of BaP-MEQ of eight carcinogenic PAHS in *Telfairia occidentalis* samples

8 PAHS	**MEF**	**Seasonal Levels**	**Bap-MEQ**	**MP**	
		Dry	**Wet**	**Dry**	**Wet**
BaA	0.082	0.0073	0.0118	5.21	4.86
Chry	0.017	0.00082	0.0014	0.58	0.58
B(b)F	0.25	0.0064	0.0103	4.54	4.24
B(k)F	0.11	0.005	0.0134	3.55	5.51
BaP	1.0	0.101	0.154	71.63	63.37
DBA	0.29	0.0119	0.0206	8.44	8.48
IND	0.31	0.0081	0.0226	5.74	9.30
B(ghi)P	0.19	0.0036	0.0093	2.55	3.83
TOTAL		**0.140**	**0.243**		

Discussion

Polycyclic aromatic hydrocarbons (PAHs) are a class of organic compounds known for their environmental persistence and toxicological significance (Sun et al., 2020). These compounds, particularly the high molecular weight (HMW) PAHs, exhibit significant carcinogenic and mutagenic potentials. The contamination of food crops such as Telfairia occidentalis raises serious public health concerns due to dietary exposure, which is a primary route of PAH intake in humans. The assessment of PAH-related carcinogenic and mutagenic risks requires the application of indices such as the benzo[a]pyrene toxic equivalency quotient (BaP-TEQ) and the benzo[a]pyrene mutagenic equivalency quotient (BaP-MEQ). These indices provide insight into the relative toxicity and mutagenicity of PAHs compared to BaP, a well-established reference carcinogen and mutagen (IARC, 2020).

The evaluation of the carcinogenic potential of eight polycyclic aromatic hydrocarbons (PAHs) in Telfairia occidentalis using the benzo[a]pyrene toxic equivalency quotient (BaP-TEQ) reveals significant seasonal variations. The total BaP-TEQ in the wet season (0.264) was higher than in the dry season (0.161), indicating an increased potential carcinogenic risk associated with consuming the vegetable during the wet season. This trend aligns with previous studies showing that PAH accumulation in vegetables is influenced by seasonal factors, particularly rainfall, which enhances the deposition and uptake of contaminants (Wang et al., 2019; Anyanwu et al., 2021)

Among the individual PAHs analyzed, benzo[a]pyrene (BaP) contributed the most to the overall BaP-TEQ, with values of 62.73 (dry season) and 5.83 (wet season). The drastic reduction in BaP-TEQ from dry to wet season may be attributed to photodegradation and microbial activity that enhance the breakdown of BaP under humid conditions (Li et al., 2022). However, despite this decline, BaP remains the most significant contributor to carcinogenicity in Telfairia occidentalis, underscoring its classification as a potent carcinogen by the International Agency for Research on Cancer (IARC, 2020).

Other PAHs with significant carcinogenic contributions include dibenz[a,h]anthracene (DBA) and indeno[1,2,3-cd] pyrene (IND). DBA exhibited an increase in BaP-TEQ from 25.47 (dry) to 26.89 (wet), reinforcing its environmental persistence and high toxicity. Similarly, IND showed a rise from 1.61 (dry) to 2.77 (wet), further indicating an enhanced carcinogenic burden during the wet season. The consistent presence of these high molecular weight PAHs in Telfairia occidentalis suggests their resistance to environmental degradation, which contributes to prolonged exposure risks (Shi et al., 2020).

Benzo[k]fluoranthene (B(k)F) also displayed a notable increase in BaP-TEQ from 2.80 (dry) to 4.62 (wet), indicating a moderate yet significant seasonal variation in its carcinogenic potential. The TEQ of benzo[b]fluoranthene (B(b)F) remained stable at 1.55 across seasons, reflecting its relatively consistent bioavailability in the environment. On the other hand, chrysene (Chry) and benzo[ghi]perylene (B(ghi)P) exhibited minimal TEQ contributions (≤ 0.3), suggesting a lesser but still present risk associated with their intake.

The seasonal differences in BaP-TEQ levels indicate fluctuating carcinogenic risks associated with Telfairia occidentalis consumption. While the wet season showed higher overall TEQ values, the dry season exhibited a more pronounced BaP contribution, suggesting different exposure dynamics. The lower BaP-TEQ in the wet season may be due to increased photodegradation and microbial breakdown, but the increased TEQ of other PAHs (e.g., DBA, IND) during the wet period highlights the sustained presence of carcinogenic PAHs in leafy vegetables (Zhang et al., 2023).

These findings suggest that long-term exposure to PAHs in Telfairia occidentalis poses significant health risks, especially for populations with high consumption rates. The consistently high BaP-TEQ values across seasons reaffirm the need for stringent monitoring and risk assessment to mitigate PAH contamination in edible crops. Given that dietary intake is a major route of PAH exposure, the observed seasonal variations emphasize the importance of controlling environmental PAH sources to reduce human health risks (Jiang et al., 2018).

BaP-MEQ of Eight Carcinogenic PAHs in Telfairia occidentalis Samples

The assessment of the mutagenic potential of eight polycyclic aromatic hydrocarbons (PAHs) in Telfairia occidentalis using the benzo[a]pyrene mutagenic equivalency quotient (BaP-MEQ) provides insight into the mutagenic risks associated with consuming this vegetable. The total BaP-MEQ was found to be higher in the wet season (0.243) than in the dry season (0.140), suggesting increased mutagenic risks during the wet season. This seasonal variation may be attributed to increased atmospheric deposition and soil contamination due to rainfall, which facilitates the mobility and bioavailability of PAHs in agricultural environments (Wang et al., 2019; Anyanwu et al., 2021).

Benzo[a]pyrene (BaP) remained the predominant PAH contributing to the overall mutagenic potential, with MEQ values of 71.63 (dry season) and 63.37 (wet season). Despite a slight decrease in the wet season, BaP still posed the highest mutagenic risk, aligning with its well-established role as a potent mutagen and carcinogen (IARC, 2020). The decline in BaP-MEQ in the wet season could be linked to enhanced photodegradation and microbial breakdown, which are more pronounced under humid conditions (Li et al., 2022).

Other PAHs that significantly contributed to the mutagenic burden included dibenz[a,h]anthracene (DBA) and indeno[1,2,3-cd]pyrene (IND), with DBA showing nearly equal MEQ values in the dry (8.44) and wet (8.48) seasons, indicating its environmental persistence. IND exhibited an increase from 5.74 (dry) to 9.30 (wet), suggesting enhanced mutagenic potential during the wet season, likely due to increased deposition from anthropogenic sources (Shi et al., 2020).

Benzo[k]fluoranthene (B(k)F) and benzo[b]fluoranthene (B(b)F) also exhibited considerable mutagenic activity. B(k)F increased from 3.55 (dry) to 5.51 (wet), while B(b)F showed a slight decline from 4.54 to 4.24. This indicates that although B(k)F may be more affected by seasonal factors, B(b)F remains relatively stable in the environment.

The seasonal variations in BaP-MEQ levels highlight differences in mutagenic risk between the dry and wet seasons. While the total BaP-MEQ was higher in the wet season, the dry season showed more pronounced contributions from BaP, reinforcing its role as a dominant mutagenic PAH. The increased MEQ values for DBA and IND in the wet season further suggest a heightened risk of DNA damage and mutagenicity, as these compounds are known for their strong mutagenic properties (Zhang et al., 2023).

Overall, the findings suggest that dietary exposure to PAHs through Telfairia occidentalis may pose significant mutagenic risks, particularly during the wet season when the total MEQ levels are elevated. These risks necessitate continuous monitoring and preventive measures to minimize PAH contamination in edible crops.

Health Implications and Risk Management

The seasonal differences in BaP-TEQ and BaP-MEQ levels suggest fluctuating carcinogenic and mutagenic risks associated with Telfairia occidentalis consumption. While wet season conditions enhance overall PAH accumulation, the dry season exhibits higher BaP dominance, altering exposure dynamics. Long-term dietary exposure to these PAHs could significantly increase cancer and mutagenicity risks, necessitating stringent monitoring and risk mitigation strategies.

Conclusion and Recommendations

The BaP-TEQ and BaP-MEQ assessments of Telfairia occidentalis indicate significant seasonal variations in both carcinogenic and mutagenic potentials of PAHs. The wet season generally exhibited higher TEQ and MEQ values, implying increased risks of PAH exposure during this period. BaP consistently emerged as the most dominant contributor to carcinogenic and mutagenic potential, alongside DBA and IND. These findings highlight the potential health risks associated with consuming Telfairia occidentalis from contaminated areas, necessitating urgent mitigation measures.

To address these concerns, the following recommendations are made:

1. Regular Monitoring: Continuous environmental and food monitoring should be conducted to assess PAH contamination levels in agricultural produce.

2. Agricultural Best Practices: Farmers should adopt protective strategies such as controlled irrigation, reduced exposure to contaminated soil, and the use of protective barriers to minimize PAH deposition on crops.

3. Public Awareness: Increased awareness programs should be implemented to educate consumers on the risks of PAH contamination in vegetables and potential dietary exposure pathways.

4. Policy and Regulation: Governmental agencies should enforce stricter regulations on industrial emissions and ensure proper remediation of PAH-contaminated sites.

5. Further Research: Additional studies should investigate PAH bio-accumulation mechanisms in different crops and explore effective remediation techniques to reduce PAH levels in food sources.
By implementing these recommendations, the risks associated with PAH contamination in edible plants can be significantly mitigated, ensuring food safety and public health protection.

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