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Aflatoxin Contamination in Animal-Derived Foods and Health Risks

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Abstract

Aflatoxins (AFs) B_1, B_2, G_1, and G_2 are important hepatotoxic mycotoxins produced by *Aspergillus flavus, A. parasiticus,* and *A. nominus.* They are converted into metabolites of AFM_1, AFM_2, B_{2a}, and aflatoxicol by cytochrome P450-related enzymes in the liver after digestion of the feed. These metabolites accumulating in the animal-derived food products such as eggs, milk, cheese, and honey cannot be destroyed by pasteurization or heating process and may influence public health negatively. Therefore, it is very important to prevent or limit the aflatoxin contamination in the animal feeds to decrease the risk of contamination of these metabolites in animal-derived foods.

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Introduction

Aflatoxins are toxic secondary metabolites produced by fungi of the genus Aspergillus, particularly *A. flavus*, *A. parasiticus*, and *A. nomius* [1]. The name "aflatoxin" was derived from the combination of "a" for the Aspergillus genus and "fla" for the species flavus and toxin meaning poison [2]. The aflatoxin problem was first recognized in 1960, when there was a severe outbreak of a disease referred to as "Turkey 'X' Disease" in the United Kingdom where more than 100,000 turkey poults and farm animals died. The cause of the disease was reported to be attributed to Brazilian peanut meal infected with A. *flavus* [3,4]. The major aflatoxins are characterized as AFB_1, AFB_2, AFG_1, and AFG_2 (based on their fluorescence under UV light, blue or green) and related chromatographic mobility during thin-layer chromatography [5]. Fungal species belonging to *A. flavus* typically produce AFB_1 and AFB_2, whereas *A. parasiticus* produces AFG_1 and AFG_2 as well as AFB_1 and AFB_2 (Figure 1).

AFB_1, the most prevalent toxin in feeds, represents the greatest toxigenic and carcinogenic threat for animals and humans [7,8]. It was reported that the toxic effects of AFB_1 were both dose and time-dependent [9]. The total aflatoxin content can be estimated from AFB_1 due to a higher correlation between AFB_1 and total aflatoxin contents [10]. AFB_1 is biotransformed by cytochrome P450-associated enzymes that generate hydroxylated metabolites such as AFM_1 and AFB_{2a} in the liver [11]. Aflatoxicol (AFL) can be formed by the reduction of AFB_1 by an NADPH-dependent cytoplasmic enzyme present in the soluble fraction of liver homogenates [12].

Aflatoxin Deposition and Clearance from Animal Tissues

Feeds contaminated with AFs were shown to result in the accumulation of the metabolites in the animal tissues including liver, adipose tissues, and animal products such as milk, meat, and eggs [13,14,15]. Those metabolites may cause potential health risks in the people because they can be carried over into the animal products. After AFs were recognized in the 1960s, the Food Drug Administration (FDA) of the USA set an action level of 30 ppb of AFs in raw or finished products [16]. In 1969, the FDA revised the action level for AFs to 20 ppb for food and feed ingredients [16]. The FDA set an action level of 0.5 ppb of AFM_1 in milk [16]. It was reported that only about 1-3% of the AFB_1 might be converted into AFM_1 of the milk [17].

Previously, feeding diet supplemented with AF was reported to result in the highest level of AF in the gizzard, kidney, and liver tissues [13]. Feeding a diet including 2500 ppb AFB_1 for 28 days was shown to cause 4.13 ppb AFB_1 deposition in the laying hens' liver [18]. It was shown that the levels of AFB_1 in the liver and kidney of chickens were significantly higher than the levels in the eggs and breast meat [19]. Residues of AFB_1 were detected in the eggs of hens fed supplemental 500µg per kg feed, at levels that ranged from 0.05 to 0.16 µg/kg [20]. Laying chicken fed diets contaminated with AFB_1 (3300 mg/kg) for 28 days was shown to produce eggs contaminated with AFB_1 [13]. Also, no aflatoxin residues were recovered from whole eggs after feeding laying chickens with aflatoxin-free diet (i.e. control diet) [13]. AFM_1, a metabolite of AFB_1, was reported to present in the eggs of laying hens fed AFB_1 contaminated feed [21]. Also, it was shown that AFM_1 and AFM_2 might be recovered in the poultry litter [22]. A study was conducted in laying hens to evaluate the effect of AFB_1 on the egg quality in laying hens fed diet supplemented with mannan-oligosaccharides (MOS) and showed that neither AFB_1 nor AFM_1 residues were found in the eggs of groups [18]. The same study also demonstrated that hepatic levels of AFB_1 were significantly lower in the group fed MOS-supplemented diet compared to the group fed MOS-excluded diet [18]. It was suggested that MOS could have an ability to adsorb and degrade AFB_1, reducing gastrointestinal absorption of AFB_1 and its levels in tissues of laying hens. In another study, synthetic zeolite was shown to have efficacy to counteract some of the toxic effects of AFs in broiler chicks [23].

Compared to the chickens, dairy cows are less sensitive to AFs due to biodegradation by rumen microorganisms [24]. In the liver, AFB_1 and AFB_2 are metabolized into AFM_1 and AFM_2, less toxic metabolites, using cytochrome P-450 associated enzymes [15,17,25]. AFM_1 in the contaminated feedstuffs may be transferred into milk as AFM_1 in the range of 0.3-6.3% [26].

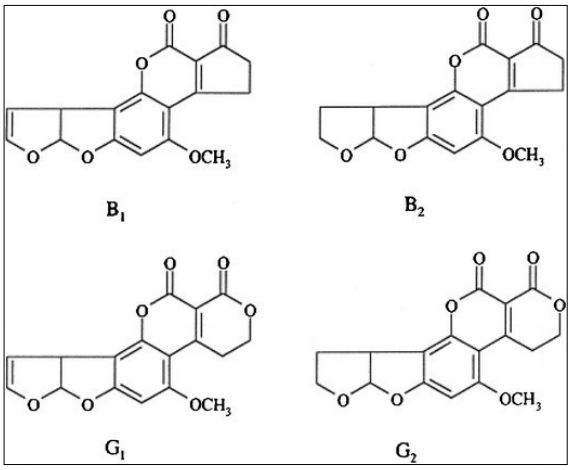

Figure 1. General structures of major aflatoxins AFB_1, AFB_2, AFG_1, and AFG_2 [6].

Table 1. International legislation on AFM_1 in milk and dairy products for human consumption [36].

Country/region	Raw milk (µg/kg)	Dairy products (µg/kg)
Argentina	0.05	0.50 (milk products)
Austria	0.05, 0.01 (pasteurized infant milk)	0.02 (butter), 0.25 (cheese), 0.4 (powdered milk)
Brazil		0.50 (liquid milk), 5.0 powdered milk
Bulgaria	0.50	0.10 (powdered milk)
Czech Republic	0.50	
Egypt	0	0
European Union	0.05	0.05
France	0.05, 0.03 (for children <3 years)	
Honduras	0.05	0.25 (cheese)
Nigeria	1	
Rumania	0	0
Switzerland	0.05	0.025 (milk whey and products), 0.25 (cheese), 0.02 (butter)
Turkey	0.05	0.25 (cheese)
US		0.50 (liquid milk), 5.0 (powdered milk)

AFM_1 is very commonly detected in milk and dairy products [27,28] and concentration in the milk was shown to increase linearly depending on the level of the AFB_1 in the feed [29]. AFB_1 levels of 20% and 13.6% of the yogurt and ayran samples were found to be exceeded the maximum tolerable limit of the Turkish Food Codex [30]. Therefore, nursing animals may be affected as a result of having milk contaminated with the toxin. Those metabolites of the AFs were reported not to be destroyed during pasteurization and thermal processing [31]. A recent study showed that 36.4% of colostrum samples were found to be contaminated with an above maximum allowable level of AFB_1 [32]. Studies showed that milk including a significant level of AFM_1 may have potential risks especially for infants and children [33]. AFM_1 concentration in the milk was reported to decline to an undetectable level after 72 hours when the intake of AFB_1 is stopped [34]. Lactating cows fed a ration including 20 ppb or more AFB_1 was reported to produce milk that exceeds the tolerance level of the toxin in the milk.

Special attention should be paid in food for infants and young children, where more restrictive levels have been regulated. Thus, limits as low as 0.1 µg kg^{-1} of AFB_1 are set for baby foods and processed cereal-based foods for infants and young children and 0.025 µg kg^{-1} for AFM_1 and 0.5 µg kg^{-1} for OTA [35]. International legislation on AFM_1 in milk and dairy products for human consumption is shown in Table 1.

Foods Contaminated with Aflatoxins and Health Risks

Aflatoxins are the hepatotoxic compounds causing health risks in the people consuming them more than the allowable amounts in the foods. As in the animals, these compounds or their metabolites may easily accumulate in the liver, kidney, and adipose tissues. It was reported that AFB_1, the most hepatocarcinogenic compound, caused cancer mainly in the liver and other organs of animals and humans [37]. After maternal exposure of AFs during pregnancy, AFB_1, AFB_1-metabolites, and AFB_1-albumen adducts were detected in cord blood of babies [38]. In a study conducted in Gambian children, it was reported that there was a relationship between impaired growth, particularly stunting and exposure to AFs [39,40]. The research suggested that ethnicity, dietary practice and socio-economic status of the individuals might influence AF-exposure significantly [41].

Attempts have been made to develop methods to remove AFs from contaminated feeds or foods by physical, chemical, and biological methods [42]. It was reported that implementing advanced agricultural technologies, good agricultural, and storage practices could mitigate the mycotoxin contaminations in the products [43]. Microwave heating, treatments with ozone, or ammonia were reported to be some of the methods used for detoxification of AFs in the foods [44,45,46]. Previously, it was shown that ozone treatment could significantly reduce the level of AFs in the red pepper [47]. Recently, it was shown that AFB_1 could be removed by ozone treatment [48]. However, the application of ozone treatment for the degradation AFs was reported to have limitations in food products because of the cost factor [49].

Conclusions

Chronic intake of AF-contaminated foods is a common problem especially in people of the developing countries. Contamination of crops with AFs in the field or storage may be controlled by implementing good agricultural and storage conditions. Also, identifying exposure of unacceptable AF levels in the feeds with reliable methods will decrease the exposure of AFs in the animals. Hence, minimizing exposure of domestic animals to moldy feed and taking precautions to prevent possible fungal growth in the products during the storage level will decrease AFs exposure in humans.

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