Modified Mycotoxins

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Mycotoxins are toxic secondary metabolites produced by filamentous microfungi on almost every agricultural commodity worldwide. After the infection of crop plants, mycotoxins are modified by plant enzymes or other fungi and often conjugated to more polar substances, like sugars. The formed—often less toxic—metabolites are stored in the vacuole in soluble form or bound to macromolecules. As these substances are usually not detected during routine analysis and no maximum limits are in force, they are called modified mycotoxins. While, in most cases, modified mycotoxins have lower intrinsic toxicity, they might be reactivated during mammalian metabolism.

modified mycotoxins metabolites risk assessment

1. Modified Mycotoxin Origin

Modified mycotoxins can be generated by plants in defense of the parasitic fungus that produces mycotoxins or by the fungus that spreads them in the host [1]. Some studies have shown the possibility that modified mycotoxins are produced during metabolic processes in animals and humans and during food processing [2][3][4][5][6]. Therefore, a distinction should be made between the origin and the modifications encountered by modified mycotoxins. The primary source is related to plant and fungus metabolism, whereas the secondary source is related to fungi and animals (including humans), mammalian metabolism, and food processing.

Plants can metabolize xenobiotic compounds, including mycotoxins, as part of their defense against pathogens [7].

Modified mycotoxins arising from plant host activity are the most widespread. They are produced via enzymatic detoxification processes, converting mycotoxins into more polar metabolites, which are transported into vacuoles for further storage or conjugated to biopolymers such as cell wall components [8][9]. *Fusarium* infection usually occurs in the field (in contrast to *Aspergillus* or *Penicillium* infections); the *Fusarium* mycotoxins deoxynivalenol (DON), zearalenone (ZEA), fumonisin B1 (FB1), T-2 Toxin, HT-2 Toxin, and nivalenol (NIV) are the most prominent targets for conjugation.

Plants can convert trichothecenes and zearalenone into polar derivatives after conjugation with sugars, amino acids, or sulphate groups [7].

Phase I reactions consist mainly of hydrolysis, reduction, and oxidation. The most prevalent reactions involve the cytochrome P450 monooxygenases (P450), which convert lipophilic toxins by oxidation to hydrophilic (excretable) metabolites [10]. Hydrolysis is catalyzed by esterases and amidases and can serve as a detoxification or activation

mechanism governing mycotoxin selectivity or resistance [11]. The plant specificity of esterase varies dramatically among species and biotypes. Moreover, the compounds from phase I reactions could have even higher toxicity [12].

Phase II is mainly characterized by conjugation; the enzymes involved can act on phase I compounds with covalent binding [4]. The main enzymes are glucosyl-, malonyl-, and glutathione-S-transferases (GSTs) [12]. These reactions led to non-toxic or less-toxic chemicals than the parent compound.

GSH (γ-glutamyl-cysteinyl-glycine) is plants' second primary detoxification mechanism [13]. The conjugation compounds are highly polar and hydrophilic and contain a side group with two carboxyls, an amine group, two peptide bonds, and a thiol.

The conjugated mycotoxins follow different metabolic patterns. GSH conjugates lead to products that differ from the parent toxicant; epoxides, lactones, or aldehyde groups form irreversible derivatives, whereas glucosyl compounds can be reversed by numerous glycosidases in plants and the digestive systems of animals.

It was observed that the level of modified mycotoxins after GSH conjugation in plants could increase after herbicide treatments stimulated the biochemical reaction [8].

After conjugation, the compounds are stored in the vacuoles (Phase III) or are irreversibly bound to the cell wall. Thus, detoxification products are concentrated and stored in the plant tissue.

Fusarium mycotoxins (DON, ZEA, T2, HT2, and NIV) are the primary targets for phase II conjugation reactions with monosaccharides, glutathione, or sulphates [4].

Zearalenone-4-glucoside (ZEA4G) and deoxynivalenol-3-glucoside (DON3G) have been detected in naturally contaminated wheat [14][15]. Oligoglycosylated DON was reported in beer, malt, and bread [16]. T-2-glucoside (T-2-G) and HT-2-glucoside (HT-2-G) were detected in naturally contaminated wheat, oats, and maize [15][17].

Glucoside conjugates of fusarenon-X (FUSX-G) and nivalenol (NIV-G) were found in artificial wheat with Fusarium spp. [1][18]. Type A trichotecene glucosides, neosolaniol-glucoside (NEO-G), and diacetoxyscirpenol-glucoside (DAS-G), were found in maize powder [19]. Contaminated wheat showed the presence of DONGSH [20]. DON3S and DON15S are obtained by sulphatase conjugation or glutathione S-transferase [21][22].

Ochratoxin A (OTA) metabolism in plants studied in in vitro trials revealed two principal metabolites, (4R)- and (4S)-4-hydroxy-OTA; moreover, β-glucosides were characterised for both isomers [23][24].

Among fungal conjugates, 3-acetyl-deoxynivalenol (3A-DON) and 15-acetyldeoxynivalenol (15A-DON) have been characterised in cereals contaminated with *F. graminearum* [4][25]. These compounds are biosynthetic precursors of DON, and subsequently a UDP-glucosyltransferase trichothecene specific converts DON into DON3G [4][26]. Fusarenon-X conjugate, a precursor of NIV, was found in infected maize [4]. The saprobic *Rhizopus* fungus can metabolize ZEA to ZEA14S [4].

Mammals can conjugate mycotoxins before excretion. DON and ZEA glucuronides and sulphate have been reported recently [27][28][29][30]. Urine samples and human and animal liver microsomes showed the presence of D3GlcA and D15GlcA [31][32]. It was also reported that the presence of OTA-acyl-GlcA, OTA-phenol-GlcA, and OTA-amino-GlcA glucuronides excreted via urine as OTα-acyl-GlcA and OTα-phenol-GlcA in urine samples [33].

2. Occurrence of Modified Mycotoxin

Modified mycotoxins have been found in food and feed, predominantly those that are cereal-based. The most frequently identified modified mycotoxin belongs to the family of the fusariotoxins and are β-linked glucose-conjugates of deoxynivalenol, nivalenol, HT-2 (DON3G, NIV3Glc, HT2Glc), zearalenone (ZEA14G, α-ZEL14G, β-ZEL14G), zearalenone-14-sulphate (ZEA14S), and fumonisins-esters (**Table 1**) [4][34][35][36][37].

Table 1. Commonly modified mycotoxins.

Toxin Family	Free Form	Modified Mycotoxin	Food and Feed Occurrence
Fusarium	Zearalenone (ZEA)	ZEA14G; ZEN14ßDG; ZEA 4-β-D-glucopyranoside; ZEA 2,4-O-β-diglucoside; ZEA14ßDGp; ZEA14S; palmitoyl ZEA	maize, wheat bran, grains, grain-based food (breakfast cereals, bread, bakery wares); and vegetable oils
	Deoxynivalenol (DON)	DON3G; DON, 3-β-D-glucopiranoside	wheat, maize, oats, barley, beer, breakfast cereals, and snacks
		DON 3-acetyl; DON 15-acetyl	corn, wheat, and rye grain,
		DON glutathione (DON-GSH); sulpho-conjugates	wheat and oats
	Nivalenol (NIV)	NIV3G; NIV 3-acetyldeoxy; NIV 15-acetyldeoxy; NIV 4-acetyl; NIV 4,7-dideoxy	wheat and corn
	Fumonisin (FB)	HFB1, N-acyl-HFB1	corn
	Trichothecenes	T-2-3α-glucoside; HT-2-3-glucoside; palmitoyl tricotecolone	wheat and oats
		NEO-G; DAS-G	maize
Aspergillus/Penicillium	Ochratoxin A	(4R)- and (4S)-4-hydroxy-OTA; and β-glucosides	tomato, potato, maize, carrots, wheat, soybean, and paprika

ZEA-16-β-D-glucopyranoside (ZEA16G), a novel modified ZEA metabolite, together with α- and β-ZEL14G conjugates (20–100% of free ZEA), were found in bread and breakfast cereals [7][38][39][40].

Animal feed naturally contaminated with ZEA showed the presence of the sulpho-conjugate ZEA14S with unique trends of mycotoxin occurrence within cultivars and local weather [41]. Other modified mycotoxins conjugated with single-sugars such as di-, tri-, and tetra-glucosides, mixed disaccharides, and malonyl-glucosides have been characterized for DON, T2, HT2, and ZEA [7][16][19][42].

DON3G has been found in wheat, maize, oats, and barley, and the resultant beer, breakfast cereals, and snacks, at relative molar proportions of 20–70% of free DON and at a concentration ranging from 2–1700 mg/kg in naturally contaminated wheat [8][43][44][45][46]. Fusarium head blight-resistant wheat produced uncharacterized products from 14C-labelled DON more effectively than susceptible wheat cultivars [47]. A survey from the Czech market showed a higher presence of DON3G than DON [48]. Similar results were found in Belgium [43].

Moreover, DON glutathione (DON-GSH) and sulpho-conjugates were found in in vitro trials on wheat and oats [20][40][41][48][49]. In addition, DON–glutathione and its processing products, DON-S-cysteine, DON-S-cysteinyl–glycine, and DON-malonylglucoside, were found [48]. *Fusarium graminearum*-inoculated or DON-treated wheat showed the presence of DON-3-sulphate and DON-15-sulphate [49]. Afterwards, other biotransformation products such as DON-hexitol were characterised (e.g., mannitol), DON-di-hexoside (e.g., glucose), 15-acetyl-DON-3-β-D-glucoside, and a DON–glutathione derivative missing two protons [22].

Fumonisin protein conjugates have been detected in maize foods; however, conjugates with starch, pectin, hemicellulose, cellulose, and lignin could be theorised, even if their exact composition is still unknown [50].

NIV3G (12–27% of NIV) and fusarenon-X-glucoside (FUSX-3-G) have been reported in wheat [18], as have T2Glc and HT2Glc in wheat and oats [51].

Trichothecenes glucosides type A neosolaniol-glucoside (NEO-G) and diacetoxyscirpenol-glucoside (DAS-G) were detected in maize powder [19].

(4R)- and (4S)-4-hydroxy-OTA and their β-glucosides were found in vegetables contaminated with OTA [14].

The limited data indicate that modified mycotoxins make up a significant fraction of the overall burden of mycotoxin contamination in foods and feeds, particularly concerning cereals' fusariotoxins.

3. Impact of Environmental Conditions on the Development of Modified Mycotoxins

Environmental conditions affect the development of mycotoxins and mycotoxicosis. Environmental changes are related to crop production, seasonality, and climate modifications. Fungi can modify their behavior to adapt to different situations. In particular, other environmental conditions could lead to a different susceptibility of crops to fungi contamination; stressed maize and figs showed more *A. flavus* contamination. Moreover, climate change could affect the ability of fungi to contaminate foods.

The increased global air and water temperatures and the melting of natural snow and ice stocks will affect the food supply chain and possibly create a more comfortable environment for fungal development. *Fusarium* prefers temperate weather ranging from 26–28 °C and water activity (aw) > 0.88, whereas *Aspergillus flavus* needs warm temperatures.

Facing climate changes, humans will have to deal with increased atmosperic CO_2, increased rainfall, desertification, and sudden changes in temperature and humidity, which will affect crop resistance and mycotoxin-producing fungi behavior. The increase in fungi pollution at temperatures and relative humidity ideal for mycotoxin production could increase parent and masked mycotoxins, setting a severe alarm on food contamination and human safety.

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