

Review

Polycyclic Aromatic Hydrocarbons in Meat Products: Formation, Detection and Regulatory Challenges

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Abstract

Polycyclic aromatic hydrocarbons (PAHs) are hazardous contaminants formed during incomplete combustion and frequently detected in smoked, grilled, roasted, and fried meat products. Their formation is influenced by temperature, smoking duration, fuel type, fat content, and the distance from the heat source. Available studies indicate that traditionally smoked and charcoal-grilled products contain higher PAH concentrations than raw or less processed meat. Under Regulation (EU) 2023/915, the maximum permitted levels are 2 µg/kg for benzo[a]pyrene (BaP) and 12 µg/kg for the sum of four marker PAHs (PAH4) in standard smoked meat products, while derogation limits of 5 and 30 µg/kg, respectively, apply to certain traditionally smoked products. Chromatographic methods enable sensitive PAH determination, with limits of detection of 0.003–0.200 µg/kg reported for high-performance liquid chromatography with fluorescence detection (HPLC–FLD) of meat products. Literature data indicate that PAH formation can be reduced through indirect smoking, appropriate fuel selection, smoke purification, prevention of fat dripping, and antioxidant-rich marinades. Overall, effective PAH control requires optimized processing technologies, sensitive analytical monitoring, and compliance with regulatory standards while preserving product quality and traditional practices. Unlike reviews focusing on individual aspects, this review integrates current knowledge on PAH formation, occurrence, health risks, analytical determination, legislation, and mitigation within a single meat-focused framework.

Keywords: polycyclic aromatic hydrocarbons; meat; benzo[a]pyrene; chromatographic analysis; mitigation strategies

1. Introduction

Meat and meat products remain essential components of the human diet due to their high nutritional value, as they contain proteins, amino acids, zinc, iron, vitamin B12, and minerals necessary for proper functioning of the human brain and overall health [1–3]. Since meat is rich in nutrients that provide ideal conditions for the growth of spoilage microorganisms, the preservation of meat and meat products is of paramount importance [4]. Today, various meat preservation methods are employed to inhibit microbial growth, delay spoilage processes, extend shelf life, and ensure the production of safe meat products for consumers [4,5]. Fundamental food processing techniques include grilling, boiling, blanching, frying, baking, roasting, stewing, drying, and smoking [6,7]. Among these, smoking is one of the oldest and most widely used methods of food preservation. This process extends the shelf life of food products and contributes to the development of sensory properties



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such as flavor, aroma, and color. Additionally, smoke exhibits antibacterial and antioxidant effects [8–10]. These effects are mainly attributed to the presence of phenolic compounds, organic acids, and carbonyl compounds generated during wood pyrolysis [8]. Furthermore, smoke contains a wide range of chemical compounds, including phenols, aldehydes, acetic acid, and polycyclic aromatic hydrocarbons (PAHs) [11]. In addition to PAHs, smoking may also lead to the formation of other potentially harmful compounds, such as heterocyclic aromatic amines (HAAs) and nitrosamines [12]. Among these contaminants, PAHs have received particular attention due to their occurrence in smoked meat products, as their concentrations of up to 200 µg/kg have been detected in smoked meat and fish [8,13]. PAHs are a large group of environmental pollutants formed during the incomplete combustion of organic materials, such as wood, petrol, and coal [14]. These processes may occur naturally or as a result of anthropogenic activities [13]. PAHs are also generated through the pyrolysis of organic materials at high temperatures [13,14]. Structurally, they consist of two or more fused benzene rings arranged in linear, angular, or cluster configurations and are composed solely of carbon and hydrogen atoms [13]. PAHs are commonly classified according to their molecular weight and the number of fused aromatic rings. Low-molecular-weight PAHs, containing two to three benzene rings, are more volatile and therefore occur predominantly in the gaseous phase. In contrast, high-molecular-weight PAHs, containing four or more rings, are less volatile, more chemically stable, and tend to adsorb onto particulate matter [13,15,16]. These compounds are generally considered of greater toxicological concern because several high-molecular-weight PAHs exhibit stronger mutagenic and carcinogenic properties than lower-molecular-weight PAHs [14,16,17]. Wenzl and Zelinkova [18] reported that elevated concentrations of PAHs in smoked food products are most commonly associated with improper smoking practices, including direct exposure of food to hot smoke, embers or open flames, excessive smoking intensity, high smoking temperatures, and insufficient distance between the product and the heat source. PAHs are known to exert carcinogenic and mutagenic effects and may adversely affect the immune system, thereby reducing overall immune resistance [13]. The United States Environmental Protection Agency (EPA) has identified 16 priority PAHs that are commonly detected in environmental samples. These include acenaphthene, acenaphthylene, anthracene, fluoranthene, fluorene, naphthalene, phenanthrene, pyrene, benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, benzo[ghi]perylene, benzo[a]pyrene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene [19]. The classification of selected PAHs according to different regulatory and analytical frameworks, including the 16 U.S. EPA priority PAHs (EPA 16), the 16 EU priority PAHs (EU 15 + 1), the sum of eight marker PAHs (PAH8), and the sum of four marker PAHs (PAH4), is presented in Table 1, adapted from [20]. In the assessment of food contamination, the PAH4 group comprising benzo[a]pyrene, benz[a]anthracene, benzo[b]fluoranthene, and chrysene is most commonly applied as an indicator of PAH occurrence and toxicity [21]. Therefore, a thorough understanding of PAH formation mechanisms, associated health risks, and mitigation strategies is essential for improving the safety of meat products [22]. Although substantial research has been conducted on PAHs in meat and meat products, current knowledge remains dispersed across studies addressing individual aspects, including formation mechanisms [23], occurrence [24], analytical methodologies [25], health implications [26,27], regulatory requirements [28], and mitigation strategies [29]. Accordingly, this review provides an updated and comprehensive overview of PAH formation, occurrence, analytical determination, regulatory requirements, health implications, and mitigation strategies, offering an integrated perspective on PAH control in meat products.

Table 1. Classification of selected PAHs according to EPA, EU, PAH8 and PAH4 priority groups.

Abbreviation	Compound	EPA Priority PAHs (16)	EU Priority PAHs (15 + 1)	PAH8	PAH4
Acy	Acenaphthylene	✓			
Acn	Acenaphthene	✓			
Ant	Anthracene	✓			
BaA	Benz[a]anthracene	✓	✓	✓	✓
BbF	Benzo[b]fluoranthene	✓	✓	✓	✓
BjF	Benzo[j]fluoranthene		✓		
BkF	Benzo[k]fluoranthene	✓	✓	✓	
BcF	Benzo[c]fluorene		✓		
BaP	Benzo[a]pyrene	✓	✓	✓	✓
BghiP	Benzo[ghi]perylene	✓	✓	✓	
Chr	Chrysene	✓	✓	✓	✓
CcdP	Cyclopenta[cd]pyrene		✓		
DBahA	Dibenz[a,h]anthracene	✓	✓	✓	
DBaeP	Dibenzo[a,e]pyrene		✓		
DBaiP	Dibenzo[a,i]pyrene		✓		
DBahP	Dibenzo[a,h]pyrene		✓		
DBalP	Dibenzo[a,l]pyrene		✓		
Flu	Fluorene	✓			
Fla	Fluoranthene	✓			
IcdP	Indeno[1,2,3-cd]pyrene	✓	✓	✓	
Nap	Naphthalene	✓			
Pyr	Pyrene	✓			
Phe	Phenanthrene	✓			
5MChr	5-methylchrysene		✓		

2. Formation, Occurrence, and Influencing Factors of PAHs in Meat Products

Several mechanisms contribute to PAH formation in meat products, including pyrolysis of organic matter, fat dripping onto the heat source, and incomplete combustion during thermal processing [30–33]. In addition, PAHs originate from both natural and anthropogenic sources [13,34]. Natural sources include forest fires and volcanic activity, whereas anthropogenic sources include industrial and residential combustion [13,35]. Since PAHs are present in water, air, and soil, they can enter the food chain [36]. Furthermore, migration from contaminated packaging materials and food additives may also contribute to the presence of PAHs in food [36]. Although environmental contamination contributes to the presence of PAHs in food, thermal processing methods such as smoking, grilling, and roasting are generally considered the major sources of PAHs in meat products because they can directly generate these compounds during processing, often resulting in higher concentrations than those originating from environmental exposure alone [20,32,37]. In meat products, however, thermal processing represents the main route of PAH formation. Three primary mechanisms are involved in the formation of PAHs in meat products.

2.1. Mechanisms of PAH Formation

The first mechanism is the incomplete thermal decomposition of organic matter, referred to as pyrolysis. This process involves free radical reactions occurring at temperatures above 200 °C, leading to the formation of precursors that can further transform into PAHs [13,37]. During thermal processing, PAHs are formed through both pyrolysis, involving the breakdown of organic compounds into smaller reactive fragments, and pyrosynthesis, where these fragments recombine into more stable polycyclic structures [30].

In a study by Zastrow et al. [38], the pyrolysis of wood during home smoking of pork resulted in BaP and PAH4 concentrations of approximately 13 and 67 µg/kg, respectively. According to Commission Regulation (EU) No 2023/915, the maximum permitted levels are 2 µg/kg for BaP and 12 µg/kg for PAH4, while higher limits of 5 µg/kg and 30 µg/kg apply to traditionally smoked meat products [39]. In addition to PAH4, broader indicators such as PAH8 are also used to assess PAH contamination in food [21]. Due to their toxicological significance, PAHs, particularly BaP, have been widely studied in food products [40,41]. These findings indicate that PAH concentrations in certain smoked products may significantly exceed the permitted limits. Similarly, Li et al. [42] investigated PAH4 and BaP levels in four traditionally smoked chicken products. The results showed that total PAH4 concentrations exceeded the regulatory limits, ranging from 30.43 to 225.17 µg/kg in smoked skin and from 18.75 to 129.54 µg/kg in smoked lean meat. Furthermore, BaP concentrations also exceeded the regulatory limits, ranging from 4.33 to 22.03 µg/kg in skin and 1.84 to 5.43 µg/kg in lean meat [42]. These findings indicate that traditional smoking processes can lead to PAH concentrations that exceed regulatory limits, particularly under uncontrolled processing conditions.

The second mechanism involves fat dripping directly onto the flame at high temperatures, generating smoke that deposits volatile PAHs, such as BaP, onto the surface of the meat [20,30,43]. When melted fat comes into direct contact with an open flame or hot charcoal, it undergoes thermal decomposition, generating volatile compounds that contribute to PAH formation. The extent of PAH formation is influenced by fat content, cooking temperature, flame intensity, cooking time, and the distance between the meat and the heat source [43–45]. Lee et al. [43] identified smoke generated from the incomplete combustion of fat drippings as the major contributor to PAH formation during charcoal grilling, confirming the important role of fat dripping in this mechanism. For example, Zhang et al. [46] reported that red meat, particularly beef, tends to produce higher levels of PAHs due to its higher fat content and increased fat dripping during thermal processing. Similar findings were reported by Rigi et al. [47], who also observed elevated PAH levels in red meat compared to white meat. Differences in PAH concentrations between studies can be attributed to variations in fat content, processing temperature, type of fuel, and duration of heat treatment. Several studies have reported elevated PAH concentrations in beef and mutton subjected to charcoal grilling, which has been attributed to their higher fat content and increased fat dripping during thermal processing [46–48]. In addition, traditionally smoked and charcoal-grilled meat products generally exhibit higher PAH concentrations than raw or less intensively processed products [42,48–50]. For example, elevated PAH levels have been reported in traditionally smoked bacon [49], smoked chicken products [42], and charcoal-grilled beef and mutton [48], highlighting the important influence of processing conditions on PAH formation.

The third mechanism involves the incomplete combustion of fuels, such as charcoal or wood, leading to the formation of PAHs that are transported by smoke and subsequently deposited onto the surface of the food [37,47,51–53]. Unlike the first mechanism, which is based on the pyrolysis of organic compounds naturally present in meat, this mechanism originates from the thermal combustion of the external fuel source. Incomplete combustion promotes the formation of PAHs, while combustion conditions, including oxygen availability and combustion temperature, strongly influence their generation and subsequent deposition onto meat products [54]. The type of fuel also plays an important role, as PAH concentrations vary according to the characteristics of the wood or charcoal used for smoking and grilling. Stumpe-Viksna et al. [55] demonstrated that the choice of wood significantly affects PAH levels in smoked meat, with apple tree and alder producing the lowest concentrations, whereas spruce produced the highest PAH content. Similarly,

Malarut and Vangnai [54] reported that wood moisture, combustion temperature, oxygen concentration, and ventilation conditions influence PAH formation during smoking. Ghorbani et al. [44] also identified the type of heat source, cooking temperature, cooking time, and distance between the meat and the heat source as important factors affecting PAH formation, and showed that charcoal cooking generally produces higher PAH concentrations than gas cooking. The PAHs formed through incomplete fuel combustion include high-molecular-weight compounds such as BaP, BaA, BbF, and Chr, which are commonly used as indicators of PAH contamination in meat products. These findings demonstrate that PAH formation through incomplete fuel combustion depends not only on the characteristics of the fuel itself but also on combustion efficiency and processing conditions, emphasizing the importance of controlling smoking and grilling parameters to minimize PAH contamination in meat products [18,20,22]. The principal pathways of PAH formation during smoking, grilling, roasting, and other high-temperature processing methods are summarized in Figure 1.

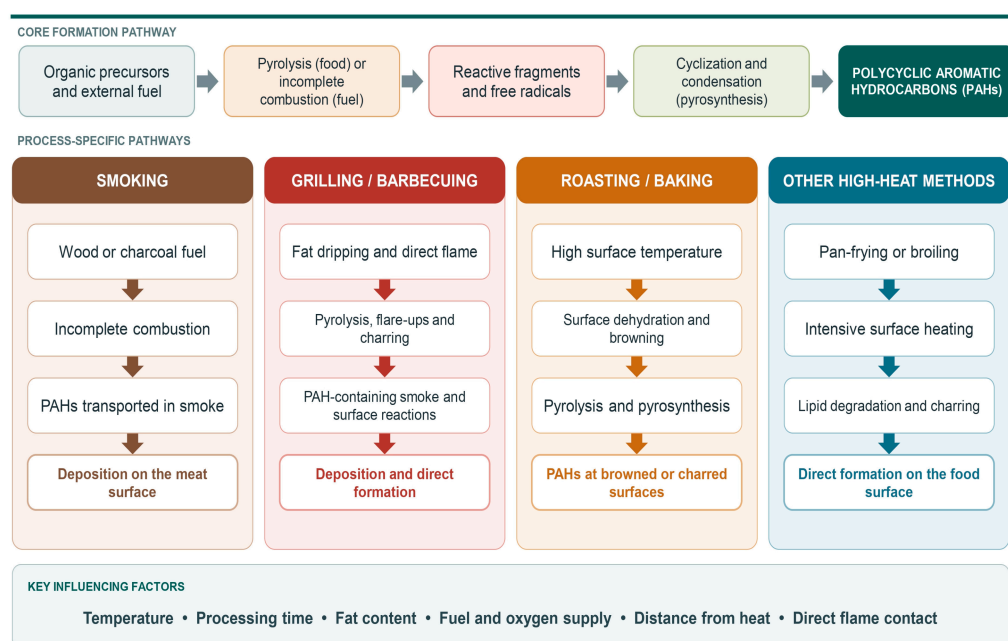


Figure 1. Schematic representation of the principal mechanisms of PAH formation during smoking, grilling, roasting, and other thermal processing methods. PAHs may originate from the incomplete combustion of wood or charcoal followed by smoke-mediated deposition, or may form directly on the product surface through pyrolysis and pyrosynthesis of lipids and other organic constituents. Created by the authors based on [30,37,43,45,54].

2.2. Sources and Processing Conditions Influencing PAH Formation

Smoking is one of the oldest preservation techniques and contributes to shelf life extension and the development of characteristic sensory properties; however, smoked products may contain elevated PAH levels. Elevated concentrations are most commonly associated with improper smoking practices, such as direct exposure of the product to hot smoke or embers and excessively intense smoking conditions [18,56,57]. Smoking techniques are generally categorized as direct or indirect, depending on whether smoke is generated within the same chamber as the product or in a separate external chamber [58,59]. Smoking processes are commonly classified according to temperature ranges, including cold (12–25 °C), warm (25–45 °C), and hot smoking (40–100 °C). In general, higher processing temperatures, particularly in warm and hot smoking, are associated with increased formation of PAHs compared to cold smoking, although deviations from this trend have been observed [59,60]. Although no single optimum temperature–time combination can be

defined for all smoked meat products, PAH formation is generally reduced by using lower smoking temperatures, shorter smoking times, indirect smoking systems, and a greater distance between the product and the smoke source. Therefore, effective PAH reduction is based on controlling the smoking process to avoid excessive combustion temperatures, direct contact with flames or embers, fat dripping onto the heat source, and prolonged exposure to dense smoke [59–61]. The type of wood used for smoke generation significantly influences PAH levels, with spruce, hazel, plum, and aspen generally producing higher concentrations than apple, alder, or maple wood [16]. The data presented in Figure 2 were extracted from the study of Stumpe-Viksna et al. [55], which evaluated total PAH concentrations in meat products smoked using different wood types, including apple, alder, alder with juniper, spruce, maple, hazel, plum, aspen, bird cherry, rowan tree, and charcoal. The figure was prepared to illustrate the influence of wood type on PAH formation during smoking. Smoke produced from wood combustion contains a wide range of chemical compounds, including phenols, aldehydes, acetic acid, and PAHs [11]. As shown in Figure 2, the highest total PAH concentrations were detected in samples smoked with spruce, while the lowest levels were observed in those smoked with apple and alder wood. These differences may be related to the higher resin content of certain wood types, which promotes more intensive soot formation during combustion and consequently increases contamination [55]. The duration of smoking can vary considerably depending on the production method. In traditional processes, smoking is often carried out over extended periods, lasting several days or even weeks, whereas in industrial production it is performed in controlled chambers and is significantly shorter. For example, Puljić et al. [62] reported that in traditional production smoking lasted approximately 20 days (around 90 h of active smoking), while in industrial conditions it lasted about three days, corresponding to roughly 12 h. Prolonged smoking duration may lead to increased PAH levels in smoked products. The distance between the food and the heat or smoke source is another critical factor influencing PAH formation. When the product is positioned too close to the heat source, dripping fat and meat juices undergo thermal decomposition, generating smoke that contributes to increased contamination. Since PAHs are associated with smoke particles, increasing the distance between the food and the smoke or heat source generally reduces their deposition and lowers PAH levels in the final product [47,59].

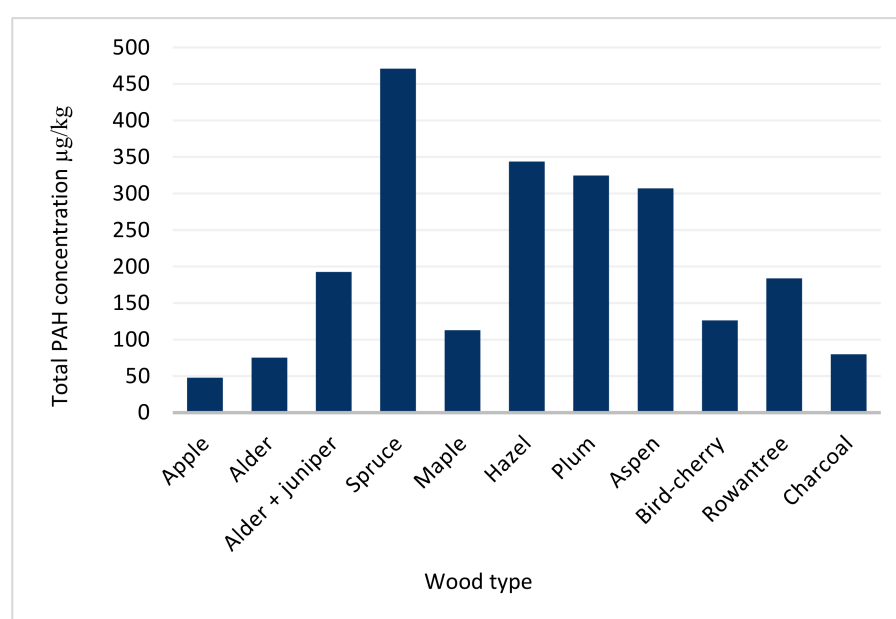


Figure 2. Influence of different wood types on total PAH concentrations in smoked meat products based on data reported by Stumpe-Viksna et al. [55].

2.3. Occurrence of PAHs in Meat Products

PAH concentrations in meat products depend on the product type and processing conditions. Smoked and charcoal-grilled products generally contain higher PAH levels than raw or less intensively processed meat products. Reported concentrations differ among studies because of variations in smoking technology, fuel type, processing temperature, smoking duration, and analytical methods. Table 2 presents representative PAH concentrations reported in different meat products; in the table, N denotes the number of analyzed samples, ND denotes not detected, and LOQ denotes the limit of quantification.

Table 2. Concentrations of detected PAHs in meat products.

Meat Products	Concentration (µg/kg)							Reference
	N	BaP	Chr	BaA	BbF	Total PAH4	Total PAHs	
Bacon, traditional smoked	10	1.87	0.47	0.75	9.47	12.56	107.57	[49]
Bacon, industrial smoked	10	0.32	4.09	3.33	9.51	17.25	100	[49]
Bacon, roasted	9	1.09 ± 0.11	-	0.66 ± 0.07	-	-	1.75 ± 0.17	[63]
Bacon, crispy	9	0.71 ± 0.12	-	0.37 ± 0.09	-	-	1.08 ± 0.21	[63]
Beef, raw	10	ND	ND	ND	ND	-	0.247	[48]
Beef, fried	10	0.117 ± 0.085	ND	ND	ND	-	1.152	[48]
Beef, charcoal-grilled	10	0.457 ± 0.306	ND	0.545 ± 0.280	0.189 ± 0.189	-	6.833	[48]
Beef, foil-grilled	10	0.092 ± 0.062	ND	ND	ND	-	1.265	[48]
Chicken, raw	5	ND	ND	-	ND	-	ND	[64]
Chicken, roasted	30	8.14 ± 0.58	2.09 ± 0.14	6.70 ± 0.46	1.28 ± 0.09	18.21 ± 1.45	29.97 ± 2.04	[65]
Chicken, grilled	30	5.96 ± 0.52	1.58 ± 0.11	4.81 ± 0.39	0.64 ± 0.05	12.99 ± 1.30	23.62 ± 1.89	[65]
Frankfurter sausage	18	<LOQ	0.64	<LOQ	<LOQ	0.64	-	[50]
Ham, smoked	6	0.4	2.6	1.6	-	-	-	[66]
Meat, roasted	30	8.19 ± 0.41	1.53 ± 0.11	6.37 ± 0.29	0.96 ± 0.08	-	28.86 ± 1.95	[67]
Meat, grilled	30	4.93 ± 0.28	1.05 ± 0.06	3.76 ± 0.19	0.61 ± 0.04	-	16.49 ± 1.33	[67]
Mutton, raw	10	ND	ND	ND	ND	-	1.09	[48]
Mutton, fried	10	0.019 ± 0.019	0.438 ± 0.438	ND	ND	-	4.06	[48]
Mutton, charcoal-grilled	10	0.030 ± 0.021	3.491 ± 1.729	2.517 ± 1.744	ND	-	26.819	[48]
Mutton, foil grilled	10	ND	0.565 ± 0.378	ND	ND	-	6.279	[48]
Pork, raw	30	ND	ND	-	ND	-	ND	[64]
Pork loin, roasted	6	0.87 ± 0.07	2.01 ± 0.17	1.42 ± 0.10	1.84 ± 0.22	6.14	7.38	[68]
Sausage	12	ND	ND	ND	ND	ND	-	[69]
Sausage, smoked	10	0.08	0.14	-	0.07	-	0.35	[64]

Note: N = number of analyzed samples; ND = not detected; LOQ = limit of quantification; - = not reported.

Evidence from studies conducted in different geographical regions demonstrates wide variation in PAH contamination. In Europe, traditionally and industrially smoked bacon from Romania contained PAH4 at concentrations of 12.56 and 17.25 µg/kg, respectively, while the corresponding sums of 15 PAHs were 107.57 and 100 µg/kg [49]. By comparison, total PAH concentrations ranged from 1.08 to 1.75 µg/kg in crispy and roasted bacon from the United Kingdom market [63] and reached 7.38 µg/kg in roasted pork loin investigated in Poland [68]. In Asia, traditionally smoked chicken products marketed in China contained 30.43–225.17 µg/kg PAH4 in skin and 18.75–129.54 µg/kg in lean meat [42], whereas a recent study from the Kurdistan Region of Iraq reported total concentrations of 16 PAHs of 7.18, 11.09, and 7.68 µg/kg in grilled beef, kebab, and grilled chicken, re-

spectively [70]. In Africa, Egyptian studies reported total PAH concentrations of 6.833 and 26.819 $\mu\text{g}/\text{kg}$ in charcoal-grilled beef and mutton [48] and 23.62–29.97 $\mu\text{g}/\text{kg}$ in grilled and roasted chicken [65]. In South America, an analysis of 205 meat products marketed in Brazil found PAH4 concentrations ranging from below the LOQ to 48.98 $\mu\text{g}/\text{kg}$ and concentrations of the sum of nine PAHs of up to 108.24 $\mu\text{g}/\text{kg}$, with the highest levels detected in smoked sausages [50]. Overall, elevated PAH concentrations were frequently reported in products subjected to traditional or direct smoking and charcoal grilling. However, these geographical comparisons should be interpreted cautiously because the studies differ in product type, processing conditions, sampling design, number of target PAHs, and analytical methodology.

3. Health Risks Associated with PAHs

The presence of PAHs in food, particularly in roasted or grilled meat, represents a potential health risk due to their known toxic effects, many of which are associated with carcinogenic activity [47]. After ingestion, PAHs are metabolized in the body and eliminated mainly through urine or feces; however, reactive intermediates may be formed during their metabolism [16]. These compounds are primarily metabolized by phase I enzymes, particularly cytochrome P450 enzymes such as cytochrome P450 1A1 (CYP1A1) and cytochrome P450 1B1 (CYP1B1), which catalyze their conversion into reactive metabolites. Although their acute toxicity is relatively low, metabolic activation can lead to the formation of compounds such as diol epoxides, radical cations, or o-quinones, which can form stable adducts with deoxyribonucleic acid (DNA), potentially resulting in mutations and promoting the development of cancer [18,71]. The metabolic activation of PAHs and the principal molecular events leading to carcinogenesis are summarized in Figure 3.

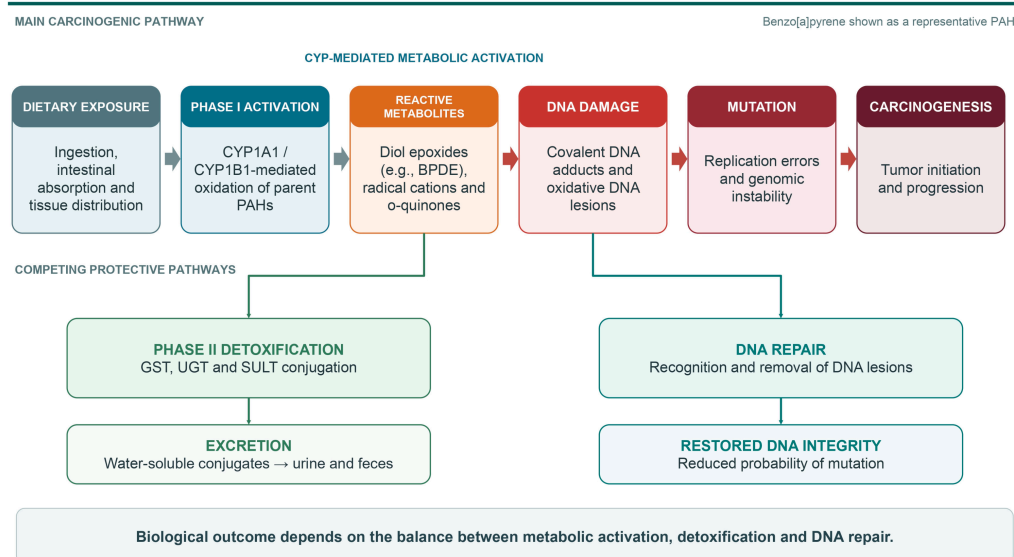


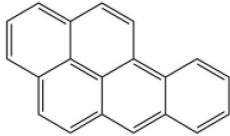
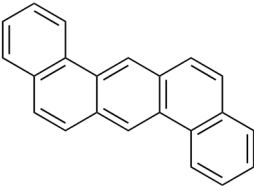
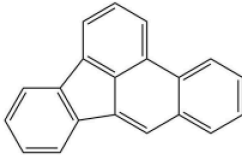
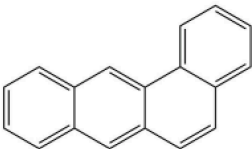
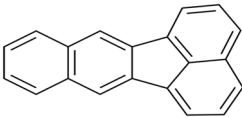
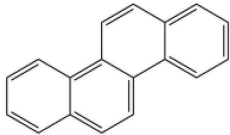
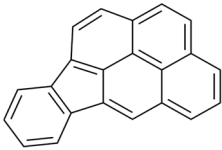
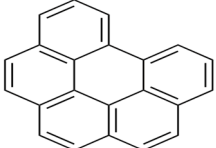
Figure 3. Simplified pathway of PAH metabolism and carcinogenesis. Following absorption, CYP1A1/CYP1B1-mediated activation produces reactive metabolites that may cause DNA adduct formation, mutations, and carcinogenesis if not detoxified or repaired. Phase II conjugation promotes their excretion. Created by the authors based on [16,18,71,72].

Because dietary PAH intake is difficult to estimate accurately, biological markers of exposure are commonly used to assess internal exposure and potential risk [73]. Biomarkers such as PAH–DNA adducts and urinary metabolites, particularly 1-hydroxypyrene, are widely used as indicators of human exposure to PAHs [74–76]. Although the consumption of grilled food in itself does not necessarily pose an immediate health risk, frequent

consumption of food contaminated with PAHs, especially when prepared by methods involving direct contact with flame, may increase long-term health risks [47]. Human epidemiological evidence has linked dietary BaP exposure with neoplastic outcomes. Sinha et al. [77] and Ferrucci et al. [78] reported increased colorectal and rectal adenoma risk with higher BaP intake, while Anderson et al. [79] and Ronco et al. [80] observed positive associations with pancreatic and breast cancer, respectively. However, these observational findings should be interpreted cautiously because dietary PAH estimates are prone to measurement error and co-exposure to other meat-derived mutagens. Several studies have reported elevated PAH concentrations in traditionally smoked meat products, particularly smoked pork and smoked chicken produced under uncontrolled smoking conditions [38,42]. Since compounds such as benzo[a]pyrene are classified by the International Agency for Research on Cancer (IARC) as carcinogenic to humans, frequent consumption of highly contaminated smoked meat products may increase long-term cancer risk [14,40,41]. In addition to carcinogenic and mutagenic effects, PAHs have been associated with oxidative stress, inflammatory responses, and adverse effects on the immune system [13,72]. To reduce consumer exposure, mitigation measures such as indirect smoking, lower smoking temperatures, prevention of fat dripping onto the heat source, appropriate wood selection, and regular monitoring of BaP and PAH4 levels have been recommended [8,21,33]. According to toxicological assessments by international organizations such as the Scientific Committee on Food (SCF), the International Programme on Chemical Safety (IPCS), and the International Agency for Research on Cancer (IARC), many PAH compounds exhibit genotoxic and carcinogenic properties. Compounds such as BaP, BaA, BbF, and DBaH are particularly notable, as there is clear evidence of their genotoxicity and carcinogenic potential. Therefore, due to the complexity of PAH mixtures, regulatory authorities have identified a group of marker compounds, particularly PAH4 (BaP, BaA, BbF, Chr), which are commonly used to assess PAH contamination in food [21,81]. Certain PAH compounds, particularly those with higher molecular weight such as BaP, are classified by IARC as Group 1 carcinogens, meaning that there is sufficient evidence of their carcinogenicity in humans [47]. Other frequently detected PAHs, such as DBaH, are classified as Group 2A (probably carcinogenic to humans), while compounds like BaA, BbF, and Chr are classified as Group 2B (possibly carcinogenic to humans), whereas BghiP is classified as Group 3 (not classifiable as to its carcinogenicity to humans) [14]. Human dietary exposure to PAHs derived from meat does not occur as individual compounds but rather as complex mixtures of multiple congeners, which makes their toxicological risk assessment more challenging and requires appropriate integrated approaches [72]. In addition to their carcinogenic and mutagenic effects, PAHs have also been associated with cardiovascular diseases, respiratory disorders, endocrine disruption, reproductive toxicity, and adverse pregnancy outcomes [13,82,83]. Epidemiological studies have further linked long-term PAH exposure with impaired lung function, asthma, reduced birth weight, and developmental abnormalities [13,82,83]. The Environmental Protection Agency (EPA), the Agency for Toxic Substances and Disease Registry (ATSDR), and various global health authorities utilize Toxic Equivalency Factors (TEFs) to estimate the cumulative carcinogenic potency of a given PAH mixture found in food or the environment [72,84]. The TEF approach, termed the relative potency factor (RPF) approach in U.S. EPA guidance [84], expresses the relative carcinogenic potency of individual PAHs compared with benzo[a]pyrene, which is assigned a reference value of 1.0 [85]. By multiplying the concentration of each detected PAH by its corresponding TEF, the overall toxic equivalent concentration of a PAH mixture can be estimated [32]. The carcinogenic potential of PAHs can be evaluated using IARC classifications and RPFs, which express the relative toxicity of individual PAHs compared

with BaP. The IARC classification, Chemical Abstracts Service (CAS) registry number, and U.S. EPA RPF values of selected PAHs are summarized in Table 3.

Table 3. IARC classification and U.S. EPA relative potency factors (RPFs) for selected PAHs.

PAH Compound	Structure	CAS	IARC Classification	Relative Potency Factor
BaP		50-32-8	1	1.0
DBaH		53-70-3	2A	1.0
BbF		205-99-2	2B	0.1
BaA		56-55-3	2B	0.1
BkF		207-08-9	2B	0.01
Chr		218-01-9	2B	0.001
IcdP		193-39-5	2B	0.1
BghiP		191-24-2	3	-

Note: BaP = benzo[a]pyrene; DBaH = dibenz[a,h]anthracene; BbF = benzo[b]fluoranthene; BaA = benz[a]anthracene; BkF = benzo[k]fluoranthene; Chr = chrysene; IcdP = indeno[1,2,3-cd]pyrene; BghiP = benzo[ghi]perylene; CAS = Chemical Abstracts Service registry number; IARC = International Agency for Research on Cancer; RPF = relative potency factor; “-” = no RPF assigned. Sources: Chemical structures and CAS registry numbers were obtained from PubChem [86]; IARC classifications were based on IARC Monographs [40]; and RPF values were obtained from U.S. EPA guidance [84].

4. Legislation

The European Union, supported by risk assessments from the European Food Safety Authority (EFSA), has established regulatory limits for PAHs in food to protect consumer health [21]. In addition to establishing maximum permitted levels of PAHs in food, international organizations play a key role in PAH risk management through hazard identification, risk assessment, environmental monitoring, and the development of scientific guidance for food safety authorities and producers [21,87]. EFSA regularly evaluates dietary exposure to PAHs and provides the scientific basis for the establishment and revision of regulatory limits and risk management measures [21]. The IARC classifies PAHs according to their carcinogenic potential, whereas the EPA identifies priority PAHs for environmental monitoring and human health risk assessment [40,41,87]. Collectively, these activities support the implementation of preventive measures and contribute to reducing human exposure to PAHs originating from anthropogenic sources [19,21]. To protect consumer health, the European Union sets maximum permitted levels for certain contaminants in food. One of the reasons for the adoption of Commission Regulation (EC) No 1881/2006 was the substantial body of research confirming the carcinogenic properties of PAHs [88]. This regulation established maximum allowable concentrations for various chemical contaminants in food, including PAHs, with the aim of maintaining their levels within toxicologically acceptable limits and reducing them in accordance with good manufacturing practices and the ‘as low as reasonably achievable’ (ALARA) principle [88]. Regulation (EC) No 1881/2006 established maximum levels for contaminants in food and was later replaced by Regulation (EU) 2023/915, which currently defines maximum levels for PAHs in smoked meat and meat products. For PAHs, EU legislation sets maximum levels for BaP and for the sum of four marker compounds (PAH4: BaP, BaA, BbF, and Chr). This approach is used because BaP alone is not considered a sufficient indicator of PAH contamination in food [39,89]. According to current legislation, the maximum permitted concentration of BaP is 2 µg/kg, while the limit for the sum of four marker PAHs is set at 12 µg/kg for standard smoked meat and meat products [39]. Achieving acceptable PAH levels in traditionally smoked products is challenging, as it often requires modifications to processing practices that may compromise sensory quality. In many European regions, traditional smoking techniques contribute to the characteristic sensory properties of valued meat products but may also produce PAH levels that exceed standard EU limits. Strict application of the standard limits to certain traditionally smoked products could create difficulties for producers and affect traditional production practices [39]. To address this issue, Article 7 of Regulation (EU) 2023/915 permits specified Member States, by way of derogation from Article 2, to authorize the placing on their national markets of certain traditionally smoked meat and meat products, provided that the applicable derogation limits are not exceeded [39]. Under this derogation, the specified traditionally smoked products may be placed on the national markets of the listed Member States provided that BaP and PAH4 do not exceed 5 and 30 µg/kg, respectively. The Member States and concerned food business operators must continue monitoring PAHs and implement good smoking practices where possible [39]. In this context, maximum levels are set at 5 µg/kg for benzo[a]pyrene and 30 µg/kg for the sum of four marker PAHs [8,39]. Regulatory approaches to PAHs vary considerably among countries and regulatory authorities. The EU establishes binding maximum levels for BaP and PAH4 in smoked meat [39], whereas Codex Alimentarius Code of Practice No. 68-2009 (CXC 68-2009) provides preventive guidance without numerical limits [90]. In the United States, the United States Department of Agriculture Food Safety and Inspection Service (USDA-FSIS) regulates meat and poultry, while the United States Food and Drug Administration (FDA) treats PAHs as process contaminants; however, no specific PAH tolerance, action level, or guidance level is listed [91–93]. Canada has no meat-specific limit,

as its value of 3 µg/kg BaP toxic equivalents applies only to olive-pomace oil [94]. Similarly, the Australia New Zealand Food Standards Code, administered by Food Standards Australia New Zealand (FSANZ), establishes no PAH limit for meat [95]. Thus, compared with the EU, these systems emphasize process control, surveillance, and risk-based assessment. The main differences among these regulatory approaches are summarized in Table 4.

Table 4. Comparison of regulatory approaches to PAHs in smoked meat and related food products.

Authority	Regulatory Instrument	Requirement Relevant to Smoked Meat	Reference
European Union	Regulation (EU) 2023/915	BaP: 2 µg/kg; PAH4: 12 µg/kg. Derogation: 5 µg/kg BaP and 30 µg/kg PAH4 for specified traditional products	[39]
Codex Alimentarius	CXC 68-2009	No numerical maximum level	[90]
United States	USDA-FSIS & FDA	No PAH tolerance, action level, or guidance level listed in the FDA transparency tool	[91–93]
Canada	Health Canada	No meat-specific maximum level; 3 µg/kg BaP toxic equivalents applies only to olive-pomace oil	[94]
Australia/ New Zealand	Australia/New Zealand (FSANZ)	No meat-specific maximum level for PAHs	[95]

Note: Regulatory instruments and publication years: European Union—Commission Regulation (EU) 2023/915 (2023, as amended); Codex Alimentarius—CXC 68-2009 (2009); United States—Federal Meat Inspection Act, Title 21 of the United States Code (U.S.C.) 601 et seq. (1906, as amended), Title 21 of the Code of Federal Regulations (CFR), Part 109 (1977, as amended), and the FDA Chemical Contaminants Transparency Tool (2025); Canada—Food and Drugs Act, R.S.C. 1985, c. F-27, and Health Canada’s Maximum Levels for Chemical Contaminants in Foods (2020, current online list); Australia/New Zealand—Australia New Zealand Food Standards Code—Schedule 19, F2015L00454, Compilation No. 6, F2024C00908 (2024). The cited United States instruments provide general regulatory authority and do not establish PAH-specific numerical limits for smoked meat. BaP = benzo[a]pyrene; FDA = U.S. Food and Drug Administration; FSANZ = Food Standards Australia New Zealand; PAH4 = sum of benzo[a]pyrene, benz[a]anthracene, benzo[b]fluoranthene, and chrysene; USDA-FSIS = U.S. Department of Agriculture Food Safety and Inspection Service.

5. Analytical Methods for PAH Determination

The analytical determination of PAHs in meat products comprises several critical steps, including sample preparation, extraction and clean-up, followed by chromatographic separation and detection. Owing to the complexity of the meat matrix and the generally low concentrations of PAHs, careful selection and optimization of each step are crucial to achieve accurate, sensitive, and reliable analytical results [96].

5.1. Sample Preparation

Sample preparation represents a critical step in the determination of PAHs in meat products due to the complex composition of the matrix, which includes high levels of lipids, proteins and water that may interfere with analytical determination [96]. Therefore, appropriate sample preparation is essential to minimize matrix effects and ensure accurate and reliable results [96,97]. Prior to extraction, meat samples are typically homogenized to obtain a representative and uniform matrix, as PAHs may be unevenly distributed within the product, particularly in thermally processed meats such as smoked or grilled products [98,99]. Homogenization increases the sample surface area and helps ensure that analytical test portions are representative of the whole sample [99]. Freeze-drying has been used in PAH analysis of meat, with recoveries of 80.1–101.1% reported for 22 PAHs in freeze-dried pork [100]. However, this finding demonstrates method performance in a

dried matrix and does not establish that freeze-drying improves recovery. Sample water content should also be considered when comparing analytical procedures. Lim et al. [98] added 5 mL of water to 2 g of homogenized sample before QuEChERS (quick, easy, cheap, effective, rugged, and safe) extraction, whereas QuEChERS protocols may differ in the solvents, salts, and buffers used for salting-out partitioning [97]. These methodological differences limit direct comparisons of recovery among studies and emphasize the need to report moisture content, water addition, and sample-preparation conditions.

5.2. Extraction and Clean-Up

Following sample preparation, the extraction of PAHs from the meat matrix represents a critical step in the analytical procedure. The determination of PAHs in food generally involves two main stages: extraction of analytes from the matrix and subsequent purification of the extract prior to instrumental analysis [96]. Conventional extraction techniques, such as Soxhlet extraction, have been widely used for PAH analysis; however, these methods are time-consuming and require large volumes of organic solvents [96,101]. Alternative extraction approaches, such as QuEChERS, have been applied for the determination of PAHs in food matrices [97,99]. QuEChERS-based methods enable simplified extraction and clean-up within a single workflow, reducing solvent consumption and analysis time while maintaining satisfactory analytical performance [97]. Typically, PAHs are extracted using organic solvents, followed by the addition of salts to induce phase separation, after which clean-up is performed using sorbents to remove co-extracted matrix components [97,99]. The clean-up step is particularly important in meat samples due to their high lipid content, which may interfere with chromatographic analysis. Therefore, various clean-up strategies, including solid-phase extraction (SPE) and sorbents such as silica, alumina, Florisil, or octadecyl-bonded silica (C18), are commonly applied to remove interfering compounds and improve analytical performance [19]. Additional extraction and microextraction techniques include magnetic solid-phase extraction (MSPE), dispersive liquid–liquid microextraction (DLLME), and ionic liquid-based extraction. In MSPE, magnetic sorbents retain the target analytes and are separated from the sample using an external magnetic field [102]. This technique has recently been combined with gas chromatography–mass spectrometry (GC–MS) for PAH determination in pizzas containing chicken or beef [102]. DLLME uses small volumes of extraction and dispersive solvents to produce fine droplets, enabling rapid analyte transfer and preconcentration [103]. Microwave-assisted extraction followed by DLLME and GC–MS has been successfully applied to PAH determination in grilled meat [103]. Ionic liquids provide alternative extraction media whose properties can be modified through the selection of their constituent ions [104]. Magnetic ionic liquids were recently used for the microextraction of five PAHs from water and were reused for five extraction cycles [104]. Because this application was demonstrated in aqueous samples, further validation is required before its use in lipid-rich meat matrices, particularly regarding matrix effects and analyte recovery.

5.3. Chromatographic Determination

Chromatographic techniques are commonly used for the separation, identification, and quantification of PAHs in meat and meat products. Among the most frequently applied instrumental methods are high-performance liquid chromatography coupled with fluorescence detection (HPLC–FLD) and gas chromatography–mass spectrometry (GC–MS) [105]. In high-performance liquid chromatography (HPLC), analytes are transported through a chromatographic column by a liquid mobile phase under high pressure. The retention time of PAHs depends on the interactions between the stationary phase, the analytes, and the mobile phase [105]. Reversed-phase columns are commonly used for PAH separation and

enable the determination of compounds with higher molecular weight. In PAH analysis, fluorescence detection (FLD) is considered more sensitive and selective than ultraviolet detection (UVD), although UVD may still be required for compounds that do not naturally fluoresce, such as cyclopenta[cd]pyrene [105,106]. Previous studies have reported limits of detection ranging from 0.007 to 0.25 ng/g for HPLC–FLD methods [105]. GC–MS is also widely used for PAH determination in food samples [105]. In GC analysis, analytes are separated after dissolution and vaporization of the sample, while an inert gaseous mobile phase transports them through the chromatographic column [19,105]. Compared with gas chromatography with flame ionization detection (GC–FID), GC–MS is more suitable for PAH determination because FID has lower sensitivity and limited selectivity, whereas MS detection enables confirmation of analyte identity based on mass spectral information [19,105,106]. In addition, MS-based methods allow the use of stable isotope-labeled PAHs as internal standards, which improves the reliability of quantitative analysis [19,106]. GC–MS is commonly operated in selected ion monitoring mode to achieve lower detection limits and more selective monitoring of target PAHs [19,106,107]. The selection of an appropriate stationary phase is also important, since several PAH groups may show co-elution; therefore, MS-compatible capillary columns such as HP-5MS, DB-5MS or related phenyl–methylpolysiloxane phases are frequently used for PAH separation [19,105]. The applicability of GC–MS to meat matrices has been demonstrated in smoked pork meat, where a modified QuEChERS–GC–MS method enabled simultaneous determination of 16 PAHs using chrysene-D₁₂ as an internal standard and provided good linearity and recoveries within the accepted validation range [108]. Overall, GC–MS represents a selective and sensitive approach for the determination of PAHs in complex food matrices, including meat and meat products [19,106,108]. Although GC–MS and HPLC–FLD are reference methods for PAH determination because of their high sensitivity and selectivity, their use may be restricted by the high costs of instrumentation and maintenance. In laboratories with limited resources, HPLC coupled with ultraviolet detection (HPLC–UVD) provides a more affordable option while maintaining adequate analytical performance for routine PAH analysis and requiring less specialized equipment [105,106,109]. Furthermore, QuEChERS-based sample preparation reduces solvent consumption and simplifies sample processing, contributing to a more sustainable analytical approach [97,99]. Selected methods and their reported validation parameters are summarized in Table 5.

From an analytical-performance perspective, HPLC–FLD generally offers greater sensitivity and selectivity than HPLC–UVD for fluorescent PAHs, whereas UVD remains useful for compounds with weak or absent fluorescence [105,106]. GC–MS provides mass-spectral confirmation and enables the use of stable isotope-labeled internal standards, although the co-elution of structurally similar PAHs may require careful column selection [19,105,106]. In smoked chicken, HPLC–FLD provided shorter retention times and better separation for most PAHs, whereas GC–MS identified more PAHs despite matrix interferences [109]. GC–MS/MS offers additional selectivity for trace analysis and has been successfully applied to the determination of 16 PAHs in grilled meat [110]. Overall, method selection depends on the target PAHs, matrix complexity, required level of confirmation, detection limits, and available instrumentation [105,106,109,110].

Table 5. Analytical methods used for PAH determination in meat and meat products and their reported validation parameters.

Analytical Method	Extraction	Clean-Up	Analyzed PAHs	Analysis Time (min)	LOD (Reported Unit)	LOQ (Reported Unit)	Linearity (r or R ² , as Reported)	Precision (RSD, %)	Recovery (%)	Sample/Reference
GC–MS	Modified QuEChERS	Integrated QuEChERS clean-up	16 EPA PAHs	30	0.27–5.74 µg/kg	0.81–17.22 µg/kg	0.99–1.00	1.7–7.6	71–120	Smoked pork meat [108]
GC–MS/MS	SPME	NR	16 EPA PAHs	34	0.02–1.66 ng/L	0.07–5.52 ng/L	0.9914–0.9993	<8.4	85.1–102.8	Grilled meat [110]
GC–FID	Liquid–liquid extraction (n-hexane/DCM) after alkaline hydrolysis	SPE (neutral silica/basic silica/acidic silica/neutral silica)	BaP, BkF, IcdP, BghiP	60.3	0.1–0.3 µg/kg	0.3–0.9 µg/kg	0.9634–0.9986	3.18–15.60	83.69–94.25	Processed meat [111]
HPLC–FLD	LLE (n-hexane) after saponification	Florisil SPE	Chr, BbF, BkF, BaP, DBahA, BghiP, IcdP	35	0.003–0.2 µg/kg	NR	NR	NR	70.1–115.7	Meat products [64]
HPLC–UVD	Soxhlet extraction with methanol/KOH saponification	Sep-Pak Florisil cartridge (SPE purification)	16 EPA PAHs	40	0.03–1.54 ng/injection	NR	NR (r = 0.99–1.00)	NR	NR	Smoked chicken [109]

Note: Values are reported as provided in the original studies and are not directly comparable because matrices, target analytes, units, and validation procedures differed. Analysis time was derived from the reported chromatographic program or retention data. LOQ was calculated as three times the reported method LOD, as specified in the original study [108]. For GC–MS/MS [110], LOD and LOQ are expressed in ng/L of working solution; for HPLC–UVD [109], LOD is expressed in ng/injection. LOD = limit of detection; LOQ = limit of quantification; RSD = relative standard deviation; R² = coefficient of determination; NR = not reported; PAH = polycyclic aromatic hydrocarbon; DCM = dichloromethane; LLE = liquid–liquid extraction; QuEChERS = quick, easy, cheap, effective, rugged, and safe; SPME = solid-phase microextraction; SPE = solid-phase extraction; GC–MS = gas chromatography–mass spectrometry; GC–MS/MS = gas chromatography–tandem mass spectrometry; GC–FID = gas chromatography–flame ionization detection; HPLC–FLD = high-performance liquid chromatography–fluorescence detection; HPLC–UVD = high-performance liquid chromatography–ultraviolet detection; KOH = potassium hydroxide. PAH compound abbreviations are defined in the Abbreviations section.

6. Strategies to Reduce PAH Formation

Since PAH contamination in smoked meat products is strongly influenced by technological conditions, mitigation strategies should primarily focus on the optimization and control of the smoking process. Kowalska et al. [8] emphasized several approaches for reducing PAH formation during smoking, including replacing direct smoking with indirect smoking, shortening smoking time, lowering smoking temperature, drying the product surface before smoking, using seasoned and bark-free wood, selecting appropriate casings or removing casings before consumption, and improving smokehouse equipment. The selection of suitable wood is considered an important measure for minimizing PAH contamination. In traditional smoking, hardwoods with lower lignin content, such as oak, apple, or alder, are generally preferred over softwoods such as spruce and pine, because softwoods burn more rapidly and may generate higher combustion temperatures, thereby contributing to increased PAH formation [33,112]. Furthermore, one of the approaches for reducing PAH concentrations involves the purification of smoke prior to its contact with food, for example by passing the smoke through filters or by cooling it. Various methods have been investigated for this purpose, with activated carbon being frequently used as an adsorbent, while some studies have demonstrated that zeolite filters may be highly effective in the removal of PAHs from smoke [11]. Zeolites are porous minerals capable of adsorbing PAHs from smoke, and their application may substantially reduce PAH concentrations while retaining most smoke aroma compounds [112]. Besides the optimization of smoking parameters, additional mitigation strategies related to product formulation and processing conditions have also been explored for reducing PAH formation in meat products. Youssef et al. [113] showed that the use of cellulose casings can reduce total PAH content in smoked sausages compared with sheep casings. Wang and Fang [114] also described strategies for preventing PAH formation caused by fat dripping, such as repositioning the direct heat source or placing a container underneath the product to collect fat droplets and prevent their contact with the heat source. Pretreatment procedures, including steaming, microwave preheating, and wrapping meat in aluminum foil or banana leaves prior to charcoal barbecuing, have been reported to effectively reduce BaP formation [20]. In addition, several studies have shown that marinating meat before grilling can reduce PAH levels, mainly due to antioxidant compounds in marinades that neutralize free radicals involved in PAH formation during thermal processing [33,51,115–118]. Marinades containing acidic components, herbs, spices, fruit juices, or plant extracts may inhibit PAH-forming reactions and reduce oxidative processes during cooking [116,117,119]. Previous studies demonstrated that ingredients such as lemon juice, thyme, garlic, onion, tea extracts, and beer-based marinades can significantly reduce PAH formation in grilled meat products due to their antioxidant activity and radical scavenging capacity [117–119]. Wang et al. [120] reported that the protocatechuic acid marinade had the strongest inhibitory effect on PAH formation, followed by gallic acid and ferulic acid marinades. Overall, these findings indicate that PAH formation in meat products can be effectively reduced by combining controlled smoking conditions, appropriate fuel selection, improved smoke purification, and the application of suitable pretreatment or formulation strategies.

7. Critical Discussion

Reported PAH concentrations in meat and meat products vary considerably among studies, reflecting differences in product characteristics and processing conditions rather than a single influencing factor [33,121]. Although traditional smoking is generally associated with higher PAH contamination [8,33], the available findings are not entirely consistent. Kowalska et al. [8] reported significantly greater PAH contamination in traditionally than in industrially smoked Polish meat products. In contrast, Tăbăran et al. [49]

found a higher PAH4 concentration in industrially than in traditionally smoked Romanian bacon (17.25 vs. 12.56 µg/kg). However, when the sum of 15 PAHs was considered, traditionally smoked bacon showed a slightly higher concentration than industrially smoked bacon (107.57 vs. 100 µg/kg) [49]. These divergent findings indicate that PAH contamination cannot be explained solely by classifying the smoking process as traditional or industrial [8,49]. Product composition and specific smoking conditions may substantially affect PAH formation [8,22,33], whereas the selected PAH marker group and analytical methodology can influence comparisons among studies [19,122]. Product composition, particularly fat content, is an important source of variability in PAH concentrations. As PAHs are lipophilic compounds, they tend to accumulate in lipid-rich tissues, whereas fat dripping onto the heat source during smoking or grilling promotes pyrolytic reactions and increases the deposition of PAHs on the product surface [22,33,121]. In addition, technological factors, including smoking or grilling conditions, processing temperature, heating duration, fuel type, and the distance between the product and the heat source, contribute to differences in PAH formation, making direct comparison of published data difficult and requiring careful interpretation of reported concentrations [22,33,121]. Methodological differences further contribute to the variability of reported PAH concentrations, as studies employ different analytical techniques, sample preparation procedures, and target compounds for PAH determination [22,122]. Furthermore, the use of different marker systems, including BaP, PAH4, PAH8, or the 16 EPA priority PAHs, may influence the interpretation of PAH occurrence and limit direct comparison among published datasets [19,122]. Geographical differences in traditional meat production practices may contribute to variations in reported PAH concentrations, as many Mediterranean dry-fermented meat products are manufactured through fermentation and prolonged ripening–drying processes, while smoking is applied with varying intensity depending on regional traditions [123–125]. In contrast, smoking represents an important preservation step for many Northern European meat products, and studies have shown that traditional smoking techniques may result in higher PAH concentrations than indirect or industrial smoking methods [126,127]. Furthermore, regional differences in smoking conditions, including the smoking method and wood type, have been associated with differences in PAH contamination profiles, highlighting the importance of geographical context when comparing results across studies [123,128]. Despite the increasing number of studies investigating PAHs in meat and meat products, current knowledge remains limited by the wide variability in processing technologies and production practices, which complicates the generalization and interpretation of published findings [22,33]. Differences in reported PAH concentrations among studies may reflect variations in raw materials, smoking conditions, and analytical methodologies rather than true differences in PAH occurrence, emphasizing the need for cautious interpretation of available data [22,33]. Future research should focus on further harmonization of analytical protocols, continuous monitoring of traditionally produced meat products, and optimization of processing conditions to improve data comparability and support more reliable risk assessment and PAH reduction strategies [8,22,33].

8. Conclusions

The presence of PAHs in meat products remains an important food safety concern, particularly because these compounds are formed during smoking, grilling, roasting, and other high-temperature processes. Their levels are influenced by processing temperature, smoking duration, fuel type, distance from the heat source, fat content, and product characteristics. Although PAH formation cannot be completely avoided, it can be substantially reduced through controlled processing conditions, appropriate wood selection, indirect smoking, prevention of fat dripping, smoke purification, and suitable pretreatment

strategies. For researchers, harmonized analytical protocols are needed to improve data comparability, while regulatory agencies should prioritize risk-based monitoring of BaP and PAH4. For the food industry, controlling smoking conditions, fuel selection, smoke exposure, and fat dripping provides practical means of reducing PAH contamination while preserving product quality.

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Abbreviations

The following abbreviations are used in this manuscript:

5MChr	5-methylchrysene
Acn	acenaphthene
Acy	acenaphthylene
ALARA	as low as reasonably achievable
Ant	anthracene
ATSDR	Agency for Toxic Substances and Disease Registry
BaA	benz[a]anthracene
BaP	benzo[a]pyrene
BbF	benzo[b]fluoranthene
BcF	benzo[c]fluorene
BghiP	benzo[ghi]perylene
BjF	benzo[j]fluoranthene
BkF	benzo[k]fluoranthene
C18	octadecyl-bonded silica
CAS	Chemical Abstracts Service
CcdP	cyclopenta[cd]pyrene
CFR	Code of Federal Regulations
Chr	chrysene
CXC	Codex Alimentarius code of practice designation
CYP1A1	cytochrome P450 1A1
CYP1B1	cytochrome P450 1B1
DBaeP	dibenzo[a,e]pyrene
DBahA	dibenz[a,h]anthracene
DBahP	dibenzo[a,h]pyrene
DBaiP	dibenzo[a,i]pyrene
DBalP	dibenzo[a,l]pyrene
DCM	dichloromethane
DLLME	dispersive liquid–liquid microextraction
DNA	deoxyribonucleic acid
EC	European Community
EFSA	European Food Safety Authority
EPA	United States Environmental Protection Agency
EU	European Union
FDA	United States Food and Drug Administration
FID	flame ionization detector
Fla	fluoranthene

FLD	fluorescence detection
Flu	fluorene
FSANZ	Food Standards Australia New Zealand
GC	gas chromatography
GC–FID	gas chromatography with flame ionization detection
GC–MS	gas chromatography–mass spectrometry
GC–MS/MS	gas chromatography–tandem mass spectrometry
HAAs	heterocyclic aromatic amines
HPLC	high-performance liquid chromatography
HPLC–FLD	high-performance liquid chromatography with fluorescence detection
HPLC–UVD	high-performance liquid chromatography with ultraviolet detection
IARC	International Agency for Research on Cancer
IcdP	indeno[1,2,3-cd]pyrene
IPCS	International Programme on Chemical Safety
KOH	potassium hydroxide
LLE	liquid–liquid extraction
LOD	limit of detection
LOQ	limit of quantification
MS	mass spectrometry
MSPE	magnetic solid-phase extraction
Nap	naphthalene
ND	not detected
NR	not reported
PAHs	polycyclic aromatic hydrocarbons
PAH4	sum of four marker PAHs
PAH8	sum of eight marker PAHs
Phe	phenanthrene
Pyr	pyrene
QuEChERS	quick, easy, cheap, effective, rugged, and safe
R ²	coefficient of determination
RPF	relative potency factor
RSD	relative standard deviation
SCF	Scientific Committee on Food
SPE	solid-phase extraction
SPME	solid-phase microextraction
TEF	toxic equivalency factor
U.S.C.	United States Code
USDA-FSIS	United States Department of Agriculture Food Safety and Inspection Service
UVD	ultraviolet detection

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