Review of PAH contamination in food products and their health hazards

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ABSTRACT

Public concern over the deleterious effects of polycyclic aromatic hydrocarbons (PAHs) has grown rapidly due to recognition of their toxicity, carcinogenicity, and teratogenicity. The aim of this review is to describe the status of PAH pollution among different food types, the route of dietary intake, measures for its reduction, and legislative approaches to control PAH. To this end, a comprehensive review is outlined to evaluate the status of PAH contamination in many important food categories along with dietary recommendations. Our discussion is also extended to describe preventive measures to reduce PAH in food products to help reduce the risks associated with human intake.

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Contents

1. Introduction

Polycyclic aromatic hydrocarbons (PAH) are defined as organic compounds that possess two or more fused aromatic rings of carbon and hydrogen atoms. They are pervasive environmental pollutants that are

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characterized by their hazardous carcinogenic and mutagenic potential (McGrath et al., 2007; Wegrzyn et al., 2006). PAHs are found ubiquitously, not only in all different environmental media (such as air, soil, and water), but also in various foods we encounter in our everyday life (Lodovici et al., 1995; Moon et al., 2010; Ishizaki et al., 2010; Ghasemzadeh-Mohammadi et al., 2012; Duedahl-Olesen et al., 2013). The primary sources of PAHs are identified as anthropogenic in origin such as the exhaust of motor vehicles, petroleum refineries, heating in power plants, combustion of refuse, deposition from sewage, oil/gasoline spills, tobacco smoke, barbeque smoke, and coke production (Christensen and Bzdusek, 2005; Moon et al., 2006). Due to the lipophilic and hydrophobic characteristics of PAHs, they tend to accumulate in the food chain (Pensado et al., 2005; Shadi et al., 2012). The occurrence of PAHs in food can be ascribed to diverse pathways that include both natural (as environmental) and synthetic sources (e.g. cooking practices and industrial food processing). On consumption, PAHs can be actively engaged in metabolic activation in human mammalian cells, diolepoxides, which have adherence to genetic material DNA, and tissues leading to mutations (Phillips, 1999). Consequently, numerous efforts have been directed to address how precisely and accurately one can quantify the PAHs that are present in food products.

PAHs have received increased attention in recent years because of the diversity of their sources. Dietary intake of PAHs is the major route of human exposure (Xia et al., 2010). In recent years, increases in concentration levels of PAH in various environmental media have been reported not only in developing but also in developed countries, accrediting to their wide spread in food chain through consumption of numerous food stuffs. Dietary intake of PAHs poses the potential health hazards to the public (Xia et al., 2010). Dietary exposure of PAH resulted in enhanced urinary elimination rates relative to occupational exposure (Buckley and Lioy, 1992). Also, PAHs concentrations consumed by local residents of Taiyuan were found to have incremental life time cancer risks, implying the significance of dietary risk (Xia et al., 2013). According to Falco' et al. (2005), the consumption of food stuffs recorded the highest contribution to the intake of pollutants. Such risk is associated with all common dietary components such as vegetables, cereals, and oils followed by consumption of open flame cooked meat (Grova et al., 2006). The burgeoning trend of processed products has led to even higher concentrations of PAHs in foodstuffs. The channelization of their formation in grilled/smoked foods is described in Fig. 1. Furthermore, PAHs also enter into packaged foods through packaging processes (e.g., treatment with polluted oils).

The quantitative assessment of PAHs in food products and human consumption patterns is a focus of interest due to their expeditious formation in food (Moon et al., 2010; Yebra-Pimentel et al., 2013). A list of PAHs commonly found in food components are shown in Table 1 with their basic properties and three letter acronyms. The common targets of analysis (as food components) include, but are not limited to, pyrene (PYR), fluorene (FLR), naphthalene (NAP), phenanthrene (PHN), benzo[b]fluoranthene (BBF), benzo[e]fluoranthene (BEF), benzo[a]fluorathene (BAF), anthracene (ANT), fluoranthene (FLT), benzo[a]anthracene (BAA), chrysene (CHY), benzo[k]fluoranthene

(BKF), dibenzoanthracene (DBA), dibenzopyrene (DBP), and indopyrene (INP) (Phillips, 1999; Srogi, 2007; Xia et al., 2010). PAHs in foods are suggested as one of the major factors contributing to skin and lung cancer (Kameda et al., 2005; Lee and Shim, 2007; Yoon et al., 2007; Zhang et al., 2009).

This article is written to describe the significance of PAH contamination in the diverse food products humans take as their main dietary components. To this end, we briefly describe important methods to control exposure through dietary intake.

2. Classification of PAH and their toxicity

The scientific community on foods (SCF) provided a comprehensive review of the toxicity of PAHs in foods in an outlook on December 2002. They placed BAP as a PAH marker for food (as shown in Table 2A). However, at the same time, they suggested the need for additional PAHs in lieu of manifestation of the PAH contamination profile. The need for grouping of other PAHs was also addressed by the EU (European Commission Recommendation No 2005/108/EC). In 2007, the European Food Safety Authority (EFSA) ruled that there was uncertainty in BAP as a reliable marker. It was in 2007 that the selection commission (2005/108/EC) had asked the member states to survey the 15 compounds in the EC regulations (208/2005). Then, the results were summarized as the 'Findings of EFSA data collection on PAHs in Food' in 2007 (Ciecierska and Obiedziński, 2013). Therefore, in succession, 18 countries submitted their analytical results of various food products for the years 2005–2007. Furthermore, in 2008, the SCF determined clear confirmation of the genotoxicity of 15 PAHs (namely, BAA, BBF, BJF, B(ghi)perylene, BKF, benzo(ghi)perylene, BAP, CHY, cyclopental(c,d)pyrene, DBA, dibenzo(ae)pyrene, dibenzo(ah)pyrene, dibenzo(ai)pyrene, INP, and MCH (5-methyl chrysene)) in the somatic cells of experimental animals (European Food Safety Authority, 2008).

As a result, the EFSA in 2008 declared that BAP is not an appropriate marker for the occurrence of PAHs in food. Therefore in 2008, a combination of two PAHs particularly under PAH2 (BAP + CHY), four specific PAHs as PAH4 (PAH2 + BAA + BBF), and PAH8 (PAH4 + BKF, BPY, DBA, and INP) were introduced as more accurate markers. EFSA also concluded that a system of eight substances (PAH8) would not provide much added value compared to a system of four substances (PAH4). Following this, the EU commission (2011) established regulation guidelines for the maximum levels of PAH4 in food matrices. EFSA deduced that such system would ensure the PAH levels in food under the restraint that do not cause health concern and that the amount of PAH can also be controlled in those samples in which BAP is not detectable, but where other PAHs are present. It was regarded that for the sum of the four substances (PAH4), the lower bound concentrations should be used as the basis for adherence of safe standards.

Earlier in 2006, the maximum PAH levels for various food products were established by European Commission regulation. In 2011, a new guideline for the maximum PAH levels (of Commission Regulation (EC) No. 1881/2006) was updated by commission regulation in 2011 (EU No 835/2011) (as shown in Table 2B). To date, the legislative
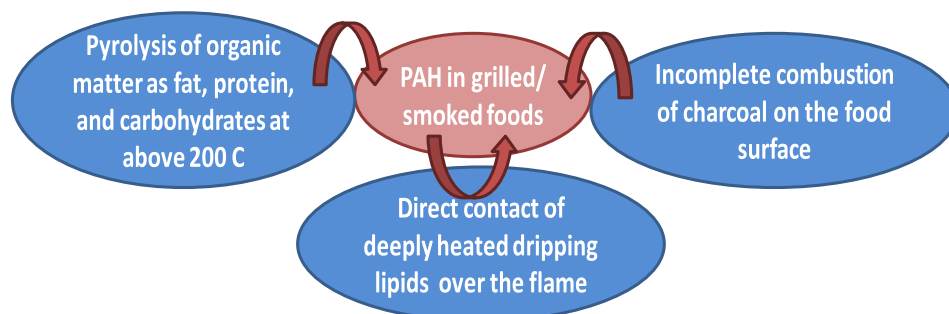

Fig. 1. Channelization of the PAH formation route in grilled/smoked food.

Table 1
List of important polycyclic aromatic compounds (PAHs) as major contaminants in food components.

Order	PAH	Abbreviation	Chemical formula	CAS number	Molecular mass (g/mol)	Toxic equivalency factor (TEF)	Structure
1	Napthalene	NAP	C10H8	91-20-3	128.17	0.001	
2	Fluorene	FLR	C13H10	86-73-7	166.22	0.001	
3	Anthracene	ANT	C14H10	120-12-7	178.23	0.01	
4	Phenanthrene	PHN	C14H10	85-01-8	178.23	0.001	
5	Fluoranthene	FLT	C16H10	206-44-0	202.26	0.001	
6	Pyrene	PYR	C16H10	129-00-0	202.25	0.001	
7	Benzo[a]anthracene	BAA	C18H12	56-55-3	228	0.1	
8	Chrysene	CHY	C18H12	218-01-9	228	0.001	
9	Benzo[a]pyrene	BAP	C20H12	50-32-8	252	1	
10	Benzo[b]fluoranthene	BBF	C20H12	205-99-2	252	0.1	
11	Benzo[e]pyrene	BEP	C20H12	192-97-2	252.31	–	
12	Benzo[j]fluoranthene	BJF	C20H12	205-82-3	252	–	
13	Benzo[k]fluoranthene	BKF	C20H12	207-08-09	252	0.01	
14	Dibenzo[a,h]anthracene	DBA	C22H14	53-70-3	278	1	
15	Indo[123-cd]pyrene	IP	C22H12	193-39-5	276	0.1	
16	Dibenzo[aL]pyrene	DBP	C24H14	191-30-0	302	–	

guidelines for PAH levels have been lacking due to the complexity of the matrices containing the food components. Therefore, there is a need for accumulation of more reliable data based on accurate quantification of the PAH contained in various foods. As such, the guidance levels are to be further refined for proper regulation of the deleterious targets, specifically 15 genotoxic PAHs as well as PAH8.

The presence of carcinogenicity in PAHs has drawn a great deal of concern. Toxic equivalency factors (TEFs^{carc}) are commonly used to quantify the toxicity or carcinogenicity of PAHs through the approximation of BAP for calculating the approximation of BAP equivalent doses (Qiao et al., 2006). This approach to risk assessment has been divided

Table 2
Administrative regulation of BAP levels in food. (A) Maximum allowable levels of BAP in regulation (EC no. 1881/year 2006). (B) Maximum levels for BAP Commission Regulation (EU) 835/August 2011.

| Order	Food commodity	Maximum level (μg/kg)
1	Oils and fats for human consumption	2
2	Smoked meat and its products	5
3	Smoked fish and the muscle meat of fish	2
4	Processed cereal-based products for infants and young children	1
5	Processed baby foods for infants and infant milk formula	1
6	Seafood including crustaceans, cephalopods (excluding crab and lobster), and molluscs	5

Order	Food commodities	BAP (μg/kg)
1	Smoked meat and smoked products	2
2	Muscle meat of smoked fish	5
3	Smoked crustaceans and crabs	2
4	Molluscs (fresh, filled, or frozen), heat treated meat products	5
5	Smoked molluscs	6
6	Oils and fats	2
7	Cereal-based products for infants and young children	1
8	Infant milk and milk formulae	1
9	Cocoa butter and chocolates	5

into two categories, carcinogenic and non-carcinogenic, by the EPA in 1984. The carcinogenic slope factor was derived from the assays on BAP, which is used for TEF evaluation of PAH. Note that BAP is the only PAH among all known toxic PAHs for the derivation of a carcinogenic factor (Qiao et al., 2006). The values of TEF for each PAH are shown in Table 1. Furthermore, the adverse effects of PAHs have been highlighted not only for a variety of cancers but also for the non-genotoxic diseases (such as diabetes mellitus (DM) and cardiovascular disorders) (Burstyn et al., 2005; Grant, 2009; Hu et al., 2015). The incremented incidence of dietary intake of PAHs has resulted in the metabolism defects due to their persistent bioaccumulation in food chain. The PAHs' resistance against degradation hinders the metabolic activity of glucose homeostasis and β-cell function to act as the potential cause of the Type 2 diabetes (T2DM) (Andersen et al., 2012; Raaschou-Nielsen et al., 2013; Alshaarawy et al., 2014). These non-genotoxic effects of the continuous environmental and dietary (charred and broiled meat) exposure has become one of the significant casual factors contribution to the development of metabolic syndrome in terms of dyslipidemia and raised blood pressure (Hu et al., 2015).

3. Sources of PAH contamination in food products

There are different pathways of PAH contamination among different foods, as shown in Fig. 2. The occurrence of PAHs in the environment became a matter of high concern because man-made activities are contributing to their existence in routes both known and unknown. Among the various routes, food is the major route of human exposure, particularly among those who do not smoke (Fig. 2). One of their commonest source pathways to raw food (i.e., fruits and vegetables) is the environmental route, pertaining to PAHs present in the soil and air. It can be significant, especially when they are growing next to industrial sources or areas with dense traffic (Fahnrich et al., 2002). In a busy traffic area, the PAH concentration in the soil layer (e.g., a depth of 5–15 cm) was found
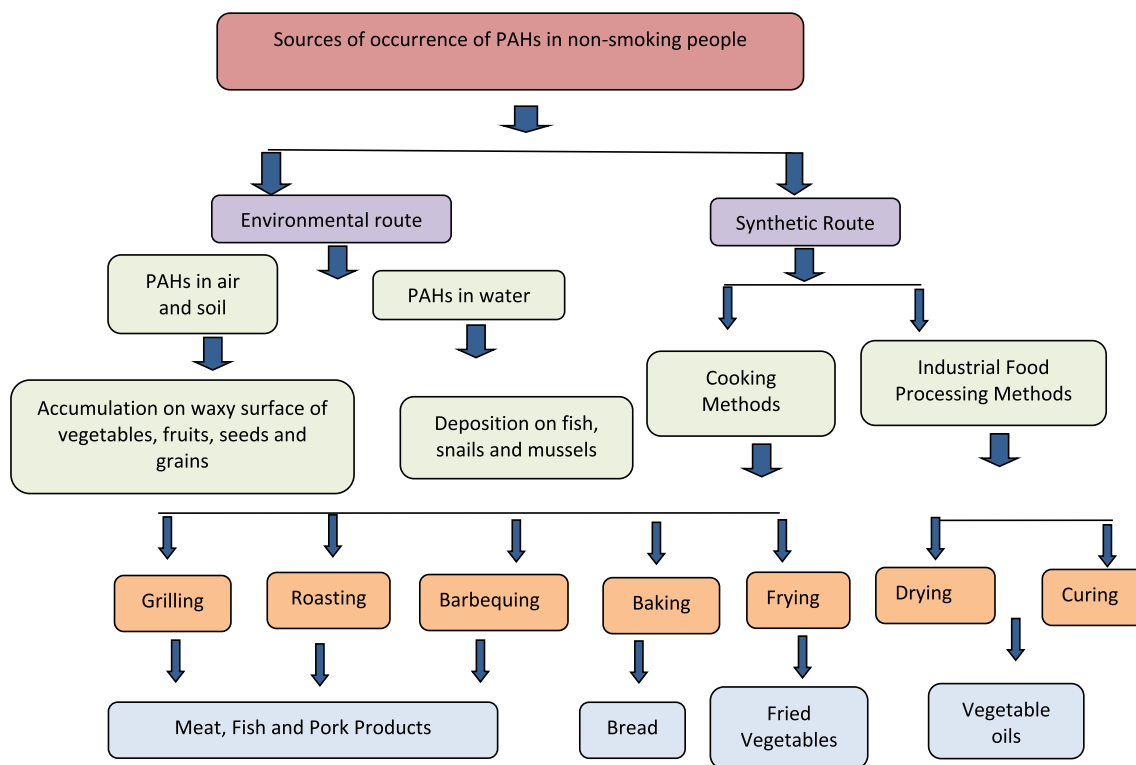
Fig. 2. Flowchart showing the pathways of PAH contamination in various food products.

at ≈ 3095 μg/kg (Bishnoi et al., 2006). Consequently, changes in their contents were also observed in the microbes growing in the soil, while the growth of other soil organisms that are not conducive to healthy vegetation is proliferating (Andersson et al., 2001). Similarly, contamination of PAH in water can also contribute to human intake of PAHs through drinking such as the direct consumption of aquatic organisms (e.g., crabs, fish, mussels, oysters, and snails) (Fahnrich et al., 2002; Ramalhosa et al., 2009; Dhananjayan and Muralidharan, 2012).

Another efficient pathway of PAH intake is majorly attributed to man-made activities in the form of cooking (grilling, roasting, and frying) and processing (performed at industrial level). The efficiency of this route is dependent on various factors such as heat source, distance of heating, design of the food device, and type of fuel, which can further help foster the production of PAHs in food products (Lijinsky, 1991; Vyrand et al., 2007). Thus, cooking processes play an inevitable role in the formation of PAH which tends to proceed with chemical distortion of the original nutrient contents in foods (e.g., proteins, carbohydrates, and lipids).

3.1. Plant-based foods

3.1.1. Cereal grains

Cereals are one of the major dietary routes of PAH exposure in humans (Orecchio and Papuzza, 2009). Bread is the most common starch product in the human diet. It is a good source of energy, minerals, and vitamins essential for human nutrition (Ciecierska and Obiedziński, 2013). Although the consumption rate of baked and packaged bread has gradually been increasing, the maximum levels of PAHs have not been established. As the baking process requires high temperature, contamination of bread inevitably takes place thereby posing threats.

The effect of toasting bread has been studied with respect to the formation of PAHs (e.g., Al-Rashdan et al., 2010). The PAHs contained in both bread types are compared in Table 1S in Supplementary information. The key representation of Table 1S has been shown through Fig. 3. No BAP was detected in any of the flour, while some low and high weight PAHs were found in the bread. For instance, heavier PAHs

(like FLT and PYR) were observed in brown and wheat flour in the range of 1.19–2.19 μg/kg and 0.71–1.66 μg/kg, respectively. Some low molecular weight PAHs (like NAP, FLR, and PHN) were also found in both white and brown wheat flour.

Alomirah et al. (2011) reported the formation of genotoxic PAHs in pita bread (Table 1S). Although the amount of BAP in Pita bread was negligible, it was found to contain low concentrations of genotoxic PAHs (PAH8) (0.94 μg/kg) and total PAHs (17.6 μg/kg). On the contrary, pita bread with meat fat dripping exhibited the highest amount of BAP (1.92 μg/kg) and PAH8 (14.0 μg/kg), indicating the probable role of meat fat on the enhancement of PAH formation (Alomirah et al., 2011). As such, the role of pyrolysis was significant as meat fat was dripped onto the heat surface (leading to incomplete combustion). The type of energy used in heating (such as electricity, wood, flame, or solar energy) can also contribute to contamination by PAHs in baked bread. Ahmed et al. (2000) reported the average PAH levels in baked bread, when cooked using solar, solid waste, and electricity. The levels of PAHs in toasted bread were also found in the range of 7.38–18.0 μg/kg (Nieva-Cano et al., 2001). Although the EU endeavored to demarcate the range of PAHs for various foodstuffs, there is a scarcity of information in the case of bread.

3.1.2. Fruits and vegetables

A wide variety of PAHs have been reported in plant-based food products. The accumulation of PAHs in plants can lead to their exposure to humans on a significant level through intake of fruits and vegetables. Indeed, the routes of their contamination are diverse enough to include atmospheric exposure, soil uptake, and autogenous biosynthesis (Guillen et al., 1997). In most cases, atmospheric exposure is identified as the dominant component of all the routes of contamination (Wild and Jones, 1992; Phillips, 1999). It is observed that leafy vegetables are usually more contaminated than stem vegetables owing to their larger, waxy, and cuticle surface area, which is vulnerable to the deposition of PAH. However, the type of pollution source or site (e.g. near industrial set-ups and highways) increases the amount on leafy
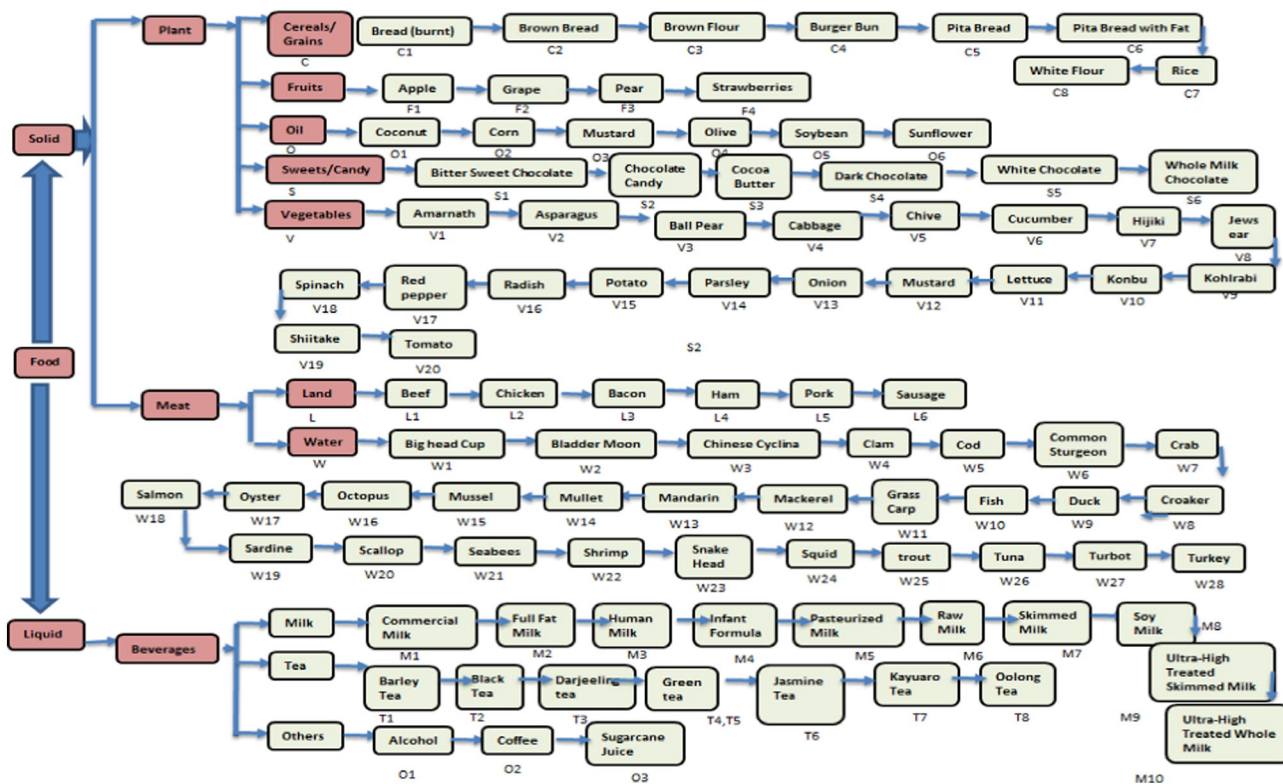
Fig. 3. Classification of food product types in relation to PAH contamination.

vegetables. At the same time, they can extend the threat to other non-leafy vegetables as well. Similarly, the root vegetables (potato, radish, and carrot) are prone to uptake PAH more easily from contaminated soil sites (due to their high potential to penetrate beneath the soil) relative to stem vegetables (cucumber, eggplant, and tomato). The PAH concentration in vegetables also depends on the physical distance from sources (industrial emissions) and meteorological conditions. It thus suggests the substantial vulnerability of vegetable tissues towards the accumulation of pollutants such as PAHs from aerial deposits. Therefore, there are three types of PAH uptake routes for vegetables and fruits: 1) contaminated soil, 2) contaminated water uptake, and 3) endogenous synthesis of PAH in tissues (Wennrich et al., 2002).

The sum concentrations of several PAHs (FLR, ANT, FLT, BAP, CHY, BAA, BKF, BBF, DBA, PYR, and INP) in strawberries and apples from an industrial town of Germany (Bitterfeld Wolfen) were 1.9–6.0 and 4.1–15 µg/kg, respectively (Wennrich et al., 2002). Likewise, the total PAH levels (FLR, PYR, BAA, BBF, BKF, and BAP) of apple, pear, and grape in the industrial city of Campinas (Brazil) were 4.05, 4, and 3.77 µg/kg, respectively. It was observed that fruits tend to be less contaminated with PAH than vegetables due to the reduced chance of soil uptake (Rojo, 2003).

Among the vegetables, broad leafy green vegetables like lettuce were reported to have a relatively high concentration of total PAHs for cabbage (9 µg/kg) and lettuce (14 µg/kg) (Rojo, 2003). Many other vegetables like tomatoes (9.50 µg/kg) were also found to have relatively high total PAHs (Rojo, 2003). In addition, Wennrich et al. (2002) also found higher total PAH levels (µg/kg) in various vegetables (lettuce 2.02–4, parsley 15–120, kohlrabi 1–4, potatoes 0.5–1.7, and tomato 0.95–1.5) along with other vegetables like mushroom and radish strips with a pyrene content of 16 µg/kg (Zougagh et al., 2004). Likewise, vegetables grown from Northern China sold in the wholesale markets of China were found to contain ANT, FLT, BAA, and BAP (6.46, 4.05, 3.33, and 4.05 µg/kg, respectively) (Zhong and Wang, 2002). They observed PAH levels in leafy (Chinese cabbage 29, cabbage 23, and celery 31 µg/kg), stem (tomato 14.81, wax gourd 15, and cucumber 12 µg/kg), and root vegetables (potato 12.54

µg/kg), which had high total PAH levels relative to fruit vegetables (eggplant 9.79 µg/kg). However, the peels of roots and fruit vegetables were found to have more PAHs in their pulp than in their core (Zhong and Wang, 2002).

As shown in Table 1S, relatively enhanced PAH content was also measured from root vegetables such as potatoes and carrots (13 µg/kg) (Ashraf et al., 2013). Bishnoi et al. (2006) observed significantly large quantities (µg/kg) of total PAHs in radish (128.5), colocasia (69), and potato (59.8). In addition, they found the maximum amount of PAH from cabbage (11.6 µg/kg) among leafy vegetables, while a relatively low concentration was seen in fruit vegetables. The greatest level of BAP was recorded in potato (3.19 µg/ kg), whereas it was BAA (3.14 µg/kg) for turnip cores (Bishnoi et al., 2006). Similarly, Zohair (2006) reported 16 PAHs in leafy vegetables, ranging from 1.22 to 12.63 µg/kg. Since leaves contain a greater surface area, they seem to accumulate more PAHs from the environment (Lin et al., 2005).

Evidence collected to date generally indicates an inclination towards the presence of more PAHs in plant-based uncooked foods (1) tubers (potato, carrot, and radish); (2) stem-based plants (cucumber, tomato, and gourds); and (3) leafy vegetables (cabbage, lettuce, and celery). The PAH content appears to be rather sensitive to environmental and regional variables (Zhong and Wang, 2002; Bishnoi et al., 2006; Zohair, 2006 Ashraf et al., 2012). Generally, PAHs of low molecular weight are engulfed by the waxy surface of leaves due to atmospheric fallout (Ashraf et al., 2013). The main reason for their occurrence is emission from man-made sources (e.g., fossil fuels) and uptake from roots through contaminated soil (Meudec et al., 2006). About 8,600 to 111,000 µg/kg of PAHs were detected from Chinese leafy vegetables (Zhang et al., 2004); it was therefore concluded that the contamination of vegetables occurs more commonly via root channels than via other pathways.

3.1.3. Oils

Fats and oils are significant dietary sources of PAHs. The occurrence of PAHs in vegetable oils is generally explained by the combined effects

of many factors and processes including the drying processes of the oil seeds (with the combustion of gases), contamination during solvent extraction, packaging material, soil burn, residue of mineral oils, relocation from contaminated water (or soil), and the raw material of vegetable oil (Amzad and Salehuddin, 2012; Standing Committee on Food Stuffs, 2001). Naturally attained vegetable oils are free of PAHs; however, their contamination proceeds via atmospheric deposition on growing crops or during technological processing (Fromberg et al., 2007). Their concentrations in refined oils are generally in the range of a few μg/kg. In the case of crude oil, it was found to exceed 2000 μg/kg of total PAHs (Moret et al., 2005). The cause of such contamination in a raw material with fatty acids (e.g., linoleic acid in grape seed oil) should probably reflect the effects of the compression of pomace (solids remaining after grape pressing) with heavy bulldozers to reduce the volume of raw material. Likewise, grinding the seeds (or dewaxing) may also facilitate the formation of heavier compounds (five condensed rings or more). Table 1S describes a recent survey of PAH contamination levels that compares various vegetable oils.

Nine PAHs were seen in corn oil, in the range of 6.40–76.08 μg L^{-1} where FLT was the highest followed by BKF (Dost and İdeli, 2012). However, the amount of BAP was 10 times higher than the legal level recommended by the European commission (Table 1S). In the case of sunflower oil, 6 PAHs were seen, but at amounts lower than those of other oils (corn and olive). On the contrary, olive contained 3 PAHs (FLT, PHN, and PYR), while BAP was absent in both olive and sunflower oil (Zougagh et al., 2004; Arrebola et al., 2006). The risk of PAH exposure due to consumption of corn oil may be due mainly to its deposition on growing corn crops, as little change in the PAH levels was seen after the refining process (Dost and İdeli, 2012). Nonetheless, some authors have put a great deal of effort into describing the possible role of refining in reducing PAHs levels. According to Teixeira et al. (2007), the contamination levels of PAHs in crude oils differed widely. However, refined oils generally contained low quantities of PAHs relative to crude ones. These authors reported the content of 15 PAHs in samples of various commercial oils (including virgin olive oil, refined sunflower oil, and soybean oil). These oils did not surpass the guideline limits recommended by the German Society of Fat Science (GSFS). Furthermore, Hossain et al. (2012) reported the effect of refining (particularly deodorization and neutralization) on the reduction of light molecular weight PAHs and found a low concentration of PAHs (NAP, PHN, FLR, PYR, CHY, BAP, and BAA) in soybean oil, mustard oil, and coconut oil. Therefore, in terms of safety prospects, the addition of a refining process is likely to help reduce the content of PAHs in vegetable oils.

3.1.4. Sweets/candy

Sweets like chocolates are relished by all age groups worldwide and are a key ingredient in most desserts (milk shakes, candies, cookies, bars, etc.). The main ingredient of chocolate is cocoa. World cocoa consumption was found be 2.83 million tonnes in the previous decade (1990 to 2000) (Medium-term prospects for agricultural commodities, 2010). The consumption of chocolate amounted to about 9.1 kg per capita in Germany as of 2007 (The Association of Germany Confectionary Industry). As the formation of PAH in these sweet products is ascribed to cocoa butter and sugar, they are also a potent source of dietary PAH intake. PAH contamination in chocolate is affected by drying, roasting, winnowing, blending, and fermenting of the cocoa seeds (The world atlas class of chocolate, 2003).

Ziegenhals et al. (2009) analyzed the concentration of PAHs in different types of chocolate at various compositions of cocoa content. The BAP concentration in all the chocolates ranged from 0.07–0.63 μg/kg. However, among the category of fluoranthenes, BBF has more levels than BKF, whereas DBP was not present in detectable amounts (≤0.07 μg/kg). It was shown that no correlation existed between the PAH contents in chocolate and the labeled cocoa contents (Ziegenhals et al., 2009). Due to the lack of information on the labels of some chocolate, it was difficult to assess the effect of cocoa butter on

PAH content (Lowor et al., 2012; Ziegenhals et al., 2009). Kumari et al. (2012) analyzed PAH levels (NAP, FLR, PHN, ACN, FLT, PYR, BAP, BAA, BBF, BKF, and DBA) in chocolate candy samples, detecting total PAH in the range of 2.7 to 235 μg/kg. The mean content of BAP was 1.62 μg/kg for all samples (n = 25), while FLT, PHN, and PYR were found in fewer samples.

Cocoa butter has long been considered as a source of contamination of PAH owing to the improper drying processes of cocoa beans (Lowor et al., 2012). However, a slight barrier effect by the cocoa shell was observed to prevent PAH formation. European No. 835/2011 amended the contamination regulation pertaining to the cocoa beans and its derivative products to a maximum concentration of 5.0 μg/kg fat for BAP and 35 μg/kg fat for PAH4 (30 μg/kg fat for PAH4 as of April 2015) (Raters and Matissek, 2014). These authors quantified the PAH contents (BAA, CHR, BBF, and BAP) in the samples of cocoa and chocolates as 12.44 μg/kg (fat) for PAH4 and 0.96 μg/kg (fat) for BAP. Thus, relatively low contamination levels of PAH were found in raw coca and semi-finished coca products. A decrease in the content of PAH in cocoa butter samples was observed from a comparative study made over an extended period (1999–2012) from 17 μg/kg (fat) (1999) to 8.8 μg/kg (fat) (2012) in terms of PAH4 (Raters and Matissek, 2014). This decrease was due to the strict code of practices for the drying and smoking processes of cocoa (Codex Alimentarius Commision, 2009). Although limited data are available for PAH content in chocolates and candies, contamination was generally detected at controlled levels.

3.2. Meat-based food products

3.2.1. Land-based products

Meat and its products are a major food consumed all over the world, serving as the most common route of human PAH intake (Alomirah et al., 2011). Barbequed meat products have been consumed by a great proportion of the population (Reinik et al., 2007). In the case of meat products, the most culpable sources of PAHs have been identified as the coke ovens of iron, steel, and aluminum heating in power plants at high temperature (Reinik et al., 2007). The contamination of meat products with PAHs usually occurs due to the combustion of organic matter during the smoking process (Farhadian et al., 2012). Hence, their contamination became worse during the smoking process when using grills made of iron and steel. In line with this observation, the consumption of grilled (or barbequed) meat was the predominant causative factor contributing to 21% of daily intake of BAP in the USA (Kazerouni et al., 2001). In the case of toxic PAHs (FLT, BBF, and BAP), Farhadian et al. (2010) found that beef products contained the maximum amount of BAP (12.5 μg/kg) among various grilled meat products (e.g., grilled chicken, chicken satay, and beef satay), as shown in (Table 1S).

BAP was classified as a possible human carcinogen by the International agency for research on cancer (Agarwal et al., 2009). Relatively high quantities of BAP were detected from pork-based meat (e.g., smoked ham, belly pork ham, and smoked wild boar ham) in the concentrations range of 0.02–17.63 μg/kg (Jira et al., 2008; Janoszka, 2011). This enhanced level of PAH is likely to reflect the increased fat content in pork products relative to chicken. FLT was not detected in the majority of chicken-based meat products when grilled; in contrast, beef satay was found to be contaminated with PAH FLT in the range of 6.22–13.8 μg/kg (Farhadian et al., 2010).

The guideline limit of BAP in grilled meat is estimated to be 10.5 μg/kg (International Agency Research on Cancer, 1993). The amount of BAP was commonly found significantly higher in the preparation of beef and chicken satay (range: 0.62–13.8 μg/kg). Farhadian et al. (2011) reported the increased presence of FLT, BBF, and NAP in satay of beef and chicken when prepared on the grills and rods. This is because high-protein foods like grilled meat products became contaminated by PAH due to the smoke produced from the incomplete combustion via grilling. Thus, direct heating in grilling process (at high temperature) fosters the direct pyrolysis of food nutrients. However,

this effect of PAH generation on direct grilling the meat products was found to be reduced if two types of pre-treatments (i.e., pre-heating and wrapping (with aluminum and banana leaf)) were applied. It was observed that no carcinogenic PAHs (BAP, BBF, and FLN) were produced in the grilled-meat samples after pre-heating with steam and micro-wave (Farhadian et al., 2011). In addition, wrapping with aluminum and banana leaf was found to reduce PAHs more effectively than pre-heating treatments (steam and microwave). Therefore, these simple pre-treatments of wrapping and pre-heating can be feasibly exercised both at homes and restaurants to practically reduce the exposure of carcinogenic PAHs to the consumers.

3.2.2. Water-based food products

Seafood has become more popular worldwide. However, this growth has attracted the concern of researchers working on the toxic effects of PAH. Consumers prefer smoked fish due to several reasons such as taste, amount of essential fatty acids, and readiness to eat. Among seafoods, some common preferences include the smoked/grilled/fried cooking of many types of fish (horse mackerel, chub mackerel sardine, mandarin fish, etc.). Along with fish, many other types of seafood are also favored such as shrimp, crab, oyster, scallop, and mussels. Although these species can be contaminated due to cooking, PAH can also be generated in their bodies via metabolic activities (from deposits through air and soil) that lead to accumulation of PAHs in the food chain (Tfouni and Toledo, 2007).

PAH levels in aquatic species have also been determined by many authors, as shown in Table 1S. FLT, PHN, and ANT were present in more than 50% of aquatic samples including mandarin fish, Japanese Spanish mackerel, and robust tongue fish (Zhang et al., 2010). The highest concentrations of FLR, PHN, and ANT were found to be as high as 58, 16, and 18 µg/kg, respectively. The concentrations of PAHs were exceptionally high for fish, shrimps, and crabs surpassing the maximum allowance levels of 30 µg/kg (established by the French government in 2000), as reported by Pensado et al. (2005). However, these authors observed that the concentrations of 12 PAHs in aquatic samples were below the guideline limit of the European Commission (EC, 208/2005). The most consumed species of fishes are horse mackerel, chub mackerel, and sardines, which belong to the pelagic food group (Ramalhosa et al., 2012a,b). These species have a global production of 150,000 tonnes per year (FAO, 2008); they are generally found to be the main species in Europe and Portugal where the annual consumption is 56 kg per year per capita (European Commission, 2010). Ramalhosa et al. (2012a,b) reported higher concentrations of naphthalene in these three species (Table 1S). This may be related to the fat content of these species which influences the accumulation of PAH as it is lipophilic in nature. The relative ordering in the fat content was in the order: sardine > chub mackerel > horse mackerel (Ramalhosa et al., 2012a,b). This result signified a positive relationship between lipid content and PAH residual levels (Xu et al., 2011). This provides further evidence that the lipid content of the tissues is the determining factor in the bio-accumulation of PAHs by fish (Basak et al., 2010; Ramalhosa et al., 2012a,b; Xu et al., 2011).

The levels of BAP in smoked Korean fish were seen in the range of 0.17–2.87 µg/kg (Cho and Shin, 2012). In contrast, total genotoxins (PAH8, chrysene, and BAA) were found in smoked fish as (11.71, 5, and 2.27 µg/kg, respectively (Alomirah et al., 2011). As many recent studies have found health related contaminants in a variety of fish, marine organisms are good parameters to assess the eco-health of an aquatic environment (Bihari et al., 2007; Serpe et al., 2010). 11). A total of 11 PAHs in mussel samples were seen in the range of 0.2–16 µg/kg (wet weight) (Alomirah et al., 2011). As such, the major threat to the health of the farmers should also be considered from contamination of their primary source of livelihood (Alomirah et al., 2011).

Among seafoods, shrimps, and squids are also delightful choices for many consumers. Perugini et al. (2007) observed that the concentration levels of several PAHs (ANT, FLT, PYR, BAA, CHY, BBF, and BKF) vary considerably between different seafoods (e.g., cephalopods (squid), crustaceans (shrimps), mussels, and fish) collected from the Gulf of Naples (depth of 30–60 cm). They observed that 72% of mussels had the highest PAH levels of 45–207 µg/kg (fresh weight). In addition, the maximum levels of heavier PAHs (BAP, DBA, and BPY) in mussels were also detected particularly during the winter (December) owing to the inflow of marine and ground water (Perugini et al., 2007). However, at the same time, the fish were not contaminated with heavier PAHs in that period. Fish are supposed to have less metabolizing activity than mussels as they can only take up PAHs in dissolved form (Perugini et al., 2007). The metabolic capacity of PAHs decreases in the order of fish > crustaceans > molluscs (Livingstone, 1994; Stegeman and Lech, 1991; Xia et al., 2012). Hence, the formation of PAHs in sea food is likely to be affected by such factors as the geographical region, presence of pollutants, and season. The levels of some key PAHs (such as ANP, BAP, DBA, ANT, FLR, BAA, BBF, and BKF) were measured from some marine organisms (cephalopods, crustaceans, and fish) in the Adriatic Sea, Italy (Perugini et al., 2007). Accordingly, Atlantic mackerel and European hake were the most heavily contaminated by the PAHs above (44.1 to 63.3 µg/kg), whereas Mediterranean mussels were only moderately contaminated in that respect. This may at least partially reflect the different uptake and metabolic rate of these invertebrate species.

Low weight PAHs are usually found in all marine organisms as they tend to bound more heavily with particulate sediments and are stored in the bottom, which leads to reduced uptake of the heavier ones by marine organisms. As winter is the period of maturation for reproductive cells (or gametogenesis), many species (e.g., oysters) release less PAHs (due to the deposition of eggs and the low metabolization of PAH in marine organisms) (Lemke and Kennedy, 1997; Perugini et al., 2007). Moreover, the rate of PAH deposition to marine organisms is suggested to be affected by the number of variables (like the spawning period, water temperature, and oxygen levels of water) (Perugini et al., 2007).

Similarly, the concentration effect of the area was also investigated by Xia et al. (2012). They analyzed seafood samples (fishes, crabs, shrimps, and oysters) collected from areas of the Mississippi Gulf Coast affected by a deep water oil spill. The effect was studied within the year of the incident (the deepwater horizon oil spill into the Gulf of Mexico in April–July 2010). Although (2 to 6-ringed) PAHs were detected in abundance just after the oil spill, their levels after about one year were considerably reduced relative to the guideline limits set by the NAOO/FDA/Gulf Coast. The concentration levels of total PAHs in fish samples were 19 µg/kg during the early period, but they decreased to 6 µg/kg in the later period. Likewise, the PAH levels in the crab and oyster samples were also reduced from 42 to 13 and 34 to 15 µg/kg, respectively. The decadence (or dissolution) of the oil spill was in fact reflected to a certain degree by variations in the PAHs.

3.3. Beverages

As beverages are instant sources of energy, they are consumed by people of all age groups. Consequently, many industries are implementing innovative methods for the production of liquid products including fresh fruit juices, instant coffee, and milk shakes. Such efforts are also extended to the fortification of infant and human milk formulations. However, all these products use sugar which in turn is procured from burnt sugarcane and its derivative products (Bettin and Franco, 2005; Tfouni and Toledo, 2007; Tfouni et al., 2007). As the PAH levels of these products are often found to be higher, it can pose a formidable threat to humans of all age groups.

Among the various beverages, milk is a key dietary item as well as a representative dairy product. Processing of milk is unique in that it undergoes a number of different stages of heat treatment depending upon the type of milk product and processing technique. The concentration of

PAHs was measured from different types of milk samples (e.g., cover raw, pasteurized, ultrahigh treated, semi-skimmed milk, and whole milk) (Naccari et al., 2011). They found contamination in raw milk owing to environmental pollution, while pasteurized and ultra-heat treated milk were affected most significantly in that respect. Interestingly, BAP was detected in all the samples, albeit at low concentrations. Moreover, ANT, PHN, and PYR were seen at high levels relative to the guidelines of the EFSA report on PAHs in food (2008) (Table 1S). Total PAHs were slightly lower in raw milk residues (5.43 μg/kg) than in pasteurized milk residues (6.52 μg/kg). In contrast, ultra-heat treated (UHT) whole and semi-skimmed (UHT) milk consistently exhibited relatively enhanced concentrations of total PAHs at 7.79 and 5.94 μg/kg, respectively (Naccari et al., 2011). The concentration of PAHs is further dependent on the content of milk fat (as PAHs) due to their lipophilic nature incorporating milk fat (Naccari et al., 2011).

Similarly, the PAH contents of the various types of milk and its products (e.g., infant milk, soy milk, skimmed milk, fat milk, custard, and yogurt) were investigated by Aguinaga et al. (2008). Although no PAHs were detected in infant milk, low concentrations of FLR and PYR were found in full fat milk at 0.83–1.04 μg/L and 0.63–1.12 ug/L, respectively. Moreover, PAH levels in skimmed milk were almost negligible, suggesting decreases during the skimming process (Kishikawa et al., 2003). From these findings, it can be inferred that consumption of milk should impose relatively minor exposure risk to PAH.

Tfouni et al. (2007) studied the effect of burnt sugar cane on PAH formation in sugarcane juice and found PAHs in 50% of the samples (Table 1S). To increase the concentration of sugar, the crops are burnt to evaporate water during the harvesting period. Concentration of BAP was found in the range of 0.013–0.021 μg/kg, which is somewhat higher than the imposed limit 0.009 μg/kg (Tfouni et al., 2007). No other human carcinogenic PAHs were detected in the samples (Serra et al., 1995; Tfouni et al., 2007).

Coffee is one of the largest selling beverages all over the world. The presence of PAHs in coffee samples was reported, as its contamination was seen to proceed either from primary green beans or through formation during the roasting step. Most PAH contamination enters through roasting, as it is an essential step for enhancing the aroma to maintain its sensory quality. Roasting can be performed by a number of options such as flame toasting, coal grilling, or toasting from a gas or electric oven (usually executed at 220–250 °C). Such processing facilitates carbonization. Orecchio et al. (2009) studied PAHs in coffee brew samples where total PAHs were between 0.52 and 1.8 μg/L; likewise, the concentration of BAP ranged from 0.008 to 0.060 μg/L. A list of the other PAHs detected includes PHN, FLT, FLR, and BKF (as shown in Table 1S).

Apart from juices and milk, alcoholic beverages are also consumed by a large population. The majority of these beverages are contaminated either due to their raw material or storage (i.e., container). Raw material is used in the form of germinated barley in beer. Beverages like whisky are stored in barrels for a long period of time. There are numerous chances that barrels are contaminated either by their wood or direct contact of the wood with the fire during the process of toasting (Chinnici et al., 2006). García-Falcón and Simal-Gándara (2005) investigated the contents of some important PAHs (BAA, BAP, BKF, BBF, and IP) on various types of alcoholic beverages from Spain (Table 1S). In the case of wines, generally low PAHs values were seen particularly for BAP. Spirits (alcoholic beverages) were found to have higher PAH content at high molecular weight; however, aging was unlikely to exert any effect on PAH formation. For some of the less aged drinks, their PAHs were unquantifiable (García-Falcón and Simal-Gándara, 2005). These authors observed that the methods employed for charring the barrels were critical enough to determine the levels of PAH contamination. The barrels made of oak wood are usually charred with the usage of direct heat (in the form of open fire). This charring of the wooden barrels has the major role to play in affecting the PAH levels of stored alcoholic beverages with aging. Oak wood has great strength and hardness and their

charring will produce the unwanted materials like PAH being released into the stored wines, brandy, whisky, etc.

Significantly high levels (536–2960 ng/L) of carcinogenic PAH were seen in mate drinks prepared from a herb infusion of roasted leaves, stems of yerba mate, or erva mate with hot or cold water (Kamangar et al., 2008). Likewise, high concentrations of PAHs (FLR, FLT, ANT, BAP, BKF, BBF, PHN, and DBA) were also found in the yerba mate commonly drunk in South America. In all the brands, BAP was found to exceed 35 μg/kg, and total PAHs were 266–2906 μg/kg (Table 1S). This enhancement in PAH levels was mainly attributed to the generation of ash from the combustion of leaves. This accounts for the need for modification of the leaf processing. In addition, the oak wood used for making barrels is supposed to be dried for a period of 1–3 years. However, toasting or drying oak with open fire can lead to the formation of PAHs (Chinnici et al., 2007). This is because toasting in open fire initiates the charring of the oak barrels. This is attributed to rise in temperature during open drying with fire to lead to liberation of materials (such as cellulose, dirt, soil, etc.) from the woods that contaminate the barrels. Also, cork which is used as wine stoppers can further lead to accumulation of PAH in alcoholic beverages (Mazzoleni et al., 2005).

4. Dietary route of PAH intake

The public's interest in dietary exposure to PAHs has been increasing in recent years owing to recognition of their toxic effects and their presence in each category of raw (and processed) foodstuffs. Prior to substantiating dietary recommendations, the route and extent of dietary exposure to PAH needs to be accurately assessed. Although the concentration data of PAH (or other toxicants) in food can be determined by proper analytical procedures, the actual information on toxicant consumption data is procured from dietary surveys conducted on the national level with individuals only as a representative in a sample (Dorne et al., 2009). The data obtained by such a procedure do not confirm the validity of an assumption set for lifetime assessment but conveys the status of a particular period. The collection of data and their evaluation is determined primarily by the type of assessment approaches (such as 24-hour recall method, questionnaires, food frequency method, dietary history, or food purchase method) (Willett, 1998). The homogeneity in the data collection pertaining to the toxicants of a particular group and the analytical techniques used are critical so that the outcome of contaminant exposure should be evaluated and implied properly only on a population set (Helsel, 2005). Quantitative assessment of toxicant exposure is usually targeted by the point estimate or probabilistic approach (Kroes et al., 2002). The description of toxicant exposure in a human population is mentioned in Fig. 4 (Dorne et al., 2009). The former is based on the assessment of two fixed levels i.e., between the consumption level data and the contaminants residue data. In contrast, in the case of the latter, the whole distribution of the consumption and concentration can be easily determined (Dorne et al., 2009).

To understand the dietary intake of food contaminants like PAHs, one needs to describe the relationship between their quantities contained in food products as well as nutritional habits (Xia et al., 2010). In spite of the health benefits of seafood, regular intake of seafood enhances the risk of exposure to toxic chemicals and contaminants (Domingo et al., 2007; Sioen et al., 2008; Moon et al., 2010). EFSA (2008) summarized that the overall dietary exposure, assuming a person of 60 kg, is about 235 ng/day (BAP), 641 ng/day (PAH2), 1168 ng/day (PAH4), and 1729 ng/day (PAH8).

Relatively few studies have been done to evaluate the relationship between PAH consumption and consistent exposure on humans (Binelli and Provini, 2003, 2004). Martí-Cid et al. (2008) studied the dietary exposure of 16 PAHs by analyzing sample foods consisting of cereals, oils, and meat products. The highest individual levels were found at PHN (29 μg/kg), NAP (25 μg/kg), FLR (13 μg/kg), BAP (1.28 μg/kg), BKF (1.31 μg/kg), and IN[123,cd]P (1.44 μg/kg).
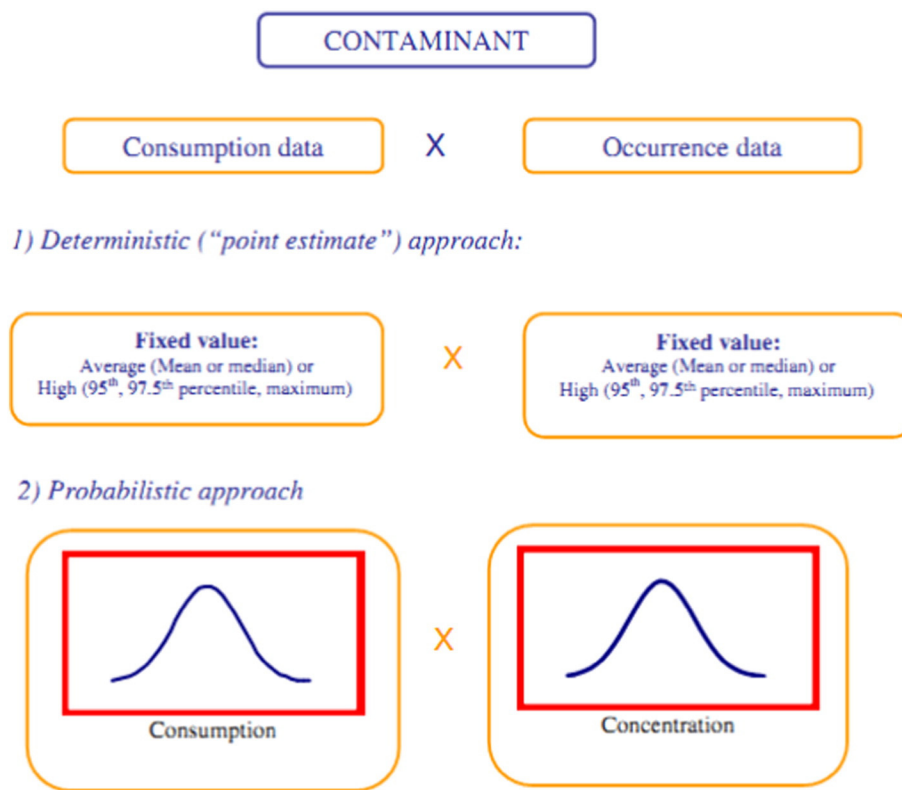
Fig. 4. Assessment of chemical toxicant exposure in human population (Dorne et al., 2009).

These levels were found to be higher than a previous survey done in 2000 (Falcó et al., 2003). Potential health risks owing to consumption of seafood in the Korean population were also assessed by Moon et al. (2010). They found that concentrations of total and carcinogenic PAHs in 26 types of common seafood (squid mackerel, croaker, catfish, tuna, oyster, mussel, crab, and shrimp) ranged from 12 to 243 µg/kg and 0.21 to 18.4 µg/kg (dry weight), respectively. The amount of PAH intake for the general population, men, and women was estimated as 15.3, 17.1 and 14 ng/kg of body weight, respectively (Moon et al., 2010). These consumption levels were found to moderately increase the potential risk of cancer in the general population (2.85×10^{-6}), men (2.93×10^{-6}), and women (2.68×10^{-6}) compared to the guideline limit (1.0×10^{-6}) by USEPA (2004). Korean men were found to have higher consumption (58.2 g/day) of seafood than Korean women (43.5 g/day) (Moon et al., 2010). Hence, total intake of PAH for the general population of Korea was estimated at 829 ng/day; this estimate is higher than one at 712 ng/day (men) and 626 ng/day (women) of Spain (Falco et al., 2005) or 231 ng/day of Kuwait (Saeed et al., 1995).

In the Taiyuan region of China, the dietary exposure of PAH was more significant from pork products than milk products (Xia et al., 2010). The results indicated that there were some age effects to show the least exposure to children (392.4 ng/d). Adults were found to have more dietary exposure (based on the order of daily consumption products such as wheat, pork, fish, vegetables, and oils) to PAH (572 ng/d). In comparison, the values for adolescents and senior males were 511 and 533 ng/d, respectively. The dietary exposure to grilled meat products has also been examined in Kuwait to cover the population of children, adolescents, and adults (Alomirah et al., 2011). They estimated the major intake of PAH8 in all three population types through total mean dietary intake. These dietary intake data were obtained from a Kuwaiti Nutrition survey based on the 24 h recall method from 683 children/adolescents (at ages 3–19 years) and 1021 adults (between 20 and 50 years). The mean dietary intakes of PAH8 for children/adolescents and adults were 84.2 and 95.7 ng/day, respectively, while those of BAP were 8.09 and 9.20 ng/day, respectively. In conclusion, grilled vegetables, whole grilled chicken, and meat chicken were found to be the leading contributors to the daily intake of PAH8 among the Kuwaiti people (Alomirah et al., 2011). Their PAH levels in children/adolescents and adults were found to be comparable with risk levels of developing cancer.

5. How to reduce PAHs in food?

There are a number of approaches to help reduce or suppress the formation of PAHs in food. The practice of preventing the contamination of PAHs into food is summarized in Table 3. By following certain practices of cooking, it is possible to substantially reduce the levels of PAHs. In addition, it is important to protect the ecological systems from which we acquire our food resources.

5.1. Cooking methods

The foremost approach for reducing PAH is the inclusion of alternative cooking methods in the initial stage of food preparation. The concentration levels of PAHs were reduced when cooking food at a lower temperature (Lijinsky and Shubik, 1964; Chen and Lin, 1997; Farhadian et al., 2010). If one can prevent the dripping of melted fat onto the heating surface of charcoal, it will helps prohibit (or suppress) the production of carcinogenic PAH within the food (Lijinsky and Ross, 1967; Farhadian et al., 2011). Moreover, if one can avoid the direct contact of high temperature during grilling (i.e., cooking on direct flame), it also reduces the formation of PAHs (Faradian et al., 2011; Chen and Lin, 1997). The use of minimum heat and keeping food to the farthest point of heating, if combined, can lower their production more efficiently (Rey-Salgueiro et al., 2008; Yebra-Pimentel et al., 2013).

As another option for reducing PAH levels, one may consider alteration of the chemical composition of food. If done properly, it could prevent the formation of carcinogenic compounds like heterocyclic aromatic amines (HCAs). For instance, this option was achieved by the addition of compounds with antioxidative properties for the reduction

Table 3
Routes for reducing PAH formation in food.

Meat and fish products	• Part of food: Usage of lean meat and fish • Exposing the area containing less fat for grilling • Avoid the direct food contact with flames during barbequing • Cooking at lower temperature • Replacement of traditional wood with grill and kiln • Use of electric or gas meat broilers over charcoal • Use of acid base marination as lemon juice before grilling
Fruits and vegetables	• Vegetation of crops at long distant areas of highways and industrial areas • Adherence of crops to horticultural area • Protection against the areas of natural emissions of volcanoes and forest fires • Washing the raw vegetables with oxidizing agents
Oil seeds and oils	• Perform drying processes of oil seeds at lower temperature • Avoid direct toasting of seeds • Refrain the direct contact of combustion with seeds
Sea food	• Industrial effluents to be disposed away from aquatic organisms • Oil spills to be prevented near coastal waters • Avoid the creosote-treated wood for mussel cultivation

of HCAs (Rey-Salgueiro et al., 2008). Recently, the treatment of lemon juice (such as acidic marination) on beef satay was found to induce a 72% reduction of PAH levels relative to oil based marination, when applied prior to the charcoal grilling of meat (Orecchio et al., 2009). The duration of marination was not critical enough to show a noticeable effect on such reduction. However, as relatively little is known about the effects of marination, more efforts are needed to explain the mechanism of such treatment. There are numerous types of functional foods (herbs, extracts of berries, ginger, garlic, etc.) that have strongly antioxidative natural properties. As such, these foods have potential to engulf carcinogenic compounds. These functional foods are the potent source of bioactive phenolic compounds (Hazra et al., 2010). These bioactive compounds are acidic in nature to engulf the carcinogenic compounds or free radicals by which the concentration of PAH and other hazardous compounds become reduced. Therefore, the marinating effect of herbs and phenolic rich compounds needs to be assessed further to combat the entry of carcinogenic PAH through smoking or grilling.

One of the major intake routes of food-related PAHs is through the consumption of vegetable oils and fats. The same contaminated oil was reported to be used in the bakery industry for the preparation of various cookies and biscuits (Yebra-Pimentel et al., 2013). Drying of oil seeds at higher temperature tends to promote the generation of PAH. It is important to avoid direct toasting or roasting of oil seeds (Yebra-Pimentel et al., 2013). Nonetheless, low temperature refining processes are suggested to render reduce contamination.

5.2. Perseverance of fruits and vegetables

Fruits and vegetables are prone to aerial deposition of PAHs because of the large waxy leaf surfaces that readily absorb PAHs (Samsøe-Petersen et al., 2002; Tao et al., 2004; Mo et al., 2009). Hence, it is desirable to grow vegetables in soils that are sufficiently distant from industrial sources. It should also be protected against forest and/or volcanic fires, as these fires render the maximum effect of PAH on crops. The effect of fire was found to be insignificant on the vegetation grown in a Spanish town, as exposure to fire was maintained at the lowest levels due to the winds blowing in a horticultural area (Rey-Salgueiro et al., 2008). In contrast, the effect of airborne emissions was more pronounced where vegetation was grown near industrial areas. The contamination level of PAHs in that area was found to be higher by about an order of magnitude than farms (Grova et al., 2002).

A large fraction of the deposited PAHs can be removed e.g., more than 50% if washed thoroughly (SCF, 2002). Similarly, the effect of washing vegetables with oxidizing agents was found to be effective to eliminate PAHs contained in carrots (Zohair, 2006). This author reported that the use of chemical reagents such as hydrogen peroxide (H_2O_2)

and potassium permanganate ($KMnO_4$) contributed significantly to the removal of PAHs in the range of 65.5–97.8% and 79.4–99.9%, respectively. As these chemicals are well known for their high redox potentials, they need to eliminate PAH efficiently. It was clearly proven that the reduction of contaminants was controlled by such factors as the type of individual PAHs and the concentration of the oxidizing agents.

5.3. Protection of aquatic organisms

Just as the contamination of PAHs on plants is transmitted through soil and air, the contamination in water bodies forces their deposition in aquatic organisms. Note that the major fraction of PAH deposition into water bodies is also explained by their transport from the atmosphere (Yebra-Pimentel et al., 2013). The load of PAH contamination in aquatic environments cannot, however, be reduced merely by efforts to clear the air. More efforts should be made to prevent incidental exposure such as oil spills and deposition of industrial effluents particularly near coastal areas (Yebra-Pimentel et al., 2013). Aquatic organisms (such as fish and mussels) were found to exhibit a strong tendency to metabolize PAHs once deposited on their outer surface (Phillips, 1999; Yebra-Pimentel et al., 2013).

5.4. Bioremediation

In the past few years, a branch of bioremediation has been spread rapidly for the treatment of contaminants such as PAHs. Bioremediation is defined as the process of biologically degrading organic wastes to a material with a harmless state (Bamforth and Singleton, 2005). In this respect, microbial degradation of PAHs has drawn a great deal of attention. Microbes are capable of employing benzene rings as their sources of carbon and energy (Juhasz and Naidu, 2000). Microorganisms have the ability to degrade the multi-ring structure of PAHs with emission of their side products (Samanta et al., 2002). The foremost step in microbial degradation is the incorporation of oxygen atoms at two or more carbon atoms of the benzene ring of the PAH. Such incorporation can then lead to the formation of cis-dihydrodiol to facilitate the conversion to dihydroxylated intermediates (Kanaly and Harayama, 2000; Samanta et al., 2002). The rate of biodegradation can then be stimulated with the adjustment of some key variables such as pH, moisture, oxygen, and chemical alteration.

5.5. Catalysts

The use of a photo-degradation approach under UV light is another important option to treat PAHs, as it can induce the conversion of PAH into less harmful components (aromatic alcohols, ketones, quinines, and ethers) (Bernstein et al., 1999; Šimko, 2005). In fact, the photolytical decomposition of PAHs was also demonstrated to be effective in the aqueous phase (Sabate et al., 2001). Oxygen plays a crucial role in the photo-oxidation of PAH (Kou et al., 2009). This is a recent research trends explored by many authors. Photo-catalytic degradation of PAHs (PHN, ACN, PYR, and BAA) was also exercised using light driven photo-catalysts (tantalum oxynitride and Pt-tantalum oxynitride) (Kou et al., 2009). The application of photo-catalysis has great potential to decontaminate PAHs in water bodies in light of their low water solubility. This property enables the delocalization of their electrons using photo-catalysts (Kou et al., 2009). In addition to photo-catalysis, biocatalysts in terms of microbial degradation can be a major route for the decontamination of PAHs, as pure bacterial strains can be employed to metabolize these substrates (Nadarajah et al., 2002). Furthermore, in the category of biocatalysts, enzymes (oxygenase, dehydrogenase, and liginolytic enzymes) can play a critical role in destabilizing the bonds in a molecule (Trejo-Hernandez et al., 2001). Lau et al. (2003) reported the use of mushroom compost for the degradation of NAP, PHN, and

BAP. These methods can be used as a feasible tool to degrade PAH in water and soil bodies.

5.6. Effect of packaging

As a means to reduce PAHs in smoked food products, the effect of packaging should be considered. Coupling of the smoking medium (solid matrix or liquid media) and packaging was proven to have a considerable effect on reducing PAHs levels. The use of a low density polyethylene (LDPE) layer over smoked meat was found to reduce PAH concentration by more than 50% from their initial levels (Chen and Chen, 2005). The presence of PAHs (e.g., BAA, BBF, and BAP) were determined in roasted duck before and after packaging with LDPE; the concentration levels of BAA, BBF, and BAP were reduced (143 to 130, 3.7 to 1.7, and 3.5 to 4.9 μg/kg, respectively) after a storage period of 24 hours (Chen and Chen, 2005). The results showed that the packaging was able to reduce the levels of PAHs. Also, immediate packaging of smoked meat is important, since the surface of the smoked/grilled foods contains most of the PAHs; therefore, instant wrapping (of smoked food) to make direct contact with the sorption (packing) material, can help decrease the retention of PAHs. This is due to the diffusion of PAH to packaging film of similar polarity (non-polar). This process starts immediately after applying the film to the food. As PAHs can be diffused into the multi layers of LDPE, packaging can be considered as a potent solution for reducing the influence of PAH from grilled/smoked foods. Moreover, polytetrafluoroethylene (PET), another important packaging material, was also seen to have good potential to reduce PAHs levels in rapeseed oil (955.1 to 315.1 μg/kg) after filling into PET cylindrical shaped receptacles (Chen and Chen, 2005).

6. Conclusion

In this review, a comprehensive survey of PAH levels in various food products has been presented. These data were also evaluated to generalize the factors and processes controlling their formation in terms of human dietary exposure. The risk factors of PAH exposure are mainly posed by human activities which pose threats to all types (sea/marine and plant) food resources. In addition to human activities, the methods or approaches involved in cooking, processing, and consumption can also exacerbate exposure to PAH. Proper methods of PAH reduction or inhibition need to be taught and adapted to globally, covering all the routes of their transfer, including use of cautious cooking methods, natural chemical agents (such as antioxidants), and prevention of contamination of vegetation in the growing stage. In addition, the applicability of various antioxidants filled with phenolic compounds was evaluated to assess their inhibitory properties for carcinogenic PAH compounds. The use of novel methods in the form of biocatalysts was intensively investigated to find the proper pathway of PAH degradation in food products and their packaging. As such, qualified approaches should be implemented so that risk analysis is communicated to regulate PAH and to maintain the quality and safety of all food products.

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