

REVIEW PAPER

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Naturally occurring contaminants in food

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Abstract

Naturally occurring contaminants are toxic substances produced by living organisms such as fungi, plants, algae, and bacteria that can enter the food chain through natural biological processes. These contaminants vary greatly in their origin, occurrence in food, and toxicological effects in humans. Depending on their chemical characteristics and exposure levels, they may cause a range of harmful health effects, from acute poisoning to chronic outcomes such as organ toxicity, genotoxicity, or carcinogenicity. Major groups of naturally occurring contaminants in food include mycotoxins, plant toxins, aquatic toxins, and biogenic amines, many of which comprise numerous compounds with different toxicological properties. While some groups, particularly mycotoxins, have been extensively studied due to their widespread occurrence and significant impact on

food safety, other natural contaminants have recently gained increasing attention. This review provides a brief overview of naturally occurring contaminants in food, focusing on their sources, occurrence in food commodities, and potential health effects.

Keywords: contaminants; mycotoxins; plant toxins; aquatic toxins; biogenic amines.

INTRODUCTION

Naturally occurring contaminants are toxic substances produced by living organisms such as fungi, plants, algae, or bacteria. In many cases, they are secondary metabolites of plants and fungi that provide ecological advantages, such as defence against predators and insects, while in other cases they are produced during algal growth or because of food spoilage. They vary greatly in their origin, occurrence in foods, and effects on human health. Depending on their chemical nature and level of exposure, these compounds may cause a variety of toxic effects ranging from acute poisoning to long-term health effects such as organ toxicity, genotoxicity, or carcinogenicity. The presence of naturally occurring contaminants in food, like other chemical or biological food contaminants, poses serious threats to public health, international and local food trade, and the overall strength of the food systems.

Several groups of naturally occurring contaminants are present in food, including mycotoxins, plant toxins, aquatic toxins, and biogenic amines, each encompassing numerous subgroups with a variety of different compounds. Most of these contaminants have been known for many years and have been extensively stud-

ied because of their potential impact on food safety and human health. Some, such as mycotoxins, remain under particular attention due to their widespread occurrence and their presence in a wide range of food and feed commodities. In recent years, however, other groups of natural contaminants have also gained increasing attention in research and food safety monitoring. This growing interest is driven by factors such as climate change, changes in dietary habits and the expansion of global food trade.

Climate change can cause, intensify, or alter the occurrence of certain food-borne illnesses and facilitate the introduction of invasive alien species [1]. It influences the occurrence, intensity, and toxicity of harmful algal and bacterial blooms, affects the spread and persistence of parasites, fungi, viruses, vectors, and invasive species, and may drive the emergence or re-emergence of hazards while increasing exposure or susceptibility and altering the levels of known risks [1]. Climate change could significantly affect the life cycle stages and development rates of toxigenic fungi, increase agricultural pests that promote fungal colonisation and growth, and alter other environmental factors such as temperature, humidity, rainfall patterns, and

plant stress, thereby influencing the occurrence and levels of mycotoxins in food [2–4].

The globalisation of the agri-food market has increased the complexity of food supply chains and has been recognised as a factor that may compromise food safety due to differences in regulations among countries and the presence of newly emerging or non-regulated compounds [5]. Additionally, in recent years, plant-based diets have become one of the most important trends in global nutrition, and consumer demand for plant-based foods is also increasing rapidly. While this trend is growing rapidly due to concerns about environmental sustainability and animal welfare, it can lead to increased exposure to mycotoxins and plant toxins.

This short review aims to provide an overview of naturally occurring contaminants in food, with a focus on their sources, occurrence in food commodities, and potential health effects. The paper summarises the main groups of these contaminants and discusses their relevance for food safety and public health.

MYCOTOXINS

Mycotoxins are toxic secondary metabolites produced by many fungal species growing on various food products such as cereals, nuts, dried fruit, legumes and spices. The growth of fungi and production of mycotoxins may occur both before and after harvest, particularly under warm and humid environmental conditions. The majority of mycotoxins are chemically stable compounds and can therefore survive various food processing procedures and can be found even in highly processed food products. To date, several hundred mycotoxins have been identified. However, only a limited number are commonly detected in food and feed and represent a concern for human and animal health. The most important among them include aflatoxins (AFs), ochratoxin A (OTA), fumonisins (FUMs), zearalenone (ZEN), and deoxynivalenol (DON) (Table 1). Next to the mycotoxins listed in Table 1, so-called emerging mycotoxins, such as enniatins, beauvericin, and fusaric acid, are also of interest.

Table 1. The most important mycotoxins, the species that produce them, and the products susceptible to contamination.

Mycotoxin	Principal producers	Commonly contaminated food products	Reference
Aflatoxins (B1, B2, G1, G2, M1, M2)	<i>Aspergillus flavus</i>	Maize and other cereals, milk and dairy products, nuts, spices, and dried fruits	[2,6]
	<i>Aspergillus parasiticus</i>		
Ochratoxin A	<i>Penicillium verrucosum</i>	Cereals, coffee, dried fruit, wine, beer, cocoa, nuts, beans, and rice	[6,7]
	<i>Aspergillus ochraceus</i>		
Deoxynivalenol	<i>Fusarium culmorum</i>	Wheat, barley, oats, maize, and rice	[6]
	<i>Fusarium graminearum</i>		
	<i>Fusarium culmorum</i>		
Zearalenone	<i>Fusarium graminearum,</i>	Wheat, barley, oats, rye, maize, rice, and sorghum	[6,8]
	<i>Fusarium verticillioides</i>		
	<i>Fusarium verticillioides</i>		
Fumonisin	<i>F. fujikuroi</i>	Maize, rice, sorghum, and beans	[6,9]
	<i>Penicillium expansum</i>		
	<i>Aspergillus clavatus,</i>		
Patulin	<i>Byssoclamys nivea</i>	Apples, pears, and grapes	[10]
	<i>Fusarium sporotrichioides</i>		
	<i>Fusarium langsethiae</i>		
T-2/HT-2	<i>Fusarium poae</i>	Wheat, barley, oats, rye, maize	[6]
	<i>Claviceps spp</i>		
Ergot alkaloids	<i>Penicillium spp</i>	Wheat, barley, oats, rye, maize, and rice	[11]
	<i>Aspergillus spp</i>		
Citrinin	<i>Monascus spp</i>	Maize and other cereals, cheeses, sausages, and fruit	[12,13]
	<i>Aspergillus spp</i>		
Cyclopiazonic acid	<i>Penicillium spp</i>	Maize and other cereals, peanuts, cheeses, hams, and sausages	[14,15]
	<i>Aspergillus versicolor</i>		
	<i>Aspergillus nidulans</i>		
Sterigmatocystin	<i>Aspergillus nidulans</i>	Maize and other cereals, cheese, nuts, and spices	[16]
	<i>Penicillium luteum</i>		

Aflatoxins

As secondary metabolites produced by certain *Aspergillus* species, AFs are potent carcinogenic, genotoxic, and teratogenic compounds found in various agricultural products, most notably in maize and other cereals, milk and dairy products, nuts, spices, and dried fruits [17,18]. Aflatoxins B1, B2, G1 and G2 (AFB1, AFB2, AFG1, and AFG2) are four naturally occurring AFs from the total of 20 identified molecules. Besides the four previously mentioned naturally occurring AFs, these fungi also produce other toxic metabolites such as aflatoxinol and sterigmatocystin [2]. Aflatoxin M1 (AFM1) is a hydroxylated metabolite of AFB1 found in milk and dairy products from livestock that have consumed contaminated feed, as well as in human milk [2]. Fungi capable of producing AFs are prevalent in hot and humid regions, and contamination of food with AFs may occur both before and after harvest. AFB1 is the most commonly detected aflatoxin in contaminated food samples, and the other three are rarely found in its absence [2].

AFs are quickly absorbed and distributed to the liver, where CYP enzymes convert AFB1, AFG1 and AFM1 to the corresponding 8,9-epoxides, which can bind to both DNA and proteins, while the 8,9-epoxides are not formed from AFB2 and AFG2 [2]. AFB1 affects reproductive and developmental parameters and impairs the immune response [2]. High exposure to AFB1 can cause acute aflatoxicosis with a high mortality rate, while chronic exposure to lower levels is associated with cirrhosis and markers of liver dysfunction [2]. AFB1 is a genotoxic and carcinogenic substance, and the liver carcinogenicity of AFB1 and AFs is considered a critical effect for the risk assessment (EFSA, 2020). According to some estimations, AFs play a contributing role in 4.6–28.2% of all global hepatocellular carcinoma cases [19]. Considering the genotoxic and cancerogenic nature of these substances, no Tolerable Daily Intake (TDI) could be established. Based on the study in rats, the EFSA Panel on Contaminants in the Food Chain (CONTAM) used model averaging to calculate a Benchmark Dose Lower Confidence Limit (BMDL₁₀) of 0.4 µg/kg body weight per day for the incidence of hepatocellular carcinoma to be used for the risk characterisation [2].

Ochratoxin A

OTA is a mycotoxin produced by certain species of the *Penicillium* and *Aspergillus* genera, primarily *Penicillium verrucosum* (in temperate climates) and *Aspergillus ochraceus* or *A. carbonarius* (in warmer climates), acting primarily as a storage mould. OTA has been detected in cereals, cereal-derived products, dried fruits, wine, grape juice, beer, tea, coffee, cocoa, nuts, spices, liquorice, processed meat, cheese, and other foods [20]. Due

to its slow elimination and excretion and low metabolic rate, OTA has the potential to bioaccumulate in the organism, leading to its accumulation in animal tissues and presence in meat and meat products, particularly in non-ruminant animals such as pigs [7].

OTA can cause a wide range of adverse health effects due to its toxicological properties, including teratogenicity, immunotoxicity, carcinogenicity, genotoxicity, neurotoxicity, and hepatotoxicity [20]. These effects have been observed mainly in animal studies, where OTA causes a range of adverse outcomes in repeated-dose experiments in mice, rats, rabbits, and pigs, including immunotoxicity, neurotoxicity, and developmental effects at maternally toxic doses [7]. The kidney is the primary target organ, and chronic oral exposure to OTA has been associated with increased incidences of kidney tumours in mice and rats [7]. OTA induces gene mutations, DNA strand breaks, and specific types of chromosomal damage both in vitro and in vivo [7]. The Tolerable Weekly Intake of 120 ng/kg body weight established by the CONTAM Panel in 2006 [21] is no longer considered valid. For the characterization of non-neoplastic effects of OTA, a BMDL₁₀ of 4.73 µg/kg body weight per day was derived from microscopic kidney lesions observed in a repeated-dose study in female pigs, while a BMDL₁₀ of 14.5 µg/kg body weight per day was identified for the characterization of neoplastic effects based on the increased incidence of kidney tumours observed in male rats [7].

Fumonisin

FUMs are a class of mycotoxins produced by *Fusarium* species, predominantly by *F. verticillioides* and *F. proliferatum*. These fungi are plant pathogens, and FUMs contamination usually starts in the field, especially at warmer temperatures accompanied by drought or rain extremes and insect damage [22,23]. FUMs are classified into four groups, and the most important group is the B-type group, particularly FB1–FB4, which differ in the number and position of hydroxy-groups in the backbone [24]. They are mainly found in maize and maize-based food and feed products, but they also occur in other staple crops, including peanuts, wine, rice, and sorghum [25]. The occurrence of FB1–FB3 is well documented in maize and maize-based products, although limited information is available for the occurrence of FB4 [24].

FUMs are suspected of being causative agents of neural tube defects and oesophageal cancer in humans, immune suppression, hepatotoxicity, nephrotoxicity, and chronic liver diseases [25]. The International Agency for Research on Cancer (IARC) has classified FB1 as a class 2B carcinogen – possibly carcinogenic to humans [26]. Toxicity assessments are mainly based on results with FB1, but FB2–4 are considered to have a similar toxicological profile and potency as FB1 [24].

The CONTAM panel established a TDI for FB1 of 1.0 µg/kg body weight per day based on increased incidence of megalocytic hepatocytes found in a mouse chronic study [24]. Based on structural similarity and the limited data available indicating a similar mode of action and toxic potencies, the CONTAM Panel decided that FB2, FB3 and FB4 should be included in a group TDI with FB1 [24].

Zearalenone

ZEA is a common mycotoxin produced by plant pathogens *Fusarium* species, such as *F. graminearum*, *F. oxysporum*, *F. equisetum*, and *F. nivale* [27,28]. ZEA is mainly found in grains, such as wheat, barley, sorghum, rice, soybean, oat, maize, and their products, including vegetable oils [4]. Several modified forms of ZEA have been identified and characterised. These metabolites are formed in the fungal, plant or mammalian organism, and they are known as phase I and phase II metabolites (α- and β-zearalenol, zearalanone, α- and β-zearalanol, and their glucosides, sulphates and glucuronides) [29].

Reproductive toxicity, hepatotoxicity, immunotoxicity, genotoxicity, and carcinogenicity are among the main manifestations of ZEA toxicity [4]. The main biological activity of ZEN is its oestrogenic effect, meaning its ability to mimic endogenous steroidal sex hormones, 17β-oestradiol, leading to numerous endocrine and reproductive dysfunctions [29]. Most of the phase I metabolites have oestrogenic activity, and the CONTAM Panel assumed dose additivity for their effect. Therefore, the CONTAM Panel found it appropriate to set a group TDI of 0.25 µg/kg body weight per day expressed as ZEA equivalents for ZEA and its modified forms based on oestrogenic activity in pigs as the critical end point [29].

Deoxynivalenol

DON is a type-B trichothecene produced by plant pathogens of the *Fusarium* genus, particularly *F. graminearum* and *F. culmorum*, typically growing on the cereals in the field, preferably in temperate climates [30,31]. The occurrence of DON and structurally related trichothecene mycotoxins 3-Ac-DON and 15-Ac-DON is common in Europe. DON is one of the most extensively studied mycotoxins worldwide, and it often co-occurs with other mycotoxins, primarily with other *Fusarium* toxins [31]. DON-3- glucoside is a modified form of DON (also called masked DON) and is the main plant metabolite of DON [31]. Other modified forms of DON have also been reported, and additional forms may be identified in future research [31]. DON and three modified forms, 3-Ac-DON, 15-Ac-DON and DON-3-glucoside, occur mainly in cereal grains such as wheat, barley, oats, rye and maize [31].

DON is commonly referred to as vomitoxin because it induces vomiting in pigs and causes gastrointestinal effects in humans. DON is linked with digestive problems, diarrhoea, reproductive problems, nutrient malabsorption, immune disorders, increased incidence of diseases, and endocrine disruption [30,31]. The CONTAM Panel established a group TDI for the sum of DON, 3-Ac-DON, 15-Ac-DON and DON-3-glucoside of 1 µg/kg body weight per day [31].

PLANT TOXINS

Plant toxins are naturally occurring plant metabolites produced by plants as protection from herbivores, insects, and microorganisms. Toxic compounds can be present in different parts of a plant, including the roots, tubers, stems, fruits, buds, and leaves. When these substances are ingested, they can potentially be harmful to human health. In some cases, plants that contain toxic compounds grow as weeds among food crops. During harvesting, their seeds or leaves may accidentally mix with the main crop. As a result, small amounts of plant toxins can sometimes be found in food products such as cereals, herbal preparations, teas, and salad crops. Examples of toxins that may occur in this way include pyrrolizidine alkaloids and tropane alkaloids. Other plant toxins occur naturally as constituents of certain plant-based foods. For instance, erucic acid can be present in some edible oils, hydrocyanic acid is found in apricot kernels, and solanine occurs in potatoes. Around 30 groups of plant toxins are regarded as major or relevant in food safety, and only the most important groups are covered here.

Tropane Alkaloids

Tropane alkaloids (TAs) are metabolites of plants from various families, including *Brassicaceae*, *Solanaceae* (e.g. mandrake, henbane, deadly nightshade, Jimson weed) and *Erythroxylaceae* (coca) [32]. More than 200 different TAs have been identified in various plants, but the most studied and predominant TAs are (-)-hyoscyamine, (-)-scopolamine and atropine (the racemic mixture of (-)-hyoscyamine and (+)-hyoscyamine) [32,33]. TAs are present throughout TA-producing plants, making contamination of cereals and other crops with parts of TA-producing plants during harvesting the primary source of TAs in food. The most common source of occurrence worldwide is the contamination of linseed, soy, millet, and buckwheat, but also maize and soy with the seeds of *Datura stramonium* and other *Datura* species [33]. Seeds and leaves of TA-producing plants are also known to contaminate teas, herbal teas, herbs, and spices [33].

Competitive antagonism of muscarinic acetylcholine receptors in both the central and peripheral nervous systems is the mode of action through which TAs

exert their pharmacological and toxicological effects [34]. Symptoms of TAs poisoning include tachycardia, muscle spasms, mydriasis, delirium and sometimes they can even cause death [34,35]. They do not exhibit chronic toxicity, and based on results showing a decreased heart rate in a human volunteer study, the CONTAM Panel established a group acute reference dose (ARfD) of 0.016 µg/kg body weight, expressed as the sum of (-)-hyoscyamine and (-)-scopolamine, assuming equivalent potency [34].

Cyanogenic glycosides

Cyanogenic glycosides (CGs) are plant-derived secondary metabolites capable of releasing hydrogen cyanide upon enzymatic hydrolysis and are extensively distributed throughout the plant kingdom as part of plant defence against herbivores and pathogens [36]. CGs contain chemically bound cyanide and occur in foods such as apricot kernels, almonds, linseed, and cassava and derived foods such as persipan and marzipan. The trivial names of cyanogenic CGs are generally derived based on the Latin names of the plants from which they were first isolated (e.g., amygdalin from almond, *Prunus amygdalus*); still, several identified CGs do not have trivial names, and currently 112 distinct CGs are known from the plant kingdom [36,37].

When plant cells are damaged, for example by chewing, CGs come into contact with their degrading enzymes, resulting in the release of cyanide, which is absorbed from the gastrointestinal tract and rapidly distributed throughout the body [38]. Acute toxicity of cyanide and CGs, based on animal studies, is characterised by dyspnoea, ataxia, arrhythmia, convulsions, loss of consciousness, reduced respiration, and death [38]. Acute cyanide toxicity is mainly caused by inhibition of oxidative phosphorylation and the resulting shift to anaerobic energy production, with the brain and heart being especially susceptible due to their high oxygen demand, leading to hypoxia, metabolic acidosis, and disruption of vital functions [38]. Repeated exposure to cyanide has been associated with histopathological alterations in the thyroid, kidney, liver, and central nervous system, as well as changes in epididymal cauda weight [38]. The reported human acute lethal oral dose of cyanide is between 0.5 and 3.5 mg/kg body weight, and the ARfD is 20 µg/kg body weight [38].

Pyrrolizidine alkaloids

Pyrrolizidine alkaloids (PAs) are plant secondary metabolites produced as part of plant defence against herbivores. More than 6,000 plant species are known to biosynthesise PAs, primarily from the families of the *Boraginaceae* (e.g. *Heliotropium* spp.), *Asteraceae* (e.g. *Senecio* spp.) and *Fabaceae* (e.g. *Crotalaria* spp.), with about 600 PAs identified [39]. PAs can occur both as free

bases and as N-oxides, which can be converted to the free base in humans and animals. [39]. Their toxicity in humans is documented in case reports of intoxication following ingestion of PA-containing herbal medicines and teas, as well as in outbreak cases, including fatalities linked to the consumption of grain contaminated with PA-containing weeds [40].

Short-term toxicity of PAs primarily affects the liver and lungs, with a particular association with the development of hepatic veno-occlusive disease [40]. Other reported effects include hepatotoxicity, developmental toxicity, genotoxicity, and carcinogenicity, with 1,2-unsaturated PAs, which are considered genotoxic and carcinogenic due to their metabolic activation to reactive pyrroles [40]. Based on that information, the CONTAM Panel identified a list of 17 PAs of importance for monitoring in food and feed, while the IARC classified lasiocarpine, monocrotaline and riddelliine as category 2B – possibly carcinogenic to humans [40]. The CONTAM Panel established a Reference Point of 237 µg/kg body weight per day for the carcinogenic risks of PAs, and concluded that exposure may pose a potential health concern, particularly for frequent and high consumers of tea and herbal infusions [40].

Erucic acid

Erucic acid is the trivial name of the fatty acid cis-13-docosenoic acid that occurs at high levels predominantly in the seeds of the *Brassicaceae* family members, e.g. rapeseed, mustard, kale, cabbage and turnips [41]. Erucic acid is also present, but at lower concentrations, in other food sources, such as fish, and there is evidence of transfer to other products of animal origin from feed [41]. With the heart as the principal target organ, myocardial lipidosis was identified as a consequence of chronic exposure to erucic acid [41]. A TDI of 7 mg/kg body weight per day for erucic acid was established [41].

AQUATIC BIOTOXINS

Aquatic biotoxins are toxins produced by algae in marine and freshwater environments. These algal toxins are generated during blooms of naturally occurring algal species [42]. Algal toxins accumulate in shellfish and fish when they consume toxic phytoplankton during blooms. Shellfish, including mussels, scallops, and oysters, are more likely to accumulate these toxins than fish. They can cause diarrhoea, vomiting, tingling, paralysis and other effects in humans, other mammals or fish [42]. They are tasteless and odourless and are not eliminated by cooking or freezing [42]. Next to the groups described in this paper, this category of natural food contaminant includes the domoic acid group, brevetoxin group, okadaic acid group, ciguatoxin group and several more.

Azaspiracid group

Azaspiracids (AZAs) are a group of shellfish toxins produced by dinoflagellates of the *Azadinium* and *Amphidoma* genera, with *Azadinium dexteroporum* known to produce the highest number of variants (up to six) among the currently identified 68 AZA toxins and their analogues [43]. AZA1, AZA2, and AZA3 are considered the most relevant due to their prevalence and toxicity [44]. AZAs can be found in various species of filter-feeding bivalve molluscs such as oysters, mussels, scallops, and clams, with mussels being the most affected species [44]. While initially identified in Ireland, there are reports of cases of AZA poisoning in coastal areas around the globe [43]. Temperatures typically used for cooking do not degrade AZAs in shellfish [44].

AZA poisoning symptoms are nausea, vomiting, diarrhoea and stomach cramps [44]. The available data on the chronic effects of AZAs in animals and humans were insufficient to establish a TDI. However, the CONTAM Panel established an ARfD of 0.2 µg AZA1 equivalents/kg body weight [44].

Saxitoxin group

Saxitoxin (STX)-group toxins are a group of closely related tetrahydropurines that have been found in filter-feeding bivalve molluscs such as oysters, mussels, scallops, and clams from various parts of the world [45]. STX can be produced by dinoflagellates in marine environments as well as by cyanobacteria in freshwater environments [46]. They are mainly produced by dinoflagellates from the genus *Alexandrium* (comprising 10 species), *Pyrodinium bahamense*, *Gymnodinium catenatum*, and *Centrodinium punctatum* [45,46]. Freshwater cyanobacteria include *Raphidiopsis brookii*, *Anabaena circinalis*, *Aphanizomenon* sp., *Raphidiopsis raciborskii*, and *Microseira wollei* [45,46]. The STX family includes various natural analogues. So far, 57 types of STX analogues have been identified, of which STX, NeoSTX, GTX1 and dc-STX seem to be the most toxic analogues [45,46].

STX-group toxins cause a syndrome known as paralytic shellfish poisoning. As a highly specific blocker of voltage-gated sodium channels, STX toxins block sodium ion influx, inhibiting nerve impulse transmission, which leads to systemic physiological dysfunctions in the nervous, respiratory, cardiovascular, and digestive systems [46]. Symptoms may appear within 30 minutes to two hours after ingestion and can vary in severity, ranging from mild tingling or numbness around the lips to incoherent speech, lack of coordination, and general weakness, progressing in more severe cases to muscular paralysis and respiratory difficulty, with death potentially occurring due to respiratory paralysis in extreme cases of poisoning [45]. Approximately 2,000 cases occur worldwide each year, with a mortality rate

of up to 15% [47]. As no data on the chronic effects of STX-group toxins in animals or humans were available to establish a TDI, the CONTAM Panel established an ARfD of 0.5 µg STX equivalents/kg body weight [45].

Cyanotoxins

Cyanotoxins are secondary metabolites produced by cyanobacteria, also known as blue-green algae, a diverse group of Gram-negative photosynthetic prokaryotes. They colonise a wide heterogeneous range of ecosystems, both freshwater and marine, and are a major component of the phytoplankton [48]. Cyanotoxins exhibit a wide range of toxicities and chemical structures. Cyanotoxins can be classified into three broad structural groups: cyclic peptides, alkaloids and lipopolysaccharides [49]. Depending on their mechanism of toxicity, cyanotoxins are classified as hepatotoxins (e.g. microcystins (MCs), nodularin), neurotoxins (e.g. anatoxin-a, homanatoxin, saxitoxin), cytotoxins (e.g. cylindrospermopsin), dermatotoxins (e.g. lyngbyatoxin), and irritant toxins (e.g. lipopolysaccharides) [49]. Produced by several genera of cyanobacteria (*Microcystis*, *Anabaena*, *Nostoc* and *Oscillatoria*), MCs are the most investigated group among cyanotoxins [49]. Presence of cyanotoxins (mostly MCs) was detected in various types of fish, mussels, crayfish, frogs, dietary supplements (e.g. *Spirulina* and *Chlorella*) and even in vegetables and rice as a consequence of water pollution [49].

The mechanism of toxicity of MCs is based on the inhibition of protein phosphatases types 1 and 2A, which leads to hyperphosphorylation of cellular proteins in the liver, causing hepatotoxicity, liver damage, massive haemorrhage and death [50]. While MCs primarily target the liver, they also affect other organs, including the gastrointestinal tract, kidneys, lungs, and heart, and there are reports of acute poisoning with MCs, mainly due to the consumption of contaminated raw drinking water [50]. A provisional TDI value of 0.04 µg/kg body weight per day was derived only for microcystin-LR, the most common, prevalent, and highly toxic variant among the microcystin family of cyanotoxins [49].

BIOGENIC AMINES

Biogenic amines (BAs) are low-molecular-weight, non-volatile, nitrogenous organic bases that are formed by amino acid decarboxylases from corresponding amino acids. These enzymes are present in spoilage microorganisms and in naturally occurring or added lactic acid bacteria involved in food fermentation [51]. Foods that may contain high levels of BAs include fish and fish products as well as fermented foods such as meat, dairy products, vegetables, beer, and wine [51]. The most important BAs found in food are histamine,

formed from the decarboxylation of histidine, and tyramine, putrescine, cadaverine, and phenylethylamine, formed from tyrosine, ornithine, lysine, and phenylalanine, respectively. The presence of high levels of toxic biogenic amines in food is undesirable and may indicate insufficient hygiene and process control. Biogenic amines are thermostable and are not destroyed by heat treatments typically applied during food processing and preparation [51].

Histamine is considered the most important member of the biogenic amines group and is responsible for scombroid poisoning (also known as histamine fish poisoning), a foodborne illness caused by the consumption of fish containing high levels of histamine. Although histamine is the predominant amine involved, other biogenic amines are also likely to play an important role in the onset of the illness. While histamine poisoning is usually associated with the consumption of scombroid-type fish, cases have also been reported following the consumption of other foods, especially cheese. Typical symptoms of histamine intoxication include rash, erythema, sweating, nausea, vomiting, diarrhoea, sensation of burning in the mouth, swelling of the tongue and face, headaches, respiratory distress, palpitations and hypotension [52]. Symptoms of histamine poisoning typically last for a few hours to a day, but in rare cases may persist for several days [52]. Tyramine causes hypertension, while other symptoms such as headache, perspiration, vomiting, and pupil dilation have also been reported and, in some cases, the consumption of foods rich in tyramine has been associated with hypertensive crises and, less frequently, with migraine episodes [51]. It has been shown that cadaverine and putrescine can enhance the toxicity of histamine [53].

An oral No Observed Adverse Effect Level (NOAEL) of 50 mg histamine has been identified, while challenge studies suggest that the threshold toxic dose is approximately 90 mg [54]. However, the exact toxic threshold remains uncertain. It should also be noted that although the NOAEL represents an appropriate hazard threshold for healthy individuals, it may not adequately protect population groups with increased sensitivity to histamine [54].

CONCLUSION

Naturally occurring contaminants represent a significant group of hazards in the food chain due to their diverse origins, widespread occurrence in various food commodities, and potential to cause adverse health effects. Next to the well-known mycotoxins, it is becoming increasingly evident that plant toxins such as glycosides and alkaloids also pose noteworthy health risks. Increased awareness and further research are needed to better understand their health impacts and

to support the development of appropriate prevention and control strategies.

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Prirodno prisutni kontaminanti u hrani

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Kratak sadržaj

Prirodno prisutni kontaminanti su toksične supstance koje proizvode organizmi poput gljiva, biljaka, algi i bakterija, a koje mogu ući u lanac hrane putem prirodnih bioloških procesa. Ovi kontaminanti se značajno razlikuju po svom poreklu, prisustvu u hrani i toksičnim efektima na ljude. U zavisnosti od njihovih hemijskih karakteristika i nivoa izlo-

ženosti, mogu izazvati različite štetne efekte na zdravlje, od akutnog trovanja do hroničnih posledica kao što su toksičnost za organe, genotoksičnost ili kancerogenost. Glavne grupe prirodno prisutnih kontaminanata u hrani uključuju mikotoksine, biljne toksine, vodene (akvatične) toksine i biogene amine, od kojih mnoge obuhvataju veliki broj jedinjenja sa različitim toksičnim svojstvima. Dok su neke grupe, posebno mikotoksini, opsežno proučavane zbog njihove široke rasprostranjenosti i značajnog uticaja na bezbednost hrane, drugi prirodni kontaminanti u novije vreme privlače sve veću pažnju. Ovaj pregledni rad daje kratak prikaz prirodno prisutnih kontaminanata u hrani, sa fokusom na njihove izvore, pojavu u prehrambenim proizvodima i potencijalne zdravstvene efekte.

Ključne reči: kontaminanti; mikotoksini; biljni toksini; vodeni (akvatični) toksini; biogeni amini.