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MYCOTOXINS IN GRAINS AND FEED – CONTAMINATION AND TOXIC EFFECT IN ANIMALS[1]

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Review paper

Abstract: Mycotoxins are secondary metabolites of moulds that represent a significant food safety issue and pose a risk to health and wellbeing of humans and animals, having a negative impact on economies all over the world. They can cause intoxication in animals and affect the less productive performance and nutritional value of animal feed. Also, mycotoxins may contaminate different types of food through directly and indirectly way and cause negative health effects in humans. To estimate the potential of their harmful effect it is necessary to know the terms of mycotoxins occurrence and frequency, toxicity and biotransformation in different animal species and humans, possibility of contamination prevention, and their inactivation or reduction in the case when the incidence is inevitable. The relatively small number of comparable data on the incidence of mycotoxins in feed from European countries show the necessity of systematic control and the application of validated analytical methods in their determination. This paper gives the overview of incidence of grains and feed contamination with most important and common mycotoxins and the evidenced toxic effects in animals.

Keywords: mycotoxins, grains, feed, contamination, mycotoxicoses, Europe

Introduction

Grains that are highly represented in human and animal diet, as also in industrial food and feed, may become contaminated by moulds that produce mycotoxins. So far, about 400 different mycotoxins were described as produced mostly by moulds from genera of *Aspergillus*, *Penicillium*, *Fusarium* and *Trichotecium*, among which a smaller number is associated with the occurrence of acute or chronic intoxications. It is very difficult to unique systematize them,

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because of their different chemical structure, biochemical pathway, origin and biological effects but most important are aflatoxin B_1, ochratoxin A, zearalenone, deoxynivalenol, fumonisin B_1 and T-2 toxin *(CAST, 2003; Bryden, 2012)*.

Grains such as maize, wheat, barley and oat, that balancing the nutrition by virtue of providing a low-fat diet, has a number of advantages. At the same time, grains are a common source of mycotoxins which under favourable conditions may produce mycotoxins before and/or during harvest, handling, shipment and storage. Conditions favourable to mould growth and mycotoxin production, such as extreme weather conditions (temperature and humidity), grain damage, presence of insects and poor storage of grain and feed, cause the inevitable occurrence of mycotoxins in a large number of samples and intoxications in larger scale. The Food and Agriculture Organization (FAO) estimated that 25% of the world crops are contaminated with mycotoxins as generally stable substances resistant to the different methods of food processing *(Pleadin et al., 2014a)*.

Contaminated feed represents the main source of mycotoxin infestation of farm animals, which get to be contaminated through parasites living on plants even prior to harvesting or on stored harvested crops *(Gareis and Wolff, 2000)*. As fodder, cereals and seeds used for feeding are inevitably in contact with yeasts and filamentous fungi, and contamination of these raw materials frequently occurs already in the field. Contamination can also occur during harvesting, transport and storage of cereals and their products, as well as due to post-harvest mishandling that can lead to rapid feed spoilage. Due to possible occurrence of mycotoxins in natural grains that are used in the production of food and feed, there is a possibility of their entrance into human food chain. Literature data show that products of animal origin, such as meat and meat products, may also contribute to the entry of mycotoxins in food, whether as a result of indirect transmission from domestic animals, which are used for the production of food of animal origin, exposed to contaminated feed materials and compounds (carryover effect), but also through the mixture of spices used in their production *(Pleadin et al., 2013; Perši et al., 2014)* or direct contamination with moulds which under certain conditions can produce mycotoxins *(Gareis and Wolff, 2000; Pleadin et al., 2015a,b)*.

This paper gives an overview of the mycotoxin contamination of grains and feed and observed toxic effects with special focus in animals for those that are considered as most important and common occurred in the European countries in the last decades.

Toxic effects and natural occurrence

Mycotoxicosis, the disease resulting from exposure to a mycotoxin, may be manifested as acute to chronic, and ranges from rapid death to tumor formation. More occult disease may occur when the mycotoxin interferes with immune

processes, rendering the patient more susceptible to infectious diseases. The general effects of mycotoxins on health and productivity of animals are dependent on dose, and generally the young of a species is more susceptible to the effects of mycotoxins than adults are *(CAST, 2003)*. Ruminants, such as cattle and sheep, are generally more resistant to mycotoxins than most animals, especially pigs, as ruminal microbial population plays a role in detoxification process. This assumption is based on the finding that rumen flora is able to convert a number of mycotoxins into metabolites that are less potent or even biologically inactive at common exposure levels *(Kiessling et al, 1984)*. In animals intended for meat production, which had consumed contaminated feed, the ingestion of mycotoxins leads to substantial degradation of meat quality *(Bonomi et al, 1994)*.

Accumulation of mycotoxins before and after grains harvesting largely reflects actual climate conditions. *Fusarium* toxins are known to be produced during cereal harvesting under high moisture conditions, whereas pre-harvest aflatoxin contamination of crops is associated with high temperatures, insect-mediated damage and prolonged drought. Chronic contamination occurs in warm, humid, tropical, and subtropical growing environments. The degree of moisture mostly depends on the water content available at the harvesting point, but also on the frequency and extensiveness of drying, aerating, and turning of the grain before and during storage, and the respiration of insects and microorganisms harbored by the stored grain *(Bryden, 2012)*. Since *Aspergillus* can tolerate lesser water activity than *Fusarium*, these contaminations may occur both pre- and post-harvesting, whereas *Fusarium* contamination is more specific for the pre-harvesting period. Stored cereals may become infested with fungi and insects; such an infestation is also affected by climatic factors such as temperature and humidity, geographical location, type of storage container, and handling and transport procedures *(Chelkowski, 1991; Krnjaja et al., 2013)*.

Aflatoxins. Among food and feed contaminants, aflatoxins are of current concern. They are known to be produced by two species of *Aspergillus* genus, specifically *Aspergillus flavus* and *Aspergillus parasiticus*, and represent highly toxic, mutagenic, teratogenic and carcinogenic compounds that exhibit an immunosuppressive activity, causing both acute and chronic toxicity in humans and animals *(EFSA, 2004)*. Among them, aflatoxin B_1 (AFB_1) is the most potent liver carcinogen known in mammals, and is classified by the *International Agency for Research on Cancer (IARC)* as Group 1 carcinogen (*IARC, 1993*).

Animals are variably susceptible to aflatoxins, depending on such factors as age, species, breed, sex, nutrition, and certain stresses. Pig, cattle, and poultry are farm animals of greatest economic concern in terms of aflatoxicosis. In all species, the evidence of disease is a general unthriftiness and reduction in weight gains, feed efficiency, immunity, and production. More conclusive evidence of aflatoxin involvement in disease includes acute to chronic liver disease with concomitant increases in specific liver enzymes in the serum. AFB_1 can cause liver

dysfunction, reduced milk production and egg production and to reduced immunity of animals. Long-term consumption of containing low concentrations of AFB_1 in feed can also result in embryo toxicity. Usually, young animals are more sensitive to aflatoxin. The clinical manifestations involve digestive disorders, reduced fertility, reduced feed efficiency and anemia. Aflatoxins not only the decline in milk production, but also the transformation of the milk containing aflatoxin M_1 and M_2 (*Dhanasekaran et al., 2011*).

Maize, as the most widely grown crop extensively used for animal feeding and human consumption, represents a particular problem. Due to its nutritional value, a high percentage of the world maize production is destined to animal feeding (*Pleadin et al., 2014b*). Earlier extensive research of commodities, feedstuffs and feed ingredients revealed the maximal AFB_1 levels in samples coming from the Northern Europe to be 60 μg/kg, in samples coming from the Central Europe to be 311 μg/kg, and in samples coming from the Southern Europe and the Mediterranean region to be 656 μg/kg (*Binder et al., 2007*). In this southeast part of Europe, in the last few years, the research performed in Serbia during 2012 also pointed towards maize contamination with AFB_1 and concluded that weather changes might be held liable for such a contamination (*Kos et al., 2013*). Data have shown that, should a grain such as maize be grown at high ambient temperature, especially during drought, such a grain becomes more susceptible to AFB_1 formation. Grains stored under high moisture/humidity (>14%) conditions and at high temperatures (>20 ºC) and/or inadequately dried, can potentially become contaminated. Grains have to be kept dry, free of damage and free of insects (*Richard, 2007*).

Zinedine et al. (2007) reported that the percentage of contamination with aflatoxins is about 66.6%, while the contamination levels of poultry feed samples ranged between 0.05 and 5.38 μg/kg for AFB_1. In study by *Pleadin et al. (2012a)* the determined mean AFB_1 concentrations were 3.22±2.21 μg/kg in poultry feed, 2.32±1.26 μg/kg in pig feed and 3.45±1.42 μg/kg in feed for calves. Further, *Pleadin et al. (2014c)* provided the evidence of high AFB_1 contamination of maize used by Croatian milk producers. In the maize sampled during 2013 AFB_1 was detected in 38.1% of samples, with 28.8% of the samples containing this toxin in levels higher than the maximal permitted levels (MPLs) and maximal observed AFB_1 level of 2,072 μg/kg (about 100 folds higher than the MPL). High maize contamination was associated with weather conditions, as the period of proceeding was extremely warm, dry, and characterised by a very low average rainfall, all of the aforementioned going in favour of mould formation and AFB_1 presence in maize (*Kos et al., 2013; Pleadin et al., 2014c; 2015a*).

Ochratoxins. Ochratoxins, of which ochratoxin A (OTA) is the most prevalent, are secondary fungal metabolites of some toxigenic species of *Aspergillus* and *Penicillium* that can be found in various feed ingredients. Several studies have shown that OTA is nephrotoxic, causing both acute and chronic

lesions of kidneys, and that it is hepatotoxic, carcinogenic, teratogenic and immunotoxic to several animal species (*Pfohl-Leszkowicz and Manderville, 2007*). Immunosuppression occurs with low concentrations of OTA, while high concentrations lead to kidney toxicity. The mechanism of action of OTA until has not yet been fully clarified but it is known that OTA poses a risk for human and animal health when ingested through contaminated food or feed. The *International Agency for Research on Cancer (1993)* has classified OTA in group 2B as a possible carcinogen to humans.

Long-term exposure of farmed animals to OTA typically results in increased mortality, poor feed conversion, poor growth rates and feed refusal (*Marquardt and Frohlich, 1992*), among which pigs are particularly sensitive *(Malagutti et al., 2005)*. OTA is mainly distributed to the kidneys, with lower concentrations in the liver, muscle and fat, and its disappearance from blood is slower than from tissues (*Perši et al., 2014*). It was found to be immunosuppressive in humans and carcinogenic and teratogenic in laboratory animals and that the DNA lesions induced by OTA *in vivo* were no longer repaired in case of repeated exposure. These effects manifest especially in a dose-dependent and dose-time related fashion *(Marquardt and Frohlich, 1992)*. OTA has been primarily recognized as a nephrotoxic mycotoxin that induces significant changes in serum parameters after ingestion in different animal species (*Kumar et al., 2007)*. Investigations in various animal species showed changes in the values of blood parameters to be observed mostly within 1-2 months of OTA exposure, and to depend on the dose applied and length of treatment (*Marquardt and Frohlich, 1992; Mir and Dwivedi, 2010*).

Literature data revealed that in raw materials OTA was detected in 22-70% of the samples ranged from 4 to 2248 μg/kg (*Griessler et al., 2010; Almeida et al., 2011; Grajewski et al., 2012; Rodrigues and Naehrer, 2012; Streit et al., 2012*). In Italy, the OTA was detected in samples of poultry feed in the range 0.04 to 6.50 μg/kg (*Schiavone et al., 2008*) and in Spain it was detected in 33% of samples of food and raw materials (*Jaimez et al., 2004*). In Croatia, OTA was detected in 39% of samples of maize (*Domijan et al., 2005*), 8% of pig feed samples with mean concentration of 1.53±0.42 μg/kg (*Pleadin et al., 2012b*) and also in low levels, all according to the recommended values, were determined in poultry, pig and calves feed (*Pleadin et al., 2012a*).

Fusarium mycotoxins. As the consumption of *Fusarium*-contaminated products may cause mycotoxicosis and induce teratogenic, carcinogenic, neurotoxic, estrogenic or immune-suppressive effects, contamination of food, feed and their ingredients can significantly affect human and animal health (*IARC, 1993*). Mycotoxins of *Fusarium* species have been found to cause major damage, especially of grains, and could frequently be associated with pre-harvest cereal contamination (*Creppy, 2002*). Their presence has traditionally been associated with temperate cereals, since these fungi require somewhat lower growth and

mycotoxin production temperatures than aflatoxigenic *Aspergillus* species (*Placinta et al., 1999*). Major *Fusarium* mycotoxins that can occur in cereal grains and cereal-based products are zearalenone (maize, wheat), deoxynivalenol (occurring mainly in wheat, maize, barley, oat and rye), fumonisins (maize) and T-2/HT-2 toxins (oat, wheat, and barley). Their biosynthesis can be affected by a number of factors including temperature, humidity, oxygen level, mechanical cereal damage and the presence of mould spores. Level of contamination is linked to climate conditions and widely varies across different world climate zones (*Sforza et al., 2006*). Maize considered to be one of the most frequent crops often contaminated with *Fusarium* mycotoxins (*Pleadin et al, 2013; Krnjaja et al., 2013*).

Zearalenone. Zearalenone (ZEA) is a secondary metabolite of *Fusarium* moulds which is very often encountered at very high concentrations, especially in maize. It is produced primarily by *F. graminearum*, which occurs naturally in high-moisture maize and has been found in moldy hay and pelleted feeds. ZEA as an estrogenic mycotoxin causes vulvovaginitis and estrogenic responses in pigs. Physiological responses in pig occur when the ZEA level in feed maize exceeds about 1 mg/kg (*Kurtz and Mirocha, 1978*). It can be transmitted to piglets in sows' milk, causing estrogenism in the young pigs.

In farm animals, especially pigs, ZEA causes hyperestrogenism causing severe reproductive and infertility issues. Female pigs are considered to be the most sensitive animal species, while poultry and ruminants show a lower responsiveness to ZEA. Its biotransformation runs along two major pathways, as follows: upon hydroxylation, phase-I metabolites α- and β-zearalenol are formed, while the conjugation of ZEA and its reduced forms with glucuronic acid and sulphate leads to the formation of typical phase-II conjugation products (*Zinedine et al., 2007*). The ratios of ZEA over its metabolites and the susceptibility to ZEA considerably vary across animal species (*Songsermsakul et al., 2006*). As ZEA ingestion by livestock may cause losses in terms of poor performance and poor animal health, and given that this mycotoxin, together with its metabolites, often occurs in plant and animal food products, its investigations are also of interest from the food safety standpoint. Studies of pharmacokinetics and metabolism indicate that following an oral administration ZEA is absorbed in a fairly large amount and can be metabolised in pig and possibly also human intestines (*Zinedine et al., 2007*).

It is widely distributed mycotoxin in different commodities and its production is also favoured by environmental conditions such as high humidity and low temperatures (10-15 °C). Data of the incidence of ZEA in grains and feed shown that it was detected in high percentage of samples (*Domijan et al., 2005; Almeida et al., 2011; Grajewski et al., 2012; Pleadin et al., 2012a; Pleadin et al., 2012c; Krnjaja et al., 2013*). Maize is the cereal at the highest risk of frequent and high-level ZEA contamination, while wheat, oat and soybean products have been

found to be contaminated only occasionally (*Zinedine et al., 2007; Placinta et al., 1999*). In raw maize ZEA was determined at the highest level of 6,492 μg/kg, reported for maize sampled in Italy (*EC, 2003*). In the study by *Binder et al. (2007)*, the highest concentration of ZEA in feed (2,348 μg/kg) was determined in the Southern Europe and the Mediterranean region.

Deoxynivalenol. Deoxynivalenol (DON) is primarily produced in cooler climates by *F. graminearum* and *F. culmorum*, and is an important contaminant of maize in many European countries (*IARC, 1993*). Epidemics of *F. graminearum* infection in crops can occur, when relatively warm temperatures and rain coincide with maize silk emergence (*CAST, 2003*), causing *Fusarium* head blight in wheat, and *Gibberella* or pink ear rot in maize. DON is the most common of this group causing animal disease and effects range from feed refusal and vomition to immunosuppression and loss of productivity. Pigs are considerably more sensitive to DON than poultry are, and cattle are quite insensitive (*Prelusky et al., 1994*).

Studies revealed that athough DON can be acutely lethal when ingested in large quantities, moderate- to low-level ingestion of the toxin can cause poor performance and altered immune function. Ruminants are relatively insensitive to DON because rumen microorganisms are able to metabolize/ detoxify this toxin whereas in pigs caused decreased body weight in animals and increase in serum IgA and IgM, cytokine tumor necrosis factor alpha and caused damage including necrosis, blood vessel thickening and hemorrhage (*Chen et al., 2008; Pinton et al., 2010*). At 1.3 mg/kg DON in diet, feed intake by growing pigs is significantly decreased, followed by complete feed refusal at 12 mg/kg and vomiting at 20 mg/kg. The most common signs of acute DON exposure are abdominal distress, increased salivation, and malaise; however, vomiting has been reported at higher dietary concentrations. Extensive lesions are not typically documented in field cases, because pigs regulate toxin ingestion by adjusting their feed intake (*Chavez and Rheaume 1986*).

In the report of experts on collection of occurrence data of *Fusarium* toxins in food and assessment of dietary intake by the population of EU Member States which included data on trichothecenes from twelve countries showed that 89% of maize samples were positive on DON (*EC, 2003*). In the report prepared by the *Joint Expert Committee on Food Additives (JECFA)* (*2001*), DON was detected in 41% samples with mean concentrations in the range 3-3,700 μg/kg. Similar data on the occurrence of these mycotoxins are also available from other European researchers. *Schothorst and van Egmond (2004)* reviewed the occurrence of *Fusarium* mycotoxins in 12 European countries and also found that the most common trichothecene mycotoxin in cereals is DON, with 57% of positive samples. Among cereals, maize showed the highest level of contamination. *Tanaka et al. (1990)* reported on contamination of 90% and 62% samples of cereals with DON with average concentrations for positive samples of 221 μg/kg. In Poland, extremely high values (up to 927 μg/kg) of DON were recorded for maize grain

(*Placinta et al., 1999*). *Binder et al. (2007)* published the results of research of this mycotoxin in Europe with regards to their geographical origin. In this research, DON was detected in 70% maize samples with the maximum level of 10.626 mg/kg in Asia and the Pacific region and 5,510 μg/kg in Europe and the Mediterranean. *Pleadin et al. (2012c)* concluded that due to the incidence of *F. graminearum*, high occurrence of ZEA in grains, and specificity of climate in this southeast part of Europe, it can be supposed that DON is often contaminant of grains and feed.

Fumonisins. Fumonisins have been found worldwide, primarily in maize, with more than 10 compounds that have been isolated and characterized. They are metabolites of moulds *Fusarium* (*F. Verticillioides*, *F. proliferatum*) and *Alternaria* and *Aspergillus niger*. Fumonisins B_1, B_2 and B_3 are the major fumonisins produced. The most prevalent is fumonisin B_1 (FB1), which is believed to be the most toxic, i.e. nephrotoxic and hepatotoxic (*Voss et al., 2007*). High concentrations of fumonisins are associated with hot and dry weather, followed by periods of high humidity. High concentrations may also occur in raw maize that has been damaged by insects.

Earlier investigations conducted on different cereals revealed high frequency of FB_1 cereals positive samples (*Domijan et al. 2005*). FB_1 is toxic and carcinogenic to rodents and there are data suggesting that fumonisins or *F. verticillioides* cause esophageal cancer or other human health problems. However, an *International Agency for Research on Cancer (IARC, 1993*) working group on fumonisins concluded that there is "inadequate evidence" for carcinogenicity in humans from oral exposure to FB_1 and a role for fumonisins in any other human disease has likewise not been proven.

Contamination of feed with FB_1 resulted in a diverse range of damage to animal tissues, including lesions to the esophagus, gastrointestinal tract, liver, lungs, and brain. In the animals hepatotoxic, nephrotoxic, neurotoxic and carcinogenic effects were observed. It is considered highly toxic to horses and pigs, while in poultry significantly higher levels generally not cause a change in the production characteristics. When the consumption of feed contaminated with large quantities of fumonisin were caused, slow growth and feed conversion, increased weight of liver, kidney and pancreas, increased levels of aspartate aminotransferase and droped leves of serum alkaline phosphatase, cholesterol and hemoglobin were observed. However, in the presence of other mycotoxins, toxic additive interactions are proven, resulting in poorer production results and the appearance of the above symptoms (*Leeson et al., 1995*).

Extremely high values of fumonisin recorded *Griessler et al. (2010)* in the analysis of samples originating from Portugal, Spain, Italy, Greece and Cyprus. Of the total of 416 samples tested, the average concentrations of fumonisin in the finished feed was 1,411 μg/kg, while in maize originating from Italy concentration

of 36,390 μg/kg was evidenced. Such high levels were not found in other grains such as barley and wheat (*Almeida et al., 2011; Rodrigues and Naehrer, 2012*).

T-2 and HT-2 toxins. T-2 and HT-2 toxins are type A of trichothecene mycotoxins produced by *F. poae, F. sporotrichioides, F. kyushuense* and *F. langsethiae* among which the major producer of T-2 and HT-2 toxins is *F. sporotrichioides* (*Richard, 2007; Creppy, 2002*). The presence of T-2/HT-2 toxin is described in the world, and toxin production is greatest in a wide temperature range (0 to 32 °C, the optimum at 5-15 °C), in the food with a moisture content of 13-22%, in hot and humid climates and in damaged grains. Due to the broad prevalence of this fungus, many different crops including maize, oat, barley, wheat, rice and soya beans can be infected with T-2 and HT-2 toxins.

T-2 toxin is a very potent cytotoxic and immunosuppressive toxin, which can cause acute intoxication and chronic diseases in both humans and animals. The symptoms of acute intoxication are nausea, vomiting, abdominal pain, diarrhea, bloody stools and weight loss. In animals, symptoms also include decreased production of milk or eggs, increased incidence of cracked eggs and oral lesions in poultry (*Morgavi and Riley, 2007*). The major effect of T-2 toxin is inhibition of protein synthesis, which leads to secondary disruption of DNA and RNA synthesis (*Richard, 2007; Creppy 2002*). The immune system is also a target of T-2 toxin, and the effect includes changes in leukocyte count, delayed hypersensitivity, depletion of selective blood cell progenitors, depressed antibody formation, allograft rejection and blastogenic response to lectins (*Creppy, 2002*). HT-2 toxin is a metabolite of T-2 toxin and is formed in microbial transformation *via* deacetylation reaction. This reaction is performed by several intestinal microorganisms in different animals (*Young et al., 2007*).

In Europe, there is very little research conducted on the occurrence of T-2 in feed, which bearing in mind the explicit toxicity of this mycotoxin needs further data collection and the legal values of the maximum allowed level need to be defined (*Vulić et al., 2011*). Earlier performed investigations of T-2 toxin by *Sokolović and Šimpraga (2006)*, over the period 1998 to 2004, determined the values of 100 to 500 μg/kg whereas in an investigation by *Vulić et al. (2011)* the highest concentrations of T-2 was determined in cattle feed (67.68 μg/kg). The highest concentration of T-2 toxin (1776 μg/kg) was determined in Northern Europe (*Binder et al., 2007*). In study by *Pleadin et al. (2012a)*, the lower mean concentration was determined in poultry feed (18.2±8.31 μg/kg) and the highest mean concentration in feed for calves (32.4±15.1 μg/kg).

Mycotoxins reduction

As the presence of moulds and/or mycotoxins in food can be dangerous for human health and represents a huge economic problem, there is a huge space for

the implementation of new methods providing for a safe food production. Methods of control can be classified into two categories: (1) prevention of mould contamination and growth, and (2) detoxification of contaminated products. The prevention of mould growth can be achieved either through pre- or post- harvesting strategies. The applied mycotoxins reduction procedure must effectively inactivate or remove the toxin, maintaining at the same time both nutritional and technological properties of the product and not generating reactive toxic products (*López-García and Park, 1998; Pleadin et al., 2014b*).

Investigation into the methods of inactivation in contaminated food and feed has revealed that pre-harvest contamination can be reduced by virtue of proper curing, drying, sorting and storage, all of the aforementioned limiting the growth of fungi. However, the implementation of unique, totipotent method of mycotoxin reduction, capable of effectively performing in any given biological material, is virtually impossible. The efficiency of the methods of reduction depends on many parameters such as the nature of food and feed, their moisture content and composition, and the level of contamination. Some studies have attempted to achieve detoxification of, or toxin inactivation in, mycotoxin-contaminated feedstuff using gamma irradiation, thermal inactivation, physical separation, microbial degradation and different chemical treatments (*Rustom, 1997*).

Methods of reduction can be divided into chemical, biological and physical. In biological reduction, microorganisms including bacteria, yeasts and acid-producing moulds are used to metabolize and inactivate mycotoxins, and *Flavobacterium aurantiacum* being the most active among them. Production of mycotoxins is also inhibited by lactic acid bacteria, *Bacillus subtilis* and many moulds. Inactivation using physical methods involves extraction with solvents, adsorption, and heat- or irradiation-based inactivation. Contamination can be reduced in stored goods using physical procedures such as color sorting, density flotation, blanching and roasting (*Pleadin et al., 2014b*). The use of chemicals to inactivate or remove mycotoxins has been studied using different chemicals such as propionic acid, ammonia, copper sulfate, benzoic acid, urea, citric acid and some other chemicals, but they have generally been labeled as impractical for application on food as they include drastic conditions in terms of temperature and pressure. Also, they are considered as unsafe because of toxic residues and unfavorable since leading to degradation of nutritional, sensory and functional properties of the product so particular chemical methods are only used for mycotoxins reduction in animal feed (*Rustom, 1997*).

Conclusion

Literature data indicate a high presence of mycotoxins in grains and feed. Special attention should be focused on the particular raw materials in order to reduce the exposure of animals to different mycotoxins that could have adverse

effects on animal health and production results, and consequently indirectly endanger human health and the occurrence of residues in products of animal origin. Data obtained for some mycotoxins suggest teratogenic and genotoxic effects in humans and animals as also their synergistic effects, however the literature does not contain sufficient relevant evidence related to that. In order to ensure safe food production and prevent economic problems of large-scale contamination, continued development and application of modern specific and selective methods in its detection and reduction of contamination are required, as well as the systematic implementation of national monitoring on a representative number of samples of grains and feed.

Mikotoksini u žitima i hrani za životinje – kontaminacija i toksično dejstvo kod životinja

J. Pleadin

Rezime

Mikotoksini su sekundarni metaboliti plesni koji predstavljaju značajan problem za bezbednost hrane, opasnost po zdravlje i dobrobit ljudi i životinja i imaju negativan uticaj na ekonomiju širom sveta. Oni mogu da izazovu trovanje životinja i da utiču na manje produktivne performanse i hranljivu vrednost hrane za životinje. Takođe, mikotoksini mogu kontaminirati različite vrste hrane kroz direktan i indirektan način i da izazovu negativne posledice po zdravlje ljudi. Da bi se procenio potencijal njihovog štetnog dejstva, neophodno je znati uslove pojave i učestalosti mikotoksina, toksičnost i biotransformaciju u različitim vrstama životinja, kod ljudi, mogućnost prevencije zagađenja, kao i njihove inaktivacije ili smanjenja u slučaju kada je pojava neizbežna. Relativno mali broj uporedivih podataka o učestalosti mikotoksina u hrani za životinje iz evropskih zemalja pokazuju neophodnost sistematske kontrole i primene validiranih analitičkih metoda u njihovom određivanju. Ovaj rad daje pregled učestalosti kontaminacije žita i hrane sa životinje sa najvažnijim i najučestalijim mikotoksinima i dokazano toksično dejstvo kod životinja.

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