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## TRANSFER OF MYCOTOXINS IN THE FOOD CHAIN

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### Abstract

Mould infection of the feed plants may produce mycotoxins in adequate environmental condition. Those mycotoxins cause health risk for farm animals and those have food safety risk concern for human consumers with animal origin foods. Occurrence of mycotoxins in animal origin foods is different because the different rate of metabolism and accumulation of mycotoxins in farm animals and their edible tissues.

Among the important mycotoxins in feed plants and consequently in animal origin foods the highest danger is the presence of aflatoxin B1 and its hydroxylated metaolite, excreted with milk or egg, aflatoxin M1, because the high carry-over rate. Mycotoxins of *Fusarium* moulds, such as deoxynivalenol, T-2 and HT-2 toxin, and zearalenone, has lower human food safety risk through animal origin foods, because of their low carry-over and accumulation rate. In contrary, another fusariotoxin, fumonisin, may arise higher food safety risk, because its absorption rate is low, but accumulate effectively in edible tissues for a long period of time, therefore fumonisins can be found in animal origin foods. Similarly, feeding farm animals with ochratoxin A contaminated diet a higher rate of accumulation can be found in some edible tissues of farm animals.

**Keywords:** mycotoxin, food chain, animal origin foods

### Mikotoxinok átvitele az élelmiszerláncban

#### Összefoglalás

A takarmánynövényeket fertőző penészek megfelelő körülmények között mikotoxinokat is termelnek, amelyek az állatok számára állategészségügyi kockázatot, míg az állati terméket fogyasztók számára élelmiszerbiztonsági veszélyt jelentenek. Az egyes mikotoxinok állati eredetű élelmiszerekben való megjelenése eltérő mértékű, amelynek oka részben az állati szervezetben való eltérő mértékű metabolizmusuk, valamint az állati szövetekben való tárolódásuk mértéke.

A fontosabb takarmánynövényekben és az állati eredetű élelmiszerekben előforduló mikotoxinok közül az aflatoxin B1 és annak hidroxilált származéka, a tejjel és a tojással kiválasztódó aflatoxin M1, jelentős élelmiszerbiztonsági kockázatot hordoz, mert az átvitel mértéke jelentős. A *Fusarium* penészek által termelt mikotoxinok, így a deoxinivalenol, a T2- és HT-2 toxin, valamint a zearalenon csak alacsony kockázatú, mert az állati eredetű élelmiszerekben nem, vagy csak kismértékben jelennek meg. A fumonizinek ugyanakkor jelentősebb kockázatot hordoznak, mert felszívódásuk mértéke ugyan kicsi, de az szervezetben hosszú időn keresztül tárolódnak, így az állati eredetű élelmiszerekben is kimutathatók. Hasonlóképpen jelentős élelmiszerbiztonsági kockázata van az ochratoxin A-val szennyezett takarmány etetésének is, mert ez is jelentős mértékben akkumulálódik egyes állati eredetű élelmiszerekben.

**Kulcsszavak:** mikotoxin, élelmiszerlánc, állati eredetű élelmiszerek



## Introduction

Mould infections and their secondary metabolites, mycotoxins, are constant concern for agriculture. Mycotoxin production is greatly influenced by weather conditions including droughts, rainfall and temperature changes (Paterson and Lima, 2011). Mycotoxin contamination occurs both during crop development and after crop maturation in cereals, but it also occurs in green fodder (Parsons and Munkvold, 2010). Cereal grains and green fodder use for nutrition of farm animals, but cereals also can use as foods for humans. There are some novel data that plant-based dietary supplement, such as medicinal herbs, also can be contaminated with mycotoxins (Veprikova et al., 2015). Intake of mycotoxin contaminated feed and food can cause health problems in farm animals (Diaz, 2005), and also human consumers (Peraica et al., 1999), therefore presence of mycotoxins in the food chain has human health implications (Bryden, 2007).

Mycotoxins are undesirable substances in feed and food commodities, but zero tolerance would be ideal because complete prevention and elimination of mycotoxins from the whole food chain is impossible (Galvano et al., 2005). Carryover of mycotoxins from plant-origin feeding stuffs to animal products, such as edible tissues, milk and egg depend on the rate of absorption, metabolism, accumulation and excretion in different farm animal species, therefore it has marked differences.

The purpose of present paper was to review the differences in the carryover rate of some frequently occurred mycotoxins, which also arise food-borne diseases in humans from feed to animal-origin foods.

### Aflatoxins

One of the most important mycotoxins is aflatoxins because of their carcinogenic activity in humans (Robens and Richard, 1992). Aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) is the most frequently found metabolite in *Aspergillus* infected plants, and its hydroxylated metabolite, aflatoxin M<sub>1</sub> (AFM<sub>1</sub>) secreted into milk and egg.

The carryover rate of AFB<sub>1</sub> in the form of AFM<sub>1</sub> is 0.3 to 6% in milk, depends on the rate of production (Galvano et al., 2005). It is important to mention from food safety point of view that AFM<sub>1</sub> remains stable during processing of milk into cheese, yogurt, cream and butter (Galvano et al., 1996). Additionally, AFM<sub>1</sub> bound to casein; therefore cheese contains higher concentration than whey. The rate of accumulation is 2.5 to 3.3 fold in soft cheese and 4.9 to 5.8 fold in hard cheese as compared to milk (Yousef and Marth, 1989).

Aflatoxin B<sub>1</sub> accumulates in the genitals of chickens, turkeys and ducks, resulting in a transfer to the egg (albumen and yolk). In regards to the carry-over to eggs, Oliveira et al. (2000) reported that feed to egg transmission of AFB<sub>1</sub> is about 5000:1.

Trucskess et al. (1983) detected aflatoxin B<sub>1</sub> and M<sub>1</sub> residues in eggs of hens fed contaminated feed. After 7 days of withdrawal only trace amounts remained in eggs. According to Wolzak et al. (1985) clearance of aflatoxin occurs faster from the albumen than from the yolk. Other studies reported that low levels of AFB<sub>1</sub> in the diet (25, 50 or 100 µg/kg) did not result measurable accumulation of AFB<sub>1</sub> or AFM<sub>1</sub> in eggs during a 60-days period (Salwa and Anwer (2009) and also not even at much higher contamination level (2.50 mg AFB<sub>1</sub>/kg), as reported by Zaghini et al., 2005).



### **Deoxynivalenol**

Deoxynivalenol (DON) is a trichothecene mycotoxin produced by *Fusarium* moulds. DON remains stable both during storage milling and the processing or cooking food and do not degrade at high temperatures (Eriksen and Alexander, 1998).

Elimination of DON is a rapid process because of biotransformation in the gastrointestinal tract, in particular ruminants, where almost 100% de-epoxydated in the rumen fluid (King *et al.*, 1984). However, free and conjugated DON was present in cow's milk, but only extremely low amounts (<4 ng/ml) were detected (Prelusky *et al.*, 1984).

Maximum tissue residues of radiolabel DON were measured at 3 hr in all tissues of laying hens and clearance of radioactivity from tissue had an average half-life of 16.83 +/- 8.2 hr. Maximum residual levels occurred in the kidneys, but it was only 60 ng DON equivalents/g (Prelusky *et al.*, 1986). Following a single oral administration of <sup>14</sup>C-DON (2.2 mg) to laying hens, only 0.087 % of total was detected in the first egg. In another study with 5.5 mg radiolabel DON/kg fed to six laying hens for 65 days (Prelusky *et al.*, 1989), radioactivity in eggs increased to a maximum level of 1.7 µg DON equivalent/egg at the 8<sup>th</sup> day of exposure and quickly dropped to negligible values when the exposure to DON ceased (Prelusky *et al.*, 1989).

The pharmacokinetics of DON swine following intragastric (0.60 mg) administration of the <sup>14</sup>C-labeled toxin showed that DON was eliminated rapidly and completely within 24 hr following a single intragastric dose, (Prelusky *et al.*, 1988). These data suggested that DON intake of farm animals do not cause measurable food safety risk to consumers.

### **T-2 toxin**

T-2 toxin is the most toxic trichothecene, which is produced by *Fusarium* moulds. T-2 toxin and its metabolites, such as HT-2 toxin, are stable compounds, both during storage milling and the processing or cooking food and do not degrade at high temperatures (Eriksen and Alexander, 1998). They are also stable at neutral and acidic pH and consequently not hydrolysed in the stomach after ingestion (Ueno, 1987).

T-2 toxin is rapidly eliminated in the faeces and urine. For example about 100% of an oral dose of T-2 toxin in cattle was eliminated within 48 hours after dosing (Feinberg and McLaughlin, 1989), therefore those are not accumulate in cattle meat or practically never occurs in milk and milk products.

The carryover of T-2 toxin to eggs was observed by Chi *et al.* (1978) in birds fed 0.25 mg radiolabel T-2 toxin/ kg bodyweight. Maximum residues in the eggs occurred 24 hours after dosing, the yolk contained 0.04% of the total dose and the white contained 0.13%.

There are no data available about carryover of T-2 or HT-2 toxin in meat or other edible tissues of farm animals, but it seems that rapid elimination resulted negligible food safety risk after intake of T-2 contaminated feed by food producing animals.

### **Zearalenone**

Zearalenone is a *Fusarium* mycotoxin which is transformed rapidly after absorption to some bioactive,  $\alpha$ -zearalenol and  $\beta$ -zearalenol, and other inactive metabolites (Malekinejad *et al.*, 2006). Otherwise excretion of zearalenone and its metabolites is also a rapid process, therefore its accumulation in meat and excretion to eggs or milk is low (EFSA, 2004).

Milk levels of zearalenone and its bioactive metabolites were determined after feeding lactating cows with zearalenone contaminated diet. It was found that zearalenone or its metabolites was found in milk only at extremely high doses of zearalenone, which means that



milk would not normally pose a human health hazard as a result of feeding rations containing ZEN to lactating dairy cows (Prelusky *et al.*, 1990). The estimated carryover rate of zearalenone to milk is 0.06% (Jouany and Diaz, 2005).

### **Fumonisin**

Presence of fumonisin B<sub>1</sub> (FB<sub>1</sub>), a major metabolite of *Fusarium moniliforme*, in corn is of great concern to both human and animal health because of its wide range of toxicity. After dietary intake of FB<sub>1</sub> only a low percentage absorb from the intestine and FB<sub>1</sub> do not metabolised in the liver (Cawood *et al.*, 1994). FB<sub>1</sub> was distributed to most tissues, the liver and kidney retained most of the absorbed material, the liver retaining more toxin than the kidney (Martinez-Larranaga *et al.*, 1999).

Investigation of milk and beef for contamination by fumonisins failed to raise any concern, because residues over the detection limit was found only after intake of extremely contaminated feed (Richard *et al.*, 1996; Smith and Thakur, 1996).

Pigs fed diets containing FB<sub>1</sub> at 2–3 mg/kg also did not show accumulation of fumonisin residues in muscle, although residues did accumulate in kidneys and liver but with extreme low carryover rate (Prelusky *et al.*, 1994).

The pharmacokinetics of FB<sub>1</sub> in laying hens showed that after intake of 2.0 mg FB<sub>1</sub>/kg bodyweight at 24 hr post-dosing showed only trace amounts in crop, liver, kidney, small intestine, and cecum, and no residues were found in eggs laid during the 24 hr post-dosing period (Vudathala *et al.*, 1994).

The low carryover rate in different animal-origin foods suggests that these low residue levels do not contribute substantially to human exposure.

### **Ochratoxin**

Ochratoxin A is produced by *Penicillium* or *Aspergillus* species. Processing of plant-origin foods decrease measurable ochratoxin A content. For instance Osborne *et al.* (1996) found that milling hard wheat to produce white flour resulted in an approximately 65% reduction, and a further 10% decrease occurred during baking.

In farm animals after intake the ochratoxin A contaminated feed it is hydrolysed to the non-toxic ochratoxin- $\alpha$  at first by gut bacteria and later in liver (Galtier, 1978).

The tissue distribution of ochratoxin A in pigs, chickens, and goats followed the order kidney > liver > muscle > fat (Harwig *et al.*, 1983).

Ochratoxin A occurrence in milk is very low, and according to some estimation eggs may contain 0.11 % of the toxin concentration present in the feed (Galvano *et al.*, 2005).

Ochratoxin A accumulation in pork is the highest among the animal-origin foods. Its carryover rate is about 3% of total intake (Verger *et al.*, 1999).

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