Review of mycotoxin reduction in food and feed: from prevention in the field to detoxification by adsorption or transformation

G. Jard[a]†, T. Liboz[a]*, F. Mathieu[a], A. Guyonvarc'h[b] and A. Lebrihi[a]

[a]*INP/ENSAT, LGC, Université de Toulouse, Castanet-Tolosan, France;* [b]*EVIALIS, Vannes, France*

Mycotoxins are secondary metabolites present worldwide in agricultural commodities and produced by filamentous fungi that cause a toxic response (mycotoxicosis) when ingested by animals. Prevention of mycotoxicoses includes pre- and post-harvest strategies. The best way to reduce the mycotoxin content in food and feed is the prevention of mycotoxin formation in the field, but this is often not sufficient, so other methods are needed. To decontaminate and/or detoxify mycotoxin-contaminated food and feed, the most prevalent approach in the feed industry is the inclusion of sorbent materials in the feed thus obtaining more or less selective removal of toxins by adsorption during passage through the gastrointestinal tract. Another reliable approach is to add enzymes or microorganisms capable of detoxifying some mycotoxins. Through a comprehensive review of published reports on the strategies for mycotoxin removal, this present work aims to update our understanding of mycotoxin removal. It provides an insight into the detoxification of mycotoxin present in food and feed. In the future, more emphasis needs to be placed on adsorption of mycotoxins in the gastrointestinal tract. Concerning the enzymatic transformation of mycotoxins, further efforts are required in understanding detoxification reactions, the toxicity of transformation products and in the characterization of enzymes responsible for transformations.

Keywords: toxicology; microbiology; HPLC; mycotoxins; animal feed; cereals

Introduction

Mycotoxins are secondary metabolites produced by filamentous fungi that cause a toxic response (mycotoxicosis) when ingested by animals. Plants can be contaminated in two ways: fungi growing either as pathogens on plants in the field or on stored plants (Glenn 2007). Not all moulds are toxigenic and while some mycotoxins are produced by a limited number of species, others may be produced by a relatively large range from several genera. Up to now, approximately 400 secondary metabolites with toxigenic potential produced by more than 100 moulds have been reported. Examples of some well-known mycotoxins are aflatoxins, fumonisins, ochratoxin, trichothecenes, zearalenone, penitrems and ergot alkaloids. The Food and Agriculture Organization (FAO) estimates that 25% of the world's agricultural commodities are contaminated with mycotoxins, leading to significant economic losses (Wu 2007). Mycotoxins are small and quite stable molecules which are extremely difficult to remove or eradicate, and which enter the feed chain while keeping their toxic properties. Consumption of a mycotoxin-contaminated diet may induce acute and long-term chronic effects in animals and humans, resulting in teratogenic, carcinogenic and oestrogenic or immune-suppressive effects. Data on mycotoxin levels causing a reduction in zootechnical performance in farm animals (ruminants, pigs and poultry) were compiled in the AFSSA report (AFSSA 2009). Direct consequences of consumption of mycotoxin-contaminated animal feed include: reduced feed intake, poor feed conversion, diminished body weight gain, increased incidence of disease (due to immune-suppression) and reduced reproductive capacity (Fink-Gremmels and Malekinejad 2007; Morgavi and Riley 2007; Pestka 2007; Voss et al. 2007).

The diversity of mycotoxin structures induces diverse toxic effects. For example, the aflatoxin structure permit the formation of DNA adducts with guanine, inducing cancerous cell formation (Bren et al. 2007). The lactone ring of aflatoxins is responsible for its toxicity (Lee et al. 1981). Fumonisins could inhibit ceramide synthase (Soriano et al. 2005), inducing adverse effect on the sphinganin/sphingosin ratio. The deamination of fumonisin B1 induces a loss of toxicity, indicating that amines play a role in fumonisin

*Corresponding author. Email: liboz@ensat.fr
†Present address: EI-Purpan, 75 voie du TOEC, Toulouse, France.

toxicity. Ochratoxins affect protein synthesis and inhibit ATP production. The toxicity of ochratoxins is associated with its isocoumarin moiety (Xiao et al. 1996). Deoxynivalenol (DON) and T-2 toxin induce apoptosis in haemopoietic progenitor cells and immune cells (Parent-Massin 2004). They also inhibit protein, ADN and ARN synthesis (Richard 2007). The epoxy structure of trichothecenes induces their toxicity (Sundstøl Eriksen et al. 2004). Zearalenone (ZEA), thanks to its conformation, is able to mimic 17β-estradiol and to bind to estrogen receptors, disrupting fertility and reproduction ability (Gaumy et al. 2001).

Prevention of mycotoxicoses includes pre- and post-harvest strategies. The best way is the prevention of mycotoxin formation in the field, but this is often insufficient and other strategies are needed. To decontaminate and/or detoxify mycotoxin-contaminated food and feed, the most prevalent approach in the feed industry is the inclusion of sorbent materials in the feed to obtain selective removal of toxins by adsorption during passage through the gastrointestinal tract, or to add enzymes or microorganisms capable of detoxifying certain mycotoxins. A new functional group of feed additives was defined by the Commission regulation (EC) No 386/2009 of 12 May 2009 as ''substances for reduction of the contamination of feed by mycotoxins: substances that can suppress or reduce the absorption, promote the excretion of mycotoxins or modify their mode of action.'' Depending on their mode of action, these feed additives may act by reducing the bioavailability of the mycotoxins or by degrading them or transforming them into less toxic metabolites.

Through a comprehensive review of published reports on the strategies for removing mycotoxins by both adsorption and transformation, this present work aims to update our understanding of mycotoxin removal and provide insights into the elimination and detoxification of mycotoxins present in food and feed. Numerous recent reports have been previously published on part of this field (Kabak et al. 2006; Schatzmayr et al. 2006; Jouany 2007; EFSA 2009; Awad et al. 2010; He et al. 2010) but none of them give an overview of mycotoxin elimination, from prevention in the field to detoxification.

Preventive strategies: controlling mould development

Moulds can develop on plants or plant products during cultivation or storage. Depending on environmental conditions, some of these moulds may produce mycotoxins leading to harmful consequences for consumers. So, a primary strategy should aim to eliminate mycotoxins by reducing mould proliferation during cultivation and storage. Magan and Aldred (2007) highlighted some interesting studies on this topic. In the following discussion, we will mention a number of strategies which could be used.

Planting

Crop varieties selection

Seeds must be free of pests and disease before planting, to ensure healthy, vigorous plants capable of withstanding attack during the growing season. A possible strategy is also to select crop varieties on their ability to resist mould attack (Clements and White 2004).

Sowing date

Sowing date is another element of crop management that has an indirect effect on the production and infection by spores because it partly determines the flowering date, together with the variety sown and prevailing weather. If the sowing date is such that flowering coincides with spore release, then more frequent and severer attacks are likely (Champeil et al. 2004). As far as is practical, crop planting should be timed to avoid high temperature and drought stress during the period of seed development and maturation (Codex Alimentarius 2003).

Pre-harvest

Suitable cultivation techniques

Initially, suitable cultivation techniques can reduce the risk of fungal contamination. For example, removal of agricultural waste is effective in preventing the contamination of the follow-on crops. Indeed, the primary reservoir of fungal inoculum is the residues from previous crops. Also, ploughing limits mould contamination (Munkvold 2003; Champeil et al. 2004). The effects of crop rotation are also significant. For example, wheat following maize in the rotation has been found to have DON concentrations six times higher than those in wheat following another cereal (wheat, barley) or soybean (Krebs et al. 2000). In that context, Codex Alimentarius (2003) established good agricultural practice guidelines for farmers for the prevention and reduction of mycotoxin contamination in cereals. For example, it is suggested to use crops such as potato, other vegetables, clover and alfalfa that are not hosts to *Fusarium* species in rotation to reduce the inoculum in the field. Drought stress, caused by a lack of water, is known to facilitate attack by mould. Lack of water allows the plant to crack and so opens passage ways through which fungal spores can enter.

Predictive models

Software applications are available to help farmers predict mycotoxin risk during the year as a function of climatic parameters (Schaafsma and Hooker 2007; Prandini et al. 2009). Fungicides should be useful whenever a predictable risk exists (Paul et al. 2008). However, risk management must take into account potential risks to consumer health linked to the presence of fungicides in food. Thus, a recent study compared the toxicity of two fungicides and two mycotoxins, ZEA and DON (Muri et al. 2009). The final results showed that DON was more toxic than both ZEA and the two tested fungicides. However, other studies have shown that conventional agriculture using fungicides induces higher risks than organic farming (Finamore et al. 2004; Schneweis et al. 2005). For example, maize from an organic farm had a 50% lower *Fusarium* infection rate than maize from a conventional farm, probably due largely to the lower intensity of cultivation, different crop rotation, ploughing and the heightened biomass activity of organic soils (Arino et al. 2007).

Avoiding insect attack

It is known that the incidence of infection from *Aspergillus flavus* and *A. parasiticus* is significantly higher in damaged than healthy kernels. Insects can act as fungal spore vectors or create critical points within the commodity mass that favour fungal growth and toxin production (Sinha and Sinha 1990). Furthermore, they attack external teguments of kernel and facilitate entry and colonization of mycotoxin-producing fungi. Therefore, treatments with insecticides are advisable to reduce these attacks.

Biocontrol techniques

Biocontrol techniques based on microorganisms are also under study (Schisler et al. 2002) in the fight against fusariotoxicosis. Different microorganisms have been proposed as bio-control agents of *A. flavus* and aflatoxin contamination in pre-harvest; for example, non-aflatoxigenic strains of the same species could be bio-competitive agents. Dorner and Cole (2002) showed that treatment of soil with non-toxigenic strains of *A. flavus* and *A. parasiticus* significantly reduced preharvest aflatoxin contamination. In addition, soil treatment with nontoxigenic strains had the beneficial carry-over effect of reducing aflatoxin contamination that occurred during storage.

Post-harvest

Storage

This step is critical in preventing mould growth and mycotoxin production in harvested feedstuffs. Various important factors should be managed to prevent crops from fungal contamination during storage (Schrödter 2004). For example, grain should be stored with less than 15% moisture content to eliminate pockets of higher moisture (Kabak et al. 2006) at low temperature (Burges and Burrel 1964). A low oxygen concentration ($<1\%$) and augmentation of carbon dioxide concentration are efficient in preventing mould development (Driehuis and Oude-Elferink 2000). Physical integrity of grains should be preserved. Mixing grains and a long-time storage should be avoided.

Despite all precautions, it may happen that stored grain will become damaged by mould. It must then be assumed that the grain could also be contaminated with mycotoxins. If the farmer has plenty of grain in store, he can afford to lose a small quantity that has turned mouldy. Ideally, the farmer must discard mouldy grain and any that is suspected of being contaminated with mycotoxins; this will include apparently clean grain in the vicinity of mouldy produce. This grain should be burnt or buried.

Sorting

Contaminated grain does not have the same color or density as safe grain. Thus, grain can be sorted according to appearance or density (Murphy et al. 1993; Guerre 2000; Afolabi et al. 2006; Kabak et al. 2006). These methods are not very specific and, in general, not exhaustive. When mycotoxin contamination is heterogeneous, the removal of the contaminated portion may reduce the level of mycotoxin in the final product (Benett et al., 1978; Osborne et al. 1996). For example, apple dissection before making apple juice led to a 95% reduction in patulin (Lovett et al. 1975). Washing food or grain can also reduce mycotoxin levels. For example, the first step in spaghetti production with wheat is washing, which removes 23% of DON (Visconti et al. 2004).

Eliminating mycotoxins from food and feed

Different methods are used to decontaminate food and feed before ingestion. Presently, regulations do not permit the decontamination of food that exceeds the concentration threshold limits. Mycotoxin reduction in food could be carried out during industrial processing (Bullerman and Bianchini 2007) or by using additives which eliminate or deactivate the mycotoxin. In all cases, decontamination processes should destroy or inactivate mycotoxins, generate no toxic products, guaranty the nutritional value of the food and induce no modification to the technological properties of the product.

Decreasing bioavailability of mycotoxins by adsorption

Principle

Since all mycotoxins are very stable substances, no physical or chemical treatment can be applied without altering the nutritional value of the grain or causing a high rise in costs. For example, ammonia or strong oxidizing agents can reduce contamination but will also reduce the nutritional value of the feed.

The most commonly used technique for reducing exposure to mycotoxins is to decrease their bioavailability by the inclusion of various mycotoxin-binding agents or adsorbents, which reduce mycotoxin uptake and subsequent distribution to the blood and target organs. These adsorbants are efficient only if the complex is stable in an animal's digestive tract, so that bound mycotoxins are channelled away via urine and faeces.

An important criterion for the evaluation of mycotoxin adsorbants is their effectiveness at different ph levels (acidic and neutral). Thus, the adsorbant must be efficient throughout the entire gastro-intestinal tract and the mycotoxin–adsorbant complex remains stable to prevent desorption of the toxin during the digestion.

The properties of both adsorbant and mycotoxin play an important role in adsorbant efficacy. Indeed, the physical structure of an adsorbant, including such features as total charge and distribution, pore size and surface accessibility, must be studied. The characteristics of mycotoxins, such as polarity, solubility, molecular size, shape and, in the case of ionised compounds, charge distribution and dissociation constants, are also very important.

Binders have been evaluated using both in vitro and in vivo systems. In vivo studies have generally used performance responses or biological markers, such as tissue residues or changes in biochemical parameters, to determine the effectiveness of binders. Numerous binders can bind aflatoxin efficiently but few could be used for other mycotoxins (EFSA 2009).

Mineral and organic adsorbants

Activated charcoal. Activated charcoal is formed by the pyrolysis of organic materials. It is a general adsorptive material with a large surface area and excellent adsorptive capacity. Thanks to its porosity, activated charcoal can adsorb the main mycotoxins in an aqueous environment. In one of the first studies to test the concept of mycotoxin binding, activated charcoal was shown to efficiently adsorb AFB1 (Decker and Corby 1980). In subsequent studies, the effects of activated charcoal have been variable. Galvano et al. (1996) showed reduced aflatoxin residues in milk of cows consuming different sources of charcoal, but responses to charcoal did not exceed that seen with a clay-based binder, a hydrated sodium calcium aluminosilicate or HSCAS (Diaz et al. 2004). Responses to charcoal suggest that it may not be as effective in binding aflatoxin as clay-based binders (Edrington et al. 1996, 1997). Activated charcoal may be suitable in binding ZEA and/or DON (Döll et al. 2004; Bueno et al. 2005; Sabater-Vilar et al. 2007). In an in vitro gastrointestinal model, activated carbon reduced the availability of ZEA, DON and nivalenol (Avantaggiatto et al. 2003, 2004). However, adsorption of ochratoxin A (OTA) had no positive effect on chicken body weight (Rotter et al. 1989). For FB1, the ratio sphinganine/shingosine was not modified in rats after charcoal was added to contaminated feed (Solfrizzo et al. 2001). For T-2 and HT-2 toxins, responses have been variable depending on the animal. For example, a 50% survival rate for mice fed with contaminated feed was observed, while a 90% survival rate was reached after the addition of charcoal (Fricke and Jorge 1990). On the other hand, there was no effect on chicken: a decrease in 20% of body weight was observed with or without treatment.

Silicate binders. Silicates are divided into subclasses according to their structure. One such group is the phyllosilicate family, characterised by a sheet-type framework. The most extensively studied of these materials is hydrated sodium calcium aluminosilicates (HSCASs), for which several reviews are available (Ramos et al. 1996; Döll and Dänicke 2004; Avantaggiato et al. 2005). A total of 80% of AFB1 could be adsorbed by HSCASs in vitro (Phillips et al. 1988) and could prevent aflatoxicosis. Positive effects of other HSCASs on *Drosophila* progeny (Sisman 2006) were also observed. Furthermore, responses to HSCASs appear to be dose-dependent (Smith et al. 1994). HSCASs are thought to absorb aflatoxin selectively during the digestive process, which renders much of the aflatoxin unavailable for absorption from the gastrointestinal tract (Kubena et al. 1990). The chemisorption of aflatoxin to HSCAS involves the formation of a complex by the β-keto-lactone or bilactone system of aflatoxin with uncoordinated metal ions in HSCAS (Sarr et al. 1990). AFB1 may react at surfaces and within the interlayers of HSCAS particles (Phillips et al. 1995, 2002, 2008). Some sorbents, such as NovaSil clay, act as selective enterosorbents since they do not affect the serum concentrations of important vitamins and nutrient minerals in humans (Afriyie-Gyawu et al. 2008).

In general, HSCAS have a low affinity for OTA (Huff et al. 1992; Galvano et al. 1998). Adsorption results for ZEA are variable: some studies have shown a decrease in chromosomic aberrations for mice (Abbes et al. 2007) and a positive effect on the length

of mink gestation (Bursian et al. 1992). Patterson and Young (1993), however, failed to see any benefit in the addition of HSCAS to pig diets containing DON. This result was confirmed in vitro by Galvano et al. (1998) and Sabater-Vilar et al. (2007). Garcia et al. (2003), using a silicate material, demonstrated reduced T-2 toxicity, while Kubena et al. (1990) did not see any effect from the addition of HSCAS. Aly et al. (2004) showed an adsorption efficiency for a mixture of AFB1 and FB1 of 95 and 85%, respectively. However, Watts et al. (2003) showed that 1% HSCAS did not protect chicks and poults receiving diets containing 1 mg DON, 5 mg moniliformin, 5 mg FB1, 100 μg AFB1, 1 mg ZEA and 0.5 mg OTA per kg of diet.

Other silicates studied include bentonites, zeolites, clinoptilolites and various others that often have not been completely characterized. Bentonite is a general clay material originating from volcanic ash and containing primarily montmorillonite as the main constituent. Montmorillonite clay is a hydrated sodium calcium aluminum magnesium silicate hydroxide. Clays are silica sheets, similar to other phyllosilicates, but contain a high concentration of water. The zeolite structure provides vacant spaces that form channels of various sizes allowing movement of molecules into and out of the structure. Thanks to this structure, zeolites present a very large specific surface (about 1000 m^2 per g of zeolite).

Bentonite and montmorillonite could adsorb from 40 to 100% of OTA in wine (Kurtbay et al. 2008). However, Lemke et al. (2001a) failed to show a positive in vivo effect on rats. Indeed, rat uterus weight increased with montmorillonite treatments, suggesting an increase in oestrogenic toxicity with treatment. A slight positive effect was observed by Carson and Smith (1983a) for the addition of bentonite in T-2 toxin-contaminated food, since 8% of body weight was recovered after treatment. A 9% body weight gain was observed for chicken treated with bentonite when their feed contained a mix of AFB1 and FB1 (Miazzo et al. 2005). Another study performed by Kubena et al. (1998) demonstrated that an HSCAS could provide protection against AFB1 but not against T-2 toxin in young broiler chicks.

Zeolites are very efficient in bovine rumen juice with 100% of AFB1 adsorbed (Spotti et al. 2005). Zeolites have not been proven to reduce the toxicity of T-2 toxin (Dvorska and Surai 2001). However, some types of clinoptilotite could not be used as a binder since its addition in presence of AFB1 caused severe lesions, which may be explained by non-specific adsorption of important compounds involved in aflatoxicosis regulation (Mayura et al. 1998).

A number of studies have examined chemically modified silicates. Döll et al. (2004) examined a chemically modified aluminosilicate that showed good binding with ZEA in vitro, confirming previous work (Lemke et al. 1998; Tomasevic-Canovic et al. 2003). Others have shown that chemical modifications have increased the binding of HSCAS with ZEA (Pimpukdee et al. 2004). Non-modified zeolites were used to adsorb AFB1, while hydrophobic zeolites (octadecyldimethylbenzyl, an ammonium-treated zeolite) were mostly used to adsorb OTA and ZEA (Dakovic et al. 2003 2005 2007). The adsorption efficiency of hydrophobic zeolites could be explained by hydrophobic interactions between OTA and ZEA and the binder.

Other mineral adsorbants. Some synthetic polymers, such as cholestryamine and polyvinylpyrrolidone, can adsorb mycotoxins, while undigestible dietary fiber has some adsorbance potential. Fibers can reduce the effects of ZEA (Tangni et al. 2006) in rats and swine and T-2 toxin in rats (Carson and Smith 1983b). Cholestyramine resin is used in human medicine for the reduction of cholesterol and functions through adsorption of bile acids. Cholestyramine has been shown to adsorb ZEA (Ramos et al. 1996; Döll et al. 2004; Avantaggiato et al. 2005) and FB1 (Solfrizzo et al. 2001). In rats consuming OTA, cholestyramine increased faecal OTA excretion (Kerkadi et al. 1998). In another in vivo study, cholestyramine did not bind OTA (Bauer 1994) and only 5% of DON could be adsorbed by cholestyramine in a dynamic digestive tract (Avantaggiato et al. 2005).

A synthetic water-soluble polymer, polyvinlypyrrolidone (PVP), has been investigated as a binder for mycotoxins. PVP is reported to bind with AFB1 and ZEA in vitro (Alegakis et al. 1999) but did not alleviate the toxicity of DON in pigs (Friend et al. 1984).

Some commercial adsorbants consist of a mixture of different adsorbants; for example, the product Standard Q/FIS allies charcoal and HSCAS. These products are, in general, tested for their capacity to adsorb various mycotoxins at the same time (Avantaggiato et al. 2007).

Limits of mineral adsorbants. Mineral adsorbants are generally efficacious against AFB1 but their efficacy varies for other mycotoxins. Moreover, in some cases, the addition of clay increases the effects of mycotoxicosis (Carson and Smith 1983a; Mayura et al. 1998; Lemke et al. 2001a). Furthermore, these adsorbants are not specific to mycotoxins and can adsorb other types of molecules, some of which are essential for fighting mycotoxicosis or in nutrition (Mayura et al. 1998).

Organic adsorbants. Humic acids are complex organic substances, a component of humus. They also have the capacity to adsorb mycotoxins, especially AFB1 and

ZEA (Jansen van Rensburg et al. 2006) but not DON (Sabater-Vilar et al. 2007).

Biological adsorbants

Due to the limitations of mineral adsorption, many studies have been conducted over the last decade on biological adsorbents, in an attempt to obtain greater efficacy and specificity while, at the same time, reduce the impact on nutritional quality compared to mineral adsorbents.

Yeast or yeast extract. One example is *Saccharomyces cerevisiae,* which has been shown to bind with AFB1 (Shetty and Jespersen 2006) and reduce the detrimental effects of AFB1 in broiler diets (Stanley et al. 1993) or on rats (Madrigal-Santillán et al. 2006). The protective effect of live yeast against aflatoxin was confirmed in rats but thermolysed yeast was shown to be ineffective (Babtista et al. 2002). In contrast, some thermolysed yeast cell walls were more efficient in adsorbing ZEA (Yiannikouris 2004). Esterified glucomannan polymer, extracted from the yeast cell wall, was shown to bind with AFB1, OTA (Bejaoui et al. 2004; Cecchini et al. 2007; Angioni et al. 2007) and T-2 toxin (Freimund et al. 2003), individually and in combination (Raju and Devegowda 2000; Aravind et al. 2003; Yiannikouris 2004; Karaman et al. 2005). Additions of esterified glucomannan at 0.5 or 1.0 $g\,kg^{-1}$ to diets containing 2 mg of total aflatoxin resulted in dose-dependent responses in broiler chicks (Basmacioglu et al. 2005). The addition of esterified glucan polymer to aflatoxin-contaminated diets of dairy cows significantly reduced milk aflatoxin residues (Diaz et al. 2004). Body weight and biochemical parameters recovered in horses after adding glucomannan to their diet (Raymond et al. 2003).

Yiannikouris et al. (2004) demonstrated the mechanism of binding yeast-modified glucan with ZEA. A glucan polymer bound both T-2 toxin and ZEA in vitro (Freimund et al. 2003), while another glucan polymer had a protective effect against the depression of antioxidant activities resulting from T-2 toxin consumed by growing quail (Dvorska and Surai 2001). A glucan polymer product also protected swine, broilers (Swamy et al. 2002b; Swamy et al. 2002a) and hens (Chowdhury and Smith 2004) against some of the detrimental effects of multiple mycotoxins, but without restoring growth rate. Aravind et al. (2003), using dietary additions of 0.5% esterified glucomannan, alleviated growth depression in broilers associated with naturally contaminated diets containing AFB1, OTA, ZEA and T-2 toxin. However, a glucan polymer product did not alleviate the toxic effects in mink consuming diets contaminated with FB1, OTA, moniliformin and ZEA (Bursian et al. 2004). The negative effects of ZEA and DON as measured by biochemical and immunological parameters in pig were alleviated by adding yeast extract enriched in glucomannan to their diet (Swamy et al. 2002b). In an in vitro experiment, 183 mg of T-2/HT-2 toxin was adsorbed per g of yeast-modified glucan (Freimund et al. 2003). FB1 adsorption by yeast or yeast extract is limited (Yiannikouris 2004); however, T-2 and HT-2 binding responses were variables on yeast-modified glucan (Freimund et al. 2003).

Lactic acid bacteria. The parietal structures of some lactic acid bacteria, propionibacteria and bifidobacteria have the capacity to bind mycotoxins (El-Nezami et al. 2000 2002a; Haskard et al. 2001; Oatley et al. 2000). The binding appears to be physical with DON, diacetoxyscerpenol, nivalenol, and other mycotoxins, associated with hydrophobic pockets on the bacterial surface (Haskard et al. 2000; El-Nezami et al. 2004). Adsorption efficiency depends on strains (Peltonen et al. 2001). Adsorption of AFB1 and AFM1 was reversible and could be performed with living or dead bacteria. Acid or heat inactivation of lactic acid bacteria increased adsorption efficiency (El-Nezami et al. 1998 2000 2002b; Pierides et al. 2000; Haskard et al. 2001; Bueno et al. 2007; Fazeli et al. 2009). OTA could also be adsorbed by lactic acid bacteria (Fuchs et al. 2002), leading to a diminution in toxicity on human hepatic cells (Del Prete et al. 2007). ZEA was adsorbed between pH 4 and pH 8 (El-Nezami et al. 2002b) and heat or acid inactivation increased adsorption efficiency (Niderkorn et al. 2006; El-Nezami et al. 1998, 2002b). Binding of FB1 and DON by *Lactobacillus rhamnosus* was not very efficient (Niderkorn et al. 2006); hydrophobic interactions are believed to be involved in the binding (Haskard et al. 2001).

Other biological materials. Fungal conidia are capable of binding mycotoxins, especially ZEA and OTA, separately or together (Jard et al. 2009). Between 29 and 60% of ZEA was eliminated, depending on the isolate and incubation time. Jard et al. (2009) suggested a hydrophobic interaction based on conidial wall characteristics and it has also been suggested that conidia could be used to decontaminate wine affected by OTA (Bejaoui et al. 2005).

Binder efficiency

Many solutions are available for binding mycotoxins but few are actually used in an industrial context. Decreasing the bioavailability of AFB1 by the inclusion of binding agents is particularly effective, as this group of toxins has a chemical structure which favors

adsorption, especially by materials of mineral origin such as clay and zeolites. OTA, ZEA and FB1 adsorption with biological adsorbents has been more extensively studied due to the inefficiency of mineral adsorbents. DON and T-2 or HT-2 toxin do not bind easily to every type of binding agents.

A binder must be effective at sequestering the particular mycotoxin(s) targetted. In some cases, it may be advantageous to bind one specific mycotoxin whereas, in others, binding multiple of mycotoxins may be the objective. A binder should significantly prevent toxicity in animals and there should be no serious side-effects or, at least, the detrimental effects should not outweigh the benefits. Costs should render its use practical and profitable. Mycotoxin residues from animals or products should not increase nor should there be any detrimental effects on the animal food product. Mycotoxins in feed should not be masked such that feed contamination cannot be verified. The binder should be physically usable in a commercial feed manufacturing context. Binder use and efficacy should be verifiable. Interaction between an adsorbent and mycotoxins must produce a very strong bond so that washing or interaction with nutrients in the digestive tract does not desorb the bound mycotoxins. Adsorption must be as specific as possible so that essential molecules will not also be bound. Most adsorbing agents, especially aluminosilicates, have been tested for their ability to bind aflatoxins; however, the mycotoxins targeted by yeast cell wall, bacteria and other adsorbing agents are more diverse.

Detoxification of mycotoxins by transformation

Transformation by physical and chemical treatment

Few physical or chemical processes can transform mycotoxins into non-toxic products. Those most studied are presented below. Kabak et al. (2006) produced an interesting detailed review dealing with this topic. He et al. (2010) reviewed the chemical and biological transformations for detoxification of trichothecene mycotoxins.

Thermal treatment. Mycotoxins are generally very stable and are rarely eliminated by thermic treatment (Kabak 2009). Little or no reduction in mycotoxin levels occurs as a result of normal cooking conditions, such as boiling, frying. For example, DON is stable at 120 °C, moderately stable at 180 °C and partially stable at 210°C (WHO 2001). Fumonisins are completely destroyed at 220 °C (Dupuy et al. 1993). Torrefaction processes could eliminate between 45 and 83% of aflatoxin content, while panification or pasteurization is inefficient in removing aflatoxin (CAST 2003). Thermal treatment of ZEA-contaminated food is ineffective (Ryu et al. 2003). The initial level of contamination, type of mycotoxin and its concentration, heating temperature and time, the degree of heat penetration, moisture content, pH and ionic strength of the food all play a significant role in toxin degradation (Rustom 1997).

Degradation by extrusion. Extrusion enables aflatoxins, DON, ZEA and FB1 to be removed from maize (Rustom 1997; Cazzaniga et al. 2001; Voss et al. 2008). Extrusion implies molecular modifications such as protein denaturation and the inhibition of the enzymatic activity.

Radiation. Most mycotoxins have complex molecular structures and are not often affected by irradiation. Radiolysis of water produces free radicals that could react with mycotoxins (Stepanik et al. 2007). AFB1 is sensitive to UV, X- and gamma-rays. Radiation of AFB1 reduces its level of contamination (Rustom 1997; Afifi et al. 2003), and gamma-radiation can also reduce microbial flora. Micro-waves can reduce aflatoxin content in peanuts (Farag et al. 1996) and trichotecenes in corn (Scott 1998).

Oxidation. Some oxidizing agents, such as ozone and hydrogen peroxide, have been used to render mycotoxin-contaminated feed harmless. Chemical oxidizing agents can react with numerous functional groups. McKenzie et al. (1998) showed that treatment of contaminated corn with electrochemically produced O_3 provided protection against AFB1 in young turkey poults. Canadas (2006) studied the efficacy of the Oxygreen® process using ozone on OTA-contaminated cereals. The Oxygreen® process reduced microbiological and mycotoxin contamination but induced some side-effects such as ADN adducts in wheat. Abd Alla (1997) demonstrated that ZEA was degraded by 83.9% using 10% H_2O_2 at 80 °C for 16 h. This was confirmed by Lemke et al. (1999), who also demonstrated that ozone could prevent the estrogenic effects of ZEA in mice. The biological activities of trichothecenes were also changed by oxidation with ozone most likely attacking the double bond in trichothecenes (McKenzie et al. 1997).

Reduction. Reducing agents (ascorbic acid, $NaHSO_3$, $Na_2S_2O_5$) decreased mycotoxin levels, particularly AFB1 and DON (Kabak et al. 2006). The sodium bisulfite-transformation of DON to DON-sulfonate, which is less toxic than DON, was reported to be an effective tool for overcoming the depressive effects of Don on feed-intake in piglets (Dänicke et al. 2005).

The reaction of FB1 with reducing sugars, such as D-glucose or D-fructose, at 65 °C for 48 h can block the primary amino group of FB1, and seems to prevent FB1-induced toxicity on cell tissue cultures of rats and swine (Fernandez-Surumay et al. 2005).

Ammoniation. Maize ammoniation, mainly used to decrease the level of aflatoxins in feed, is an efficient method for detoxifying feed and has been in use for several years (Park et al. 1988). This process is particularly effective against AFB1 when carried out at high temperature and pressure. One of the degradation products is AFD1, which is less toxic than AFB1. However, this costly method is ineffective against other mycotoxins and can damage food quality due to excessive ammonia level in the food involved (Huwig et al. 2001).

Alkalization. Under alkaline conditions, mycotoxins structure can change; for example, the 12-13-epoxy group of DON can be opened (Bretz et al. 2006).

Acidification. Treatment with strong acids destroyed the biological activity of AFB1, converting it to the hemi-acetal form (Heathcote and Hibbert 1978). Treatment with HCl (pH 2) has been shown to reduce AFB1 levels by 19.3% within 24 h (Doyle et al. 1982).

Deamination. Adding $NaNO_2$ to an aqueous medium deaminates FB1 and so reduces its toxicity (Lemke et al. 2001b).

Transformation by microorganisms and enzymes

The use of chemical or physical processes to decontaminate food is limited by high costs, loss of food's nutritional quality, poor efficiency, low specificity and consumer reticence toward chemical methods. Thus, scientists have come to favor the detoxification of mycotoxins by biological transformation, which can be defined as the degradation or enzymatic transformation of mycotoxins (by full microorganisms or enzymes) to less toxic compounds.

A wide range of microorganisms belonging to the bacteria, moulds and yeasts have shown a capacity to biotransform mycotoxins. Such microbes act in the intestinal tract of animals prior to resorption of the mycotoxins.

AFB1 transformation. Removal and detoxification of AFB1 by transformation have been investigated for many years but, unfortunately, few studies have led to the identification of the transformed product. One of the first bacteria studied for its capacity to remove AFB1 was *Flavobacterium aurantiacum* (also known as *Nocardia corynebacteriodes*). Its crude extract was shown to remove AFB1 (Ciegler et al. 1966; Hao and Brackett 1989). Further studies have shown that the transformation did not lead to a toxic product (Lillehoj et al. 1967) and that an intracellular enzyme was involved (Smiley and Draughon 2000). Guan et al. (2008) succeeded in obtaining S*tenotrophomonas maltophilia* isolates on selective medium containing only coumarin (chemical component of AFB1 nucleus) as the carbon source and showed that it is capable of transforming AFB1. *Bacillus subtilis* was also able to detoxify AFB1-contaminated feed and thus facilitate animal growth rate (Kubo 1996; Petchkongkaew et al. 2008). Actinomyceta, such as *Mycobacterium fluoranthenivorans*, were shown to remove AFB1 from contaminated media (Hormisch et al. 2004; Teniola et al. 2005). Teniola et al. (2005) succeeded in isolating extracellular enzymes from *Rhodococcus erythropolis*, responsible for the transformation of AFB1. Moreover, Alberts et al. (2006) observed the elimination of toxicity using the Ames test after transformation. Nakazato et al. (1990) and later Shantha (1999) observed the removal of AFB1 by moulds but no transformation product was observed. Extracellular enzymes from the macroscopic fungus *Pleurotus ostreatus* were shown to be able to cleave the AFB1 lactone ring leading to a loss of toxicity (Motomura et al. 2003), while *Armillariella tabescens* was able to open the AFB1 difuran ring (Liu et al. 1998, 2001).

OTA transformation. Certain bacteria, moulds, yeasts and plants are able to transform OTA, with many being able to transform OTA into OTα (Galtier and Alvinerie 1976; Wegst and Lingens, 1983; Kiessling et al. 1984; Hwang and Draughon 1994; Skrinjar et al. 1996; Abrunhosa et al., 2002; Péteri et al. 2007), a less toxic compound. This transformation leads to the formation of phenylalanine (Figure 1). Moulds like *Aspergillus*, *Rhyzopus* and *Penicillium* spp. are particularly effective at removing OTA (Varga et al. 2000; Abrunhosa et al. 2002; Bejaoui et al. 2006). *Aureobasidium pullulans* was used as a biocontrol agent in wine, preventing OTA accumulation in grapes and decreasing aspergillosis symptoms (De Felice et al. 2008). Plants, such as wheat and maize (Ruhland et al. 1996), or fungi, such as *P. ostreatus* (Engelhardt 2002), are capable of removing OTA but no transformation products have been identified. *Trichosporon mycotoxinivorans* is a microorganism that has been developed into a commercial product for detoxifying OTA in animal feed (Molnar et al. 2004). Total transformation of OTA to OTα occurred in 2.5 h. Moreover, Schatzmayr et al. (2003, 2006)

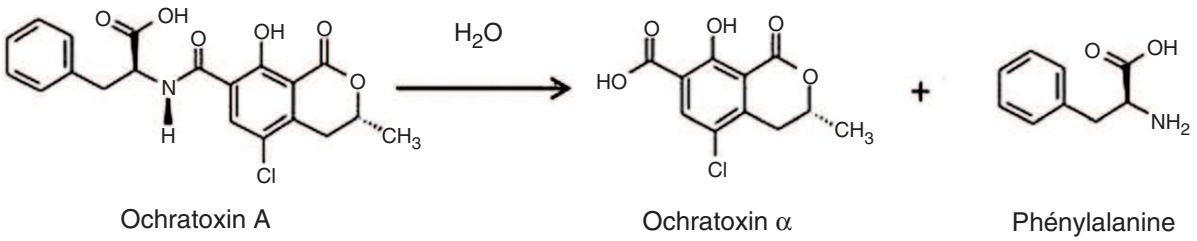
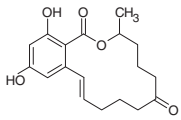
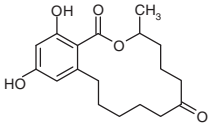
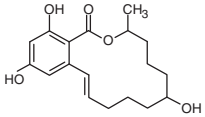
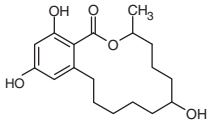
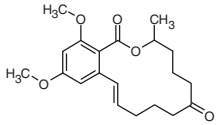
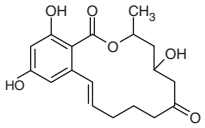
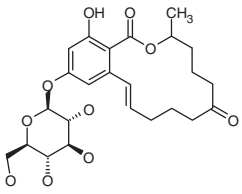


Figure 1. Transformation of ochratoxin A into ochratoxin α and phenylalanine.

showed that the toxic effects of OTA could be alleviated by the addition of this yeast to chicken diet. The main microorganisms which transform OTA into Otα use carboxypeptidases (EC 3.4.17.1) and purified enzymes able to cleave OTA have been characterised (Stander et al. 2000; Abrunhosa et al. 2006).

ZEA transformation. ZEA can be transformed into an oxidised compounds, such as zearalanone, hydroxyl compounds, such as α- and β-zearalenol, methyl compounds, gluco- or sulfo-conjugates and hydrolysed compounds (Table 1) by bacteria, yeasts, moulds or plants (El-Sharkawy and Abul-Hajj 1987a,b 1988). The transformation of ZEA to α-zearalenol (McMullen 1977; Kiessling et al. 1984; Kamimura 1986; Böswald et al. 1995) does not lead to actual detoxification since this product has oestrogenic activity even higher than ZEA (Fitzpatrick et al. 1989; Minervini et al. 2005). The proteic sequence of a ZEA esterase transforming ZEA into decarboxylated ZEA from a *Rhodococcus* or *Nocardia* spp. has been patented for insertion in transgenic plants (Duvick and Rood 2000; Karlovsky et al. 2003). *Trichosporon mycotoxinivorans* is also able to decarboxylate ZEA (Molnar et al. 2004; Vekiru et al. 2010). Some conjugates can also be formed from ZEA, such as ZEA-glucoside (Kamimura 1986; El-Sharkawy and Abul-Hajj 1987a) and ZEA-sulfate (El Sharkawy et al. 1991; Plasencia and Mirocha 1991). Recently, Jard et al. (2010), using a MCF-7 cell line, showed that ZEA sulfonation leads to a reduction in oestrogenic toxicity. This confirms the study done by Plasencia and Mirocha (1991), using the uterus weight test, showing a decline in toxicity by ZEA sulfonation. However, it has not been proven that sulfonation or glycosylation leads effectively to detoxification insofar as hydrolysis of this conjugate could occur in the digestive tract. Moreover, plants are able to transform ZEA into ZEA-glycoside (Schneweis et al. 2002) leading to masked mycotoxins which could not be measured but could be released by feeding with contaminated plants. Other degradation products have been observed, including decarboxylated and hydrolysed compounds (El-Sharkawy and Abul-Hajj 1988; Duvick and Rood 2000; Kakeya et al. 2002; Takahashi-Ando et al. 2002;

Table 1. Chemical structure of zearalenone metabolites.

ZEA and its metabolites	Chemical structure
ZEA	
Oxydised Compounds Zearalanone	
Hydroxyled and methyl compounds a-β zearalenol	
a-β zearalanol	
Methoxy-ZEA	
Hydroxy-ZEA	
Gluco- and sulfo-conjugates ZEA-4-β-glucopyranoside	

(continued)

Table 1. Continued.

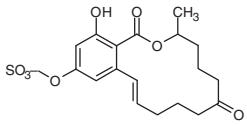
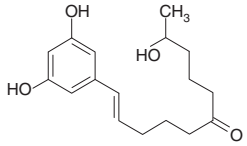
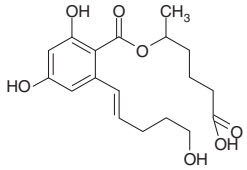
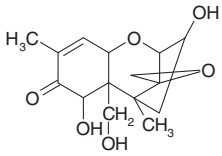
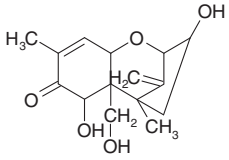
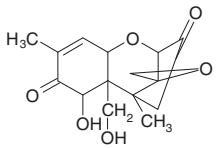
ZEA and its metabolites	Chemical structure
ZEA-4-sulfate	
Hydrolysed compounds	
Decarboxylated ZEA	
Hydroxylated ZEA	

Table 2. Chemical structure of deoxynivalenol metabolites.

DON and its metabolites	Chemical structure
DON	
De-epoxy DON	
Ketonic compound	

Karlovsky et al. 2003; Igawa et al. 2007). The corresponding gene of this enzymatic activity was cloned and transferred to different organisms, such as yeast (Takahashi-Ando et al. 2005) and plants (Kakeya et al. 2002; Takahashi-Ando et al. 2002; Igawa et al. 2007). Utermark and Karlovsky (2007) showed that ZEA lactonohydrolase from *Clonostachys rosea* (El-Sharkawy and Abul-Hajj 1988) prevents the growth inhibition observed for others types of fungi growing on ZEA-contaminated media. Gromadzka et al. (2009) showed that this fungus could be used as a biocontrol agent for mycotoxin production in cereals. *Pseudomonas* sp. of soil bacteria are able to remove ZEA (El-Deeb 2005); the product was not identified but was assumed to be less toxic than ZEA. Megharaj et al. (1997) showed that a mixed culture from soil was able to remove ZEA but no transformation product was observed. An unidentified bacterium from pig intestine was also able to remove ZEA (Kollarczik et al. 1994). Some microorganisms have the capacity to remove ZEA producing different derived compounds. For example, El Sharkawy et al. (1991) identified 26% of ZEA-sulfate and 18% of α-ZOL after a ZEA transformation by *Rhyzopus*. An enzyme from *Pseudomonas* sp., responsible for the transformation into a less toxic product, is encoded by a plasmid (Skrinjar et al. 1996). Cheng et al. (2006) studied an enzyme able to transform ZEA but its origin and transformation product(s) are unknown.

Trichothecenes transformation. Trichothecenes transformation by microorganisms was reviewed by Zhou et al. (2008) and He et al. (2010). The 12,13-epoxy ring of DON and T-2 toxin seems to be the part of the molecule responsible for the toxicity. By opening this ring, the mycotoxin become less toxic (Swanson et al. 1988; Zhou et al. 2008).

Some studies report a transformation of DON by mixed cultures of microorganisms but few of these have been identified. The two main metabolites are de-epoxidised DON (Yoshizawa et al. 1983; King et al. 1984; Côté et al. 1986; Swanson et al. 1988; He et al. 1992; Kollarczik et al. 1994; Young et al. 2007) and 3-keto-DON (Shima et al. 1997; Binder and Binder 2004; Young et al. 2007). The chemical structures of these DON metabolites are presented in Table 2. Both are less toxic than DON; for example, Shima et al. (1997) observed weaker immunosuppressive activity for 3-keto-4-DON produced by *Agrobacterium-Rhizobium* E 3-39 than for DON.

The *Eubacterium* sp. strain BBSH 797 has been developed into a commercial product – Mycofix plus (Biomin®) – for detoxifying trichothecenes in animal feed (Schatzmayr et al. 2006). An enzyme known as MDE (mycotoxin-degrading enzyme), whose origin is not specified, is able to degrade DON with ZEA (Cheng et al. 2006). Some transformation products have not yet been identified (Binder and Binder 2004; Völkl et al. 2004; Guan et al. 2009) but, among these transformations, one was done using a bacterium isolated from fish intestine, which achieved 100% removal in 96 h.

Few studies exist concerning the transformation of T-2 and HT-2 toxins. Some metabolites were identified to be de-acetylated and de-epoxidised T-2 (Table 3). Some microorganisms were able to transform T-2 toxin into HT-2 toxin but this did not lead to

Table 3. Chemical structure of metabolites of T-2 toxin and HT-2 toxin.

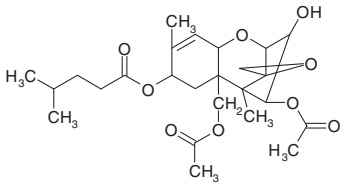
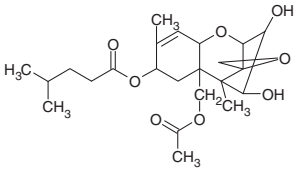
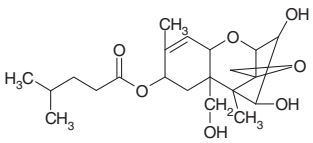
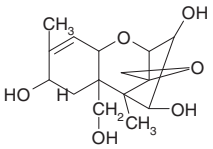
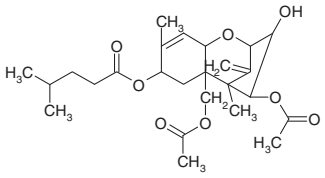
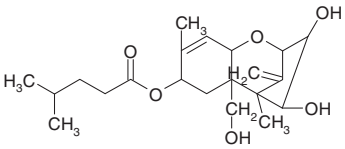
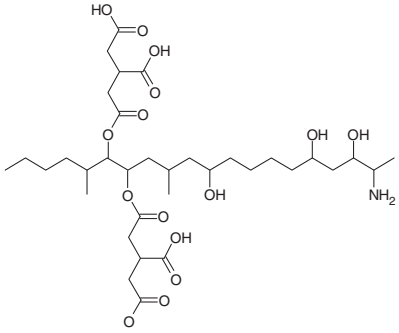
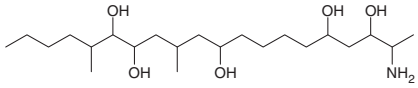
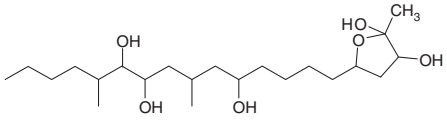
T-2 or HT-2 toxin and their metabolites	Chemical structure
T-2 toxin	
De-acetylate molecules HT-2 toxin	
T-2triol	
T-2tetraol	
De-epoxydised molecules De-epoxy T-2 toxin	
De-epoxy T-2 triol	

Table 4. Chemical structure of fumonisin B1 metabolites.

FB1 and its metabolites	Chemical structure
FB1	
Polyolamine (AP1)	
2-OP1	

detoxification (Swanson et al. 1988; Fuchs et al. 2002). Bacteria isolated by enrichment from contaminated soil or water can transform T-2 toxin into T-2 triol and T-2 tetraol (Beeton and Bull 1989). A similar transformation was observed for carboxyl esterase isolated from rat liver (Johnsen et al. 1986). Some moulds can transform T-2 toxin but the transformation products have not been identified (Jesenska and Sajbidorova 1991). The metabolism of T-2 toxin was elucidated by Swanson et al. (1988) and Beeton and Bull (1989). It includes a sequence of different steps: the molecule is first transformed into HT-2 toxin by deacetylation. A second deacetylation was observed to transform HT-2 toxin into T-2 triol, 20 times less toxic than the T-2 toxin (Ueno et al. 1983). Finally, this molecule is transformed into T-2 tetraol. Other metabolites observed are the de-epoxidised metabolites cited previously (Table 3).

Fumonisin B1 transformation. The primary amine of FB1 confers its toxicity; thus, deamination of this molecule greatly reduces its toxicity. Very few studies have been done on the biological degradation of FB1. The main microorganism capable of degrading FB1 is the black yeast, *Exophiala spinifera*, and the various metabolites produced by this yeast are presented in Table 4. The transformation of FB1 into AP1 is performed by an extracellular carboxylesterase. This enzyme has been cloned and was shown to be efficient in transgenic maize, as the plant became resistant to fumonisin (Duvick et al. 2003). Other enzymes are involved in FB1 degradation and some derived products have been characterised (Blackwell et al. 1999). A patent was taken out in 2003 (Duvick et al. 2003) for an esterase capable of detoxifying fumonisins.

Conclusion

Although there are many publications on the removal of mycotoxins by adsorption and transformation, their

application in the detoxification process has been limited.

Chemical treatment for the detoxification of aflatoxins using ammonia is the only application currently licensed in the United States. Chemical treatment is not allowed within the European Union for products used by humans (EU Commission Regulation 2001) as recourse to chemical transformation may lead to toxic derivatives in the treated products. These side-effects limit their use in the human and animal food chains (Kabak et al. 2006) and with the decreased interest in chemical transformations, interest has been increasingly focused on adsorption or transformation of mycotoxins. Adsorption by yeast cell walls added to feed is used most frequently in the industrial context. Legislation is changing to allow the marketing of mycotoxin binders whereas, up to now, these products have been sold for other applications, such as increasing antioxidising activity. It has proved difficult for governments to legislate on this topic without encouraging suppliers to sell contaminated commodities. Mycotoxin adsorption is well documented and tested in vitro and in the gastrointestinal tract, which has enabled us to better determine the stability of the binder–mycotoxin complex (Avantaggiatto et al. 2004, 2007).

Transformation of mycotoxins for detoxification purposes is not much used in industry, which may be due to lack of information on transformation mechanisms, the toxicity of products derived from transformation or the effect of transformation reactions on the nutritional value of food and feed. In some cases, transformation products have not been identified and so cannot be used in industrial processes. The stability of transformations and their potential side-effects needs to be investigated further.

The use of a microorganisms for detoxifying mycotoxins in feed requires the observance of certain conditions:

- The first step is identification of a microorganism's degradation potential;
- Each product must be identified and its toxicity tested using different methods if possible;
- Derived products must be non-toxic;
- Biotransformation must be fast and microorganisms must be efficient under different oxygen or pH conditions, especially when transformation takes place during digestion;
- Microorganisms must be non-pathogenic;
- Microorganisms must be active in a complex environment and not inhibited by nutrients;
- The efficiency of the transformation must be assessed *in vivo*.

The use of microbial detoxification agents (living cells) in the human food and animal feed industries is diminishing due to regulatory, toxicological and consumer considerations. Thus, applications of detoxification enzymes and genes may represent alternative detoxification methods. Some interesting studies have been carried out using molecular engineering techniques to obtain genes (Takahashi-Ando et al. 2004) which can be cloned and incorporated into microorganisms to produce recombinant enzymes suitable for industrial-scale enzyme production and purification (Althali and Deeb 2009).

Chemical transformation is no longer acceptable for detoxifying mycotoxin-contaminated food and feed. Mycotoxin binding may be a feasible industrial solution due to its relative low cost compared to biotransformation. However, this technique can lead to negative side-effects. Indeed, adsorption could be reversible as a mycotoxin could desorb in the digestive tract, and adsorption can be non-specific, thus decreasing overall nutritional value (Phillips et al. 2008). Mycotoxin transformation appears to offer some hope as a technique for increasing the quality of food and feed. Studies involving biotransformation have led to encouraging results but further studies are needed to ensure the safety of this method. The transformation of mycotoxins in food and feed promises to be a very reliable technique for enhancing food safety and eliminating all negative effects of the mycotoxins present.

Acknowledgements

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