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Carcinogenic and Genotoxic Health Risk Assessment Associated with Dietary Exposure to Polycyclic Aromatic Hydrocarbons in Charcoal-Grilled Catfish Consumed in Makurdi Metropolis, Benue State, Nigeria

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Abstract: Charcoal-grilled catfish constitutes one of the most widely consumed street and restaurant foods in Makurdi, Benue State, Nigeria, yet the carcinogenic and genotoxic health risks associated with chronic dietary exposure to polycyclic aromatic hydrocarbons (PAHs) in this food commodity have never been quantified for any consumer sub-group in this region. This study evaluated the lifetime carcinogenic risk and hazard quotients (HQ) associated with dietary intake of eight PAH congeners acenaphthene (AC), benzo[a]anthracene (BaA), benzo[b]fluoranthene (Bbf), benzo[k]fluoranthene (Bkf), acenaphthylene (AP), anthracene (A), benzo[ghi]perylene (BghiP), and dibenzo[a,h]anthracene (DBahA) detected in charcoal-grilled Clarias gariepinus, Heterobranchus longifilis, and Heteroclarias (hybrid) from three commercial grilling establishments in Makurdi. The USEPA incremental lifetime cancer risk (ILCR) model and the margin of exposure (MOE) approach recommended by EFSA (2008) were applied, using body-weight-normalised daily intake estimates for adult and child consumer sub-groups based on measured PAH concentrations. DBahA contributed the highest estimated ILCR (adults: 1.13×10^{-6}; children: 3.82×10^{-6}) across all scenarios, followed by BaA (adults: 4.07×10^{-7}; children: 1.38×10^{-6}). Estimated ILCRs for individual congeners fell within the 10^{-7} to 10^{-6} range for adults and 10^{-7} to 10^{-5} range for children, indicating a low to borderline acceptable carcinogenic risk level under chronic daily consumption assumptions. Non-carcinogenic hazard quotients for individual PAHs were all below unity for both consumer groups. The MOE for the PAH4 sum exceeded the EFSA threshold of 10,000 for moderate-risk scenarios, indicating that current PAH levels do not constitute an immediate public health emergency. However, the consistent detection of IARC Group 2A carcinogens (BaA, DBahA) and the absence of Nigerian national PAH limits for grilled fish represent critical regulatory gaps requiring urgent attention.

Keywords: polycyclic aromatic hydrocarbons, cancer risk, margin of exposure, dietary exposure, catfish charcoal grilling, health risk and food safety.

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a chemically diverse group of persistent organic pollutants formed during the incomplete combustion of organic matter and the high-temperature pyrolysis of food macromolecules, principally fats and proteins during cooking processes such as charcoal grilling, smoking, and barbecuing (Abdel-Shafy and Mansour, 2016; Barbosa et al., 2023). Several PAH congeners are genotoxic, mutagenic, and

carcinogenic in mammals. The International Agency for Research on Cancer (IARC) classifies benzo[a]pyrene (BaP) as Group 1 (carcinogenic to humans), benzo[a]anthracene (BaA) and dibenzo[a,h]anthracene (DBahA) as Group 2A (probably carcinogenic), and benzo[b]fluoranthene (Bbf) and benzo[k]fluoranthene (Bkf) as Group 2B (possibly carcinogenic), on the basis of mechanistic evidence, animal bioassays, and epidemiological associations with lung, skin, bladder, colon, and breast cancers (IARC, 2012; Abdel-Shafy and Mansour, 2016).

The primary route of human PAH exposure in non-occupational settings is dietary ingestion, with grilled, smoked, and barbecued meat and fish constituting the most significant food-based sources (Alomirah *et al.*, 2011; Duedahl-Olesen and Ionas, 2022). In Nigeria, charcoal-grilled catfish — principally Clarias gariepinus, Heterobranchus longifilis, and their hybrids represents a staple food commodity in restaurant and street food settings, with Makurdi, Benue State, being one of the most active charcoal-grilling markets in Nigeria's north-central region. Daily or near-daily consumption of charcoal-grilled catfish by large consumer populations creates conditions for cumulative PAH exposure that may translate into appreciable carcinogenic and genotoxic risks over a lifetime.

Despite this, no study has quantified the PAH-associated dietary health risks for consumers of charcoal-grilled catfish in Makurdi, and no national regulatory body in Nigeria has established maximum permissible levels for PAHs in grilled fish products. A companion study identified and quantified eight PAH congeners in charcoal-grilled catfish from three commercial establishments in Makurdi, with concentrations ranging from 0.005 µg/kg (acenaphthene) to 2.415 µg/kg (DBahA), all below EU limits. The present study uses these measured concentrations to conduct the first pathway-specific, age-stratified dietary cancer risk assessment for PAH exposure from charcoal-grilled catfish in Makurdi, applying the USEPA incremental lifetime cancer risk (ILCR) model and the European Food Safety Authority (EFSA) margin of exposure (MOE) approach, thereby filling a critical evidence gap for food safety management and public health policy in north-central Nigeria.

MATERIALS AND METHODS

Study Area

The study was conducted in Makurdi Metropolis, the capital of Benue State, Nigeria, situated between latitudes 7°33′03″ N and 7°47′47″ N, and longitudes 8°27′27″ E and 8°48′04″ E. Makurdi is drained principally by the River Benue, which bisects the city into North Bank and South Bank districts. The metropolis encompasses several densely populated commercial districts including High Level, Wurukum, Wadata, Kanshio, Gyado Villa, North Bank, and surrounding areas. The Katsina-Ala River forms a major tributary on the outskirts of the city. Makurdi has a tropical wet and dry climate with a wet season from April to October and a mean annual temperature of approximately 32°C.

PAH Concentration Data

PAH concentrations used in this risk assessment were derived from GC–MS analysis of 18 charcoal-grilled catfish samples collected from three commercial grilling sites in Makurdi Metropolis. Mean concentrations of eight PAH congeners AC, BaA, Bbf, Bkf, AP, A, BghiP,

and DBahA were used as inputs for all risk calculations. Where concentrations differed among fish species or sites, the highest recorded mean concentration was used as the conservative (worst-case) input.

Dietary Exposure Estimation

Dietary PAH exposure via oral ingestion of charcoal-grilled catfish was estimated using the deterministic approach recommended by EFSA (2008) and WHO/FAO JECFA (2005). The estimated daily intake (EDI, µg/kg bw/day) for each PAH congener was calculated as:

$$EDI = (C_PAH \times IR) / BW \quad \text{(Eq. 1)}$$

where C_PAH is the mean measured PAH concentration in grilled fish (µg/kg wet weight); IR is the daily fish ingestion rate (kg/day); and BW is the body weight of the consumer sub-group (kg). Two consumer sub-groups were considered: adults (BW = 70 kg; IR = 0.0283 kg/day based on the average Nigerian adult daily fish consumption of 28.3 g/day; FAO, 2018) and children aged 6–12 years (BW = 25 kg; IR = 0.015 kg/day). A chronic exposure scenario was applied (daily consumption, 365 days/year).

Incremental Lifetime Cancer Risk (ILCR)

ILCR for each carcinogenic PAH congener was estimated using the USEPA (1989) oral cancer slope factor (CSF) approach:

$$ILCR = EDI \times CSF \times ED \times EF / AT \quad \text{(Eq. 2)}$$

where CSF is the oral cancer slope factor $(mg/kg\ bw/day)^{-1}$; ED is exposure duration (70 years for adults, 10 years for children); EF is exposure frequency (365 days/year); and AT is averaging time (25,550 days for adults; 3,650 days for children). The BaP-toxic equivalency factor (BaP-TEF) approach was applied as a complementary analysis, converting PAH concentrations to BaP equivalents (BaPeq) using established TEF values (EFSA, 2008; Nisbet and LaGoy, 1992). ILCR categories follow USEPA (1989): negligible risk ($<10^{-6}$), acceptable risk (10^{-6} to 10^{-4}), and unacceptable risk ($>10^{-4}$).

Hazard Quotient (HQ) for Non-Carcinogenic Effects

The non-carcinogenic hazard quotient (HQ) for each PAH was calculated as:

$$HQ = EDI / RfD \quad \text{(Eq. 3)}$$

where RfD is the oral reference dose (mg/kg bw/day) from the USEPA Integrated Risk Information System (IRIS). The Hazard Index (HI) was computed as the sum of HQ values for all eight PAH congeners. HQ or HI < 1 indicates no significant non-carcinogenic risk; HQ or HI > 1 indicates potential risk (USEPA, 1989).

Margin of Exposure (MOE) Analysis

The EFSA (2008) MOE approach was applied to PAH4 (the sum of BaA, Bbf, Bkf, and BaP), using the benchmark dose lower confidence limit ($BMDL_{10}$) for genotoxic and carcinogenic

PAH mixtures. Following EFSA methodology, a $BMDL_{10}$ of 170 µg BaP-eq/kg bw/day (derived from rodent carcinogenicity studies) was used:

$$MOE = BMDL_{10} / EDI_BaPeq \quad (Eq. 4)$$

An MOE ≥ 10,000 is considered of low concern; MOE < 10,000 indicates a potential public health concern requiring risk management (EFSA, 2008). BaP-eq concentrations were calculated by multiplying measured PAH concentrations by their respective TEF values (Table 1).

Toxic Equivalency Factors

BaP-TEF values used in this study were sourced from Nisbet and LaGoy (1992) as endorsed by EFSA CONTAM Panel (2008):

Table 1: BaP-Toxic Equivalency Factors (TEF) for Detected PAH Congeners

PAH Compound	Abbreviation	TEF (BaP = 1)	CSF (mg/kg/day)$^{-1}$
Acenaphthene	AC	0.001	N/A (non-carcinogen)
Benzo[a]anthracene	BaA	0.1	0.70 (USEPA IRIS, 2018)
Benzo[b]fluoranthene	Bbf	0.1	0.11 (USEPA IRIS, 2018)
Benzo[k]fluoranthene	Bkf	0.1	0.11 (USEPA IRIS, 2018)
Acenaphthylene	AP	0.001	N/A (non-carcinogen)
Anthracene	A	0.01	N/A (non-carcinogen)
Benzo[ghi]perylene	BghiP	0.01	N/A (non-carcinogen)
Dibenzo[a,h]anthracene	DBahA	1.0	12.0 (USEPA IRIS, 2018)

TEF values from Nisbet and LaGoy (1992) as endorsed by EFSA (2008). CSF = oral cancer slope factor from USEPA IRIS database (2018). N/A = no established carcinogenicity.

RESULTS

BaP-Toxic Equivalents and Dietary Exposure

The BaP-equivalents (BaPeq) and estimated daily intakes (EDI) for each detected PAH congener are presented in Table 2. DBahA generated the highest BaPeq contribution (mean concentration × TEF 1.0 = 2.252–2.415 µg BaPeq/kg), reflecting both its high concentration and its TEF of 1.0 (equitoxic to BaP). BaA contributed the second highest BaPeq (mean 0.805–0.857 × TEF 0.1 = 0.081–0.086 µg BaPeq/kg), followed by Bbf and Bkf (TEF 0.1 each). AC, AP, BghiP, and A contributed minimally to BaPeq totals (TEF ≤ 0.01).

The combined PAH4 BaPeq for adult consumers ranged from 2.38 to 2.58 µg BaPeq/kg fish per day, yielding an EDI of 0.00096–0.00104 µg BaPeq/kg bw/day for adults (BW = 70 kg, IR = 0.0283 kg/day) and 0.00143–0.00155 µg BaPeq/kg bw/day for children (BW = 25 kg, IR = 0.015 kg/day), under daily consumption assumptions.

Table 2: BaP-Toxic Equivalents and Estimated Daily Intakes (EDI) for Detected PAH Congeners (Worst-Case Scenario: Highest Site Mean)

PAH	Max. conc. (µg/kg)	TEF	BaPeq (µg/kg)	EDI Adult (µg/kg bw/d)	EDI Child (µg/kg bw/d)	% BaPeq total
AC	0.052	0.001	5.2×10^{-5}	2.1×10^{-8}	3.1×10^{-8}	<0.01
BaA	0.857	0.1	0.0857	3.46×10^{-5}	5.14×10^{-5}	3.52
Bbf	1.100	0.1	0.110	4.44×10^{-5}	6.60×10^{-5}	4.52
Bkf	1.492	0.1	0.149	6.02×10^{-5}	8.95×10^{-5}	6.12
AP	2.250	0.001	0.00225	9.09×10^{-7}	1.35×10^{-6}	0.09
A	2.452	0.01	0.0245	9.90×10^{-6}	1.47×10^{-5}	1.01
BghiP	2.412	0.01	0.0241	9.74×10^{-6}	1.45×10^{-5}	0.99
DBahA	2.415	1.0	2.415	9.75×10^{-4}	1.45×10^{-3}	99.3
TOTAL BaPeq	—	—	2.432	9.82×10^{-4}	1.46×10^{-3}	100

BaPeq = benzo[a]pyrene equivalent. EDI = Estimated Daily Intake. Adult: BW = 70 kg, IR = 0.0283 kg/day. Child: BW = 25 kg, IR = 0.015 kg/day.

Incremental Lifetime Cancer Risk (ILCR)

ILCR values for individual carcinogenic PAH congeners are presented in Table 3. DBahA contributed the highest ILCR across all consumer groups and scenarios, with an adult ILCR of 1.13×10^{-6} and a child ILCR of 3.82×10^{-6}, placing both estimates within the 10^{-6} to 10^{-5} range (low to borderline acceptable risk per USEPA). BaA generated the second highest adult ILCR (4.07×10^{-7}) and child ILCR (1.38×10^{-6}). Bbf and Bkf contributed adult ILCRs of approximately 8.1×10^{-8} and 1.1×10^{-7} respectively. The cumulative ILCR from all carcinogenic congeners combined was 1.66×10^{-6} for adults and 5.56×10^{-6} for children, both within the low acceptable risk range (10^{-6} to 10^{-4}) but above the negligible risk threshold of 10^{-6}.

Table 3: Incremental Lifetime Cancer Risk (ILCR) for Carcinogenic PAH Congeners via Dietary Ingestion of Charcoal-Grilled Catfish in Makurdi

PAH	CSF $(mg/kg/d)^{-1}$	EDI Adult (µg/kg/d)	EDI Child (µg/kg/d)	ILCR Adult	ILCR Child	Risk Level
BaA	0.70	3.46×10^{-5}	5.14×10^{-5}	4.07×10^{-7}	1.38×10^{-6}	Low
Bbf	0.11	4.44×10^{-5}	6.60×10^{-5}	8.14×10^{-8}	2.76×10^{-7}	Negligible
Bkf	0.11	6.02×10^{-5}	8.95×10^{-5}	1.10×10^{-7}	3.74×10^{-7}	Negligible
DBahA	12.0	9.75×10^{-4}	1.45×10^{-3}	1.13×10^{-6}	3.82×10^{-6}	Low
Cumulative ILCR	—	—	—	1.66×10^{-6}	5.56×10^{-6}	Low

ILCR risk categories (USEPA 1989): Negligible $<10^{-6}$; Low 10^{-6}–10^{-4}; Unacceptable $>10^{-4}$. CSF = Cancer Slope Factor from USEPA IRIS (2018).

Non-Carcinogenic Hazard Quotients

Non-carcinogenic HQ values for all eight PAH congeners are presented in Table 4. All individual HQ values and the composite Hazard Index (HI) were below 1.0 for both adult and

child consumer groups, indicating no significant non-carcinogenic risk from dietary exposure to any individual PAH or to the total PAH mixture at the measured concentrations. The highest individual HQ was recorded for DBahA in children (HQ = 0.041×10^{-3}, negligible), consistent with the low EDI relative to the non-carcinogenic reference doses. These findings indicate that non-carcinogenic systemic effects from PAH ingestion through grilled catfish consumption, including hepatotoxicity, immunotoxicity, and reproductive toxicity, are unlikely to be manifested at the consumption rates modelled here.

Table 4: Non-Carcinogenic Hazard Quotients (HQ) for PAH Congeners via Dietary Ingestion of Charcoal-Grilled Catfish in Makurdi (Adults and Children)

PAH	RfD (mg/kg/d)	EDI Adult	HQ Adult	HQ Child	Risk Level
AC	6.00×10^{-2}	2.10×10^{-8}	3.5×10^{-10}	5.2×10^{-10}	Negligible
BaA	Not established	3.46×10^{-5}	—	—	(carcinogen, ILCR used)
Bbf	Not established	4.44×10^{-5}	—	—	(carcinogen, ILCR used)
Bkf	Not established	6.02×10^{-5}	—	—	(carcinogen, ILCR used)
AP	6.00×10^{-2}	9.09×10^{-7}	1.5×10^{-8}	2.2×10^{-8}	Negligible
A	3.00×10^{-1}	9.90×10^{-6}	3.3×10^{-8}	4.9×10^{-8}	Negligible
BghiP	3.00×10^{-2}	9.74×10^{-6}	3.2×10^{-7}	4.8×10^{-7}	Negligible
DBahA	7.30×10^{-5}	9.75×10^{-4}	1.3×10^{-2}	2.0×10^{-2}	Negligible (<1)
HI (Total)	—	—	<0.02	<0.03	Acceptable (HI <1)

RfD values from USEPA IRIS (2018). HQ = EDI/RfD. HI = sum of all HQ values. HQ or HI < 1 indicates no significant non-carcinogenic risk.

Margin of Exposure (MOE) Analysis

MOE values for the total BaPeq intake are presented in Table 5. Using the EFSA-recommended $BMDL_{10}$ of 170 µg BaPeq/kg bw/day, the calculated MOE was 173,469 for adults and 116,438 for children under the conservative (worst-case site) daily consumption scenario. Both values substantially exceed the EFSA threshold of 10,000, indicating that PAH exposure from charcoal-grilled catfish consumption in Makurdi is of low public health concern under current measurement conditions, and does not warrant an immediate risk management intervention. Under a more moderate consumption assumption (three servings per week rather than daily), MOE values increase approximately 2.3-fold, further reinforcing the low-concern classification.

Table 5: Margin of Exposure (MOE) Estimates for Dietary PAH Exposure via Charcoal-Grilled Catfish in Makurdi

Consumer Group	Total BaPeq in fish (µg/kg)	EDI BaPeq (µg/kg bw/d)	$BMDL_{10}$ (µg/kg bw/d)	MOE	EFSA Threshold
Adults (daily)	2.432	9.82×10^{-4}	170	173,100	10,000
Children (daily)	2.432	1.46×10^{-3}	170	116,400	10,000
Adults (3×/week)	2.432	4.20×10^{-4}	170	404,800	10,000
Children (3×/week)	2.432	6.26×10^{-4}	170	271,600	10,000

$BMDL_{10}$ = benchmark dose lower confidence limit for 10% incidence of cancer in rodent studies (EFSA, 2008). MOE > 10,000 = low public health concern.

DISCUSSION

Carcinogenic Risk from DBahA and BaA

The finding that DBahA is the dominant contributor to ILCR (adult: 1.13×10^{-6}; child: 3.82×10^{-6}) reflects both its consistently high concentration in charcoal-grilled catfish from Makurdi and its high cancer slope factor (CSF = 12.0 mg/kg/day^{-1}; USEPA IRIS, 2018) and TEF value of 1.0 — the highest of the EFSA-recognised PAH indicators, equitoxic to BaP on a molar basis. DBahA is an IARC Group 2A probable human carcinogen, classified on the basis of its potent genotoxicity in mammalian cell systems and carcinogenicity in rodent models at multiple tissue sites (skin, lung, and colon; IARC, 2012; Ferreira Azevedo et al., 2025). Its consistent detection across all three catfish species and all three sampling sites in this study, coupled with its negligible volatility and high lipophilicity (log Kow ≈ 6.8), underlines both its bioaccumulation potential in fish tissue and its persistence in the human gastrointestinal tract after ingestion.

BaA's contribution to cumulative ILCR (adults: 4.07×10^{-7}; children: 1.38×10^{-6}) is attributable to its relatively high CSF (0.70 mg/kg/day^{-1}) and its classification as a Group 2A carcinogen. Although BaA concentrations in this study (mean 0.785–0.857 µg/kg) were substantially lower than those reported for traditionally smoked Nigerian fish (Akpambang et al., 2009; Adeyeye et al., 2020), the chronic nature of daily grilled catfish consumption in Makurdi creates cumulative exposure conditions not captured by single-meal assessments. These findings are consistent with Benson et al. (2017) and Dwumfour-Asare et al. (2023), who documented low but cumulative BaA and DBahA risks in West African grilled and smoked fish.

The Role of DBahA as an Underregulated Priority Carcinogen

A critical implication of this study is the dominant role of DBahA in driving PAH-associated cancer risk, despite its absence from the EU PAH4 regulatory marker group (which comprises BaA, Bbf, Bkf, and BaP) and from the WHO single-marker system (BaP). The EFSA CONTAM Panel (2008) noted that DBahA is among the most potent of the eight PAH indicators (PAH8 group), but it is frequently undetected in studies using less sensitive analytical platforms. The present study detected DBahA in all 18 samples at concentrations of 2.127–2.415 µg/kg, representing the single most abundant PAH congener. This finding is consistent with Ferreira Azevedo et al. (2025), who demonstrated that high-molecular-weight PAH isomers including DBahA exhibit distinct genotoxic mechanisms from BaP, including cytotoxicity and oxidative stress-induced DNA damage, at environmentally relevant concentrations.

The absence of DBahA from PAH4 regulatory limits in both the EU framework and the draft Nigerian food safety guidelines for grilled products means that the most toxicologically significant PAH in this study's samples is entirely unregulated in the Nigerian food safety context. This represents a regulatory gap that, if unaddressed, may result in systematic underestimation of cancer risk to catfish consumers across Nigeria's north-central region.

Children as a Priority Vulnerable Group

Children consistently exhibited higher ILCR values (approximately 3.4-fold higher than adults for DBahA) under equivalent exposure assumptions, reflecting their lower body

weight relative to ingestion rate. The child ILCR for DBahA (3.82×10^{-6}) approaches the upper boundary of the 'low risk' range (10^{-6} to 10^{-4}; USEPA, 1989) and warrants precautionary attention, particularly given that children in Makurdi may begin dietary exposure to grilled catfish at ages as young as three to five years. Moreover, PAH exposure during childhood and adolescence is associated with disproportionate health effects relative to equivalent adult exposures, including genotoxic impacts on rapidly dividing cells, prenatal neurological effects, and epigenetic modifications that may manifest as cancer risk decades later (Perera *et al.*, 2014; Rengarajan *et al.*, 2015).

The reproductive and developmental toxicity of PAHs including benzo[a]pyrene-mediated induction of male infertility and embryotoxic effects of BaA documented in animal models (Bolden *et al.*, 2017; Smith *et al.*, 2007) further supports the classification of children and reproductive-age adults as priority sub-groups for exposure monitoring and dietary counselling in catfish-consuming communities.

MOE Analysis and Regulatory Implications

The MOE values computed in this study (>116,000 for all scenarios) substantially exceed the EFSA threshold of 10,000, indicating that current PAH exposures from charcoal-grilled catfish do not constitute an immediate or unmanageable public health risk for the Makurdi population under measured concentrations. This finding is consistent with PAH risk assessments in other West African grilled fish contexts, where Bandowe *et al.* (2014) and Dwumfour-Asare *et al.* (2023) similarly computed MOE values above 10,000 for charcoal-grilled tilapia and catfish, contrasting with MOE values below 10,000 for traditionally smoked products. Nevertheless, the MOE approach is inherently conservative and does not account for cumulative exposure from multiple PAH-contaminated dietary sources (cooking oils, smoked foods, atmospheric deposition on fruits and vegetables) that may substantially reduce effective margins in Nigerian urban populations (Tong *et al.*, 2018; Li *et al.*, 2019). A compound dietary PAH intake assessment incorporating all major Nigerian PAH sources would likely produce lower MOE values and may shift risk classifications upward.

Mitigation Strategies for PAH Reduction

Based on the mechanistic literature and the results of this study, several evidence-based mitigation strategies can be recommended for charcoal-grilled catfish vendors in Makurdi. First, increasing the fish-to-charcoal distance to 15–20 cm and pre-heating charcoal fully before placing fish on the grill substantially reduces PAH deposition during the highest-smoke phase of combustion (Rose et al., 2015; Barbosa et al., 2023). Second, marination with antioxidant-rich ingredients such as garlic, onion, and spice combinations containing diallyl disulfide has been shown to reduce 16 EPA PAHs by up to 39% in comparable protein matrices (Farhadian *et al.*, 2011; Wongmaneepratip *et al.*, 2019). Third, wrapping fish in aluminium foil during grilling significantly reduces PAH deposition by intercepting smoke contact with the food surface (Chen and Chen, 2016; Sampaio et al., 2021). Transition toward cleaner fuel alternatives including liquefied petroleum gas (LPG), electric grills, or certified low-emission briquettes would represent the most comprehensive structural intervention for reducing PAH risk at population scale (Dutta et al., 2022; Jelonek et al., 2020).

CONCLUSIONS

This study presents the first dietary carcinogenic risk assessment for PAH exposure from charcoal-grilled catfish in Makurdi, Nigeria, integrating measured PAH concentrations with the USEPA ILCR model and EFSA MOE approach for adults and children. DBahA, an IARC Group 2A probable human carcinogen unregulated under current EU PAH4 and Nigerian food safety frameworks, was identified as the dominant driver of carcinogenic risk, contributing approximately 68% of total cumulative ILCR across consumer groups. Cumulative ILCRs fell within the low acceptable risk range (10^{-6} to 10^{-4}) for both adults and children under daily consumption assumptions, and MOE values exceeded the EFSA threshold of 10,000 by more than one order of magnitude, indicating that current exposures do not constitute an immediate public health emergency. Nevertheless, the consistent detection of multiple IARC Group 2A and 2B carcinogens, the absence of national Nigerian regulatory limits for PAHs in grilled fish, and the higher relative risk for children together underscore the urgent need for: (i) establishment of Nigerian national maximum limits for PAHs including DBahA in charcoal-grilled fish; (ii) periodic PAH monitoring of commercial grilling establishments by food safety authorities; (iii) promotion of evidence-based mitigation practices among vendors; and (iv) targeted health communication to consumers, particularly parents and caregivers of young children.

Conflict of Interest

The authors declare no conflict of interest.

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