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B[a]P in food products: Sources, dietary exposure, and toxicity

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ABSTRACT

Benzo(a)pyrene (B[a]P), a highly carcinogenic polycyclic aromatic hydrocarbon (PAH), enters the food chain via environmental pollution, industrial activities, and cooking at high temperatures. This review compiles research on B[a]P contamination in plant-based foods, animal products, and beverages, emphasizing its widespread presence, particularly in smoked, grilled, and processed items. The data indicate that dietary exposure to B[a]P differs notably by region, with cereals, meats, and smoked foods serving as primary sources. Mechanistically, B[a]P triggers oxidative stress, DNA damage, and prolonged inflammation through metabolic activation by cytochrome P450 enzymes (CYP1A1, CYP1B1), resulting in mutations in critical oncogenes such as TP53 and Ki-Ras. Although regulatory thresholds for B[a]P in food are established, enforcement is often inconsistent, with some products surpassing safe limits. This study highlights the pressing need for stronger food safety regulations, increased public education, and innovative risk-reduction approaches, such as diets rich in antioxidants and healthier cooking methods, to mitigate health threats posed by B[a]P, including cancer, cardiovascular issues, and neurological damage.

KEYWORDS: *Benzo(a)pyrene, Dietary exposure, Toxicity, Oxidative stress, DNA damage.*

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1. INTRODUCTION

The widespread occurrence of foodborne illnesses and contamination incidents has had a profound impact on public health, safety, and property. A report from EU member states documented 20,017 human cases and 3,086 foodborne outbreaks in 2020, highlighting the severity of the issue [1]. The term “persistent organic pollutants” (POPs) refers to a broad category of organic contaminants present in environmental systems. These pollutants originate from both anthropogenic and natural sources and have posed a global concern since the end of World War II [2]. POPs are primarily produced through the incomplete combustion of organic substances and include compounds such as polycyclic aromatic hydrocarbons, polychlorinated chemicals, dioxins, dibenzo-dioxins, as well as polychlorinated furans and dibenzofurans [3].

Polycyclic aromatic hydrocarbons (PAHs) represent a major group of environmental pollutants, primarily generated by the incomplete combustion of fossil fuels and biomass [4]. These compounds often accumulate in food, particularly due to heat processing methods. The International Agency for Research on Cancer has classified various PAHs as carcinogenic to humans, placing them in Group 1 (known), Group 2A (probable), or Group 2B (possible) carcinogens. Given their mutagenic and carcinogenic properties, dietary intake of PAHs poses a notable public health concern [5]. Benzo[a]pyrene (B[a]P), a specific type of PAH composed of five fused benzene rings, is a solid compound, contrasting with benzene, which is liquid. Owing to their hydrophobic characteristics, PAHs with two or more benzene rings form highly stable molecular configurations [6].

Incomplete combustion of carbon-rich substances such as coal, tobacco, wood, and petroleum-based products results in numerous polycyclic aromatic hydrocarbons (PAHs), including benzo[a]pyrene. This compound was first extracted from coal tar and recognized as a strong carcinogen as early as 1933 [7]. Elevated B[a]P levels have been detected in industrial waste, cigarette smoke, diesel emissions, charcoal-cooked foods, and as a byproduct of various high-temperature processes [8]. The International Agency for Research on Cancer has categorized B[a]P as a Group 1 carcinogen, indicating it is carcinogenic to humans. Moreover, B[a]P ranks eighth out of 275 chemicals on the Agency for Toxic Substances and

Disease Registry's (ATSDR) Priority List of Hazardous Substances and has been identified at 524 hazardous waste locations on the National Priorities List [9].

Sources of Benzo(a)pyrene

Benzo(a)pyrene is considered one of the most hazardous polycyclic aromatic hydrocarbons (PAHs) due to its high toxicity. In the European Union, B[a]P levels in ambient air are regulated under Directive 2004/107/EC, which establishes a target value of 1 ng/m³ in PM10, averaged annually [10]. The primary sources of B[a]P emissions are anthropogenic, including fossil fuel combustion, pyrolysis, and the burning of biomass through incomplete combustion processes [11]. Typical PAH concentrations in urban outdoor air range from about 1 to several dozen nanograms per cubic meter (ng/m³), with the highest B[a]P levels—up to several dozen ng/m³—detected in road tunnels and cities with extensive coal and fuel use for heating. Approximately 20% of Europe's population is exposed to B[a]P concentrations above the EU's target limit of 1 ng/m³, while about 7% reside in areas with levels below the safer threshold of 0.12 ng/m³ [6]. On average, individuals spend around 90% of their time indoors, where they may be exposed to pollutants emitted from various sources such as building materials, furniture, electronics, toys, carpets, paints, household chemicals (e.g., glues, cleaning agents, pesticides), and indoor combustion activities like cooking, heating, and smoking [11].

The presence of benzo[a]pyrene (B[a]P) in soils, vegetation, and natural water bodies is largely attributed to increasing anthropogenic activity. B[a]P's potential to accumulate in plants and move through soil is influenced primarily by the soil's adsorption capacity, as well as the compound's physicochemical characteristics, particularly its water solubility and its tendency to transfer into the soil solution [12]. The estimated environmental persistence of B[a]P varies by medium: it has a half-life of less than 1 to 6 days in the air, under 1 to 8 hours in water, but can persist for over 5 to 10 years in sediments and more than 14 to 16 months in soil before complete degradation [6]. Table 1 outlines the regulatory standards for maximum allowable B[a]P concentrations in different food categories. (A) Maximum levels as defined by Regulation (EC) No. 1881/2006. (B) Revised limits set by Commission Regulation (EU) No. 835/2011, introduced in August 2011.

Benzo[a]pyrene standards for surface waters differ across regions. In the European Union, Directive 2013/39/EU establishes an annual average limit of 0.17 ng/l and a maximum allowable concentration of 270 ng/l [13]. The U.S. Environmental Protection Agency (EPA) recommends a stricter range of 0.12 to 0.13 ng/l to safeguard human health**.** In China, B[a]P is classified as a carcinogen with no safe exposure threshold; the regulatory limit was initially set at 2.5 ng/l in 1988 (GB 3838-1988) and later revised to 2.8 ng/l in 1999 [14].

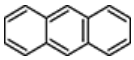
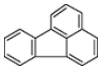
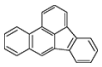
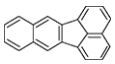
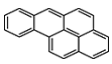
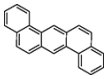
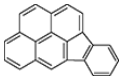
Table 1. Administrative Regulation of BAP Levels in Food [15].

A.	**Food commodity**	**Maximum level (µg/kg)**
	Fats and oils used in the human diet	2
	Smoked meat and its products	5
	Smoked fish and the muscle meat of fish	2
	Processed cereal-based products for infants and young children	1
	Seafood, including crustaceans, cephalopods (excluding crab and lobster), and molluscs	5
	Processed baby foods for infants and infant milk formula	1
B.	**Food commodities**	**BAP (µg/kg)**
	Smoked meat and smoked products	2
	Muscle meat of smoked fish	5
	Smoked crustaceans and crabs	2
	Molluscs (fresh, filled, or frozen), heat-treated meat products	5
	Smoked molluscs	6
	Oils and fats	2
	Cereal-based products for infants and young children	1
	Infant milk and milk formulae	1
	Cocoa butter and chocolates	5

While food processing techniques improve flavor and shelf life, they can also lead to the formation of carcinogenic substances. Consumption of processed meats—such as those that are salted, cured, fermented, or smoked—has been associated with an increased risk of colorectal cancer. Additionally, foods preserved with salt, including pickled vegetables and dried fish, may elevate the risk of stomach cancer [16]. Table 2 highlights the International Agency for Research on Cancer (IARC) classification system, which categorizes compounds based on their carcinogenic potential: Group 1 (carcinogenic to humans), Group 2A (probably carcinogenic to humans), Group 2B (possibly carcinogenic to humans), and

Group 3 (not classifiable regarding carcinogenicity in humans). Polycyclic aromatic hydrocarbons (PAHs) are distributed across these groups—for instance, benzo[a]pyrene is listed in Group 1, dibenzo[a,h]anthracene in Group 2A, chrysene in Group 2B, and pyrene in Group 3 [17].

Table 2. IARC classification: group 1 = carcinogenic to humans, group 2A = probably carcinogenic to humans, Group 2B = possibly carcinogenic to humans, Group 3 = not classifiable as to carcinogenicity to humans.

Compound	Structure	Molecular Weight	IARC PAHs
Acenaphthene		154	3
Acenaphthylene		152	Not assessed
Anthracene		178	3
Fluoranthene		202	3
Naphthalene		128	2B
Phenanthrene		178	3
Pyrene		202	3
Benz[a]anthracene		228	2B
Benzo[b]fluoranthene		252	2B
Benzo[j]fluoranthene		252	2B
Benzo[k]fluoranthene		252	2B
Benzo[ghi]perylene		276	3
Benzo[a]pyrene		252	1
Chrysene		228	2B
Dibenz[a,h]anthracene		278	2A
Indeno[1,2,3—cd]pyrene		276	2B

Benzo(a)Pyrene toxicity

Benzo[a]pyrene is recognized as the most extensively studied and among the most potent carcinogenic polycyclic aromatic hydrocarbons (PAHs), frequently serving as a reference compound in toxicological research. A significant proportion of sporadic cancer cases are linked to environmental exposures, particularly to chemical pollutants. One such contaminant of major concern is benzo[a]pyrene, a widespread environmental toxin found in air, water, soil, sediments, living organisms, cigarette smoke, coal tar, vehicle emissions, and industrial discharges [18].

In foods subjected to high-temperature processing, benzo[a]pyrene exhibits carcinogenic, teratogenic, and immunotoxic properties. The Aryl Hydrocarbon Receptor (AHR), which is broadly expressed across various cell types, initiates programmed cell death upon activation by specific ligands. This receptor plays a central role in the metabolic processing of B[a]P . AHR is also key to maintaining cellular equilibrium by regulating essential functions such as cell proliferation, differentiation, gene expression, movement, and inflammation [19]. Figure 1 illustrates the pathway by which B[a]P enters the cell, binds to AHR, translocates to the nucleus, and causes DNA damage, which can lead to mutations and potentially cancer.

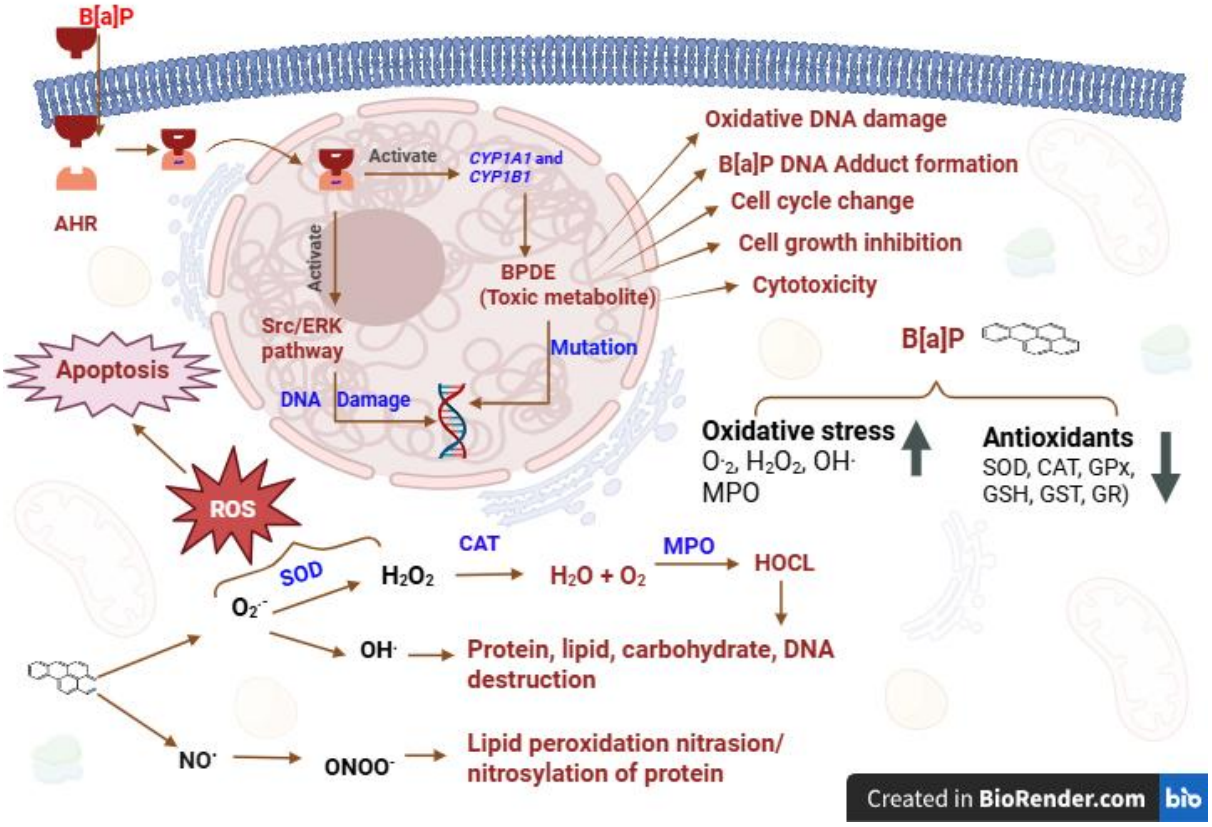


Figure 1. Molecular Mechanism of Benzo[a]pyrene (B[a]P)-Induced Oxidative Stress, DNA Damage, and Cellular Toxicity In its inactive form, AHR remains in the cytoplasm associated with chaperone proteins such as HSP90 and XAP2. Upon binding with B[a]P, a high-affinity exogenous ligand, the AHR complex translocates to the nucleus [20]. Inside the nucleus, the B[a]P-AHR complex forms a dimer with the AHR nuclear translocator (ARNT). This heterodimer then attaches to xenobiotic response elements (XREs) within the promoter regions of specific genes, including CYP1A1 and CYP1B1 [21].

1.1. Genotoxic and Metabolic Pathways

AHR activation by B[a]P upregulates cytochrome P450 1A1, IA2, and 1B1 enzymes (e.g., CYP1A1, CYP1A2, and CYP1B1) [22], which metabolize B[a]P into reactive intermediates such as benzo[a]pyrene-7,8-dihydrodiol-9,10-epoxide (BPDE). BPDE forms DNA adducts, causing mutations and genomic instability [21]. BPDE-DNA adducts act like roadblocks, causing mutations in the DNA as shown in Figure 1. These mutations can turn normal cells into cancer cells. Specific Mutations in the Ki-Ras and Ha-Ras genes help control cell growth. Mutations in these genes (like G→T or G→A changes) can make cells grow out of control, leading to tumors [23]. The TP53 gene acts like a "brake" for cancer, but mutations in TP53 (like G→T changes) can disable this brake, allowing cancer to develop [24].

1.2. Non-Genotoxic Pathways: Inflammation and Oxidative Stress

BaP-AHR activation lowered the levels of some important proteins (C/EBPα, PPARγ, FABP4, PGC-1α, and PPARα) that help in fat storage and energy use. At the same time, it raised the levels of other proteins (NF-κB, MCP-1, and TNF-α) that cause inflammation [20]. The 9,10-diol is the metabolite of B[a]P, which generates oxidative stress, and its metabolites can damage DNA, proteins, and lipids. This is particularly evident in the case of BPDE, which causes extensive DNA damage. Nrf2 activation leads to the upregulation of antioxidant enzymes, which is a direct response to combat oxidative stress [25].

1.3. Disruption of Lipid Metabolism

BaP suppresses adipogenic factors (PPARγ, C/EBPα, FABP4) and lipid oxidation mediators (PGC-1α, PPARα) via AhR, reducing fat storage and energy production in white adipose tissue (WAT). Impaired lipid metabolism leads to elevated serum triglycerides, cholesterol, and LDL-C while decreasing HDL-C. This dysregulation is linked to atherosclerosis and fatty liver diseases [20].

2. OXIDATIVE STRESS

Several endogenous and exogenous processes produce reactive oxygen species (ROS), and their adverse effects are deactivated by antioxidant defence. The disproportion between ROS production and their removal causes oxidative stress. Oxidative stress plays a significant role in various diseases, including cancer, chronic obstructive pulmonary disease, atherosclerosis, and Alzheimer's disease. B[a]P is metabolized in the body to form highly reactive intermediates, including epoxides and dihydrodiols, which can generate ROS such as superoxide anions (O_2^-), hydroxyl radicals (·OH), and hydrogen peroxide (H_2O_2) [26]. These ROS can damage cellular macromolecules, including lipids, proteins, and DNA, leading to oxidative stress. B[a]P exposure has been shown to impair the activity of key antioxidant enzymes, such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX), which are critical for neutralizing ROS [27]. Additionally, B[a]P can reduce the levels of non-enzymatic antioxidants, such as glutathione (GSH), further compromising the cell's ability to mitigate oxidative stress. B[a]P can activate pro-oxidant pathways, such as the aryl hydrocarbon receptor (AhR)-dependent pathway, which leads to the production of ROS and the downregulation of antioxidant defenses [28]. For example, in human endothelial progenitor cells, B[a]P activates the NF-κB pathway, which promotes ROS generation and inflammatory responses. Table 4. Illustrates the oxidative Stress biomarkers in different biological systems exposed to B[a]P. Due to its high metabolic activity, the liver is a primary target of B[a]P toxicity. B[a]P-induced oxidative stress in hepatocytes leads to lipid peroxidation, glutathione depletion, and DNA damage [29]. The activation of cytochrome P450 enzymes (CYP1A1) in the liver plays a key role in the bioactivation of B[a]P, resulting in the formation of reactive metabolites that contribute to oxidative stress [28]. B[a]P exposure has been linked to neurotoxicity, particularly in the hippocampus and striatum of rats. Oxidative stress in these brain regions is associated with decreased activities of antioxidant enzymes (SOD, CAT, and GPX) and increased lipid peroxidation, leading to impaired neurobehavioral functions such as learning and memory [30].

Table 4. Oxidative Stress Biomarkers in Different Biological Systems Exposed to B[a]P

Biological System	Oxidative Stress Biomarkers	Reference
Common carp (*Cyprinus carpio*) intestine	Increased GSH, malondialdehyde, and SOD activity	[27]
Rat (Hippocampus)	Decreased SOD activity and increased MDA levels	[30]
Human endothelial progenitor cells (EPCs)	Increased ROS production and NF-κB activation	[29]
Human Skin	Reduced ROS and protein peroxidation	[31]
Zebrafish (Embryos)	Reduced mitochondrial respiration and ATP turnover	[32]

GSH: Glutathione; MDA: Malondialdehyde; NF-Kb: Nuclear Factor kappa-light-chain-enhancer of activated B cells; ROS: Reactive oxygen species; SOD: Superoxide dismutase.

B[A]P TOXICITY IN VARIOUS ORGANS

Benzo[a]pyrene is a common polycyclic aromatic hydrocarbon compound readily produced during food processing and can enter the body through various sources. B[a]P was found to induce cell death in HL-7702 human normal liver cells, enhance intracellular reactive oxygen species (ROS) levels, and arrest the cell cycle at the S phase. B[a]P resulted in cell death through two programmed cell death types: autophagy and pyroptosis [33]. B[a]P treatment led to significant increases in catalase (CAT), glutathione peroxidase (GPx), superoxide dismutase (SOD), and glutathione S-transferase (GST), along with glutathione (GSH) levels in the lungs. However, the levels of glutathione disulfide (GSSG) and malondialdehyde (MDA) were also significantly increased in a time- and dose-dependent manner. DNA damage in the lungs showed the highest levels at 24 hours after B[a]P treatment and increased with higher B[a]P doses. Histopathological changes in the lungs included thickening of the alveolar wall, compressed alveoli, and severe inflammatory cell infiltration [34]. B[a]P exposure significantly increased the serum levels of urea and creatinine in rats compared to the control group. This suggests that B[a]P exposure impairs renal function by decreasing the glomerular filtration rate. Additionally, B[a]P treatment was found to increase oxidative stress markers like reactive oxygen and nitrogen species, lipid peroxidation, and myeloperoxidase activity in the kidney tissue. This oxidative damage likely contributes to the observed decline in renal

function [35]. B[a]P may induce neurotoxicity, including dysregulation of neurotransmitter systems, oxidative stress, and epigenetic changes. In rodents and zebrafish demonstrate that prenatal or early postnatal exposure to B[a]P can lead to long-term changes in multiple behavioral domains, such as locomotor activity, anxiety profile, and cognitive abilities [36].

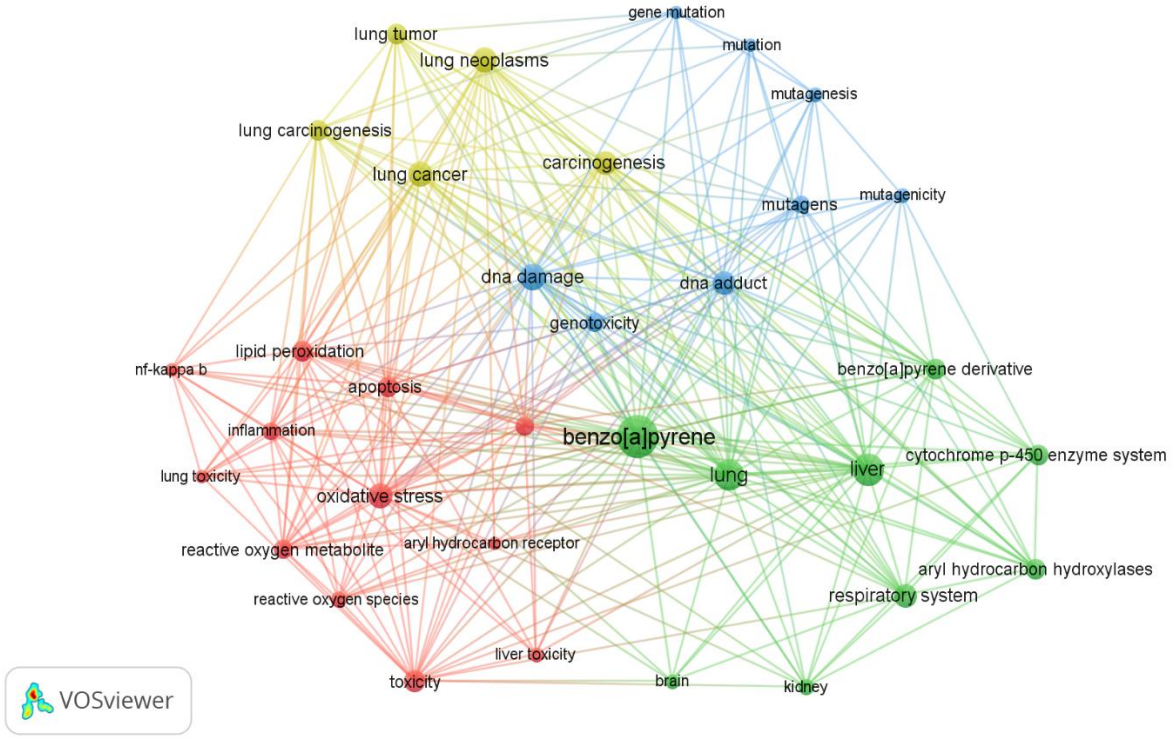


Figure 2. VOS viewer software is used for network analysis of keyword co-occurrence.

A total of 1,676 articles were retrieved from the Scopus database for this bibliographic analysis, covering benzo[a]pyrene toxicity, with keyword combinations centered around "benzo[a]pyrene," "oxidative stress," "toxicity," "liver," "lung," "kidney," “DNA damage” and related terms. The VOSviewer software was used to analyze all the extracted keywords. Twenty-two key co-occurrence terms were selected to construct a network visualization map, as illustrated in Figure 2. In this map, each cluster is represented by a distinct color and size corresponding to the frequency of co-occurrence, while the straight lines represent the strength of association between the keywords. Four main clusters were identified through co-occurrence analysis: (i) Oxidative stress, apoptosis, aryl hydrocarbon receptor, and inflammation pathways related to benzo[a]pyrene exposure (red cluster), (ii) Carcinogenesis and lung neoplasms (yellow cluster), (iii) DNA damage, mutagenesis, and genotoxic effects (blue cluster), and (iv) Metabolic pathways involving the liver, lung, kidney, brain, and respiratory system (green cluster). This keyword-based text mining study highlights the toxicological effects of benzo[a]pyrene, particularly its capacity to induce oxidative stress and damage in vital organs such as the liver, lungs, and kidneys. Figure 2 illustrates the interconnected biological processes and organ-specific impacts associated with benzo[a]pyrene toxicity, offering insights into its role in disease pathogenesis.

3. DIETARY EXPOSURE OF B[A]P

The dietary intake of B[a]P varies significantly across different populations and regions. In China, the total daily intake of B[a]P was estimated to be 124.55 ng/person/day, with fried chicken being the highest contributor. Similarly, in Italy, the total dietary intake of PAHs, including B[a]P, was calculated to be 3 µg/day/person, with cereals, meat, and vegetables being major contributors [37].

B[a]P was detected in numerous foodstuffs, and detection levels depend on the cooking mode, food processing, and pseudo-curing with smoke flavouring agents. During the cooking process (grilled, pan-fried, boiled, and broiled), heating temperature and cooking time highly affected B[a]P [38]. Fried and grilled cooking methods and the widespread consumption of certain dishes may expose the population to elevated levels of potentially carcinogenic compounds, such as benzo[a]pyrene. B[*a*]P commonly occurs in many food products, such as olive oil (2.19 ± 0.2 µg/kg), Alwana olive oil (31.3 ± 0.3 µg/kg), milk (0.06–2.09 µg/kg), and meat/fish-based baby foods (0.00–1.66 µg/kg)**.** B[*a*]P has also been

determined in traditionally smoked goat cheeses, such as Wallahian-style cheese (0.85 ± 0.255 μg/kg) or smoked "Ritta" (17.0 ± 5.10 μg/kg), charcoal-grilled chicken with and without marinating at 270 °C (1.19 ± 0.31 μg/kg; 2.22 ± 0.13 μg/kg), oysters (1.26 ± 1.22 μg/kg), mussels (0.24 ± 0.18 μg/kg), fresh shellfish (0.31 ± 0.42 μg/kg), Iranian bread samples (mean 0.1 μg/kg), and cucumbers (4.35 ng/kg) [6].

4. B[A]PYRENE CONTAMINATION IN AGRICULTURAL PRODUCTS

Benzo[a]pyrene contamination in agricultural products is a significant concern due to its carcinogenic properties and widespread presence in the environment. B[a]P, a polycyclic aromatic hydrocarbon (PAH), is found in various food products, particularly those that are smoked or grilled [6]. A survey of vegetable oils from canned vegetables and fish revealed that 15% of oil samples from vegetable products exceeded the 2 μg/kg level for B[a]P, with the highest concentration (11.3 μg/kg) noted in oil from grilled mushrooms [39]. Interestingly, the contamination levels of B[a]P in agricultural products can be influenced by various factors. For instance, the duration of storage for rapeseed can significantly affect PAH levels in the resulting refined oil. Raw materials processed shortly after harvesting showed higher contamination levels compared to those stored in silos for several months. Additionally, the wood used in smoking can impact B[a]P contamination in food products. A study on smoked pork sausages found that plum, alder, and birch wood resulted in higher PAH concentrations than commonly used beech wood [40].

4.1. Wheat, rice, and barley

Crops can absorb PAHs from polluted soil as a primary food source. Consuming these contaminated crops facilitates the transfer of PAHs through the food chain. Generally, the major ways of PAH uptake by plants are through the root and the epidermis of the aerial parts (foliar uptake) [41].

Benzo[a]pyrene was detected in roots, straw, and grains of winter wheat. In soil, low-molecular-weight (LMW) PAHs dissipated faster than high-molecular-weight PAHs (B[a]P) during wheat growth stages. This disparity arises because LMW PAHs, with lower lipid solubility and higher water solubility, are more accessible to microbial degradation, while HMW PAHs bind strongly to lipophilic soil organic matter (SOM), reducing bioavailability and impeding removal [42].

The bioavailability and phytotoxic effects of benzo[a]pyrene on wheat vary significantly across soil types, red soil primarily inhibits shoot growth, likely due to reduced nutrient uptake and oxidative stress in aerial tissues, brown soil affects shoot growth and germination rate, attributed to physicochemical interactions between B[a]P, soil organic matter, and microbial activity, and black soil causes the most severe inhibition, impairing shoot growth, root elongation, and germination, likely due to higher organic matter content enhancing B[a]P retention and bioavailability [43].

Benzo[a]pyrene, the marker used for evaluating the carcinogenic risk of PAHs in food, in some studies, B[a]P was not detected in the rice grain samples [44]. However, an increase in B[a]P concentration decreased rice biomass, indicating that rice was sensitive to B[a]P exposure. The toxicity threshold (EC10) of B[a]P for rice ranged from 1.64 to 37.32 mg/kg across the 10 different soil types [45].

Controlled B[a]P exposure trials were systematically compared with field experiments in Shenyang, China, to assess air pollution impacts on rice crops. Under strictly regulated conditions (air and sterile quartz sand media), B[a]P accumulation in rice tissues remained negligible, with no significant difference in grain B[a]P content between controls and treatments, excluding root media as a contamination pathway. In contrast, field-exposed plants exhibited substantially elevated B[a]P levels—grains (7.33 fold), husks (9.21 fold), and stems/leaves (27.10 fold)—compared to controlled environments. These findings confirm atmospheric deposition as the primary B[a]P source in aboveground rice tissues, directly driving cereal crop contamination [46]. The observed levels of benzo[a]pyrene in different foods are listed in Table 3.

The concentration of benzo[a]pyrene in the barley plant roots increased with the increasing levels of B[a]P contamination in the soil. The B[a]P concentration in the plant roots ranged from 1.0 ± 0.1 μg/kg in the control and background variants to 23.4 ± 1.2 μg/kg in the variant with 40 MPC (maximum permissible concentration) of B[a]P in the soil. This indicates that the roots were an active source of B[a]P accumulation in the barley plants [45].

Table 3. Benzo[a]pyrene concentration in various foods.

Food items	B(a)P concentration (μg/kg)	Reference
Wheat	1	[46]
	0.91 (μg/100 m^3)	[46]
	19.3	[47]
Rice stem and leaf	185.0 and 111.1	[46]

Barley	1.8	[46]
	30	[48]
Mixed Vegetables	0.01–12.4	[49]
Tomatoes	10.649–21.774	[50]
Leafy Vegetables	0.59–0.77	[51]
Leafy Vegetables	60.5–312	[52]
Leafy Vegetables	532–2261	[53]
Brassica juncea	5.5	[54]
Banana peel	0.8348	[55]
Apple	0.01–2.3	[49]
Refined maize and rapeseed oil	1.04 and 0.93	[56]
Olive Oil	0.03–0.95	[57]
Soybean oil and Corn oils	Not detected – 6.1, and 1.6 – 58.9	[58]
Peanut oil	Not detected	[59]
Black tea	3.96 – 209.36	[60]
Green tea	4.30 – 24.82	[60]
Red tea	7.00 – 18	[60]
White tea	0.8 – 27	[60]
American roasted Arabica Riada coffee	12.52	[61]
green coffee Arabica Rio	Not detected	[61]
Cocoa-Based Drinks	53.2	[62]

4.2. Vegetables and fruits

According to the US Environmental Protection Agency (USEPA), fruits and vegetables have lower concentrations of PAH when compared to processed and unprocessed meat and meat products. The minimum and maximum recommended limits are 0.01 and 0.5 μg/kg [63]. Atmospheric deposition is a significant pathway for B[a]P contamination in vegetables. PAHs, including B[a]P, can be deposited on plant surfaces from polluted air. Leafy vegetables, such as lettuce and spinach, are more prone to contamination due to their large surface area [51]. The benzo[a]pyrene contents of vegetables grown in areas far from industrial plants are lower compared to those grown near industrial areas or in the proximity of emitters [49].

A maximum B[a]P level of 1 mg/kg fresh weight for vegetable foods is permitted. Benzo[a]pyrene is a significant pollutant that poses toxicity risks to vegetables, particularly Solanum lycopersicum L. (tomato) [63]. For lettuce, the toxicity threshold (EC10) values ranged from 1.55 to 7.49 mg/kg, for oilseed rape, the EC10 values ranged from 0.92 to 11.92 mg/kg, EC10 values ranged from 1.59 to 18.75 mg/kg for cucumber and tomato, the EC10 values ranged from 0.50 to 15.24 mg/kg across10 different soil types [45]. As the accumulation of B[a]P increased in plant tissues, severe damage to physiological and biochemical indices occurred. Significant increases were observed in malondialdehyde (MDA), proline, and the activities of antioxidant enzymes like superoxide dismutase (SOD), peroxidase (PRX), ascorbate peroxidase (APOX), and glutathione peroxidase (GP), indicating enhanced oxidative stress [63].

Sources of PAHs in fruits are attributed to atmospheric deposition. Benzo(a)pyrene was commonly detected in all fruit samples, with the highest levels found in peels, indicating that the outer layers of fruits might accumulate more B[a]P [51], a highly carcinogenic polycyclic aromatic hydrocarbon (PAH), was detected in grapes at a concentration of 1.82 μg/kg and in pineapple at 0.22 μg/kg [64]. Some studies have shown that olive fruits can be contaminated with B[a]P, particularly when grown in areas with high levels of air pollution from traffic or industrial activities. However, olive oil generally shows low B[a]P contamination [65]. Dried fruits, such as dried plums and apricots, have been found to contain moderate levels of PAHs, including B[a]P, likely due to environmental contamination during the drying process [66]. While B[a]P exposure is primarily associated with cooked and smoked foods, certain fruits have shown potential for mitigating their toxic effects. These fruits exhibit antimutagenic and antioxidant properties that can reduce the genotoxicity of B[a]P and other PAHs like Psidium guajava leaf extract showed significant antioxidant and antimutagenic activities against various mutagens, including B[a]P, with the methanolic fraction inhibiting over 70% mutagenicity at 80×10^{-3}μg/kg concentration [67].

4.3. Oils

B[a]P can enter the food chain through various pathways, including environmental contamination, industrial processes, and food processing. In the context of oils, B[a]P contamination can occur during the production process, storage, and refining of oilseeds and oils. Regulation (EU) No 835/2011 sets the maximum levels in oils and fats intended for direct human consumption or used as an ingredient in food at 2 μg/kg for benzo(a)pyrene. Environmental factors such as air pollution and soil contamination can also contribute to B[a]P levels in oilseeds, which are subsequently processed into

edible oils [68]. Cooking/indirect pyrolysis generates various types of PAH, and recent studies show considerable PAH content in vegetable oils that are processed using frying and roasting techniques at high temperatures. For example, the roasting of sesame seeds has been proven to promote the generation of B[a]P, raising concerns about sesame oil safety [69]. Processing factors, such as drying, refining, and frying, can further contribute to B[a]P contamination. For instance, high-temperature drying of oilseeds and refining processes can lead to the formation or concentration of PAHs, including B[a]P. The storage conditions of oils and oilseeds also play a role in B[a]P contamination. Improper storage can lead to the accumulation of PAHs due to environmental exposure or microbial activity [68]. Additionally, the type of oil and its production process significantly influence B[a]P levels. For example, virgin olive oils tend to have higher B[a]P levels compared to refined oils due to the minimal processing involved [70]. Several studies have assessed the health risks associated with B[a]P intake through edible oils. The Target Hazard Quotient (THQ) and Margin of Exposure (MOE) are commonly used metrics for risk assessment. For example, in Tehran, the mean THQ for adults and children was 0.0006 and 0.0028, respectively, indicating a low non-cancer risk for adults but a borderline risk for children [71].

5. ANIMAL-BASED FOOD PRODUCTS OR PROTEIN FOOD

B[a]P formation in meat products is primarily linked to the incomplete combustion of organic materials during cooking. Processes like grilling and smoking, which involve high temperatures and pyrolysis of fats and proteins, are significant sources of B[a]P [72]. The type of fuel used (e.g., charcoal, wood) and cooking duration also play crucial roles in B[a]P formation. Grilling and barbecuing methods involve direct flame exposure, leading to higher B[a]P levels due to fat dripping onto heat sources, which pyrolyze and release PAHs. Traditional smoking, especially with wood or charcoal, can lead to B[a]P contamination. The duration and type of wood used significantly influence B[a]P levels. Some studies suggest that marinating meat before cooking can reduce B[a]P formation by creating a protective barrier on the meat surface [73]. Charcoal-grilled beef burgers showed higher B[a]P levels than oven-cooked ones, with concentrations ranging from 15.83 to 82.64 μg/kg [74]. Smoked beef sausages had B[a]P levels between 0.30 and 1.14 μg/kg [73]. Smoked pork samples, especially those from traditional methods, often exceeded EU maximum limits. For instance, Baltic smoked pork samples had B[a]P levels ranging from 0.05 to 166 μg/kg [75]. Fried chicken and smoked turkey showed significant B[a]P levels, with concentrations up to 5.25–5.55 μg/kg in fried chicken and 0.30–1.14 μg/kg in smoked turkey [73]. The EU has established maximum permissible levels for B[a]P and PAH4 (a group of four PAHs including B[a]P) in smoked meats. For example, the maximum limit for B[a]P in smoked meats is 5 μg/kg [75]. FAO/WHO Codex Alimentarius Commission has set guidelines for B[a]P in smoked foods, with recommended 10 μg/kg limits for certain products [76].

6. BEVERAGES

The production process, including roasting, brewing, and packaging, can significantly influence B[a]P levels in beverages. Alcoholic beverages, particularly those aged in charred barrels, can contain B[a]P due to the charring process. The traditional charring produces higher levels of PAHs compared to convective toasting. For instance, in whisky and brandy, B[a]P levels were found to be below 10×10^3 ng/m^3, which is relatively low compared to other foodstuffs like smoked meats [77]. In tea and coffee, B[a]P levels are influenced by the production process. For instance, in Chinese tea, the total PAH concentration ranged from 136.99 to 462.51 μg/kg, with B[a]P contributing significantly to the carcinogenicity of the samples [78]. In coffee, B[a]P was found to be absent in green coffee beans but present in roasted coffee, with concentrations ranging from 0.47 to 12.5 μg/kg. The roasting process was identified as a critical factor in B[a]P formation [61]. The toxicity levels of Benzo[a]pyrene in milk are generally low, but they can vary depending on regional factors, environmental contamination, and processing methods. In raw milk and other dairy products, B[a]P concentrations can vary significantly. For example, a study in Nigeria reported B[a]P concentrations in raw milk ranging from 0.35 to 0.42 μg/kg [79].

7. CONCLUSION

The widespread presence of benzo(a)pyrene in food poses a significant yet often overlooked threat to public health. Studies reveal alarming levels of B[a]P in smoked, grilled, and processed foods, with some products—such as smoked meats and certain oils—exceeding regulatory limits. This carcinogenic compound enters the food chain through environmental pollution, industrial processes, and high-temperature cooking, leading to chronic exposure in populations worldwide. Upon ingestion, B[a]P undergoes metabolic activation via cytochrome P450 enzymes, generating reactive intermediates like BPDE that cause DNA damage, oxidative stress, and inflammation. These mechanisms contribute to cancer development, metabolic disorders, and other chronic diseases. Despite existing regulations, enforcement remains inconsistent, particularly in regions with limited food safety oversight. To address this issue, a multi-pronged approach is necessary, including stricter food safety policies, public education on safer cooking methods, and dietary strategies to counteract B[a]P's harmful effects. Future research should focus on innovative mitigation techniques, such as bioremediation and antioxidant-rich dietary interventions, to reduce exposure risks. Ultimately, proactive measures—spanning regulation, technology, and consumer awareness—are essential to minimizing B[a]P impact and protecting long-term health.

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