Chapter

Polycyclic Aromatic Hydrocarbons (PAHs) and Their Importance in Animal Nutrition

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Abstract

Polycyclic aromatic hydrocarbons (PAHs) formed as a result of incomplete combustion of organic compounds. It contains compounds that cause toxic, teratogenic, mutagenic and carcinogenic damage, such as heterocyclic aromatic amines, benzene and formaldehyde. PAHs can be found in industrial wastes, garbage, cigarette smoke, pesticides and flue gases and can contaminate air, water, soil and food. Although more than 100 PAH compounds are detected in nature, it is accepted that 16 PAH compounds have more harmful effects. It is important to determine the PAH exposure levels of feeds used in animal nutrition, since the contamination of feed plants and factory feeds with PAH compounds will indirectly affect human health. In this study, the physical and chemical properties of PAHs and their effects on animal production and indirectly on human health were compiled.

Keywords: benzo[a]pyrene, animal nutrition, exposure, polycyclic aromatic hydrocarbons (PAHs), feeds

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are pollutants generated by the pyrolysis or incomplete combustion of carbon-containing material, fossil fuels such as coal and other organic matter including food at high temperatures under oxygen-deficient conditions and are defined as organic lipophilic compounds that contain two or more benzene rings [1, 2]. The contamination of food with these compounds generally results from water, air and soil pollution, but PAHs may also be produced during food processing at high temperatures [3]. Owing to their chemical structure, PAHs have carcinogenic and mutagenic effects. The association of PAHs with cancer was first described in 1775 by Percivall Pott, surgeon of St. Bartholomew's Hospital in London, upon his diagnosis of scrotal cancer in chimney sweepers. This was the first observation of environmental factors being a possible cause of cancer. Later, scrotal cancer was also detected by Bell and Volkmann in workers employed in the paraffin manufacture industry in Germany and Scotland, and thereby, the observation of Pott was confirmed. In subsequent research on laboratory animals and humans, chemicals containing benzo[a]pyrene, such as tar, oil, smoke and fume, were identified as being rich in PAHs [4, 5]. Today, PAHs are produced either anthropogenically through industrialisation, the increased use of fossil fuels, waste deposition and tobacco consumption, or naturally, as a result of

forest and brush fires and volcanic eruptions [1, 6, 7]. In modern-day life, people are exposed to these compounds mostly through the consumption of contaminated water and food and the inhalation of polluted air. Exposure to PAHs increases with the consumption of heat-treated food as well as by smoking [2]. Three different mechanisms have been reported for the production of PAHs in food. The first involves the generation of PAHs as a result of the pyrolysis of organic matter such as carbohydrates, fat and protein at high temperatures (500–900°C) [8]. The second mechanism involves the generation of volatile PAHs as a result of the dripping of melting fat onto hot coal from food cooked on coal fire. These volatile PAHs contaminate the surface of cooked food at much higher levels with increased smoke generation. In particular, when meat is char-grilled, melting fat drips onto hot coal, resulting in pyrolysis, and the contact of grilled meat with smoke results in the accumulation of the volatile PAHs in the lipid components of the food. Owing to the lipophilic structure of PAHs, the water and fat content of food dictates the rate of transport of these compounds to food [2]. The third mechanism of PAH production is the incomplete combustion of coal, which eventually results in the contamination of the food surface with the generated pollutants [3].

After being discharged into the atmosphere, polycyclic aromatic hydrocarbons are either carried away from the source of emission or naturally settle in soil and water. The absorption of gaseous chemicals by plants is one of the main pathways of PAHs entering the Agri foodstuffs chain. Thus, PAHs enter the food chain of humans either directly, through the consumption of plant products such as cereals and vegetables, or indirectly, by the consumption of animal products such as milk and meat [4, 8]. The direct consumption of contaminated food and feed by livestock is the main course of organic pollutants entering the animal body. This results in animal products consumed by humans, such as meat and milk, being contaminated. Overall, 88–98% of cases of exposure to PAHs are foodborne [1, 8].

PAHs are contaminants which get in whole parts of the environment: atmosphere, waters and soils. This means that there is a risk that can be directly contaminated to plant and animal products. Considering this existence of PAHs and the risks for public health and animals associated with the exposures, the aim of this paper is to review and underline current information on the features, destinies and hazards associated with the presence of these compounds in the feeds and animal nutrition.

2. Methodology

In the present review, researches on PAHs are briefly summarised based on literatures. In this methodology, the physical and chemical properties and the most important components of PAHs are defined. For this purpose, the effects of PAHs, which have the environmental hazards, on feed and animal production are investigated. As a result, the potential risks of PAHs on human and animal health are summarised.

3. Polycyclic aromatic hydrocarbons (PAHs)

3.1 The physical and chemical properties of PAHs

Incomplete combustion does not produce a single type of PAH but results in the generation of a complex array of combustion products [5]. On the other hand, it is possible to produce PAHs as pure compounds for research purposes. PAHs,

which are produced in the form of pure compounds, are either colourless or of light yellow, white or green colour, have a light, pleasant odour and are found in solid state. Except for research purposes, these pure compounds have no further area of use [4].

PAHs, which have carcinogenic and mutagenic effects, are classified according to the number of benzene rings found in their structure. Those containing less than four benzene rings are classified as light PAHs, whilst those with more than five benzene rings are classified as heavy PAHs. Besides their classification, PAHs are also named after the number of benzene rings they possess, such that compounds with two benzene rings are referred to as naphthalene and those with three benzene rings are named as anthracene and phenanthrene. Compounds with a greater number of benzene rings have specific names [6, 9]. Light PAHs include naphthalene, acenaphthene, acenaphthalene, fluorene, anthracene and phenanthrene, whilst heavy PAHs include pyrene, fluoranthene, benzo[a]pyrene, chrysene, benzo[b] fluoranthene, indeno[1,2,3-cd]pyrene, benzo[k]fluoranthene, dibenzo[a,h] anthracene and benzo[g,h,i]perylene [4, 10]. The carcinogenicity and mutagenicity of heavy PAHs such as dibenzo[a,h]anthracene and benzo[a]pyrene are stronger than those of light PAHs [11].

The physical and chemical properties of polycyclic aromatic hydrocarbons vary with the molecular weight of the compounds [2]. Increased molecular weight is associated with reduced water solubility. PAHs with a high molecular weight are capable of enduring without evaporating, given their low solubility and volatility. Lowly volatile PAHs become even less volatile with an increase in the number of benzene rings in their structure. An increase in the molecular weight of PAHs is associated with higher boiling and melting points and lower vapour pressure. The majority of PAHs have a boiling point above 300°C and a melting point below 250°C [4, 12].

While more than a hundred PAHs have been detected in nature, among these, only 16 (**Table 1**) have been described as primary pollutants, in view of their greater toxicity and carcinogenicity [13]. This description of the primary pollutants was made by the US National Priority List (NPL) and is based on the extensive information available on these compounds, the more serious side effects, the greater residual risks, and the higher levels of detectability in hazardous dumpsite analysis [4].

3.2 PAHs in products of plant and animal origin

Environmental pollution caused by increased industrial production has resulted in the contamination of several food products, including vegetables, milk products, fruits, tea, oils, coffee, smoked meat and cereals with PAHs. Contaminated soil, water and air are known to be the main PAH contamination sources for food. Furthermore, PAHs may also contaminate food products by means of smoke generation, grilling on charcoal, processing, improper cooking methods and the use of feed additives [3, 14]. Cereals (corn, wheat, barley and oat) in industrialised regions enhance PAH levels in comparison with more outlying regions. Grain samples from a heavily industrialised region included 10 times more PAHs than samples from areas far from industry. The growth of rye near an autobahn resulted in PAH pollution, which reduced lightly 7–25 m far off from the way [15]. PAHs disrupt the growth and development of plants, which eventually reduces the overall biological activity in the ecosystem. This reduced activity also restricts productivity. Given their lipophilic nature, PAHs are deposited particularly in the double layer of membranes in plants. Plants that grow in regions with high levels of PAHs in the soil and air also contain high levels of PAHs [6, 16–18]. Thus, plants can be used to

Compounds	Chemical formula	Molecular mass (g/mol)	Toxic equivalency factor (TEF)
Naphthalene (Np)	C10H8	128.17	0.001
Phenanthrene (Phn)	C14H10	178.23	0.001
Benzo[a] pyrene (BaP)	C18H12	252	1
Fluoranthene (Flu)	C16H10	202.26	0.1
Benzo[a]anthracene (BaA)	C18H12	228	0.1
Benzo[b] fluoranthene (BbF)	C20H12	252	0.1
Indeno[1,2,3-cd] pyrene (IcdP)	C22H12	276	0.1
Benzo[e]pyrene (Bep)	C20H12	252	-
Fluorene (Flr)	C13H10	166.22	0.001
Dibenzo[a,h]anthracene (DbA)	C22H14	278	1
Pyrene (Pyr)	C16H10	202.25	0.001
Anthracene (Ant)	C17H10	178.23	0.01
Benzo[k] fluoranthene (BkF)	C20H12	252	0.01
Benzo[j]fluoranthene (BjF)	C20H12	252	-
Chrysene (ChY)	C18H12	228	0.001

Table 1.
Polycyclic aromatic hydrocarbon compounds evaluated as priority pollutants [13].

detect environmental pollution with PAHs. This also indicates that plants serve as a point of entry for PAHs into the food chain [17]. PAHs enter the human body either by the consumption of contaminated plants or by the consumption of products from animals fed on contaminated plants [1, 8].

Moreover, environmental factors may also cause the contamination of oilseeds with PAHs, such that these compounds pass into vegetable oils during the processing of oilseeds [19]. The European Food Safety Authority (EFSA) has pointed out to meat and meat products as another important source of the daily exposure of consumers to PAHs. The level of PAHs produced in meat and meat products varies with the fat content and oxygen concentration of meat, the type and temperature of the heat source used for processing, the distance maintained between the food product and heat source and the duration of processing. The direct contact of food with flames, extended heat treatment and high temperatures during processing particularly increase PAH levels [2, 10].

A previous study on the synergistic toxicity of PAHs with other pollutants investigated the combined effects of fly ash and sulphur dioxide (SO_2) on cucumbers. It was observed that neither fly ash nor sulphur dioxide showed effect alone, but when combined, the two caused severe chlorosis, which is a plant disease that manifests as the yellowing of leaves [20]. As a result, the active organic substances found in fly ash were claimed to be PAHs [17].

3.3 The effects of PAHs on human and animal health

The significant role of environmental factors in the development of cancer, one of the major diseases of the modern day, has been well acknowledged. Chemicals originating from hazardous substances, including industrial wastes, flue gases, litter, pesticides and tobacco smoke, pollute the environment, and by contaminating air, water, soil and food, these chemicals threaten human health [4]. PAHs,

including sulphur dioxide, pesticides, insecticides and nitric oxide, are carcinogenic and toxic to humans [9].

The toxicity of PAHs is not related to molecular size, but rather to the chemical structure of molecules. Generally, a carcinogenic effect is induced by the binding of PAH metabolites to deoxyribonucleic acid (DNA) [11, 12]. Once having entered the human body, PAHs cause DNA mutation. It is considered that benzo[a]anthracene and benzo[a]pyrene are particularly carcinogenic to animals and humans, respectively; thus, they are used as model compounds in cancer research [21, 22]. To exemplify, upon exposure to tobacco/cigarette smoke, benzo[a]pyrene diol epoxide adducts bind covalently to several guanine positions of the DNA p53 gene in the bronchial epithelial cells and cause cancer-inducing mutations.

Due to the potential danger posed by PAHs, food and environmental contamination risks are of high importance for human health. The fumes of fossil fuels, tobacco smoke, fruits, vegetables, bread, cereals, meat, processed and salted products and milk all contain PAHs. Moreover, the grilling or high-temperature cooking of meat and other food products increases the level of PAHs in food [4, 23]. As PAHs are generated in the form of a complex mixture of compounds, humans are most likely to be exposed to multiple PAHs at the same time. The amount of PAHs entering the human body may vary with eating, drinking, dermal contact with contaminated material and the presence of other chemical substances [24]. PAHs may enter all body tissues that contain fat. In the human body, fat is mainly deposited in the kidneys and liver. Small amounts of fat are also deposited in the spleen, ovaries and adrenal glands [4].

In order to eliminate PAHs, the human body renders them water-soluble, and this process, which involves oxidative metabolism, generates highly productive diol epoxide derivatives. These diol epoxide derivatives chemically react with DNA. Eventually, the chemical binding of PAHs to DNA causes cancer [25]. Furthermore, biological research on the placenta has shown that PAHs cause predisposition to the lung, liver, nervous system and lymphatic tissue tumours in children [3, 26]. Low IQ and childhood asthma have been reported to be associated with prenatal exposure to high levels of PAHs [27]. The Centre for Children's Environmental Health has reported that exposure to PAH pollution during pregnancy may result in adverse effects, leading to preterm labour, cardiovascular anomalies and low birth weight [12]. It is indicated that, upon PAH exposure, cancer-induced DNA damage is detected in the umbilical cord blood of babies, which may be followed by growth retardation and behavioural disorders that may increase between 6 and 8 years of age [27]. In view of these data, EFSA has stated that these compounds are potentially genotoxic and carcinogenic to humans and constitute a priority group for the assessment of health risks [12, 28].

Experimental studies on PAHs have demonstrated that animals known to have suffered from short- and long-term exposure to PAHs present with body fluid disturbances, immunity disorders and cancer of the urinary bladder, skin and lungs [4]. Following the subcutaneous and intraperitoneal injection of benzo[a]pyrene to newborn mice throughout the first 15 days after birth, it was observed that liver and lung tumours developed within a period of six months [29]. Pregnant mice exposed to very high levels of benzo[a]pyrene have been reported to display dystocia, low birth weight and other pregnancy-related problems [3, 4]. Furthermore, it is indicated that nitro-PAHs cause leukaemia and tumours of the colon and milk glands [30]. In several other research studies conducted in animals, it has been reported that exposure to PAHs, within a time frame extending from foetal development to adulthood, is highly associated with cancer development [3, 4, 26].

3.4 Previous research on PAHs

In a study conducted by Gutiérrez and Vega [1] in industrial farms located near industrial sites, the primary PAHs detected in cow's milk were reported as acenaphthene (Ace) (0.25 mg/g^{-1}), acenaphthylene (Acy) (0.32 mg/g^{-1}) and fluoranthene (Fla) (0.22 mg/g^{-1}). The most probable sources of these compounds have been suggested as contaminated grass and fuel combustion. The study reported that the milk concentrations of the 16 different PAHs detected did not exceed the dietary intake level set by the United States Environmental Protection Agency (USEPA) (25 mg/g^{-1}) and suggested that the pollutants posed a limited health risk to the animal and human populations in the study location.

Bechtel and Waldner [8] investigated the correlation between the immune functions of an annual beef cattle population, atmospheric levels of polycyclic aromatic hydrocarbons and PM_{10} oil and natural gas facilities. By analysing blood samples collected from beef cattle, the researchers determined the potential correlation between exposure to atmospheric fine particles (particles of 1 μm diameter or PM_{10}), polycyclic hydrocarbons and immune system functions. They placed herds of the annual beef cattle population at various distances to the industrial facilities located in areas producing large amounts of oil and natural gas. The researchers assessed immune system sufficiency, based on levels of B-lymphocytes and subtypes of T-lymphocytes (CD4, CD8 and WC1) in peripheral blood (n=469) and systemic antibody levels produced in response to vaccination (n=469). In this study, the mean PAH levels detected in the ambient air the animals breathed were low, and those measured at the highest levels were naphthalene (geometric mean 5.6 ng/m^3; geometric standard deviation 38) and 1-methylnaphthalene (geometric mean 2.2 ng/m^3; geometric standard deviation 12). The researchers reported not to have detected any statistically significant correlation between exposure to any of the pollutants detected in the ambient air and the immune system functions of the animals.

In another study investigating the level of exposure of dairy cattle and fattening pigs to PAHs, samples taken inside and outside the pens, in which the animals were housed, were analysed. The results of the study showed that the exposure level of the dairy cattle to PAHs was 86 times higher than the exposure level of the fattening pigs and revealed the main source of PAHs as feed for both species. The same study reported that the share of PAH intake, by means of water consumption and inhalation, in the total PAH load was negligible (**Table 2**) [31].

Samples	∑PAH		∑Cancerogenic PAH		Py		BaP		Cancerogenic PAH %	
	Cow	Pig	Cow	Pig	Cow	Pig	Cow	Pig	Cow	Pig
Indoor air (ng/m3)	56.0	25.0	4.3	1.3	7.0	3.0	0.5	0.2	7.0	5.1
Feed mixtures (μg/kg)	128.0	82.0	34.0	2.3	1.8	7.2	0.6	0.3	12.0	4.0
Water (ng/l)	38.0	100.0	11.0	51.0	2.3	8.5	0.1	6.7	8.7	9.2
Barn dust (μg/kg)	4475.0	676.0	162.0	38.0	33.6	26.0	1.2	1.1	17.0	9.0

Py: average pyrene concentration; BaP: average benzo(a) pyrene concentration (% carcinogenic. PAH).

Table 2.
PAHs contamination from cows and pigs barns [31].

In their research investigating the presence of PAHs in the tissues and internal organs of pigs and cattle, Ciganek and Neca [32] analysed specimens taken from the liver, lungs, kidneys, eyeballs (lens and vitreous body), muscles and fat tissue. Analyses revealed the presence of PAHs in the following internal organs and tissues of pigs and cattle at the indicated levels in ng/g, respectively: liver (3.8, 2.7), lungs (4.6, 5.4), kidneys (5.4, 6.3), fat tissue (0.05, 0.11), muscle tissue (3.6, 5.1), lens (57.9, 16.3) and vitreous body (14, 6.4). The most common PAHs detected in the specimens were phenanthrene, pyrene, naphthalene and fluorenone. As a result, in this study, no statistically significant difference was observed between the PAH levels detected in the organ and tissue samples of the pigs and cattle. However, the PAH levels detected in the tissue samples of animals of the same species housed in the same pen were found to significantly differ.

In the studies carried out to reduce the contamination of PAHs in the soil, there are also results that alfafa, ryegrass and Juncus subsecundus plants reduce the concentration of pyrene and phenanthrene compounds in the soil [33–35]

4. Conclusion

In conclusion, the production of feedstuff, both naturally and from industrial by-products, for use in animal husbandry, poses the risk of exposure to PAHs. This risk points out to the need for conducting PAH analyses in feedstuff used for the production of animal products, which have an important place in the food chain of humans. It is evident that the number of available studies on the impact of PAHs on the environment and animal health is rather limited [12, 18, 36]. In this context, it is considered that determining the impact of PAHs on human nutrition and health by fully demonstrating the adverse effects of PAHs on animal nutrition and health, as well as developing remediation strategies, is of utmost importance.

Conflict of interest

The authors declare no conflict of interest.

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