Original Research Article

Health Risk Assessment of Polycyclic Aromatic Hydrocarbons (PAHs) from Barbecued Beef and Chicken within Abuja and Environs

ABSTRACT

Aims: Polycyclic Aromatic Hydrocarbons (PAHs) in heat-treated meat constitute a risk to human health globally. This study determined the concentrations of PAHs in commonly consumed barbecued meat (suya) in Abuja, Nigeria and evaluated the health dangers related to their intake.
Methodology: Analysis was performed using Gas Chromatography-Mass Spectrometry, while health risks were estimated using models proposed by the European Commission, the European Food Safety Authority, and the United States Environmental Protection Agency.
Results: The results showed different levels of 13 PAHs in meat products, with the highest concentration in barbecued beef. The mean concentrations of individual PAHs ranged from not detected (ND) to 0.220 mg/kg. The total PAH concentrations (mg/kg) in various meat samples were 1.358, 1.031, 0.969, 0.687, and 0.733 for barbecued beef, basted barbecued chicken, non-basted barbecued chicken, control beef, and chicken, respectively. Total carcinogenic PAH levels (mg/kg) were 0.357, 0.281, 0.175, 0.119, and 0.259 for barbecued beef, basted barbecued chicken, non-basted barbecued chicken, control beef, and chicken, respectively. Benzo[a]pyrene (B[a]P) and PAH4 concentrations in barbecued samples, were well above the maximum levels stipulated by the European Union, suggesting health risks. But they were undetected in non-basted barbecue chicken. The total daily dietary exposure was higher for barbecued beef compared to barbecued chicken and non-basted barbecued chicken. Hazard quotients and indexes were<1 in all barbecued samples. The margin of exposure (MOE) for the indicators of PAHs in barbecued chicken and non-basted barbecued chicken was greater than 10,000 implying low concern for consumer health. However, the MOEs for (B[a]P) and PAH8 in barbecued beef were less than 10,000, implying that dietary exposure may pose a significant health risk and so require risk management actions.
Conclusion: Basting of meat during barbecuing may increase PAH formation, including carcinogenic PAHs, therefore, vendors should be enlightened on control measures.

Keywords: Polycyclic aromatic hydrocarbons, barbecued beef, and chicken, risk assessment, Federal Capital Territory, Abuja, basting, non-basted chicken.

1. INTRODUCTION

Commercial preparation and sale of regular suya in Nigeria are unregulated, and there is no standard set by the food regulatory bodies on the maximum levels of PAHs allowed in food; so the quality sold is dependent on the experience of each producer, popularly called 'Mai Nama', his discretion, and also on the desire or request of the consumer. Some of them, both out of ignorance or intentionally because of greed for money, cook and sell burnt suya on request to the buyers. Furthermore, they use a technique known as basting during cooking to add or enhance flavor in the meat as well as prevent unwanted loss

of moisture. Basting involves cooking meat with its juice or fat, melted butter, fat or oil which is periodically poured or sprayed over the meat while it is cooking above a heat source [1]. It has the potential to boost the formation of heat-induced contaminants such as polycyclic aromatic hydrocarbons in barbecued meat if there is no proper control. This is because of the pyrolysis of sprayed fat, which may drip into the heat source, a condition that produces volatile PAHs that will, in turn, adhere to the surface of the meat as the smoke rises and thus increase the concentration of PAHs in meat [2].

PAHs are organic compounds formed during incomplete combustion or pyrolysis of organic substances like wood, oil, etc. [3]. Seven pyrogenic PAHs in the International Agency for Research on Cancer (IARC) groups 1 (benzo[a]pyrene), 2A (dibenzo[a, h]anthracene), and/or 2B (benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, and indeno[1,2,3-cd]pyrene) have been reasonably estimated by the United States Environmental Protection Agency [4] and the IARC to be human carcinogens based on sufficient evidence of carcinogenicity in animal studies [5]; and are therefore used to assess carcinogenic risk. In 2008, at the request of the European Commission, the EFSA Panel on Contaminants in the Food Chain (CONTAM PANEL) reviewed available data on the occurrence and toxicity of PAHs with a particular focus on the suitability of benzo[a]pyrene alone as a marker for PAH contamination in food. The panel adopted PAH4 and PAH8 as better indicators of PAH occurrence than only B[a]P [6]. Furthermore, the European Commission provided guidelines for PAHs in food by setting maximum levels of 0.005 mg/kg for benzo[a]pyrene and 0.030 mg/kg for the sum of PAH4 in heat-treated meat and meat products including barbecued meat [7].

Production and sale of burnt meat, as well as using basting in barbecuing as observed in Abuja, have the potential to expose consumers to PAHs including carcinogenic PAHs formed in the suya. This research thus became necessary because of the risk of cancer associated with dietary exposure to PAHs in burnt meat [8], coupled with the lack of sufficient data on the profile and concentrations of PAHs in suya sold in Abuja. The aim of the study, therefore, is to assess the levels of 16 priority PAHs in suya meat and the possible health risks associated with human exposure to these PAHs. The results from this study will contribute to the existing data on PAHs in barbecued meat sold in Abuja, providing valuable insights into potential health risks associated with dietary exposure to these PAHs.

2. MATERIALS AND METHODS

2.1 Study area

The study areas are all in the Federal Capital Territory, Abuja (Fig. 1), in the North Central region of Nigeria. There are six area councils in the FCT, Abuja, including Abuja municipal (AMAC), Abaji, Bwari, Gwagwalada, Kuje, and Kwali [9]. Three area councils (AMAC, Bwari, and Gwagwalada) were selected for this study. Sections of the towns with suya spots, markets, and heavy business activities were specifically chosen for sampling.

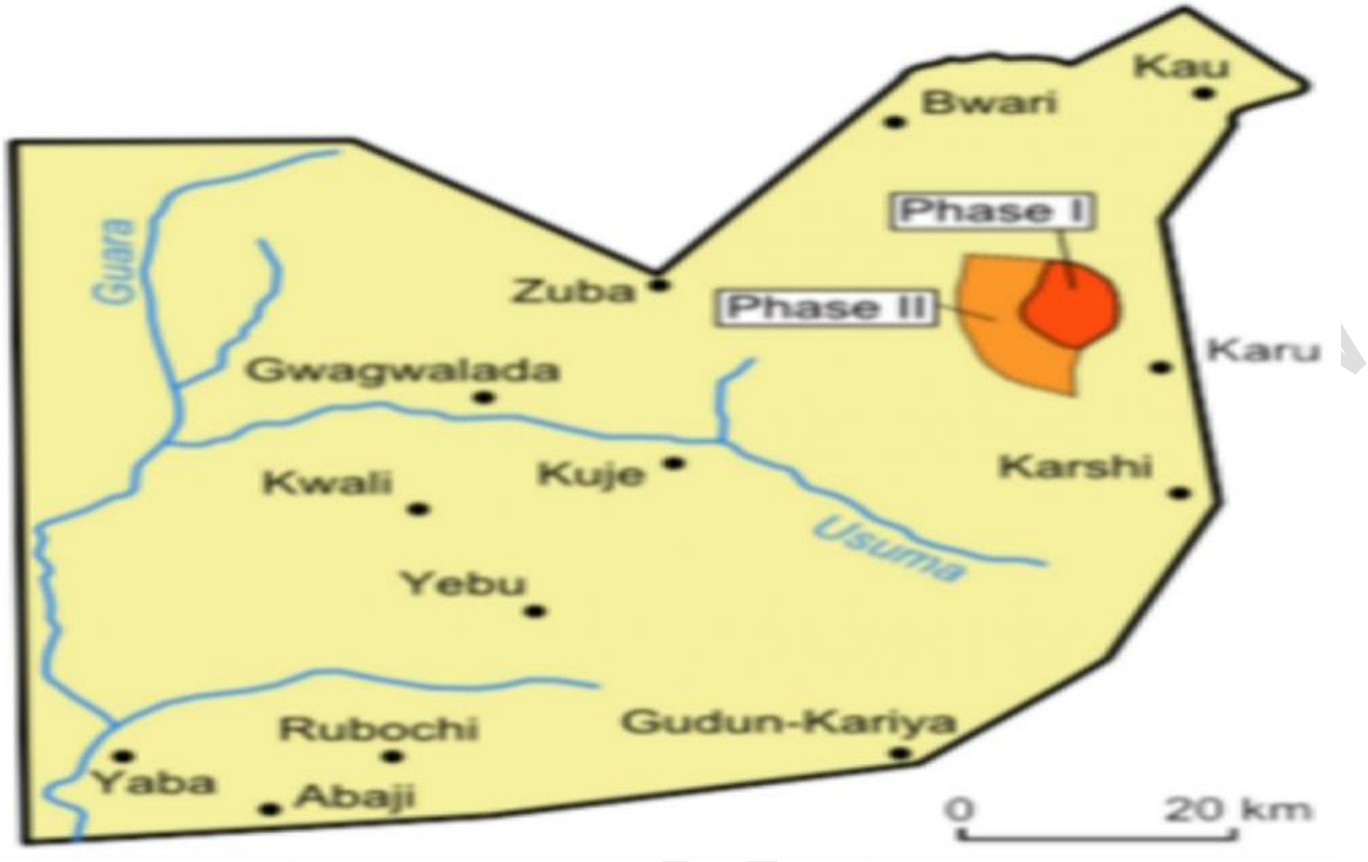

Fig. 1. Map of Federal Capital Territory, Abuja showing the study areas (Phases I and II on the map represent AMAC) (Source: [9]).

2.2 Chemicals and Standards

The standard aggregate of sixteen US EPA priority PAHs in acetonitrile was obtained from Sigma-Aldrich (Bellefonte, PA, USA). The PAHs include Naphthalene (Naph), Acenaphthylene (Acy), Acenaphthene (Ace), Fluorene (Fluo), Phenanthrene (Phen), Anthracene (Anth), Pyrene (Pyr), Fluoranthene (Flt), Chrysene (Chr), Benzo[a]anthracene (B[a]A), Benzo[K]fluoranthene (B[k]F), Benzo[b]fluoranthene (B[b]F), Benzo[a]pyrene (B[a]P), Benzo[ghi]perylene (B[ghi]P), Dibenz[ah]anthracene (D[ah]A), and Indeno [1,2,3-cd]pyrene (I[cd]P). The standard was diluted as required to working standard solutions in (4+1) hexane and acetone (0.05, 0.1, 0.25, 0.5, 1, and 2 µg/mL of calibration standards), and stored in dark glass vials at 4℃. Acetone (HPLC grade) with 99.99 % purity was acquired from Fischer Scientific, United Kingdom. Acetonitrile (HPLC grade) (purity > 95%) was from Merck, Japan. n-hexane (98.0 %), $MgSO_4$ (>98%) for clean-up, and sodium chloride (NaCl) (99.5%) were obtained from Merck, Germany. $MgSO_4$ anhydrous grit (coarse) was obtained from Sigma- Aldrich, USA. Disodium hydrogen citrate sesquihydrate ($C_6H_6Na_2O_7.1.5H_2O$) was purchased from Sigma-Aldrich, Germany. Trisodium citrate dehydrates ($Na_3C_6H_5O_7.2H_2O$) were from Sigma Aldrich, Japan. Bondesil Primary Secondary Amine (PSA) sorbent was purchased from Agilent Technologies, USA, while C-18 sorbents were purchased from Supelco, USA.

2.3 Sample Collection and Preparation

Barbecued beef and chicken (suya) samples and uncooked samples were randomly purchased from different vendors within the study areas in the FCT. Sample preparation included shredding and blending using a Waring 800 EG food blender to obtain a homogenized sample. The samples were placed in bottles, wrapped in aluminum foil, and kept at a temperature of -4^oC in the freezer [10], pending the determination of PAHs.

2.4 Analysis of PAHs in the barbecued samples

Before analysis, all glassware was carefully cleaned with soap and water, thoroughly rinsed with acetone, and dried at 90°C to prevent cross-contamination. Vials were capped, and flasks and concentration tubes were covered with aluminum foil to prevent airborne contamination. The Quick, Easy, Cheap, Effective, Rugged, and Safe (QuEChERS) methodology, which is the European Standard EN 15662:2008 citrate buffered procedure [11], was utilized for sample extraction and clean-up, while GC-MS was used for instrumental analysis. These are described below.

2.4.1 Extraction and Clean-up

About 5g of homogenized sample was added into a 50 mL QuEChERS Teflon extraction/centrifuge tube, followed by the addition of 10 mL of distilled water. The tube and its contents were shaken on a vortex shaker for 1 minute to mix thoroughly. This was followed by the addition of 10 mL of acetonitrile (HPLC grade) containing 100 µl (0.1 mL) of 0.5 µg/mL of surrogate standard PCB 153, shaken vigorously on a vortex shaker, and the tubes were allowed to stand for 15 minutes. Next, 6.5 g of QuEChERS pre-mixed extraction salts containing (4 g of anhydrous magnesium sulfate (coarse), 1 g of sodium chloride, 0.5 g of disodium hydrogen citrate sesquihydrate and 1 g of Trisodium citrate dihydrate) were added, tubes caped, vigorously shaken for 1 minute for quick dispersion of the salt into the homogenized sample, vortexed for 3 minutes, and centrifuged for 5 minutes at 3000 rpm.

Aliquot (6 mL) of the supernatant (clear organic phase) was taken using a pipette and transferred into the QuEChERS 15 mL dispersive solid phase (dsp) clean-up Teflon centrifuge tube containing 1.2 g of the clean-up salts made up of (900 mg magnesium sulfate (fine), 150 mg primary secondary amine (PSA), and 150 mg C18 sorbents). The tube and contents were capped and again shaken, vortexed for 5 minutes, and centrifuged as above. The supernatant liquid was transferred into a 10 mL graduated glass test tube with a cap, and 5 % formic acid was added to it for stability (10 µL/mL of extract). The contents were shaken on a vortex shaker for one minute. Extract (1 mL) was transferred into a round bottom flask, evaporated at 40^{o}C to near dryness on a rotary evaporator, reconstituted in 1 mL of (4+1) hexane + acetone, and subsequently, transferred to an autosampler amber vials ready for analysis by GC-MS.

2.4.2 Gas Chromatography-Mass Spectrometry (GC-MS) Analysis of PAHs

The extracts were analyzed using a GC-MS system (consisting of a gas chromatography GC-2010 Plus (Shimadzu) coupled to MS QP2010 Ultra (Shimadzu)) fitted with a VF5 MS cross-linked capillary column of 30m × 0.25µm x 0.25mmID. The GC Oven Temperature Program was 60^{o}C (hold 1 min) to 200^{o}C at 10^{o}C/min (hold 2 min) to 300^{o}C at 10^{o}C/min (hold 3 min). The injection mode was splitless, injection temperature of 250^{o}C and an injection volume of 1µL. The ion source temperature of the MS was set to 200^{o}C, the interface temperature to 250^{o}C, and the solvent cut-off time was 3 minutes (solvent delay). Calibration standards and extracts (1µL) were injected into the GCMS and analyzed under the operating conditions. The Reference Standards and samples were run in Selected Ion Monitoring (SIM) mode. For each target analyte, the characteristic primary ion was selected with additional qualifying ions for quantitation. All standards and sample extracts were analyzed in triplicate under the same operating GCMS conditions. The retention time and area of each observable PAH peak were measured for all 16 US EPA priority PAHs.

2.5 Evaluation of Human Health Risks

The health risks associated with exposure to PAHs in barbecued meat were estimated by comparing the concentrations of PAHs in the meat with regulatory bodies' guidance values. The daily dietary exposure (DDE) to PAHs was calculated and used to determine the associated cancer and non-cancer risks to human health, using models provided by regulatory authorities. The toxicological parameters are listed in Table 1.

Table 1. Toxicological Parameters for Risk Assessment

Parameters	Unit	Value	Reference
The concentration of each congener (C)	mg/kg	Table 4	Table 4
Beef consumption rate (BCR)	Kg/AME/day	0.0078	[12]
Poultry consumption rate (BCR)	Kg/AME/day	0.0010	[12]
Adult body weight (BW)	Kg	70	[13]
Oral Slope Factor (SF)	mg/kg/day	Table 5	[14]
Reference dose (RfD)	mg/kg/day	Table 5	[14]
$BMDL_{10}$ (benzo[a}pyrene)	mg/kg BW-day	0.07	[6]
$BMDL_{10}$ (PAH2)	mg/kg BW-day	0.17	[6]
$BMDL_{10}$ (PAH4)	mg/kg BW-day	0.34	[6]
$BMDL_{10}$ (PAH8)	mg/kg BW-day	0.49	[6]

$BMDL_{10}$ is the benchmark dose lower confidence limit at a 10% incidence level. PAH2 (Benzo[a]pyrene and chrysene); PAH4 (Benzo[a]pyrene, chrysene, Benzo[a]anthracene and Benzo[b]fluoranthene); PAH8 (PAH4, Benzo[k]fluoranthene, Benzo[ghi]perylene), Dibenz[a, h]anthracene, and Indeno[1,2,3-cd]pyrene

2.5.1 Determination of Daily Dietary Exposure (DDE)

The FAO/WHO-proposed approach was used to determine the chronic (lifetime) daily dietary exposure (DDE) to PAHs in barbecued meat [15]. This was determined by multiplying the respective PAH concentration in each meat by the consumption rate (CR) of an adult of average body weight, which in Nigeria is 70 kg [13] (Table 1). The mean daily consumption rate for meat products in Nigeria for an average adult population is shown in Table 1 [12]. Daily dietary exposure (DDE) was then assessed for individual PAH congeners, total PAHs, PAH4, PAH8, and carcinogenic PAHs in each sample using equation (2.1),

$$DDE=\Sigma(CxCR)/BW \quad (2.1)$$

Where,

DDE = daily dietary exposure; C = concentration of PAHs in meat (mg/kg); CR = meat consumption rate (kg/Ame-day); BW = body weight (kg) of an adult consumer.

2.5.2 Non-carcinogenic risk characterization

Using the hazard quotients (HQs) and hazard index (HI) methods, risks connected to dietary exposure to non-carcinogenic PAHs were calculated [4].

$$HQ=DDE/RfD \quad (2.2)$$

Table 5 provides pertinent RfD values (mg/kg/day) for identified PAHs. As illustrated in equation (2.3), the hazard index (HI) was calculated by adding up all hazard quotients.

$$HI= \Sigma[HQ_1+HQ_2+ \text{---}HQ_n] \quad (2.3)$$

Where DDE = daily dietary exposure; RfD = reference dose; HQ = hazard quotient; HI = hazard index

2.5.3 Determination of cancer risks

2.5.3.1 Indicators of PAH Occurrence and Toxicity

B[a]P and PAH4 were used based on the decision of the CONTAM panel to adopt them as indicators of PAH occurrence and toxicity in food [6]. PAH4 was determined as the sum of four different PAHs including B[a]P, B[a]A, B[b]F, and Chr using equation 2.4. Therefore, concentrations of B[a]P and PAH4 in meat products were compared with the maximum levels provided by the European Union Commission Regulation, No. 1327/2014 [7].

$$\text{PAH4 Indicator } \Sigma(B[a]P+B[a]A+B[b]F+Chr) \quad (2.4)$$

2.5.3.2 The Margin of Exposure (MOE)

The margin of exposure (MOE) characterization approach was used to determine the risk from exposure to carcinogenic and genotoxic substances. The MOE is calculated by dividing the toxicity estimate ($BMDL_{10}$) obtained from animal experiments by the daily dietary exposure according to Equation 2.5 [16]. $BMDL_{10}$ is the dose at which a small but measurable adverse effect is first observed [17]. The magnitude of MOE is an indicator of the level of concern, but it is not a precise quantification of risk; the larger the MOE, the smaller the potential risk posed by exposure to the selected compound [18]. MOE values less than 10,000 indicate potential concern for human health [6].

$$MOE=BMDL_{10} /DDE \quad (2.5)$$

Where $BMDL_{10}$ is the benchmark dose lower confidence limit at a 10% incidence level. Values (mg/kg BW per day) of 0.07, 0.17, 0.34, and 0.49 have been calculated for BaP, PAH2 (Benzo[a]pyrene and chrysene), PAH4, and PAH8, respectively [6, 14]; MOE = margin of exposure; DDE = daily dietary exposure.

2.6 Data analysis

The chromatogram and data obtained from GC-MS analysis were processed using the computer-based GC software GCMS Solutions provided by Shimadzu Corporation. The results were then transferred to MS Excel version 15.0 program for further analysis. Descriptive statistics were applied to the data, and mean values and standard deviations were calculated for individual PAHs, total PAHs (∑PAHs), total carcinogenic PAHs (∑CPAHs), etc. A statistical test of significance was conducted on the mean concentrations, using a significance level of $p = .05$. Additionally, a correlation coefficient (r) analysis was performed to examine the relationship between different groups of meat samples at $P \neq 0$.

3. RESULTS AND DISCUSSION

The concentrations of PAHs in barbecued and control samples are presented in Table 2. Table 3 displays the daily dietary exposure (DDE) of PAHs in barbecued meat (mg/kg/day). Non-carcinogenic risk assessment results are shown in Table 4, while Table 5 presents the margin of exposure values for indicators of PAH occurrence and toxicity in barbecued meat.

The statistical test of significance on the means of concentrations at $P = .05$ yielded a P-value of .17, which is greater than .05. This suggests that there may not be a significant difference between the mean concentrations of PAHs in the meat samples. The correlation coefficient analysis revealed a strong positive correlation (r = .85) between the occurrence of PAHs in regular barbecued beef and barbecued chicken. Additionally, moderate positive correlations were observed between the occurrence of PAHs in non-basted barbecued chicken and both barbecued beef (r = .66) and barbecued chicken (r = .54). The correlations between raw beef and raw chicken with other meat samples were generally weaker. These findings indicate the existence of common factors influencing the occurrence of PAHs in barbecued beef and

chicken. Further investigation using other models was conducted to explore these factors and their potential impact on human health.

3.1 PAH concentrations in uncooked meat

The concentrations of PAHs in uncooked meat were generally low. Total concentrations of PAHs were higher in raw chicken than in beef, and this slight difference may be attributed to a difference in fat content [19]. The values of the more toxic PAH B[a]P, PAH4, PAH8, and carcinogenic PAHs in raw chicken were also higher than in raw beef. In addition, the concentration of B[a]P in raw chicken (0.112 ± 0.062 mg/kg) was higher than in barbecued chicken (0.060±0.011 mg/kg). This may be attributed to the thermal decomposition of PAHs when meat is heated at high temperatures. The order of occurrence of PAHs based on the number of rings they have is as follows: 2 rings> 3 rings >4 rings > 6 rings >5 rings, with a percentage occurrence (according to the number of individual PAHs detected in each ring group) of 100 >80 > 60 > 50 > 38. This portrays the dominance of the LMW PAHs over the HMW PAHs. Five out of the six LMW PAHs were very prominent in the two samples, with an incident ratio of 55. 6% compared to the pyrolytic HMW PAHs which were mostly undetected. These are in agreement with the findings that PAH formation occurs during meat processing at high temperatures and that HMW PAHs are pyrogenic [20]. A study also reported different concentrations of PAHs in raw chicken and raw beef [21].

3.2 PAHs in barbecued meat

Barbecued beef displayed the highest total PAHs (1.358 mg/kg), with mean individual concentrations ranging from 0.069 to 0.220 mg/kg. Also, the level of total carcinogenic PAHs was highest in barbecued beef, followed by barbecued chicken (Table 2). These high concentrations could be linked to the specific conditions of the barbecuing process and the meat composition transforming at high temperatures [22]. This could have implications for human health, as dietary exposure to PAHs has been associated with an increased risk of cancer.

The non-basted chicken contains lower levels of PAHs, with B[a]P, PAH4, and many other analytes undetected. These higher PAHs shown in the regular basted meat compared to the non-basted chicken may be attributed to the pyrolysis of the sprayed fat that drips into the heat source if there is no proper control. This will subsequently produce volatile PAHs that in turn adhere to the surface of the food as the smoke rises, increasing PAH formation [2, 20]. This indicates that avoiding basting meat during barbecuing will most likely reduce PAH formation and levels. PAH levels and profiles observed in this report are in harmony with those reported in other studies [10, 23].

The order of occurrence of PAHs in the barbecued meat is as follows: 2 rings >3 rings > 4 = 6 rings> 5 rings, with % occurrence (based on the number of individual PAHs detected in each ring group) of 100% > 87% > 67%=67% > 42%, respectively. Generally, lower molecular weight PAHs were detected more frequently (88.9 % relative to the total number of LMW PAHs) but at lower concentrations. This is consistent with the environmental occurrence of these contaminants and with the report of [24].

These results indicate that the concentration of PAHs varies between different types of meat and cooking techniques. For example, the total concentration of 16 PAHs (∑PAH16) is higher in the regular suya (basted barbecued beef (1.358±0.057 mg/kg) and basted barbecued chicken (1.031±0.058 mg/kg)) compared to non-basted chicken (0.969±0.056 mg/kg), control beef (0.687±0.043 mg/kg), and control chicken (0.733±0.047 mg/kg). The concentration of carcinogenic PAHs (∑CPAHs) is also higher in these regular barbecued beef (0.357±0.053 mg/kg) and barbecued chicken (0.281±0.053 mg/kg) compared to non-basted chicken (0.174±0.044 mg/kg), control beef (0.119±0.030 mg/kg), and control chicken (0.259±0.048 mg/kg). These results suggest that barbecuing meat with basting may increase the concentration of PAHs, including carcinogenic PAHs, compared to other cooking techniques or uncooked meat. They also show that removing basting could be adopted as a risk management measure. This explains why there is a strong positive correlation between sources of PAH in regular barbecued beef (BB) and chicken (BC).

Table 2.Mean concentrations (mg/kg) of PAHs in barbecued meat (suya) sold in FCT Abuja

PAHs	BB	BC	NBC	CB	CC
□ Naphthalene	0.069±0.009	0.078±0.016	0.114±0.075	0.070±0.002	0.087±0.023
□ Acenaphthylene	0.082±0.021	ND	0.061±0.053	ND	ND
□ Acenaphthene	0.076±0.009	0.084±0.013	0.077±0.018	0.062±0.002	0.045±0.039
□ Fluorene	0.085±0.046	ND	0.070±0.077	0.087±0.037	0.085±0.034
□ Phenanthrene	0.220±0.100	0.182±0.006	0.179±0.038	0.123±0.016	0.131±0.004

□ Anthracene	0.091±0.014	0.074±0.004	0.073±0.006	0.040±0.035	0.041±0.035
□Pyrene	0.118±0.034	0.105±0.006	0.103±0.012	0.091±0.006	0.087±0.002
□ Fluoranthene	0.140±0.008	0.118±0.043	0.115±0.100	ND	ND
•Chrysene	0.133±0.016	0.117±0.006	ND	ND	0.076±0.066
•Benzo[a]anthracene	ND	ND	ND	ND	ND
•Benzo[k]fluoranthene	0.089±0.028	0.104±0.001	0.105±0.002	ND	0.069±0.060
•Benzo[b]fluoranthene	ND	ND	ND	ND	ND
•Benzo[a]pyrene	0.065±0.006	0.060±0.011	ND	0.049±0.043	0.112±0.062
□ Benzo[gh]perylene	0.120±0.035	0.109±0.050	ND	0.095±0.082	ND
•Dibenz[ah]anthracene	ND	ND	ND	ND	ND
•Indeno[1,2,3-cd] pyrene	0.070±0.004	ND	0.070±0.003	0.070±0.005	ND
Total PAH16 ∑PAH16	1.358±0.057	1.031±0.058	0.969±0.056	0.687±0.043	0.733±0.047
Total PAH2 ∑PAH2	0.198±0.048	0.177±0.040	ND	0.049±0.035	0.188±0.026
Total PAH4 ∑PAH4	0.198±0.064	0.177±0.056	ND	0.049±0.025	0.188±0.056
Total PAH8 ∑PAH8	0.477±0.054	0.390±0.055	0.175±0.042	0.214±0.039	0.257±0.046
Total Carcinogenic PAHs ∑CPAHs	0.357±0.053	0.281±0.053	0.174±0.044	0.119±0.030	0.259±0.048
Total Non-Carcinogenic PAHs ∑NCPAHs	0.1001±0.047	0.750±0.057	0.794±0.049	0.568±0.043	0.476±0.048
Low molecular weight PAHs ∑LMWPAHs	0.623±0.057	0.418±0.067	0.576±0.045	0.382±0.042	0.389±0.046
High molecular weight PAHs ∑HMWPAHs	0.735±0.056	0.613±0.055	0.393±0.052	0.305±0.041	0.344±0.046

BB = barbecued beef; BC = barbecued chicken; NBC = non-basted barbecued chicken; CB = control beef; CC = control chicken; □ = non-carcinogenic PAHs, •= carcinogenic PAHs. Values are mean ± standard deviation for three replicates, (n = 3)

3.3 Determination of Daily Dietary Exposure (DDE)

The results in Table 3 indicated variations in DDE values between different types of meat. For example, the total DDE of 16 PAHs was higher in barbecued beef (1.51×10^{-4} mg/kg/day) compared to barbecued chicken (1.47×10^{-5} mg/kg/day) and non-basted barbecued chicken (1.38×10^{-5} mg/kg/day). The DDE values (mg/kg/day) for individual PAHs ranged from 0.000 to 2.45×10^{-5}, 0.000 to 2.60×10^{-6}, and 0.000 to 2.56×10^{-6} in beef suya, chicken suya, and non-basted chicken suya respectively.

Comparing DDE values to RfD values can indicate whether exposure to a substance is within safe levels. For instance, the DDE of naphthalene in barbecued beef is lower than the RfD of naphthalene, suggesting that dietary exposure to naphthalene in barbecued beef is within safe levels. The DDE values for all the PAHs were also below the reference values (RfD) and the cancer slope factors (CSF) for the respective individual PAHs as well as for the total PAHs as depicted in Table 3. Additionally, among the samples, the sum of the values of DDE to PAHs from beef suya was highest, and this may be related to high adult consumption rates of beef (0.0078 kg/Ame/day) as well as a high level of PAHs occurrence in it. This indicates that regular intake of beef suya will likely result in a higher risk of exposure to PAHs in it, especially carcinogenic PAHs [25]. Furthermore, results also show that estimated dietary exposure was lowest in non-basted chicken compared to basted beef and chicken, suggesting a lower risk of dietary exposure to PAHs than basted beef and chicken. These results imply that high consumption of regular basted barbecued meat may increase exposure to PAHs, including carcinogenic PAHs. It also suggests that exposure to multiple PAHs can have cumulative effects on human health since not all PAHs have established RfD values, e.g., phenanthrene.

Table 3. Estimated Daily Dietary Exposure (DDE) of PAHs in barbecued meat (mg/kg/day)

PAHs	BB	BC	NBC	RfD (mg/kg/day)	CSF (mg/kg/day)
Naph	7.69×10^{-6}	1.11×10^{-6}	1.63×10^{-6}	2.00×10^{-2}	NA
Ace	9.14×10^{-6}	0.00×10^{-0}	8.71×10^{-7}	2.00×10^{-2}	NA
Ace	8.47×10^{-6}	1.20×10^{-6}	1.10×10^{-6}	6.00×10^{-2}	NA
Fluo	9.47×10^{-6}	0.00×10^{-0}	1.00×10^{-6}	4.00×10^{-2}	NA
Phen	2.45×10^{-5}	2.60×10^{-6}	2.56×10^{-6}	3.00×10^{-1}	NA
Anth	1.01×10^{-5}	1.06×10^{-6}	1.04×10^{-6}	3.00×10^{-1}	NA
Pyr	1.32×10^{-5}	1.50×10^{-6}	1.47×10^{-6}	3.00×10^{-2}	NA
Flt	1.56×10^{-5}	1.69×10^{-6}	1.64×10^{-6}	4.00×10^{-2}	NA
Chr	1.48×10^{-5}	1.67×10^{-6}	0.00×10^{-0}	NA	7.30×10^{-3}
B[a]A	0.00×10^{-0}	0.00×10^{-0}	0.00×10^{-0}	NA	7.30×10^{-1}
B[k]F	9.92×10^{-6}	1.49×10^{-6}	1.50×10^{-6}	NA	7.30×10^{-2}
B[b]F	0.00×10^{-0}	0.00×10^{-0}	0.00×10^{-0}	NA	7.30×10^{-1}
B[a]P	7.24×10^{-6}	8.57×10^{-7}	0.00×10^{-0}	NA	7.3×10^{-0}
B[ghi]P	1.34×10^{-5}	1.56×10^{-6}	0.00×10^{-0}	4.00×10^{-2}	NA
D[ah]A	0.00×10^{-0}	0.00×10^{-0}	0.00×10^{-0}	NA	7.3×10^{0}
I[cd]P	7.80×10^{-6}	0.00×10^{-0}	1.00×10^{-6}	NA	7.30×10^{-1}
∑DDE					
PAH16	1.51 x 10-4	1.47×10^{-5}	1.38×10^{-5}		
∑DDE					
PAH2	2.21×10^{-5}	2.53×10^{-6}	0.00×10^{-0}		
∑DDE					
PAH4	2.21×10^{-5}	2.53×10^{-6}	0.00×10^{-0}		
∑DDE					
PAH8	5.32×10^{-5}	5.58×10^{-6}	2.50×10^{-6}		
∑DDE					
CPAHs	3.98×10^{-5}	4.02×10^{-6}	2.50×10^{-6}		
∑DDE					
NCPAHs	1.12×10^{-4}	1.07×10^{-5}	1.13×10^{-5}		

∑DDE			
LMWPAHs	6.94×10^{-5}	5.97×10^{-6}	8.20×10^{-6}
∑DDE			
HMWPAHs	8.20×10^{-5}	8.77×10^{-6}	5.61×10^{-6}

RfD = reference dose; CSF = cancer slope factor; RfD of anthracene was used as a surrogate for phenanthrene as there was no value for it.

3.4 Non-carcinogenic risk characterization in humans

Table 4 shows the results of non-carcinogenic risk assessment for various polycyclic aromatic hydrocarbons (PAHs) in barbecued meat. The assessment is based on the calculation of hazard quotients (HQs) and hazard indexes (HI) [4]. The results specify that HQ values for all PAHs in barbecued meat are less than 1, suggesting that exposure to individual PAHs from consuming these types of meat is within safe levels. The HI values are also less than 1, implying that the combined dietary exposure to PAHs from barbecued meat is within safe levels. This also complements a report by [26]. Despite the estimated minimal danger of exposure, it is still crucial to regularly evaluate the levels of PAHs in barbecued meat to prevent any future harm to people's health.

Table 4. Non-carcinogenic risk assessment for PAHs in barbecued meat

Hazard quotients (HQs)				
PAHs	**Code**	**BB**	**BC**	**NBC**
Naphthalene	Naph	3.85×10^{-4}	5.55×10^{-5}	8.15×10^{-5}
Acenaphthylene	Acy	4.57×10^{-4}	0.00×10^{-0}	4.36×10^{-5}
Acenaphthene	Ace	1.41×10^{-4}	2.00×10^{-5}	1.83×10^{-5}
Fluorene	Fluo	2.37×10^{-4}	0.00×10^{-0}	2.50×10^{-5}
Phenanthrene	Phen	8.17×10^{-4}	8.67×10^{-5}	8.53×10^{-5}
Anthracene	Anth	3.37×10^{-5}	3.53×10^{-6}	3.47×10^{-6}
Pyrene	Pyr	4.40×10^{-4}	5.00×10^{-5}	4.90×10^{-5}
Fluoranthene	Flt	3.90×10^{-4}	4.23×10^{-5}	4.10×10^{-5}
Chrysene	Chr	NA	NA	NA
Benzo[a]anthracene	B[a]A	NA	NA	NA

Benzo[k]fluoranthene	B[k]F	NA	NA	NA
Benzo[b]fluoranthene	B[b]F	NA	NA	NA
Benzo[a]pyrene	B[a]P	NA	NA	NA
Benzo[ghi]perylene	B[ghi]P	3.35×10^{-4}	3.90×10^{-5}	0.00×10^{-0}
Dibenz[a,h]anthracene	D[ah]A	NA	NA	NA
Indeno[1,2,3-cd]pyrene	I[cd]P	NA	NA	NA
Hazard index (HI)	∑HQs	3.24×10^{-3}	2.97×10^{-4}	3.47×10^{-4}

3.5 Determination of cancer risk

3.5.1 Indicators of PAH Occurrence and Toxicity

The result in Table 2 shows that values (mg/kg) of the toxic B[a]P and PAH4 in beef (0.065 and 0.198), and in chicken (0.060 and 0.177), were well above their respective maximum permissible levels of 0.005 mg/kg and 0.03 mg/kg [7]. The implication is that frequent consumption of this meat may have adverse effects on human health. This calls for serious concern. On the other hand, non-basted barbecued chicken contained no B[a]P or PAH4. It also had lower levels of ∑CPAHs compared to barbecued beef and chicken. This may be attributed to the non-inclusion of basting while the meat was cooking over the heat source. This also implies no dietary risk to human health.

3.5.2 Margin of Exposure

Table 5 shows the margin of exposure (MOE) for adults to various polycyclic aromatic hydrocarbons (PAHs) in barbecued meat. The MOE is a measure used to assess the potential health risk of exposure to a substance. The results of MOE for benzo[a]pyrene (BaP), PAH2, and PAH8 in barbecued beef (9668, 7692, and 9210) are less than 10,000. This suggests that dietary exposure may pose a significant health risk and therefore require risk management actions [6]. The value of MOE for PAH4 in beef is high, probably because only two of PAH4 were detected in the sample. However, the MOEs for all indicators in barbecued chicken were above 10,000, revealing low concern for consumer health.The MOEs for all indicators except PAH8 in non-basted barbecued chicken were not applicable.

Table 5. The margin of exposure (MOE) of adults to PAHs in barbecued meat

PAHs	BB	BC	NBC
BaP	9668	81680	NA
PAH2	7692	67193	NA
PAH4	15382	134387	NA
PAH8	9210	87971	196000

NA = not applicable

4. CONCLUSION

In summary, the findings suggest that concentrations of PAHs vary between different types of meat and cooking techniques. Barbecuing meat with basting increases PAH levels, including carcinogenic PAHs, compared to non-cooked meat or other cooking techniques. This explains why there is a strong positive correlation between sources of PAH in barbecued beef and chicken.

Although the estimated dietary exposure to PAHs from barbecued meat was below safe levels, regular consumption may increase the risk of exposure, especially to carcinogenic PAHs. Suya vendors must be educated on safer and alternative techniques of barbecuing meat. Barbecuing without basting could be adopted as a risk management measure to reduce PAH formation and levels.Further research is needed to provide insights into their effectiveness and practical application in commercial settings.

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