

Human Health Risk Assessment of Polycyclic Aromatic Hydrocarbons in Red Meat and Chicken From Southwest Iran

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Abstract

Introduction: Polycyclic aromatic hydrocarbons (PAHs) are harmful environmental pollutants with potential carcinogenic effects.

Methods: This study assessed the health risks of PAHs from red meat and chicken consumption in southwest Iran by analyzing 16 PAH compounds in 28 samples (14 red meats and 14 chickens) using gas chromatography-mass spectrometry (GC-MS). The hazard quotient (HQ) index was used to determine noncarcinogenic risk, and the incremental lifetime cancer risk (ILCR) index was used to assess carcinogenic risk. The results were analyzed using Excel and SPSS software.

Results: The mean total PAH concentration was higher in red meat (22.207 µg/kg) than in chicken (18.53 µg/kg), with Benz (a)anthracene having the highest maximum concentration (17.04 µg/kg). The average benzo (a)pyrene (BaP) concentration in Lamb and veal meat samples exceeded the European Union maximum limit (2 µg/kg). The results indicated that HQ was 0.01, and the highest ILCR was 2.2×10^{-5} , both found for children consuming chicken thigh. These values are well below the safety thresholds of 1 and 1×10^{-4} , respectively, showing no significant cancer risk from red meat and chicken consumption.

Conclusion: Although the consumption of these foods indicates a low risk level, chronic exposure to multiple PAHs, especially in children, warrants caution. Therefore, regular monitoring and assessment are recommended.

Keywords: Polycyclic aromatic hydrocarbons (PAHs), Red meat, Chicken, Health risk assessment

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Introduction

Polycyclic aromatic hydrocarbons (PAHs) result from the incomplete combustion of organic materials (1–3). They are present in industrial waste, trash, cigarette smoke, and chimney gases, and they have the potential to contaminate air, water, soil, and food (4–6). Detecting PAHs in the environment is of great importance due to their widespread presence and potential health risks (7).

Exposure of animals to PAHs is a significant concern due to the potential health risks and adverse effects on animals (8). This exposure can occur in animals such as dairy cows and fattening pigs through consuming contaminated water and feed (3). The levels of PAH exposure in animal feed have been determined, as contamination of feed crops and factory feed with PAHs indirectly affects human health (3,9). These harmful compounds have been detected in various animal tissues, including the kidney, liver,

lungs, muscles, adipose tissue, and even the eyeball (10). When parts of these animals are marketed as food, the accumulated PAHs can enter the human organism (5).

Given that meat (livestock and poultry) is the primary type of meat consumed by humans today, its quality is closely monitored and verified. Meat products are key providers of lipids, carbohydrates, proteins, vitamins, minerals, and water for humans (11,12). Toxic compounds like PAHs need to be examined due to their significant health risks (13) as PAHs are major carcinogenic compounds found in food products, including meat (14,15).

Accordingly, numerous studies have been conducted to examine the factors influencing the formation of PAHs in red or white meat products and to assess their harmful and carcinogenic effects worldwide (16–23) and in Iran (8,11,24–26).

Their findings suggest that the presence of PAHs in



(red or white) meat products is influenced by both the environmental levels of these pollutants (8,13,15,16,27) and the physiological and ecological characteristics of the meat item (6,19,22,23,28). Additionally, the amount of PAHs ingested from food depends on various cooking methods and potential contamination from manufacturing and packaging materials (2,11,18,24–26,29–33).

Based on the literature, on the one hand, the primary focus has typically been on measuring PAH compounds in processed meats and in cooked, fried, and grilled meat, revealing a significant gap in research on measuring PAH compounds in raw red meat and chicken. On the other hand, in industrial environments like Khuzestan province in southwest Iran, where food sources are exposed to carcinogenic pollutants such as PAHs, it is crucial to examine red meat and chicken for contamination before processing. These two factors underscore the need for this study to assess the health risks associated with dietary PAH exposure from consuming red meat and chicken in southwest Iran.

Materials and Methods

Sampling

Sampling procedure

Initially, 28 samples (14 chicken and 14 red meat) were collected from poultry and butcher shops in three cities (Shushtar, Dezful, and Andimeshk) of Khuzestan in southwest Iran (Figure 1) in May 2022.

The sample size was determined a priori using a standard formula for estimating a mean with specified precision [$n = (Z^2 \times sd^2) / d^2$]. A stratified random sampling approach was employed to ensure the sample was representative of the entire community. The stratification was based on urban zones with varying population densities (e.g., high, medium, and low density) within each city. First, a list of all registered poultry and butcher shops was obtained for each city. Then, shops were categorized according to the population density of their location. A predetermined number of shops were then randomly selected from each population-density stratum using a computer-generated random number sequence. This method ensured that our sample comprehensively represented the different

demographic contexts across the studied cities. All samples were promptly transported to the lab in an insulated cooler with ample ice and stored in a freezer at -70°C until analysis.

Sample preparation

The liquid-liquid extraction method was employed to extract PAH compounds, which were subsequently analyzed using gas chromatography-mass spectrometry (GC-MS).

In liquid-liquid extraction, 10 g of each sample was spiked with internal standards and extracted five times by sonication with a 50 mL hexane-dichloromethane mixture (1:1, v/v) for 15 minutes at room temperature. Copper granules were added to the samples to remove elemental sulfur for 24 hours. The extracts were then concentrated and purified using open-column chromatography, which included 5 g of activated silica, 10 g of neutral aluminum oxide, and 1 g of anhydrous sodium sulfate in ascending order. Finally, the extracts were concentrated to 5 mL under a gentle stream of N_2 .

Standard preparation and analysis

Standard preparation

A GC-MS system (GC Agilent 7890 and MS Agilent 5975, USA) was used to determine PAH concentration using a separating column (HP-5, 30 m $\times$ 0.25 mm ID, and 0.25 μm). The furnace heating cycle was started at 60°C for 1 min and gradually increased ($5^\circ\text{C}/\text{min}$) to 290°C . The furnace temperature remained at 290°C for 2 min to remove the remaining analytes from the column. Measurements were done in triplicate to obtain reliable results.

Method validation

The method was validated by assessing the limit of detection (LOD), limit of quantification (LOQ), linearity, accuracy, and precision. The LOD and LOQ were determined according to the European Commission's guidelines for 16 PAHs (34). Linearity was evaluated using a five-point calibration curve (0.5 to 20 $\mu\text{g}/\text{kg}$), which demonstrated excellent correlation for all analytes with correlation coefficients (R^2) exceeding 0.99. Accuracy and precision

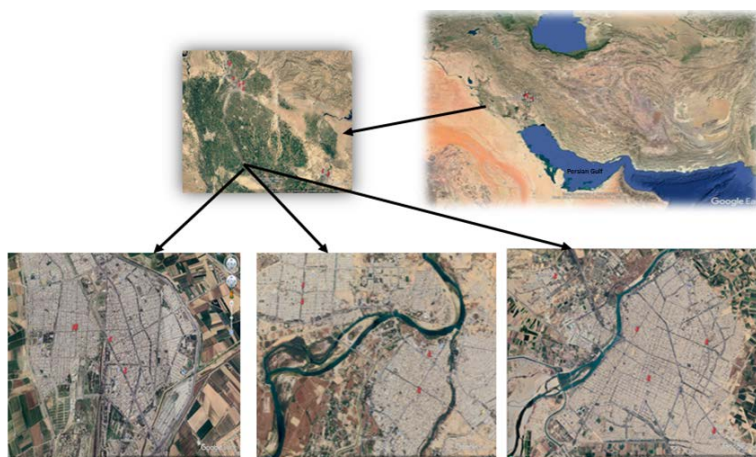


Figure 1. Study zones and sampling points

were determined through recovery experiments. Blank samples were spiked with PAHs at concentrations ranging from 0.5 to 10 µg/kg. To assess these parameters, each spiked concentration level was prepared and analyzed as three independent samples ($n=3$). The method demonstrated good accuracy, with mean recovery rates ranging from 84.2% to 93.4%. The precision of the method, expressed as the relative standard deviation (RSD) of the triplicate measurements, was consistently below 8.3% across all spiking levels. These results confirm that the method is reliable and fit for purpose.

Statistical analysis

All analyses were performed using SPSS version 22. The data distribution was first assessed to determine if it was normal or non-normal. The Mann-Whitney test compared PAH concentrations between chicken and red meat, while the Kruskal-Wallis test evaluated differences in PAH levels across various meat types (veal, lamb, chicken thigh, and chicken breast). The Wilcoxon one-sample test compared PAH concentrations in samples to the WHO standards. The use of non-parametric tests was necessitated by the non-normal distribution of the PAH concentration data, which was confirmed using the Shapiro-Wilk test ($P<0.05$). Furthermore, the data exhibited heteroscedasticity, as assessed by Levene's test. Given these conditions and the relatively small sample size, non-parametric tests were deemed the most appropriate analytical method. A significance level of 5% was set, with P values <0.05 indicating significant differences. A heat map analysis was created and visualized using the ClustVis software (<https://biit.cs.ut.ee/clustvis/>), an online tool that employs Euclidean distance.

Risk assessment

To estimate a non-carcinogenic hazard of PAHs in chicken and meat samples, chronic daily intake (CDI) was calculated by the following equation:

$$CDI = \frac{C \times IR \times EF \times ED}{BW \times AT} \quad (1)$$

In this context, C represents the concentration of PAHs in meat and chicken (mg/kg), and IR_i denotes the daily intake of meat and chicken. We used values of 16.2 g/day for red veal, 11.3 g/day for lamb, and 65.4 g/day for chicken, which were derived from local dietary intake data for Khuzestan (35). ED refers to the exposure duration to PAHs, which is 70 years for adults and 6 years for children. EF is the frequency of PAH exposure, assumed to be 350 days per year (36). BW is the average body weight, with adults averaging 70 kg and children 15 kg (WHO). AT is the average time of exposure, calculated as 25,550 days for adults (70 years $\times$ 365) and 2,190 days for children (6 years $\times$ 365 days) (36,37).

Health risk assessment based on the non-cancer risk hazard was calculated using Eq. (2).

$$HQ = \frac{CDI}{RfD} \quad (2)$$

Where HQ is the hazard quotient for exposure to pollutants via dietary intake, CDI is chronic daily intake (mg/kg day), and RfD is the reference dose for exposure to pollutants via dietary intake (mg/kg day). HQ values less than 1 indicate no significant risk, while values equal to or greater than 1 suggest a potential health risk (USEPA 2017) (38).

The total toxic equivalent quotient (TEQ) determined the carcinogenic risk of PAHs (39). Carcinogenic risk for PAH mixture compounds was determined using the toxicity equivalency factor (TEF) and BaP equivalent dose (BaPeq) (Table 1) (40).

Therefore, the toxicity equivalent quotient was determined by the sum of TEF multiplied by each PAH concentration based on Eq. (3), EPA (2010) (41).

$$\Sigma TEQ = \Sigma (C_i \times TEF_i) \quad (3)$$

where C is the concentration of an individual PAH, and TEF_i is the toxicity equivalency factor in Chicken and red meat.

The incremental lifetime carcinogenic risk (ILCR) for BaP in chicken and red meat was also calculated using Eq. (4).

$$ILCR = CDIBaP \times SFBaP \quad (4)$$

In this calculation, $ILCR$ represents the incremental lifetime cancer risk, CDI is the chronic daily intake of BaP (mg/kg day⁻¹), and CSF is the oral cancer slope factor for BaP, which is 7.3 (mg/kg day⁻¹) (42).

According to the USEPA (2016), the cancer risk is considered negligible if $ILCR < 10^{-6}$. When $ILCR > 10^{-4}$, the cancer risk is deemed unacceptable. When $ILCR$ falls between 10^{-6} and 10^{-4} , the cancer risk is considered tolerable for consumers (42,43).

Results

Validation study

The complete results of the method validation, including LOD, LOQ, recovery, RSD, and R^2 for all 16 PAHs, are presented in Table 2.

The method demonstrated high sensitivity, with average LOD and LOQ values of 0.85 µg/kg and 2.80 µg/kg, respectively. The accuracy, determined through recovery

Table 1. PAHs and their toxic equivalent factors (TEFs)

PAHs	TEF	PAHs	TEF
Benzo (a)pyrene (BaP)	1	Anthracene (A)	0.01
Dibenz (a, h)anthracene (DahA)	1	Naphthalene (NA)	0.001
Benzo (k)fluoranthene (BkF)	0.1	Acenaphthylene (AC)	0.001
Indeno (1,2,3-cd)pyrene (IcdP)	0.1	Acenaphthene (ACE)	0.001
Benz (a)anthracene (BaA)	0.1	Phenanthrene (PHE)	0.001
Benzo (b)fluoranthene (BbF)	0.1	Fluorine (FLO)	0.001
Chrysene (CHR)	0.01	Pyrene (PYR)	0.001
Benzo (g, h, i)perylene (BghiP)	0.01	Fluoranthene (FL)	0.001

experiments, showed an overall mean recovery of 88%, with individual compound recoveries ranging from 85% to 92.9%. The precision, expressed as the relative standard deviation (RSD), was within acceptable limits. Furthermore, all calibration curves exhibited strong linearity, with correlation coefficients (R^2) greater than 0.99.

PAH concentration in red meat and chicken

The average level of PAHs in all samples (including chicken breast, chicken thigh, veal, and lamb) was 20.37 $\mu\text{g/kg}$, with

Table 2. Median concentration, LOD, LOQ, recovery rate, and R^2 for the PAHs in the samples

Compound	Median ($\mu\text{g/kg}$)	LOD ($\mu\text{g/kg}$)	LOQ ($\mu\text{g/kg}$)	RSD	Recovery	R^2
NA	2.75	0.09	0.30	5.2	85.0	0.993
AC	0.915	0.35	1.16	3.9	91.0	0.997
ACE	1.16	0.25	0.84	6.3	84.5	0.991
FLO	1.62	0.60	1.97	7.4	90.3	0.999
PHE	0.355	0.18	0.60	6.2	84.2	0.996
A	0.000	0.21	0.69	3.8	89.9	0.994
FL	0.815	0.54	1.78	4.6	87.1	0.991
PYE	2.065	0.21	0.69	5.1	93.4	0.997
B (a)A	0.000	0.97	3.21	4.7	86.3	0.990
CHR	1.585	1.92	6.34	6.6	85.9	0.997
B (b)F	0.000	1.31	4.31	7.3	88.4	0.997
B (k)F	0.000	1.06	3.51	3.8	85.2	0.994
B (a)P	1.315	1.51	4.98	4	87.3	0.993
DahA	0.015	1.39	4.57	7.8	89.2	0.994
IcdP	1.490	0.89	2.93	8.3	91.3	0.999
BghiP	0.900	2.12	6.98	6.9	92.9	0.996

a standard deviation of 6.46, reflecting a moderate degree of variability in PAH concentrations. The minimum observed level of PAHs was 0.00 $\mu\text{g/kg}$, while the maximum reached 17.04 $\mu\text{g/kg}$, representing the range of PAH concentrations in the dataset. A heat map was employed to classify the PAH analysis process using a cluster dendrogram illustrated in Figure 2.

As shown in Table 3, the lowest average concentration among all PAH compounds was for B (b)F, at 0.00 $\mu\text{g/kg}$ in nearly all samples.

An independent Mann-Whitney U test was performed to evaluate the impact of two meat types (chicken versus red meat) on PAH compound levels. The test revealed a significant difference in PAH levels between chicken and red meat, as shown in Figure 3.

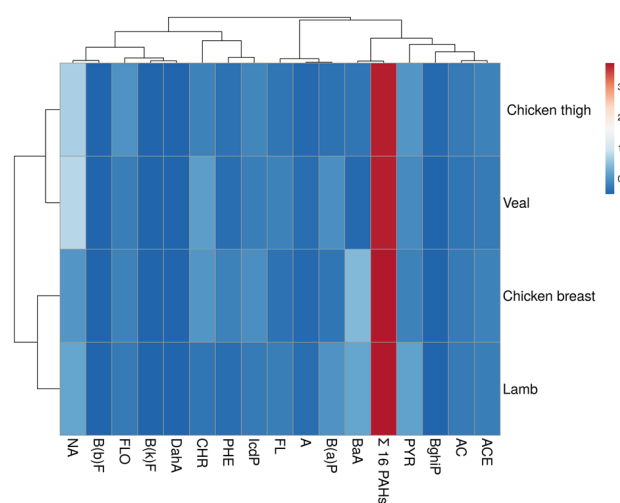


Figure 2. Heat map of PAHs in different meat samples

Table 3. Descriptive analysis of PAHs based on meat types

Meat types				
PAHs	Chicken breast	Chicken thigh	Lamb	Veal
NA	2.237 ^a (1-3.6) ^b	5.232 (0.74-11.9)	3.565 (2.01-9.77)	6.725 (2.04-13.06)
AC	0.935 (0.9-2.3)	1.019 (0.08-1.85)	1.199 (0.39-1.89)	0.959 (0.26-1.92)
ACE	1.279 (0.6-2.8)	1.381 (0.8-2.5)	1.493 (0.41-2.67)	1.158 (0.25-2.33)
FLO	1.283 (0.04-2.6)	2.268 (1.5-3.3)	1.159 (0.06-2.58)	1.367 (0.17-2.38)
PHE	1.320 (0.0-5.6)	0.670 (0.0-3.1)	0.554 (0.0-1.45)	0.452 (0.0-0.82)
A	0.236 (0.0-0.7)	0.211 (0.0-1.4)	0.609 (0.0-1.44)	0.489 (0.0-2.09)
FL	0.561 (0.2-1.6)	0.834 (0.24-2.22)	1.409 (0.82-1.91)	1.671 (0.35-6.77)
PYR	1.264 (0.8-1.7)	2.408 (1.06-3.72)	3.266 (1.71-4.49)	2.196 (0.05-5.34)
B (a)A	3.897 (0.0-9.7)	0.921 (0.0-6.4)	3.564 (0.0-17.04)	0.327 (0.0-2.28)
CHR	2.263 (1.1-3.7)	1.446 (0.0-3.9)	1.047 (0.0-1.88)	3.31 (0.6-7.21)
B (b)F	0.002 (0.0-0.01)	0.001 (0.0-0.006)	0.00 (0.0-0.0)	0.00 (0.0-0.0)
B (k)F	0.027 (0.0-0.16)	0.071 (0.0-0.26)	0.004 (0.0-0.31)	0.003 (0.0-0.02)
B (a)P	0.741 (0.2-1.97)	0.621 (0.09-1.7)	2.463 (1.24-3.42)	2.492 (1.28-5.05)
DahA	0.072 (0.0-0.18)	0.097 (0.0-0.2)	0.022 (0.0-0.1)	0.059 (0.0-0.21)
BghiP	0.082 (0.0-0.18)	0.174 (0.001-0.38)	0.162 (0.05-0.35)	0.041 (0.0-0.11)
IcdP	1.832 (0.0-3.04)	1.652 (0.95-2.2)	1.106 (0.67-1.57)	1.543 (1.35-1.81)
Σ16 PAHs	18.032 (4.8-39.8)	19.030 (5.38-45.3)	21.622 (7.36-48.2)	22.792 (6.35-51.4)

^a (average)

^b (ranges)

A Kruskal-Wallis test was conducted to examine the effect of different meat types on PAH levels, revealing significant differences among the samples ($r = 0.627$, $P < 0.05$).

Risk assessment of PAHs from red meat and chicken

The toxicity equivalency quotient (TEQ) of BaP was evaluated to estimate the suggested health risk, as shown in Table 4.

The outcome showed that red meat had a total TEQ_{BaP} (3.02 µg/kg) higher than chicken (1.40 µg/kg).

Non-carcinogenic and carcinogenic risk

The Hazard Quotient (HQ) was calculated to assess the risk

of non-cancerous effects, as shown in Table 5.

An HQ greater than or equal to one ($HQ > 1$) indicates a potential risk of non-cancerous effects. If the HQ value is below one, indicating the absence of non-cancerous risk.

As shown in Table 5, the ILCR model was utilized for the health risk assessment in southwest Iran caused by dietary PAH exposure. The U.S. Environmental Protection Agency (USEPA) has defined the acceptable ILCR range as less than 10^{-6} . Regarding the overall uncertainty in the lifetime cancer risk model, a one-in-a-million chance of additional human cancer over a 70-year lifetime from exposure to PAHs ($ILCR = 10^{-6}$) is an acceptable level, while a one-in-a-ten-thousand chance of exposure to PAHs ($ILCR = 10^{-4}$) or greater is a serious level (2).

Discussion

PAH concentration in red meat and chicken

Our analysis revealed a distinct profile of PAH contamination (Table 3). The compounds formed two clear clusters, with the first—comprising NA, B (a)A, and CHR—representing the most significant contaminants. Notably, NA was the most prevalent compound, with the highest mean concentration in veal (6.72 µg/kg) and the lowest one in chicken breast (2.23 µg/kg) across all samples, while potent carcinogens like B (b)F and B (k)F were undetectable. This specific profile, dominated by lower molecular weight PAHs like NA, suggests a contamination source distinct from high-temperature pyrolysis, potentially pointing to environmental absorption or low-temperature processes. This pattern aligns with the findings from raw buffalo meat in this region (8) and various smoked meats (2,30), indicating that NA's prevalence may be a common feature of pre-cooked meat contamination in certain agricultural regions, rather than a result of cooking methods.

A key finding was that the potent carcinogen B (a)P exceeded the EU regulatory limit (2 µg/kg) in red meat, particularly in lamb (2.46 µg/kg) and veal (2.49 µg/kg). This

Table 4. TEQ (toxic equivalent) concentration for individual PAHs

PAH Compounds	Veal	Lamb	Chicken thigh	Chicken breast
NA	6.85E-06	3.67E-06	5.27E-06	2.49E-06
AC	9.20E-07	1.16E-06	1.10E-06	9.94E-07
ACE	1.11E-06	1.44E-06	1.46E-06	1.35E-06
FLO	1.36E-06	1.32E-06	2.29E-06	1.35E-06
PHE	4.47E-07	4.94E-07	7.68E-07	1.50E-06
A	5.70E-06	5.76E-06	2.46E-06	2.02E-06
FL	1.80E-06	1.41E-06	9.28E-07	5.98E-07
PYR	2.18E-06	3.15E-06	2.57E-06	1.29E-06
BaA	2.54E-07	2.77E-06	1.07E-06	4.23E-06
CHE	3.52E-05	9.56E-06	1.29E-05	2.09E-05
BbF	0.00E+00	0.00E+00	1.01E-07	1.73E-07
BkF	3.10E-07	5.23E-07	8.12E-06	3.10E-06
BaP	2.68E-03	2.62E-03	6.98E-04	7.32E-04
DahA	2.84E-04	1.03E-04	5.24E-04	4.18E-04
BghiP	3.76E-07	1.50E-06	1.93E-06	9.17E-07
IcdP	1.53E-04	1.10E-04	1.61E-04	1.82E-04
ΣTEQ	3.18E-03	2.86E-03	1.42E-03	1.37E-03

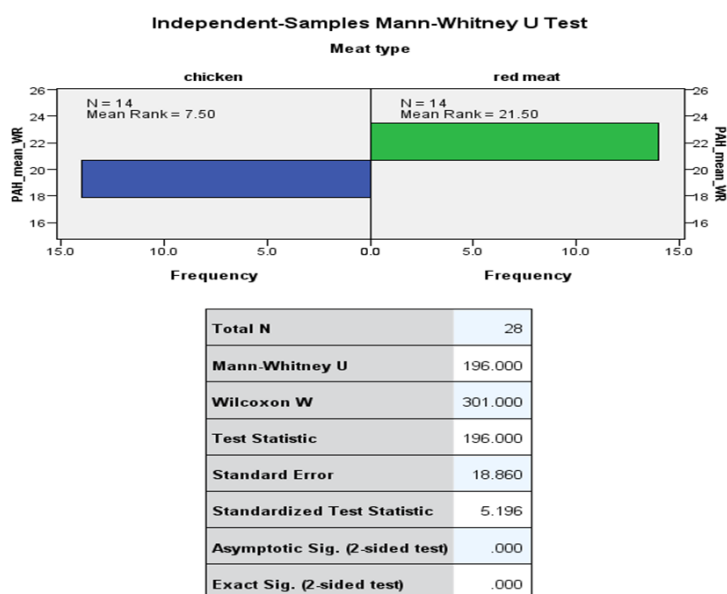


Figure 3. Independent-Samples Mann-Whitney U Test of meat types

Table 5. CDI, HQ, and ILCR of PAHs in the samples

	CDI		HQ		ILCR	
	Adult	Children	Adult	Children	Adult	Children
Veal	6.66667E-07	1.66667E-06	0.0024	0.005633333	5.375632400E-06	1.254159564E-05
Lamb	6.66667E-08	3.33333E-07	0.0015	0.001266667	3.366693257E-07	2.869250400E-06
Chicken thigh	1.26667E-06	3.06667E-06	0.0044	0.0103	9.706964899E-06	2.265166303E-05
Chicken breast	1.23333E-06	2.93333E-06	0.0043	0.009933333	9.367366605E-06	2.185919430E-05

is a critical public health insight, as it demonstrates that regulatory concerns exist even in raw, unprocessed products from this region. The consistency between our results and those of the study by Zare et al. (8) in local buffalo strongly implies a regional, pre-slaughter contamination source, likely from contaminated feed, water, or environmental exposure in southwestern Iran, as supported by other local environmental studies (3,9,11,18,20,23,25,33,44).

The maximum mean of $\Sigma 16$ PAHs was detected in veal at 22.8 $\mu\text{g}/\text{kg}$, while the minimum was in chicken breast at 18.03 $\mu\text{g}/\text{kg}$. The lowest average B (a)P concentration was in chicken thigh at 0.621 $\mu\text{g}/\text{kg}$. At the same time, lamb and veal samples had higher average concentrations of 2.46 and 2.49 $\mu\text{g}/\text{kg}$, respectively, exceeding the EU maximum allowable level of 2 $\mu\text{g}/\text{kg}$. Interestingly, our findings are consistent with those of the study by Zare et al, who reported elevated levels of $\Sigma 16$ PAHs and B (a)P in buffalos from the southwestern region of Iran (8). These findings suggested that animal exposure to PAHs is likely due to the consumption of contaminated water and feed (3,8,9).

When contextualizing our results, it is essential to note that they reflect PAH levels in raw meat, filling a significant gap in a literature dominated by studies on cooked and processed products. As expected, our $\Sigma\text{PAH}16$ concentrations were consistently and substantially lower than those reported in grilled, smoked, or fried products (11,18,20,23,25,33,44) where high-temperature cooking is a major generative source of PAHs.

More revealing are the comparisons with other raw or minimally processed meats. The levels we found were higher than those in kebabs from Tehran (24) but lower than in processed sausages and burgers (16,26,27). This variability suggests that pre-slaughter factors (like regional pollution and farming practices) can cause raw meat contamination to vary significantly, sometimes reaching levels comparable to lightly processed products. Furthermore, the specific PAH profile we observed—different from the profiles generated by grilling or smoking—reinforces the conclusion that the contamination in our samples originated from the animals' environment, not from food preparation.

Our statistical analysis (red meat mean rank: 21.50 vs. chicken: 7.50) and quantitative data confirm that red meat carries a significantly higher PAH burden than chicken, with a total $\Sigma\text{PAH}16$ of 22.207 $\mu\text{g}/\text{kg}$ compared to 18.53 $\mu\text{g}/\text{kg}$. This disparity is likely due to fundamental differences in animal husbandry; ruminants like cattle and sheep have longer lifespans and are often pasture-grazed, leading to greater cumulative exposure to environmental PAHs from soil, feed, and air, compared to poultry raised in controlled

settings with shorter lifespans.

This pattern is consistent with the broader literature. Mohammadi et al. also found higher PAHs in grilled beef than chicken, and Domingo et al. have consistently reported that white meats generally accumulate fewer persistent organic pollutants than red meats (16,26,27). More critically, our study adds a new layer of concern: we identified an alarmingly high maximum concentration of the potent carcinogen Benz (a)anthracene (17.04 $\mu\text{g}/\text{kg}$) in red meat. This highlights that the risk is not only one of overall burden but also of specific, high-potency carcinogens, underscoring a targeted health risk from red meat in this region.

The highest average PAH concentrations were observed in veal (22.8 $\mu\text{g}/\text{kg}$) and lamb (21.61 $\mu\text{g}/\text{kg}$), while the lowest were in chicken products, suggesting a clear ranking: veal > lamb > chicken. This pattern is consistent with studies of raw meats, which attribute higher levels in ruminants to their longer lifespan and grazing habits, leading to greater environmental exposure and bioaccumulation compared to poultry (19,29,45,46). The contrasting finding that grilled chicken can have higher PAH levels than grilled beef (16) does not contradict our results but rather highlights the fundamental difference between endogenous contamination in raw meat and exogenous contamination from cooking. In raw meat, the source is environmental (feed, soil, air), favoring accumulation in red meat. During grilling, the source is the cooking process itself, where factors like fat content and dripping can make chicken a significant PAH source.

Risk assessment of PAHs from red meat and chicken

The observed rank order of toxic equivalents (ΣTEQ)- veal (3.18 $\mu\text{g}/\text{kg}$) > lamb (2.86 $\mu\text{g}/\text{kg}$) > chicken thigh (1.42 $\mu\text{g}/\text{kg}$) > chicken breast (1.37 $\mu\text{g}/\text{kg}$)- provides a direct measure of the relative carcinogenic potency across meat types. This gradient strongly suggests that the increased risk is intrinsically linked to the animal's species and rearing practices, rather than to cooking methods (since the meat was raw). The significantly higher potency in ruminants (veal and lamb) likely reflects their longer lifespan and grazing-based diet, which lead to greater cumulative exposure to environmental PAHs from contaminated soil, feed, and airborne particulates in the region (29,47–54). This pattern underscores that pre-slaughter contamination in southwestern Iran is a critical public health issue. The findings necessitate targeted investigations into local environmental sources—such as industrial emissions, vehicle exhaust, and agricultural practices—that contribute

to the bioaccumulation of high-potency PAHs in the food chain, particularly in grazing livestock.

Non-carcinogenic and carcinogenic risk

According to Table 5, the HQ for both age groups across different types of meat was below one, indicating no significant risk of adverse effects. Overall, the findings of the present study are consistent with risk assessment frameworks and related studies in this area (5,45,47,49,50,55). This indicates that, despite the diversity in meat types and regional differences, the calculated non-carcinogenic risks from unprocessed meat fall within a range generally considered acceptable for both age groups based on current risk assessment guidelines.

Our analysis revealed a critical public health insight: despite red meat having higher PAH concentrations, the ILCR was the highest for chicken products (thigh: 2.26×10^{-5} ; breast: 2.18×10^{-5}) in children. Although the ILCR for the two groups (children and adults) is below the maximum acceptable risk level (10^{-4}), the potential for chronic exposure, especially in children, warrants a cautious interpretation. This counterintuitive finding is a direct consequence of the risk assessment framework, where higher consumption rates relative to lower body weight in children disproportionately elevate risk, even from matrices with lower contaminant levels. This heightened vulnerability underscores the importance of developing dietary guidelines aimed at limiting children's cumulative exposure to PAHs from all meat products, even those perceived as lower-risk.

When contextualized within the global literature, the cancer risk estimated in our study aligns with the lower end of the spectrum reported for various food categories. Our ILCR values are consistent with those found in similar products, such as those reported by Khalili et al. in Iran and Jiang et al. for grilled meats in China, which also fell below the 10^{-4} threshold (2,25).

However, a critical pattern emerges from this comparison: our risk estimates for raw meat are consistently and substantially lower than those reported for processed, grilled, or smoked meat products (11,17,22,30). This consistent disparity underscores that cooking and processing methods—such as grilling, frying, and smoking—are dominant factors in elevating PAH-related cancer risk, often generating high levels of potent compounds not present in raw tissue. Consequently, our findings establish an important baseline for the region, demonstrating that the intrinsic risk from pre-slaughter PAH contamination in raw meat is low, but this risk can be significantly amplified by subsequent food preparation practices.

Limitations and future directions

This study has limitations that should be considered when interpreting the results. The most notable is the relatively small sample size ($n=28$), which may affect the generalizability of the findings to the broader population across the three cities. Furthermore, the one-season sampling design offers a crucial snapshot but

cannot account for potential seasonal variations in PAH contamination due to factors like changes in animal feed or husbandry practices.

Despite these limitations, a key strength of this work is the use of measured PAH concentrations from locally sourced meat, providing a more accurate foundation for exposure assessment than studies relying on literature values. To build upon this foundation, future studies should prioritize: larger, multi-season sampling campaigns to establish robust annual averages and understand temporal trends; and the inclusion of advanced methodological parameters, such as measurement uncertainty, to further enhance analytical rigor. Addressing these points will enable a more precise and comprehensive characterization of the health risks associated with PAH exposure in this population.

Conclusion

Southwestern Iran revealed a significant baseline level of contamination and distinct profiles between meat types. While the incremental lifetime cancer risk was below the regulatory threshold of 10^{-4} for all samples, the findings underscore several key points for public health. Despite limitations including the sample size, use of international dietary parameters, and one-season sampling, the data consistently show that potent carcinogens like Benz (a) anthracene are present in red meat at concerning levels, and that children face a disproportionately higher risk.

These results translate into three critical recommendations: First, public health authorities should implement regular monitoring programs for PAHs in raw meat to establish baseline trends and ensure early detection of rising levels. Second, consumer awareness campaigns should emphasize that safer cooking methods (e.g., avoiding direct flame, using marinades) are essential to prevent the significant additional PAH formation that occurs during grilling or frying. Finally, environmental and agricultural agencies must investigate and mitigate sources of industrial and environmental contamination to reduce the pre-slaughter burden of PAHs in the food chain. This multi-pronged approach is essential to protect public health, particularly for vulnerable groups like children, from cumulative PAH exposure.

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Competing Interests

The authors have no relevant financial or non-financial interests to disclose.

Ethical Approval

Ethical approval for this study was obtained from the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (Ethical code: IR.AJUMS.REC.1401.033). This research did not involve human participants, and no informed consent was required. Additionally, no animals were used in this study.

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References

- Bansal V, Kumar P, Kwon EE, Kim KH. Review of the quantification techniques for polycyclic aromatic hydrocarbons (PAHs) in food products. *Crit Rev Food Sci Nutr* 2017;57(15):3297-312. doi:10.1080/10408398.2015.1116970
- Jiang D, Wang G, Li L, Wang X, Li W, Li X, et al. Occurrence, dietary exposure, and health risk estimation of polycyclic aromatic hydrocarbons in grilled and fried meats in Shandong of China. *Food Sci Nutr* 2018;6(8):2431-9. doi:10.1002/fsn3.843
- Şahin T, Dalğa S, Ölmez M. Polycyclic aromatic hydrocarbons (PAHs) and their importance in animal nutrition. In: Kukovics S, ed. *Animal husbandry*. IntechOpen; 2022. doi:10.5772/intechopen.101816
- Abdel-Shafy HI, Mansour MS. A review on polycyclic aromatic hydrocarbons: source, environmental impact, effect on human health and remediation. *Egypt J Petrol* 2016;25(1):107-23. doi:10.1016/j.ejpe.2015.03.011
- Rey-Salgueiro L, García-Falcón MS, Martínez-Carballo E, Simal-Gándara J. Effects of toasting procedures on the levels of polycyclic aromatic hydrocarbons in toasted bread. *Food Chem* 2008;108(2):607-15. doi:10.1016/j.foodchem.2007.11.026
- Sampaio GR, Guizellini GM, da Silva SA, de Almeida AP, Pinaffi-Langley AC, Rogero MM, et al. Polycyclic aromatic hydrocarbons in foods: biological effects, legislation, occurrence, analytical methods, and strategies to reduce their formation. *Int J Mol Sci* 2021;22(11):6010. doi:10.3390/ijms22116010
- Aralu CC, Okoye PC, Abugu HO, Eboagu NC, Eze VC. Characterization, sources, and risk assessment of PAHs in borehole water from the vicinity of an unlined dumpsite in Awka, Nigeria. *Sci Rep* 2023;13(1):9688. doi:10.1038/s41598-023-36691-3
- Mostajabi E, Moghainami SR, Jazayeri F, Guodarzi BG, Zare MA. Polycyclic aromatic hydrocarbons (PAHs) traceability in old buffalos living in south west part of Iran. *Arch Razi Inst* 2006;61(2):105-10. doi:10.22092/ari.2006.103790
- Kan CA, Traag WA, Hoogenboom LA. Voorkomen van PAK's in voer, omgeving van dieren, melk en zuivelproducten alsmede een oriënterende studie in melkvee. Lelystad: Animal Sciences Group; 2003.
- Ji J, Jiang M, Zhang Y, Hou J, Sun S. Polycyclic aromatic hydrocarbons contamination in edible oils: a review. *Food Rev Int* 2023;39(9):6977-7003. doi:10.1080/87559129.2022.2131816
- Samiee S, Fakhri Y, Sadighara P, Arabameri M, Rezaei M, Nabizadeh R, et al. The concentration of polycyclic aromatic hydrocarbons (PAHs) in the processed meat samples collected from Iran's market: a probabilistic health risk assessment study. *Environ Sci Pollut Res Int* 2020;27(17):21126-39. doi:10.1007/s11356-020-08413-z
- Shariatifar N, Ebadi Fathabad A, Madihi S. Antibacterial activity of aqueous and ethanolic extracts of *Echium amoenum* on food-borne pathogens. *J Food Saf Hyg* 2016;2(3-4):63-6.
- Wang J, Zhang H, Cai J, Li J, Sun B, Wu F. Effects of different carbon substrates on PAHs fractions and microbial community changes in PAHs-contaminated soils. *Environ Pollut* 2023;324:121367. doi:10.1016/j.envpol.2023.121367
- Deutsch-Wenzel RP, Brune H, Grimmer G, Dettbarn G, Misfeld J. Experimental studies in rat lungs on the carcinogenicity and dose-response relationships of eight frequently occurring environmental polycyclic aromatic hydrocarbons. *J Natl Cancer Inst* 1983;71(3):539-44. doi:10.1093/jnci/71.3.539
- Falcó G, Domingo JL, Llobet JM, Teixidó A, Casas C, Müller L. Polycyclic aromatic hydrocarbons in foods: human exposure through the diet in Catalonia, Spain. *J Food Prot* 2003;66(12):2325-31. doi:10.4315/0362-028x-66.12.2325
- Domingo JL, Nadal M. Human dietary exposure to polycyclic aromatic hydrocarbons: a review of the scientific literature. *Food Chem Toxicol* 2015;86:144-53. doi:10.1016/j.fct.2015.10.002
- Duan X, Shen G, Yang H, Tian J, Wei F, Gong J, et al. Dietary intake polycyclic aromatic hydrocarbons (PAHs) and associated cancer risk in a cohort of Chinese urban adults: inter- and intra-individual variability. *Chemosphere* 2016;144:2469-75. doi:10.1016/j.chemosphere.2015.11.019
- Mastanjević K, Kartalović B, Petrović J, Novakov N, Puljić L, Kovačević D, et al. Polycyclic aromatic hydrocarbons in the traditional smoked sausage *Slavonska kobasica*. *J Food Compos Anal* 2019;83:103282. doi:10.1016/j.jfca.2019.103282
- Perelló G, Martí-Cid R, Castell V, Llobet JM, Domingo JL. Concentrations of polybrominated diphenyl ethers, hexachlorobenzene and polycyclic aromatic hydrocarbons in various foodstuffs before and after cooking. *Food Chem Toxicol* 2009;47(4):709-15. doi:10.1016/j.fct.2008.12.030
- Santos C, Gomes A, Roseiro LC. Polycyclic aromatic hydrocarbons incidence in Portuguese traditional smoked meat products. *Food Chem Toxicol* 2011;49(9):2343-7. doi:10.1016/j.fct.2011.06.036
- Turesky RJ. Mechanistic evidence for red meat and processed meat intake and cancer risk: a follow-up on the international agency for research on cancer evaluation of 2015. *Chimia (Aarau)* 2018;72(10):718-24. doi:10.2533/chimia.2018.718
- Xia Z, Duan X, Qiu W, Liu D, Wang B, Tao S, et al. Health risk assessment on dietary exposure to polycyclic aromatic hydrocarbons (PAHs) in Taiyuan, China. *Sci Total Environ* 2010;408(22):5331-7. doi:10.1016/j.scitotenv.2010.08.008
- Zachara A, Gałkowska D, Juszczak L. Contamination of smoked meat and fish products from Polish market with polycyclic aromatic hydrocarbons. *Food Control* 2017;80:45-51. doi:10.1016/j.foodcont.2017.04.024
- Eshaghi Gorji M, Ahmadvani R, Moazzen M, Yunesian M, Azari A, Rastkari N. Polycyclic aromatic hydrocarbons in Iranian kebabs. *Food Control* 2016;60:57-63. doi:10.1016/j.foodcont.2015.07.022
- Khalili F, Shariatifar N, Dehghani MH, Yaghmaeian K, Nabizadeh Nodehi R, Yaseri M, et al. Polycyclic aromatic hydrocarbons (PAHs) in meat, poultry, fish and related product samples of Iran: a risk assessment study. *J Environ Health Sci Eng* 2023;21(1):215-24. doi:10.1007/s40201-023-00854-1
- Mohammadi M, Valizadeh-Kakhki F. Polycyclic aromatic hydrocarbons determination in grilled beef and chicken. *Polycycl Aromat Compd* 2018;38(5):434-44. doi:10.1080/10406638.2016.1236824
- Domingo JL, Nadal M. Carcinogenicity of consumption of red and processed meat: what about environmental contaminants? *Environ Res* 2016;145:109-15. doi:10.1016/j.envres.2015.11.031
- Domingo JL, Nadal M. Carcinogenicity of consumption of red meat and processed meat: a review of scientific news since

- the IARC decision. *Food Chem Toxicol* 2017;105:256-61. doi:10.1016/j.fct.2017.04.028
29. Farhadian A, Jinap S, Abbas F, Sakar ZI. Determination of polycyclic aromatic hydrocarbons in grilled meat. *Food Control* 2010;21(5):606-10. doi:10.1016/j.foodcont.2009.09.002
 30. Li G, Wu S, Wang L, Akoh CC. Concentration, dietary exposure and health risk estimation of polycyclic aromatic hydrocarbons (PAHs) in youtiao, a Chinese traditional fried food. *Food Control* 2016;59:328-36. doi:10.1016/j.foodcont.2015.06.003
 31. Onyango AA, Lalah JO, Wandiga SO. The effect of local cooking methods on polycyclic aromatic hydrocarbons (PAHs) contents in beef, goat meat, and pork as potential sources of human exposure in Kisumu city, Kenya. *Polycycl Aromat Compd* 2012;32(5):656-68. doi:10.1080/10406638.2012.725195
 32. Shamsedini N, Dehghani M, Samaei M, Azhdarpoor A, Hoseini M, Fararouei M, et al. Health risk assessment of polycyclic aromatic hydrocarbons in individuals living near restaurants: a cross-sectional study in Shiraz, Iran. *Sci Rep* 2022;12(1):8254. doi:10.1038/s41598-022-12040-8
 33. Terzi G, Celik TH, Nisbet C. Determination of benzo[a]pyrene in Turkish döner kebab samples cooked with charcoal or gas fire. *Ir J Agric Food Res* 2008;47(2):187-93.
 34. European Commission. Opinion of the Scientific Committee on Food on the Risks to Human Health of Polycyclic Aromatic Hydrocarbons in Food. Brussels: European Commission; 2002. Available from: https://ec.europa.eu/food/fs/sc/scf/out153_en.pdf. Accessed December 4, 2002.
 35. Statistical Centre of Iran. Available from: <https://ssis.sci.org.ir/>.
 36. Office of Emergency and Remedial Response, US Environmental Protection Agency. Risk Assessment Guidance for Superfund, Part A. Human Health Evaluation Manual. Vol 1. Washington, DC: Office of Emergency and Remedial Response, US Environmental Protection Agency; 1989.
 37. Sinkakarimi MH, Mansouri B, Azadi NA, Maleki A, Davari B. Assessment of heavy metals in chicken meat distributed in Sanandaj, Iran, and calculating the food consumption risk. *J Mazandaran Univ Med Sci* 2017;26(146):128-38.
 38. Integrated Risk Information System (IRIS). Toxicological Review of Benzo[a]pyrene—Executive Summary (Final Report). IRIS; 2017.
 39. Nisbet IC, LaGoy PK. Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). *Regul Toxicol Pharmacol* 1992;16(3):290-300. doi:10.1016/0273-2300(92)90009-x
 40. Saito E, Tanaka N, Miyazaki A, Tsuzaki M. Concentration and particle size distribution of polycyclic aromatic hydrocarbons formed by thermal cooking. *Food Chem* 2014;153:285-91. doi:10.1016/j.foodchem.2013.12.055
 41. Benford D, Dinovi M, Setzer RW. Application of the margin-of-exposure (MoE) approach to substances in food that are genotoxic and carcinogenic eg: benzo[a]pyrene and polycyclic aromatic hydrocarbons. *Food Chem Toxicol* 2010;48 Suppl 1: S42-8. doi:10.1016/j.fct.2009.09.039
 42. Roudbari A, Rafiei Nazari R, Shariatifar N, Moazzen M, Abdolshahi A, Mirzamohammadi S, et al. Concentration and health risk assessment of polycyclic aromatic hydrocarbons in commercial tea and coffee samples marketed in Iran. *Environ Sci Pollut Res Int* 2021;28(4):4827-39. doi:10.1007/s11356-020-10794-0
 43. United States Environmental Protection Agency (USEPA). Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Washington, DC: USEPA; 2005.
 44. Chen YH, Xia EQ, Xu XR, Li S, Ling WH, Wu S, et al. Evaluation of benzo[a]pyrene in food from China by high-performance liquid chromatography-fluorescence detection. *Int J Environ Res Public Health* 2012;9(11):4159-69. doi:10.3390/ijerph9114159
 45. Aygün SF, Kabadayi F. Determination of benzo[a]pyrene in charcoal grilled meat samples by HPLC with fluorescence detection. *Int J Food Sci Nutr* 2005;56(8):581-5. doi:10.1080/09637480500465436
 46. Rose M, Holland J, Dowding A, Petch SR, White S, Fernandes A, et al. Investigation into the formation of PAHs in foods prepared in the home to determine the effects of frying, grilling, barbecuing, toasting and roasting. *Food Chem Toxicol* 2015;78:1-9. doi:10.1016/j.fct.2014.12.018
 47. Kamalabadi M, Kamankesh M, Mohammadi A, Hadian Z, Ferdowsi R. Contamination and daily intake of polycyclic aromatic hydrocarbons in Iranian bread samples. *Polycycl Aromat Compd* 2020;40(4):1187-95. doi:10.1080/10406638.2018.1534747
 48. Ranjbar Jafarabadi A, Mashjoor S, Riyahi Bakhtiari A, Jadot C. Dietary intake of polycyclic aromatic hydrocarbons (PAHs) from coral reef fish in the Persian Gulf - human health risk assessment. *Food Chem* 2020;329:127035. doi:10.1016/j.foodchem.2020.127035
 49. Barzegar G, Rezaei Kalantary R, Bashiry M, Jaafarzadeh N, Ghanbari F, Shakerinejad G, et al. Measurement of polycyclic aromatic hydrocarbons in edible oils and potential health risk to consumers using Monte Carlo simulation, southwest Iran. *Environ Sci Pollut Res Int* 2023;30(2):5126-36. doi:10.1007/s11356-022-22446-6
 50. Huang M, Penning TM. Processing contaminants: polycyclic aromatic hydrocarbons (PAHs). In: Mortarjemi Y, Moy G, Todd E, eds. *Encyclopedia of Food Safety*. Cambridge: Academic Press; 2014. p. 416-23. doi:10.1016/b978-0-12-378612-8.00212-2
 51. Zhao Z, Zhang L, Cai Y, Chen Y. Distribution of polycyclic aromatic hydrocarbon (PAH) residues in several tissues of edible fishes from the largest freshwater lake in China, Poyang Lake, and associated human health risk assessment. *Ecotoxicol Environ Saf* 2014;104:323-31. doi:10.1016/j.ecoenv.2014.01.037
 52. Hernández ÁR, Boada LD, Almeida-González M, Mendoza Z, Ruiz-Suárez N, Valeron PF, et al. An estimation of the carcinogenic risk associated with the intake of multiple relevant carcinogens found in meat and charcuterie products. *Sci Total Environ* 2015;514:33-41. doi:10.1016/j.scitotenv.2015.01.108
 53. Zhang Y, Chen X, Zhang Y. Analytical chemistry, formation, mitigation, and risk assessment of polycyclic aromatic hydrocarbons: from food processing to in vivo metabolic transformation. *Compr Rev Food Sci Food Saf* 2021;20(2):1422-56. doi:10.1111/1541-4337.12705
 54. Lawley R, Curtis L, Davis J. *The Food Safety Hazard Guidebook: The Royal Society of Chemistry*; 2012. doi:10.1039/9781849734813
 55. Haiba NS, Asaal AM, El Massry AM, Ismail I, Basahi J, Hassan IA. Effects of “doneness” level on PAH concentrations in charcoal-grilled beef and chicken: an Egyptian study case. *Polycyclic Aromatic Compounds* 2021;41(3):553-63. doi:10.1080/10406638.2019.1602062