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REVIEW ARTICLE

Food Safety

Polycyclic aromatic hydrocarbons and food safety: A review

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Summary: Polycyclic aromatic hydrocarbons (PAHs) are of petrogenic or pyrogenic origin. Of the known few hundred PAHs, 16 are identified to be toxic, of which 8 are teratogenic. Of the 8, 4 are carcinogens. Among them, Benzo[*a*]pyrene (BaP) possesses the highest carcinogenicity and is used as a marker in toxicity studies. PAHs of petrogenic origin contaminate soil, water and air. They may occur in air associated with dust particles. PAHs of pyrogenic origin are generated during burning of wood and other organic matter. They are also produced from lipids and carbohydrates during heating of foods. PAHs are mostly identified with grilling, smoking, frying, roasting and toasting of foods. Among the foods, formation of PAHs is studied mostly in association with grilling of fish and meat, where contacts occur between the dripping lipids and naked flames. The generated PAHs get deposited back on fish and meat muscles. PAHs in smoke are deposited on food surfaces on exposure to contaminated air from the vehicle fumes and smoke curing of foods. PAHs are formed during mechanical expulsion of edible oils. Presence of markers, carcinogenic PAHs and total PAHs in different foods and their extent of formation and deposition is discussed in relation to food safety hazards and health implications. Among the local foods and food preparation habits, the role of coconut oil, smoke curing of foods and formation of PAHs even under mild conditions of heat such as manufacture of black tea are reviewed. Recommendations for future actions, research and implementing food regulations to minimize PAHs in foods are presented.

Keywords: Carcinogens, deep frying, PAHs, smoke curing.

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a group of organic compounds containing two or more aromatic rings linked in linear, angular or clustered forms. Mainly they contain carbon and hydrogen in their structures. A few PAHs containing nitrogen, oxygen and sulphur are derived from coal tar and petroleum products. Distillation and fractionation of coal tar and petroleum products, and operation of engines release PAHs to the atmosphere (Dabestani & Ivanov, 1999). Airborne PAHs tend to cause lung cancer. Among several hundreds of the PAHs, 16 implicated with toxic properties to humans are identified by International Agency for Research on Cancer (WHO, 2010). The PAHs are formed during incomplete combustion of organic matter including foods. They may be formed directly in foods during processing or cooking or may contaminate foods from the environment along the food chain (Figure 1).

The PAHs are produced as mixtures of compounds in all situations. Their presence in uncooked or unprocessed foods indicates environmental contamination of unprotected foods (Phillips, 1999). Food processing generates more PAHs than what is deposited from the environment. PAHs are lipid soluble, making them easily available for absorption through gastrointestinal tract when ingested with foods. WHO (2010) indicates that there is notable exposure to Benzo[*a*]pyrene (BaP) and other PAHs among the populations consuming toasted bread and grilled meats frequently. In Europe, high concern on PAHs associated with olive oil and smoked meats is evident in 1110 research publications during 1997 to 2010 (Sun *et al*., 2019). The publications recognized formation of PAHs during food processing, presence of high concentrations in edible oils, and in smoked foods.

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In Sri Lanka, the exposure of humans to PAHs would be predominantly through food, and to a lesser extent through the smoke in firewood kitchens, food processing kilns and vehicle emissions. The exposure through marine or inland fish is non-existent as the country is not engaged in coal or petroleum industries or major chemical extraction processes. Probable relationship between the high incidence of lung cancer in Sri Lanka (National Cancer Control Programme, 2021) and the exposure to PAHs through air is yet to be investigated.

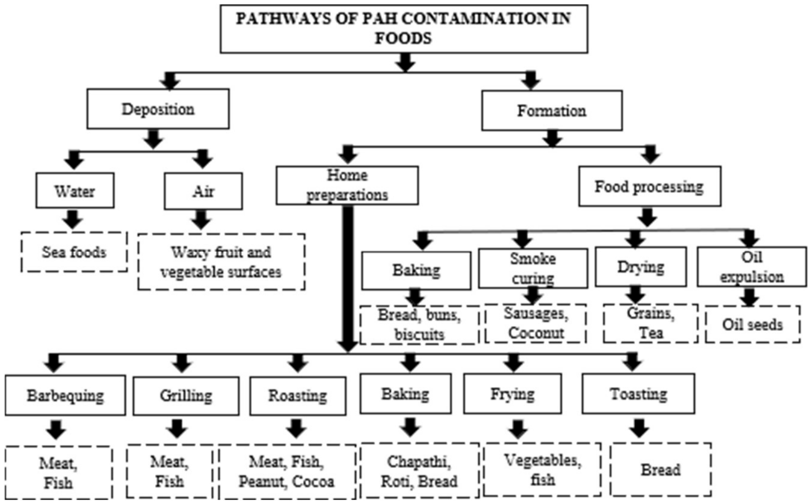

Figure 1: Pathways of entry of polycyclic aromatic hydrocarbons into human food chain

Chemistry and toxicity

There are a few hundred PAH compounds and their derivatives. They are pyrolytic products of organic matter such as wood and petroleum derivatives exposed to temperatures in the range 350–1500 °C under limited access to oxygen (Abdel-Shafy & Mansour, 2016). The chemical pathways generating PAHs and soot from simple molecules during combustion are well understood (Richter & Howard, 2000). Petrogenic PAHs may form at low temperatures but under high pressure in the earth crust. The PAHs exhibit low solubility in water and high solubility in the lipids. Their lipophilic nature leads to easy association with foods and interactions with cellular systems in the human body. In general, up to 16 PAHs have been identified by different authorities as hazardous to humans (Table 1). Zelinkova and Wenzl (2015) reviewed the hazardous PAHs identified by the United States Environmental Protection Agency, the International Agency for Research on Cancer, the Scientific Committee on Food, the Joint FAO/WHO Expert Committee on Food Additives, the International Programme on Chemical Safety, and the European Food Safety Authority (EFSA, 2008).

The research on hazards associated with PAHs in foods initially focused on the total PAHs and the BaP. Of the PAHs in Table 1, BaP is the most carcinogenic and is used as a marker to assess the carcinogenicity of foods. Due to variations in the carcinogenic activity among PAHs, it is difficult to quantify the collective hazardous nature of them in foods. With time, more hazardous clusters of 2 (BaP & Chr) as carcinogenic, 4 (BaA, BaP, BbFA & Chr) as mutagenic, and 8 (BaA, BaP, Chr, BkFA, BbFA, IP, DBahA & BghiP) as toxic were recognized. They are expressed as sum of 2 (Σ_2), sum of 4 (Σ_4) and sum of 8 (Σ_8) in discussing the food safety hazards (Table 1).

Generally, the PAHs by themselves are non-reactive towards biological molecules in living systems. The metabolic activation of PAHs leads to notable toxic reactions (Hongtao, 2002). The PAHs may also react with sulphur and nitrogen oxides in the environment mediated by solar energy, increasing their toxicity (Naccari *et al.*, 2011). Increased hazards due to synergistic effects among the PAHs and their derivatives may occur. The diols and epoxides of PAHs are powerful oxidizing agents capable of inducing biological damage to cells. Wilkinson *et al.* (1993) reported activation of anthracenes by active oxygen. BaP diols and epoxides undergo bio-transformations to bind covalently to DNA, initiating mutations and tumor formation in the animal and human cells (Dabestani & Ivanov, 1999).

Table 1: PAH compounds identified to be associated with food safety hazards

Name (Abbreviation)	Hazard*	IARC carcinogenicity	(Rings) mol wt.	Sum$^{\Delta}$
Benzo[*a*]anthracene† (BaA)	m/g/c	2B	(4) 228	8, 4
Benzo[*b*]fluoranthene† (BbFA)	m/g/c	2B	(5) 252	8, 4
Benzo[*j*]fluoranthene (BjFA)	m/g/c	2B	(5) 252	
Benzo[*k*]fluoranthene† (BkFA)	m/g/c	2B	(5) 252	8
Benzo[*g,h,i*]perylene† (BghiP)	m/g	3	(6) 276	8
Benzo[*a*]pyrene† [BaP]	m/g/c	1	(5) 252	8, 4
Chrysene† [Chr]	m/g/c	2B	(4) 228	8, 4
Cyclopenta[*c,d*]pyrene (CPP)	m/g/	3	(5) 226	
Dibenzo[*a,h*]anthracene† (DBahA)	m/g/c	2A	(5) 278	8
Dibenzo[*a,e*]pyrene (DBaeP)	m/g	3	(6) 302	
Dibenzo[*a,h*]pyrene (DBahP)	m/g/c	2B	(6) 302	
Dibenzo[*a,i*]pyrene (DBaiP)	m/g/c	2B	(6) 302	
Dibenzo(*a,l*)pyrene (DBalP)	m/g/c	2A	(6) 302	
Indeno(*1,2,3-cd*) pyrene† (IP)	m/g/c	2B	(6) 276	8
5 methyl chrysene (MCH)	m/g/c	2B	(4) 242	
Benzo[*c*]fluorene (BcFL)	m/g	3	(4) 216	

†Identified by US EPA as priority 16 PAHs causing human health risks including Acenaphthene, Acenaphthylene, Anthracene, Naphthalene, Fluorene, Phenanthrene, Fluoranthene and Pyrene (Hussar *et al*., 2012). * m = mutagenic; g = genotoxic; c = carcinogenic.

Sum$^{\Delta}$ Indicate the group of 8 or 4 identified to use in expressing combined carcinogenic effect in interpreting test reports. It appears that sum of 8 does not give additional advantage over sum of 4 and there are situations where BaP is absent, but Chrysene present in foods.

IARC classified carcinogenicity as 1 = highly carcinogenic, 2A = probably carcinogenic, 2B = possibly carcinogenic and 3 = not classifiable as carcinogenic to humans, 4 = probably not carcinogenic to humans

Kazerouni *et al.* in 2001 have established correlation coefficient of 0.87 between BaP and total PAHs, and 0.98 between Σ_8PAHs and BaP after testing many foods, justifying the use of BaP as a marker. However, Kelly *et al.* (2021) examining the atmospheric exposure of humans to PAHs and the cancer risk indicated that only 11% of cancer is associated with BaP and the rest is with the other PAHs. They do not justify the use of BaP as a marker. The PAHs reach the organs of the human body through inhalation, skin absorption and ingestion of food, taking different routes and undergoing different interactions making it difficult to apply information from inhalation to that of dietary exposure.

Some PAHs may be present as vapour in the air or absorbed into dust and other particles forming solid particulates. PAHs with 2-3 rings tends to be present predominantly in the vapour phase and those with 5 or more rings in particulate phase. Their molecular nature governs absorption or deposition in foods. The PAHs with 2-3 rings tend to get adsorbed by waxy surfaces in plants. The PAHs originating from petroleum sources could also enter the food chain through the water and fishes that consume them, or the plants that may adsorb them, or absorb from soil. Filter feeding mollusks tend to accumulate more PAHs (Afolabi *et al*., 1983). Moriji *et al*. (2019) identified bioaccumulation of 16 PAHs in fish at concentrations in the region of 11 - 4208 ng/L in water. However, PAHs in fish muscles is reported to get metabolized leaving no toxic residues to enter the human food chain. Tolerance limits are therefore not established in food regulatory systems for PAHs in fresh fish (Zelinkova & Wenzl, 2015). Stringent tolerance limits are set for foods containing lipids or fried in lipids, which may generate PAHs on exposure to high temperatures.

The regulatory limit for PAHs varies in different countries. The Regulations of the European Commission (EC) stipulates maximum limits for BaP and Σ_4 consisting of benzo[*a*]pyrene, benzo[*a*]anthracene, benzo[*b*]fluoranthene and chrysene (European Commission, 1998). The EC regulations are revised regularly based on new evidence from EU countries. Current regulations are summarized in Table 2.

Table 2: Regulatory limits of European Commission

Food group	Regulatory limits in µg/kg BaP & Σ_4 (products)	EC regulations
Cereals	1 &1 (Processed cereal foods)	835/2011 (BaP & Σ_4)
Fats & oils	2 & 10 (Except cocoa butter and coconut oil)	
	2 & 20 (Coconut oil)	
Cocoa	5 & 30 (Fats from cocoa beans and derived products with certain exceptions)	1933/2015 (BaP & Σ_4)
	5 & 15 (Fibre and fibre products as food ingredients)	
Smoked/barbequed	5 & 30 (Smoked meat and products, smoked fish and products)	1327/2014 (BaP & Σ_4)
	5 & 30 (Smoked sprats, mollusks, heat treated meat products)	835/2011 (BaP & Σ_4)
	6 & 35 (Smoked bivalve mollusks)	
Special foods	1 &1 (Infant formulae, processed cereal based foods for infants)	
Banana	2 & 20 (Banana chips)	1933/2015 (BaP & Σ_4)
Supplements	10 & 50 (Containing botanicals, royal jelly, spirulina)	
Herbs	10 & 50 (Dried herbs)	
Spices	10 & 50 (Dried spices except cardamum and smoked capsicum species)	1255/2020 (BaP & Σ_4)
Drinking water	0.010 & 0.10 µg/L	835/2011 (BaP & Σ_4)

Table 3: Regulatory approach and limits in other systems and countries

System/ country	Regulatory approach / limits	Reference
Australia	Drinking water – BaP 0.01 ng/L	NHMRC, 2011
Brazil	Smoke flavourings – 30 µg/kg, Olive oil – 2.0 µg/kg, Drinking water – BaP 0.70 µg/L	Sampaio *et al*., 2021
Canada	Olive pomace oils – BaP 3.0 µg/kg, Drinking water – BaP 0.04 µg /L	Health Canada (cited 2022)
China	Paddy rice, wheat, smoked or baked meat and aquatic products – BaP 5 µg/kg, Fats oils and oil emulsions – BaP 10 µg/kg	CFDA, 2017
Codex	No regulatory limits. Recommend code of good practices	CAC/RCP 68, 2009
India	Smoked fish – BaP 5 µg/kg	Indian Standard, 2016
USA	Drinking water – BaP 2.0 ng/L	US EPA (cited 2022)

EC also has regulations on the use of smoke liquids as flavouring agents in foods. The producers of smoke liquid are required to provide analytical information on 15 PAHs recognized by EC and the presence of BaP and BaA at concentrations below 10 and 20 µg/kg respectively (European Commission, 2003). The regulatory approach and the limits in other countries are summarized in Table 3.

Quantification of PAHs require well designed sampling techniques as they are not distributed homogenously in the foods. The glassware needs to be cleaned with special techniques to prevent cross contaminations.

The test samples need purifications using solid phase cartridges (SPE) prior to quantitation. Among the several SPE cartridges, the QuEChERS procedure has gained recognition (Gratz *et al*., 2011). The quantitation of PAH uses GC-MS or LC-UVA or fluorescence detector techniques more commonly, with application of validated test methods practiced with strict laboratory quality control.

Biological effects

The biological effects are caused by the interference of PAHs with the cellular membranes and their enzymes. The PAHs can cause carcinogenic, mutagenic and immunosuppressive actions. Acute toxicity due to ingestion of the PAHs is almost non-existent. There is much evidence on the chronic effects from dietary PAHs. The PAHs being lipophilic are absorbed by the cells. BaP with the highest lipophilic character could possibly enter chylomicrons and reach many body tissues. The PAHs undergo different biochemical reactions depending on the number of rings in the molecule and the conformational features. PAHs get modified into more active metabolites including epoxides, dihydrodiols and phenols. The metabolites may interact with Glutathione (GSH) or glucuronic acid in cells. The epoxides and dihydrodiols of the PAHs produced during cellular metabolism carry higher capacity to bind with DNA and cellular proteins leading to cell damage, mutations and cancer (Armstrong *et al*., 2004). The genotoxic and carcinogenic effects through the epoxide and -diol derivatives of PAHs are well documented (Tarantini *et al*., 2011). Interaction of the PAHs appears to cleave DNA molecules affecting cellular biochemical activities. The mechanisms used by each of the PAHs in cleaving DNA is shown to vary depending on the presence or absence of light (Hongtao, 2002).

The PAHs may follow mechanisms similar to tumor promoter 12-O-tetradecanoylphorbol-13-acetate in addition to serving as tumor initiators (Baird *et al*., 2005). Shen *et al*. (2022) followed metabolomics with a focus on proteomics to explore BaP related intracellular responses through the viability and migration of KYSE-150 human oesophageal cells. They showed that BaP impedes the migration of oesophageal cells causing serious cellular damage even at short term exposure. The study suggests initiation of cancer risk at the cellular entry point of BaP to the human body through oral route. In contrast, Li *et al*. (2011) reported detoxification of PAHs through o-methylation of PAHs catechols in humans. Both biochemical activities may be happening in the body depending on the dosages of exposure and other factors specific to target organs and cells of the human body.

Van Grevenynghe *et al*. (2003) reported that several PAHs with known toxicological trends possess the capacity to impair differentiation of blood monocytes into functional macrophages *in vitro*. The same effect was not observed with the non-toxic Benzo[*e*]pyrene which is structurally close to BaP. The authors suggest that impairing of functional macrophages formation by PAHs may cause immunotoxicity leading to weakening of the human immune defense system. The concentrations of PAHs used in this study were of the same order as that reported in charcoal-broiled foods. Controlling the entry of PAHs to human body through foods appears to be an important step in protecting the immune systems.

The first step in carrying out risk assessments due to PAHs is through the exposure studies. Phillips (1999) reported that 96.2 % PAHs enter the human body through the diets of non-smoking persons of the age group 19–50 years. Representative mean dietary BaP intakes of 4 ng/kg bw/d with a high level of 10 ng/kg bw/d were established by WHO/FAO Joint Expert committee on Food Additives and Contaminants (JEFCA, 2005). Chronic effects such as reduced immune functions, cataracts, kidney and liver damages, breathing problems and asthma like symptoms are reported with the exposure to PAHs (Abdel-Shafy & Mansour, 2016).

There is much research on exposure of humans to PAHs from the air, especially through coal, coke, other industries and directly to soot and shale. After examining research data for 40 years, Mastrangelo *et al*. (1996) suggests that the safety levels established for air pollution does not seem to reflect real risk. Exposure to smoke containing PAHs associated with food preparations, mainly cooking, appears to be far below that of the industrial exposure situations in triggering hazards. Exposures to PAHs through use of firewood appears insignificant in the light of above information. Unfortunately, while the smoke gets detected sensorily by humans easily, the high amounts of the PAHs produced within foods is not. It is known that the amounts of PAHs deposited by smoke and visible on copra is 6–7 times less than PAHs generated during expulsion of coconut oil, which is not visible.

Contamination of PAHs in foods

The PAHs may be deposited in foods from the air and smoke or generated during heat processing or cooking. Ingestion of BaP by humans is reported to be in the range of 2-500 ng/d against inhalation of 10-50 ng/d (Singh *et al*., 2016). The foods form a main pathway of exposure of humans to the PAHs, other than the workers engaged in industries emanating PAHs. The grilled foods and edible oils constitute major sources of PAHs in human diet. There is much research on PAHs in grilled fish and meat. Edible oils acquire PAHs pyrolytically during mechanical expulsion or frying of the foods.

Table 4: PAHs reported in foods (µg/kg)

Food	BaP	Σ_4 PAH	Σ_8 PAH	Total PAH	Reference
Olive oil from canned tuna	1.9			11.3	Moret *et al*., 2005
Oil from grilled mushroom cans	11.3			47.1	
Charcoal grilled pork	2.9			Σ_{15} 144.7	Food and Environmental Hygiene Department, 2004
Charcoal barbequed beef	12 (median)			Σ_{15+1} 221 (median)	Rose *et al*., 2015
Donor (kebab) meat	nd	2.21	3.17	Σ_{16} 6.08	Sahin *et al*., 2020
Donor chicken	nd	2.45	3.15	Σ_{16} 4.42	
Meat balls	0.70	1.95	2.74	Σ_{16} 4.45	
Grilled chicken	nd	2.13	3.68	Σ_{16} 4.91	
Grilled fish	0.73	3.30	5.13	Σ_{16} 7.26	
Gas grilled pork	< 0.1			Σ_{15} 5.7	Food and Environmental Hygiene Department, 2004
Electric oven roasted pork	< 0.1			Σ_{15} 7.2	
Blended oil	2.04	Σ_3 5.34			Olatunji *et al*., 2014
Sunflower seed oil	1.71	Σ_3 5.90			
Vegetable oil	0.5				Siddique *et al.,* 2021b
Sunflower oil	0.4				
Sesame oil	0.2				
Market edible oils Iran	0.90–11.33	3.51–84.03	7.41–117.12	129.28–195.4	Yousefi *et al*., 2018
Chocolate	0.22	1.75		Σ_{15} 2.58	Ziegenhals *et al*., 2009
Fresh grated coconut	nd		1		Wijeratne *et al*., 1996a
Desiccated coconut	nd		11		
Copra (smoke dried)	3		102		
Parings (smoked)	5		109		
Copra meal	1		68		
Crude coconut oil	12		359		
Amaranth oil	1.32	10.5		Σ_{19} 101.6	Ciecierska & Obiedzinski, 2013b
Linseed oil	nd	0		Σ_{19} 115.0	
Common flax oil	nd	1.21		Σ_{19} 170.9	
Camelina oil	nd	2.47		Σ_{19} 41.3	
Pumpkin oil	15.7	35.0		Σ_{19} 234.3	
Sesame oil	1.23	4.25		Σ_{19} 30.1	
Poppy oil	nd	1.71		Σ_{19} 23.4	
Mustard oil	nd	2.73		Σ_{19} 35.0	
Safflower oil	nd	2.93		Σ_{19} 53.7	
Blackseed oil	1.98	15.84		Σ_{19} 221.2	
Walnut oil	nd	3.15		Σ_{19} 46.0	
Borage oil	1.0	4.12		Σ_{19} 66.2	
Evening primrose	nd	4.04		Σ_{19} 68.9	
Breast milk				Σ_{13} 0.84–696	Ssepuya *et al*., 2022
Milk raw	0.27		5.4		Naccari *et al*., 2011
Pasteurized milk	0.27		6.5		
Skimmed milk UHT	0.35		5.9		
Whole milk UHT	0.25		7.8		
Mullet fish (fresh)	4			Σ_{16} 65	Tongo *et al*., 2018
Prawn (fresh)	2			Σ_{16} 106	
Crabs (fresh)	4			Σ_{16} 57	
Fish (unprocessed)			Σ_6 9.5–31.2	Σ_{15} 17–70	Dhananjayan & Muralidharan, 2012
Cocoa butter				Σ_{19} 16–74	Ciecierska, 2020
Chocolate candy	1.62	8.16		Σ_{14} 67.62	Kumari *et al*., 2012
Chocolate	0.22	1.75		Σ_{15} 2.85	Ziegenhals *et al*., 2009
Tea leaves		25–115			Duedhal-Olesen *et al*., 2015a
Black tea	51	218			Zachara *et al.,* 2018
Green tea	13	76			
Red tea	13	90			
White tea	12	57			
Black dry tea		54.3			Phan *et al*., 2020
Herbal dry tea		16.4			
Oolong dry tea		14.2			
Green dry tea		6.6			

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Food	BaP	Σ_4 PAH	Σ_8 PAH	Total PAH	Reference
Black tea	9.42			Σ_{18} 23.32	Tran-Lam *et al*., 2018
Fried noodles	3.6			Σ_{18} 9.6	
Fried snacks	0.93			Σ_{18} 1.74	
Dried vegetables	1.2			Σ_{18} 1.93	
Cherry coffee	4.52			Σ_{18} 15.0	
Stove grilled meat	1.08			Σ_{18} 1.43	
Coffee		2.2–5.1			Duedhal-Olesen *et al*., 2015a
Bread				Σ_{19} 1.59–13.6	Ciecierska & Obiedziński, 2013a
Bread		0.19–0.46			Kacmaz, 2016
Breakfast cereals		0.10–0.87			
Jujube dried			2.32		Kim *et al*., 2020
Milk bread			1.25		
Black sesame			1.17		
Barbequed pork		195			Duedahl-Olesen *et al*., 2015b
Barbequed chicken		0.1			
Controlled barbeque		< 0.1			

nd – Not detected

The PAHs entering foods are grouped as 'light' and 'heavy' PAHs. Light PAHs (Naphthalene, Acenaphthene, Anthracene, Fluorene, Phenanthrene, and Pyrene) consists of 2-4 member rings and heavy PAHs (BaA, BaP, BbFA, BkFA, Chr, DBahA, BghiP, and IP) consists of 5 or more rings. The groups may best be described as low molecular weight and high molecular weight PAHs following information on Table 1. The hazardous PAHs in foods belongs to the heavy group.

The exposure of food lipids to high temperatures generates PAHs. In grilling, the melting lipids from meat and fish muscles drips direct on to flames producing PAHs. The PAHs get deposited back on the muscles. The formation of PAHs depends on the temperature, the distance of the foods from the flame and other factors. Comparing fuel sources in grilling fish and meat, BaP concentrations of 2.4–31.2 µg/kg were reported with woodfire, against laboratory grilling with charcoal producing 0.7–2.8 µg/kg (Akpambang *et al*., 2009). Adeyeye and Ashaolu (2020) reported high concentrations of PAHs in commercially smoked catfish and grilled meat in Nigeria. They observed BaP and Σ_4PAHs concentrations above the EU tolerance limits of 2 and 12 µg/kg respectively.

Fish consume microplastics and nano-plastics in the seabed. Petrogenic BaP in the sea bottoms get absorbed into microplastics readily. While the nano-plastics by themselves have been shown non-toxic to brine shrimps and zebrafish, they were shown to serve as vectors to spread BaP in brine shrimps and zebrafish embryos causing toxicity (Martínez-Álvarez *et al*., 2022). Entry of PAHs to the marine fish chain through microplastics may provide a pathway for exposure of humans to BaP. Increasing pollution of rivers and oceans with the microplastics pose an increasing challenge through PAHs in the aquatic food chain.

In a study on 15 PAHs in five fish types from Mumbai harbour in India, total PAHs of 17–70 µg/kg and carcinogenic PAHs (Σ BaA, BbFA, BkFA, BaP, IP, and DBahA) 9.5–31.2 µg/kg were reported (Dhanajayan & Muralidharan, 2012). The authors postulated exposure of the general population to 1.77–10.70 ng/kg bw /d. Of the 15 PAHs examined in this study, only 6–10 PAHs were observed in any of the 5 types of fish examined, suggesting variations in PAH accumulation among different fish types from the same contaminated water. The harbours tend to be polluted with petrogenic PAHs due to movement of ships and being physically protected regions of water. Dhananjayan and Muralidharan (2012) indicated that the exposure of persons to PAHs in fish from Mumbai is less than the values reported in Spain (626–712 ng/d), Kuwait (231 ng/d), and Korea (13.8–16.7 ng/kg bw/d). These variations are probably associated with both accumulation rates of PAHs and consumption rates of fish by the respective human populations. In the sea water of Cimandari river estuary in Indonesia, the PAHs concentrations were reported to be low. Of the 15 PAHs examined, the highest value reported for low molecular weight PAHs was 0.0096 µg/kg and for high molecular weights was 0.0021 µg/kg (Helfinalis *et al*., 2021). Estuary is not polluted by discharge of fuel waste from ships.

A study examined 16 PAHs in 30 samples of fish of 3 species from the Niger Delta. The Niger Delta has 360 and 246 oil wells onshore and offshore respectively, allowing petrogenic contamination of waters. The concentrations of PAHs were reported to vary from nondetectable to 171 µg/kg within Niger Delta (Nwaichi & Ntorgbo, 2016). The study recognized areas having PAHs of pyrogenic and petrogenic sources separately, based on the ratios of different PAHs in fish samples. Similar results for fresh mullet fish, prawns and crabs from Bonny River in Niger Delta are given in Table 4 (Tongo *et al*., 2018). The presence of PAHs in bile of fish from Atlantic coastal waters near Columbia and in contaminated zones suggest it as a pathway for entry of petrogenic PAHs to the human food chain (Johnson-Restrepo *et al*., 2008). The Indian, Nigerian, Columbian and Indonesian situations indicate that the major contaminations by PAHs in waters and the fish, are of petrogenic origin.

The contamination of Maldive-fish (cured tuna, traditionally produced in Maldives; local name: *Umbalakada*) and fried cutlets by PAHs have been reported in Sri Lanka (Wijeratne *et al*., 1995). Jinadasa *et al*., (2020) have observed unsafe concentrations of PAHs in fish and fishery products. They suggest the need to establish good manufacturing guidelines for fish products in Sri Lanka. The contaminations in both situations arise from deposition of smoke during fish preparations.

The PAHs get dissolved in lipids of the milk readily. Milk may generate PAHs on exposure to heat during processing. Ssepuya *et al*., (2022) reported Σ_{13}PAHs in concentrations of 3–696 µg/kg from Kampala and 0.84–88 µg/kg from Nakaseke in Uganda in breast milk. They attributed at least 33% of variation of Σ_{13}PAHs in breast milk to the types of fuels used in the kitchen. Exposures are suggested to be linked to the extent of PAHs in home prepared foods, as high concentrations of PAHs were observed in households consuming charcoal grilled and fried foods in Uganda. Though increasing Σ_8PAHs were reported with higher lipid content and higher temperatures of treatment of milk in Table 4, the BaP concentrations reported did not show notable differences related to heat treatments (Naccari *et al*., 2011). The presence of the PAHs shown in the breast milk in this study also suggests possible pathways beginning with animal feed materials.

Junmin *et al*., (2021) examining 86 market vegetable oils for 16 PAHs, reported that 37 and 34 oil samples contained BaP and Σ_4PAHs respectively at concentrations above the EC tolerance limits. They postulated that the high contaminations of PAHs observed in peanut oil and sesame oil are due to heat processing of the raw materials prior to oil expulsion. In a study of 40 samples of olive oils in Brazil, up to 164 µg/kg of BaP is reported with almost all tested samples giving positive results (Marcos *et al*., 1996). The reported concentrations probably indicate pyrolytic PAHs formed during the expulsion of oils.

Of the 43 samples of market edible oils identified as frying, sunflower, corn and canola in Iran, 12 samples contained BaP above the EC tolerance limit of 2 µg/kg. Of the PAHs observed in all oils, 65% were light PAHs. The results are summarized in Table 4 (Yousefi *et al*., 2018). The authors did not recognize a serious food safety hazard through the market oils. Fromberg *et al.* (2007) reports that PAHs in the sunflower oil is 50% light PAH and in the rapeseed oil it is 70%. The coconut oil produced from smoke cured copra contained 47–81% light PAHs (Wijeratne *et al*., 1996a). The light PAHs are not implicated in food safety hazards. A higher tolerance limit is established by EC for total PAHs in coconut oil, to accommodate high BaA and Chr is notable.

On the analysis of 296 samples of edible oils in India including coconut, groundnut, hydrogenated vegetable, linseed, mustard, olive, palm, refined vegetable, rice bran, safflower, sesame, soybean, and sunflower oils, Pandey *et al.* (2004) reported that 262 (88.5%) were contaminated with PAHs. Of the contaminated oils 66.4% had total PAHs above the tolerance limit of 25 µg/kg. Among them, olive oil showed the highest contamination of 624 µg/kg and the lowest of 40.2 µg/kg was refined vegetable oils. It is not clear whether the refining is chemical or physical. Physical refining adsorbs only a part of the PAHs in edible oils in the bleaching step, whereas chemical refining destroys the PAHs totally. In a similar study examining 28 oil samples, notably high contamination of olive oils with light PAHs (53.0–105.6) µg/kg is reported (Speer *et al*., 1990). In a study on coconut, safflower, evening primrose and linseed oils, 96% samples contained more than 15 µg/kg of Σ_4PAHs. Of them, the BaP concentrations exceeded tolerance limit in 12% samples and Σ_4PAHs in 28% samples (Silva *et al*., 2018). In contrast to the observations in USA, where bread is considered a major source of BaP (Kazerouni *et al*., 2001), vegetable oils appear to be the major contributor of PAHs in most Asian and African diets.

Roasting of cocoa beans at 160 °C was reported to produce predominantly non-carcinogenic light PAHs. Reduction of the roasting temperature to 140 °C with extended exposure time has not reduced the concentrations of PAHs notably (Ciecierska, 2020).

In the rice plants, high concentrations of PAHs have been observed in roots with low concentrations in the grains and leaves, suggesting low translocation of PAHs from soil to grains (Tao *et al*, 2006). It is not known whether the low molecular PAHs are metabolized in shoots of rice. Metabolism of phenanthrene by millets have been observed in pot experiments (Tarigholizadh *et al*., 2022). Based on the trends in PAHs contaminations associated with boiling of other foods, cooked rice consumed globally may not be an alarming contributor of PAHs to human diet. BaP concentrations of up to 11.9 µg/kg with mean of 3.6 µg/kg have been detected in fried noodles in Hanoi (Tran-Lam *et al*., 2018).

Martena *et al*. (2011) examined botanicals supplementing foods for BaP and Σ_4PAHs. Recognizing high variations of the PAHs in different botanicals, they summarized an overall lower mean of 3.37 µg/kg during 2003–2009 for Σ_4PAHs. They also reported high concentrations of BaP in dong quai, green tea and valerian. The black tea is kiln dried during manufacture against green tea, which is steam treated. In a study on market teas in USA, Adisa *et al*. (2015) reported PAHs in all the tea samples, except one green tea sample. They reported the presence of 5–12 PAHs out of 18 PAHs tested in the teas. The concentrations of BaP reported in black tea was four-fold higher than that of the green, red and white teas (Table 4) from Polish market (Zachara *et al*., 2018). Studies in several countries on BaP and Σ_4PAHs concentrations in different types of teas showed high concentration of PAHs in the black tea suggesting process induced contamination (Zachara *et al*., 2018). They reported that the transfer of PAHs from the black tea to the tea infusions is of the order of 0.5%, whereas the comparable results by other authors were around 1.5%. Lin *et al.* (2006) observed 51.6% of the PAHs transfer to infusions during brewing. They also report 30.4% reduction of the transfer of PAHs, on washing the black tea before brewing. Phan *et al*., (2020) observed 1.9%, 15.4% and 40.7 % transfer of Σ_4PAHs in black, herbal and green teas respectively from the processed leaves to the infusions. Girelli *et al.* (2017) observed lower transfer of PAHs to tea infusion from market tea in Italy compared to Chinese and Indian observations. Transfer of PAHs from black tea to the infusion appears to be less with lower contact time between tea leaves and infusion (El-Aty *et al*., 2014). The tea oils facilitate transfer of PAHs by solubilizing them.

Plant materials may get contaminated with PAHs from the air, or in contact with the soils carrying PAHs of petrogenic and pyrolytic origins. A summary of BaP in fruits and vegetables from USA and Italy is reported in Table 1.15 of the IARC monograph 92 (WHO, 2010). The reported values of BaP vary from 0.003–0.52 µg/kg. They probably represent PAHs adsorbed on to surfaces from the environment.

The concentrations of PAHs summarized in Table 4 from different sources indicate the difficulty in recognizing trends or taking a statistical approach in predicting the contamination patterns. Though the test methods for PAHs have been well defined and practiced with high degree of quality control, the conditions leading to formation of the PAHs during food processing vary widely. In test reports there is less uniformity in the examination of PAHs as a group, because authors use different criteria such as Σ_4, Σ_8 and total PAHs. The definitions of total PAHs tend to vary. A review by Singh *et al*. (2016) on PAHs in processed foods has tabulated information up to 2015. His work also shows the difficulty of recognizing food associated or process linked trends of contaminations.

Foods carrying unacceptable concentrations of PAHs are rejected or notified by the EU countries. The notifications over a two-year period ending on 18 September 2022 is given in the Table 5 (RASFF, 2022).

Table 5 indicates that a variety of foods in international trade continue to be hazardous due to PAHs.

Table 5: Notifications and rejections of foods at EU country borders due to presence of PAHs.

Code	Notification/rejection	Origin
2021.519	PAHs in reduced fat cocoa powder	Slovakia
2021.563	BaP & Σ_4PAHs in smoked sardinella	Senegal
2021.662	BaP and PAHs in sausages	Slovakia
2021.687	BaP & PAHs in sausages	Slovakia
2021.716	PAHs in Green wheat, roasted	Turkey
2021.563	BaP & Σ_4PAHs in smoked sardinella	Senegal
2022.043	PAHs in sausages	Portugal
2022.140	BaP & PAHs in cabamos sausages	Poland
2022.221	PAHs in lutein powder	Netherlands
2022.260	BaP & PAHs in paprika powder	Spain
2022.260	BaP & PAHs in refined sunflower oil	Ukraine
2022.307	PAHs in turmeric	India
2022.458	BaP & PAHs tea powder	China
2022.477	PAHs in sliced garlic	Germany
2022.541	PAHs coconut oil	Philippines

Formation of PAHs in foods

Formation of the PAHs during food processing and home food preparations leads to food safety hazards. The home food preparations do not come under food regulatory controls. It leaves room for hazards. The lipid concentrations in foods, exposure to heat, the methods of heating, the proximity of heat source to the food, and the contaminated frying oils contribute to PAHs in the foods.

Singh *et al*. (2016) reviewing PAHs in processed foods observed higher concentrations of PAHs in deep fried and grilled foods. The deep fried and grilled foods form excellent delivery systems of PAHs to the human body tissues. Chen & Chen (2001) observed decreasing order of the PAHs formed from methyl esters of linolenate, linoleate, oleate to stearate in model systems. They reported higher concentrations of PAHs in the soyabean oils than canola and sunflower oils. The fatty acid composition of edible oils appears to influence the formation of PAHs.

Purcaro *et al*. (2006) reported no notable increase of BaP when olive oils and peanut oils containing 8.1 and 0.1 µg/kg BAP were heated at 180 °C up to 25 h. Barranco *et al*. (2004) had similar observations on heating of Spanish fatty foods. Kim *et al.* (1993) reported increase of BaP in the soybean oil on heating up to 200 °C for 10–20 h. Dost and Ideli (2012) reported 2 to 8-fold increase of the PAHs during barbequing of meat and fish using corn, sunflower and olive oils containing 0.44–99 µg/kg of BaP. In several less commonly used edible oils, formation of light Σ_4PAHs (Phenanthrene, Anthracene, Fluorene, and Pyrene) appears to be predominant (>70%) compared with the carcinogenic Σ_4PAHs (Ciecierska & Obiedzinski, 2013b).

Smoke curing of coconut kernels to produce copra and mechanical expulsion of oil from the copra is a major industry in coconut producing countries. The concentrations of PAHs in coconut products (Table 4) reflect the individual effects of copra curing, expulsion and chemical refining of coconut oil (Wijeratne *et al*., 1996a). Wijeratne *et al*. (1998) observed more than 10-fold increase of Σ_8PAHs (367 µg/kg) than predicted (24 µg/kg) in oils mechanically expelled from copra. The increase is shown to be due to pyrolysis during expelling the oil in screw press at 140–160 °C, by experimentally expelling oil from desiccated coconuts free of the PAHs. This may be happening during the mechanical expulsion of other edible oils, too. Water jacketed expellers maintained at 50 °C are used in extracting the virgin coconut oil to prevent pyrolysis leading to formation of PAHs. Figure 2 indicates the gradual increase in formation of the PAHs starting around 55 °C on exposure of coconut oil in laboratory ovens at temperatures from 40–250 °C (Wijeratne *et al*., 1998). The exact cut off temperature cannot be established as convection currents in oil and other compositional conditions, too contribute to the reactions. Manually extracted coconut milk is boiled to break the emulsion in home preparation of coconut oil. During boiling, the liquid near the bottom of the pot reaches temperatures around 170 °C. The sediment gets charred and is high in PAHs. It is consumed as a delicacy (*Thelka*) mostly by children (Wijeratne *et al*., 1996b).

Copra is smoke cured, anticipating reduced fungal spoilage and aflatoxin production on storage. This assumption needs validation as both aflatoxins and BaP are class 1 carcinogens that require equal concern (Samarajeewa, 2003). Laboratory experiments comparing liquid smoke produced by burning coconut shells (containing PAHs) and Zesti^{TM} liquid smoke (free of PAHs) showed that suppression of fungal growth in grated coconuts was not due to the PAHs in smoke, but other chemicals (Rodrigo *et al*., 1995).

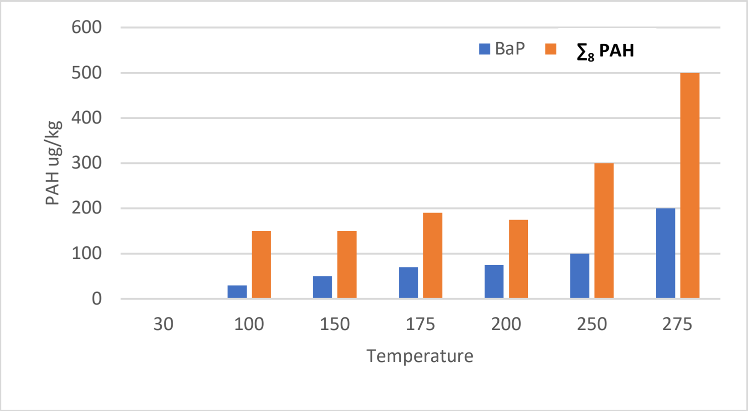

Figure 2: Formation of PAHs in coconut oil on exposure to different temperatures

A study describes increased formation of PAHs on three successive frying of fish with the same oil (Chukwujindu *et al*., 2020). The concentrations of Σ_{22}PAHs in the unused oils were in the range of 43.3–2107 µg/ kg, whereas those in the used oil ranged from 40.8–4090 µg/kg. The increase of PAHs appears to depend on the type of oil and also the type of fish.

In comparing the PAHs in 60 samples of BBQ pork, roasted pork and roasted duck collected from sales points, grilled pork showed highest concentrations with lower concentrations in the gas grilled or electric oven roasted meats (Table 4). Incomplete combustion during grilling may be contributing more to the formation of PAHs (Food and Environmental Hygiene Department, 2004). In controlled experiments with steam heated meat, carcinogenic Σ_3PAHs were not detected and the concentrations of total PAHs [Σ_{15}] observed were below the tolerance limit. It is known that the lipids in meats tend to drip on to fire undergoing pyrolysis to produce PAHs, which gets deposited on the meat. The lipids reaching the flame tend to reach temperatures of 500 °C or above instantly producing PAHs. A study comparing in-house preparations (shallow frying on electric hot plate, grilling in electric oven, barbequing in charcoal & wood chips, roasting in electric oven and toasting) showed no recognizable increase of PAHs [Σ_{27+1}] compared with the raw materials (Rose *et al*., 2015). Experiments in this study were conducted under well-defined conditions. However, BaP was produced in most foods when barbequed using charcoal and wood chips together. Rose *et al*. (2015) have observed highest concentrations of Σ_8PAHs in the barbeques using charcoal as fuel. The values PAHs were higher when the distance between charcoal and food was 4 cm against 7 cm. Halagarda and Wojciak (2022) reviewing the health and safety aspects of European meat products showed that the use of uncontrolled smoking in traditional /conventional products tend to cause heavier contamination with the PAHs. The overall observations suggest that the concentrations of PAHs in home prepared foods, where conditions are not well controlled, could be above the tolerance limits.

Siddique *et al*., (2021a) reported no further increase of the BaP after poaching, boiling, frying, grilling, microwaving and oven roasting using 4 types of oils containing BaP concentrations within the tolerance limits (Table 4). They detected fluorene (non-toxic) as the main PAH formed during frying. Their experiments were conducted under defined conditions, not allowing over exposure of the rabbit muscles to heat with the edible oils maintained below 140 °C in deep frying. Another study by Siddique *et al.* (2021b) examining the impact of conventional oil on frying of rabbit meat reported that PAHs are outside the toxic Σ_8. The PAHs outside toxic Σ_8 are of low carcinogenic relevance.

Baking exposes products to high temperatures. Most bakery products are rich in carbohydrates and low in lipids. Bread dough may get contaminated with PAHs from the environment or due to pyrolysis during baking. Bread was found to contain Σ_{19}PAHs at concentration range of 1.59 to 13.6 µg/kg in different locations of the bread and in relation to the baking temperatures (Ciecierska & Obiedziński, 2013a). In this study from 1.07 to 3.65 µg/kg Σ_{19}PAHs have been found in the dough even before baking. Authors suggest the concentrations of PAHs detected in baked products are of low health significance, considering probable dietary exposure. Comparable PAHs concentrations were observed in bread and breakfast cereals in bakery and cereal products in Turkish markets (Kacmaz, 2016). Research is needed on formation of PAHs in some of the flour-based sweets and foods prepared by frying in oils, or direct heating on pans respectively in the South Asian countries.

Roasting of coffee results in formation of PAHs due to heat while developing the aroma of consumer preference. Compared with hot air roasting, the superheated steam roasting of arabica coffee at 250 °C has shown to contain less light PAHs. Superheat treatment by steam was done in a fluidized bed drier (Rattanarat *et al*., 2021). Kinetic models were developed to predict formation of PAHs, namely Py, BaA, Chr and Anthracene, where good correlations between predictions and formation of PAHs on roasting at 180–260 °C for 5–10 min. were observed (Houessou, 2008).

Kim *et al*. (2020) reported Σ_8PAHs from several agricultural commodities in South Korea. Martorell *et al.* (2010) reported PAHs in randomly collected food groups in Spain. High levels of total PAHs were detected in the meat and meat products (38.99 µg/kg), followed by oils and fats (18.75 µg/kg), and dairy products (7.57 µg/kg). These concentrations were less than the values reported previously.

Prevention and removal

Preventing formation or deposition of the PAHs is the most rational approach to ensure food safety. The traditional smoking techniques are currently replaced by spraying of smoke flavours on foods to ensure consumer satisfaction avoiding hazards from PAHs. It is practical to monitor PAHs in smoke liquids prior to their application on foods.

Essumang *et al*., (2014) described modification of traditional smoke kilns using a filter system for PAHs. The filter was able to reduce the total PAHs by 21–69%. They reported mean BaP concentrations below the tolerance limit of 2 µg/kg in fish smoked by the modified kiln. Mechanisms to minimize PAHs during smoke curing by marinades, fuel-wood types, low fat raw materials, and the use of smoke filters are developed now to reduce food safety hazards (Dutta *et al*., 2022). While preventing contact of the meat with the flames during barbequing, the removal of drippings and smoke was reported to reduce Σ_4PAHs by 48–89% and 41–74% respectively in ready to consume meats (Lee *et al*., 2016). Controlled barbequing could notably reduce the PAHs in meats (Duedahl-Olesen *et al*., 2015b). This aspect is reviewed by Afe *et al*. (2020) projecting possible re-engineering approaches in food preparations for the future.

Edible oils are refined by deodorizing and bleaching to remove pigments and other constituents originating from the raw materials. Physical refining removes the PAHs in edible oils only partially. Olatunji *et al.* (2014) reported presence of three PAHs BaP, BkF and Phe (Phenanthrene) in sunflower oils and mixtures of sunflower and soya oils at concentration ranges of 5.3–7.7 µg/kg. They report reduction of Σ_{13} PAHs from the range 10–316 to 3–69 µg/kg in the soybean oil on refining (neutralization and deodorization). Rojo Carmago *et al*., (2012) described the possibility of reducing PAHs up to 88% by physical refining. The percent reduction seems to depend on the initial concentrations of PAHs in the foods. Chemical refining is far more effective in reducing the PAHs in edible oils as the alkali interacts with PAHs chemically, making them hydrophilic to be removed in the aqueous fraction.

The PAHs possess the capacity to absorb energy in the ultraviolet (wave length: 320 - 400 nm) and visible (400 nm) regions generating reactive species. This effect may be enhanced using catalysts and oxygen to degrade the PAHs. There is much interest currently to carry out catalysed photodegradation of soil PAHs in solid phase. Wang *et al.,* (2009) describe application of iron oxide to photodegrade pyrene. They describe a photo-fenton-like system operating with added hydrogen peroxide effectively to degrade PAHs in soil. An effort to degrade PAHs in coconut oil was made using moving layers of oil in a cascade set up used previously for solar degradation of aflatoxins in coconut oil (Samarajeewa *et al.*, 1985). The pilot experiments worked effectively (Wijeratne *et al.*, 1997). It is also noted that the molecules of PAHs activated by light have their toxicity increased 100-fold than in the dark (Hongtao, 2002) forming active toxic derivatives. The energized molecules tend to breakdown readily in their active phase. The principle may be applied with sunlight and catalysts to remove PAHs in the edible oils.

In smoke drying of coconut kernels, a variety of plant-based fuels are used in the industrial kilns. A comparison of smoke produced from different parts of coconut palm, burned under the same conditions and absorbed into grated coconut (free of PAH) showed high variations in content and concentrations (Figure 3). It is interesting to note that Sri Lanka uses only coconut shells as fuel for copra curing, whereas the fuels differ in other countries.

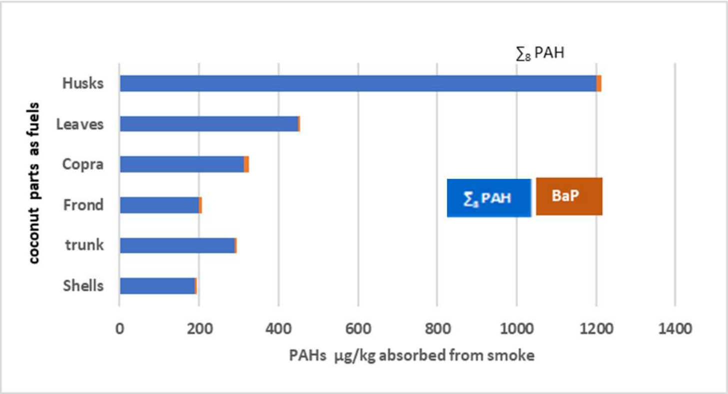

Figure 3: PAHs absorbed into grated coconut when exposed to smoke generated by different parts of the coconut palm used commonly as fuels (Rodrigo *et al.*, 1999)

Smoke flavourings are sprayed on meat products to provide consumer satisfaction preventing contaminations by the PAHs. Analysis of 18 commercial liquid smoke flavourings have shown total PAHs concentrations in the range of 6.3–43.7 µg/kg and carcinogenic PAHs concentrations of 0.3–10.2 µg/kg. Since the amounts of liquid smoke sprayed on to foods are of the order of few millilitres, food safety hazards cannot be expected (Gomma *et al.*, 1993). Smoke flavourings appear to be a better option as the most lipophilic PAHs do not appear in the condensates of watery smoke liquids (McDonald, 2015).

There are new approaches to use microorganisms to remediate PAHs and their toxicity from soil and other sources including food. This is an emerging area to minimize hazards (Kanaly & Haryama, 2000: Patel *et al.*, 2020; Premnath *et al.*, 2021).

Preventing food contaminations with the PAHs by environmental and industrial controls appears to be a need to address food safety hazards. Shift from smoke drying of copra using coconut shell to more mild drying using charcoal that was established in Sri Lanka is one such approach. Some of the photochemical methods could also possess prospects to eliminate PAHs in the foods.

Recommendations

1. The Environmental and Occupational Health Division of the Ministry of Health and Environment Authority in Sri Lanka need to establish limits for PAHs in air quality in smoke houses and major cities as the PAHs are proven to cause lung cancer. Incidence of lunger cancer is the highest among cancers in Sri Lanka.
2. Research to recognize occupational exposure levels of persons to smoke as a probable cause of lung cancer needs to be initiated.
3. Research to assess formation of the PAHs during frying of foods is needed.

4. There should be strict regulatory measures introduced to test the edible oil imports for PAHs or get accredited certification from exporting countries on the PAHs.
5. Restaurants need to shift barbequing of fish and meat from direct flame fires to gas or electric heating combined with spray of liquid smokes free of PAHs.
6. The coconut administration needs to assist the industry to move away from smoke copra kilns to indirect heating methods.
7. Considering the research evidence and methods of food manufacture, preparation and consumption patterns in Sri Lanka, regulatory limits given in Table 6 to ensure the safety of market foods from PAHs is suggested.

Table 6: Suggested regulatory guidelines for PAHs in foods in Sri Lanka

Foods	BaP µg/kg	Σ_4 PAHs µg/kg	Notes
Chemically refined coconut oil	2	10	Chemical refining removes all PAHs in the oil totally
Virgin coconut oil	2	10	Extraction is carried out below 55 °C to prevent formation of PAHs
Crude coconut oil and Coconut parings	10	50	Expulsion leads to formation of PAH at the screw press operating around 140 °C
Oil fried foods	5	10	Fraction of PAHs would be absorbed into foods during frying
Vegetable oils	2	10	Imported oils are expected to be chemically refined
Palm oil	2	10	
Desiccated coconut	2	10	There is no deposition or production of PAHs in the process
Food for children and sick using roasted rice	2	10	Minimize over-roasting of rice.
Spices (whole or powdered)	10	50	They are not smoke dried and used in small quantities in foods
Tea (Black)	10	50	PAHs are formed during kilning of black tea
Smoke cured fish, meat and sausages	5	12	Smoke deposition itself and action of heat on lipids in fish or meat produce PAHs

CONCLUSION

Of the PAHs, 16 compounds have been recognized scientifically to bring in food safety hazards in humans. They may be of petrogenic origin entering the food chain through fish. PAHs also may be airborne as a part of vehicle fumes, distillation of petroleum products and burning of wood and similar organic matter. PAHs are generated during contact between edible lipids and hot surfaces during roasting and frying or contacts between naked flames and fatty tissues of meat during grilling. BaP and some of the PAHs interfere with cellular metabolism in the body leading to interactions with DNA leading to mutagenicity. Controlled exposure of foods to heat during manufacture and cooking could reduce the PAHs to acceptable levels.

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