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Abstract:	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 that 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

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	<p>update our understanding of mycotoxin removal. It could provide insight in the detoxification in mycotoxin present in food and feed. In the future, more emphasis should be placed on adsorption of mycotoxins in the gastrointestinal tract. Concerning the enzymatic transformation of mycotoxins, further effort is required in understanding detoxification reactions, the toxicity of transformation products and in the characterization of enzymes responsible for the transformations.</p>

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

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Abstract

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 that 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 could provide insight in the detoxification in mycotoxin present in food and feed. In the future, more emphasis should be placed on adsorption of mycotoxins in the gastrointestinal tract. Concerning the enzymatic transformation of mycotoxins, further effort is required in understanding detoxification reactions, the toxicity of transformation products and in the characterization of enzymes responsible for the transformations.

KEYWORDS: mycotoxins; decontamination; adsorption; binding agents; sequestering agents; transformation; detoxification.

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 field or on stored plants (Glenn, 2007). All moulds are not toxigenic and while some mycotoxins are produced by only a limited number of species, others may be produced by a relatively large range from several genera. Up to now, there have been reported approximately 400 secondary metabolites with toxigenic potential produced by more than 100 moulds. Examples of some well-known mycotoxins are aflatoxins, fumonisins, ochratoxin, trichothecenes, zearalenone, penitremes 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 in mycotoxin contamination 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). Fumonisin could inhibit the ceramide synthase (Soriano *et al.*, 2005) inducing adverse effect on the sphinganine/sphingosin ratio. The deamination of fumonisin B1 induces a loss of toxicity indicating that amine play a role in fumonisin toxicity. Ochratoxins affect the protein synthesis and inhibit the 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 17 β -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 not sufficient 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 more or less 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 as "substances for

1 reduction of the contamination of feed by mycotoxins: substances that can suppress or
2 reduce the absorption, promote the excretion of mycotoxins or modify their mode of action”
3 was defined by the Commission regulation (EC) No 386/2009 of 12 May 2009. Depending on
4 their mode of action, these feed additives may act by reducing the bioavailability of the
5 mycotoxins or by degrading them or transforming them into less toxic metabolites.
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9 Through a comprehensive review of published reports on the strategies for removing
10 mycotoxins by both adsorption and transformation, this present work aims to update our
11 understanding of removal of mycotoxins and it could provide insight in the elimination and
12 detoxification in mycotoxin present in food and feed. Numerous recent reports have been
13 previously published on part of this field (Kabak *et al.*, 2006; Schatzmayr *et al.*, 2006;
14 Jouany, 2007; EFSA, 2009; Awad *et al.*, 2010; He *et al.*, 2010) but none of them propose an
15 overview on mycotoxin elimination from prevention in fields to mycotoxin detoxification.
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23 **Preventive strategies: controlling mould development**

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

37 *Crop varieties selection*

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

45 Sowing date is another element of crop management that has an indirect effect on the
46 production and infection by spores because it partly determines the flowering date, together
47 with the variety sown and prevailing weather. If the sowing date is such that flowering
48 coincides with spore release, then more frequent and severer attacks are likely (Champeil *et al.*,
49 2004). As far as practical, crop planting should be timed to avoid high temperature and
50 drought stress during the period of seed development and maturation (Codex alimentarius,
51 2003).
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59 **Pre-harvest**

60 *Suitable cultivation techniques*

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

22 *Predictive models*

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

43 *Avoiding insect attack*

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

54 *Biocontrol techniques*

55 In the fight against fusariotoxicosis, biocontrol techniques based on microorganisms are also
56 under study to fight against (Schisler *et al.*, 2002). Different microorganisms have been
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1 proposed as bio-control agents of *A. flavus* and aflatoxins contamination in pre-harvest; non-
2 aflatoxigenic isolated strains of the same species could be optimal bio-competitive agents.
3 For example, Dorner and Cole (2002) showed that treatment of soil with nontoxigenic strains
4 of *A. flavus* and *A. parasiticus* significantly reduces preharvest aflatoxin contamination. In
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9 addition, soil treatment with nontoxigenic strains had the beneficial carry-over effect of
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15 reducing aflatoxin contamination that occurred during storage.
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17 **Post-harvest**

18 *Storage*

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20 This step is critical in preventing mold growth and mycotoxin production in harvested
21 feedstuffs. Various important factors should be managed to prevent crops from fungal
22 contamination during storage (Schrödter, 2004). For example, grain should be stored with
23 less than 15% moisture content to eliminate pockets with higher moisture (Kabak *et al.*,
24 2006) at low temperature (Burgess and Burrell, 1964). A low oxygen concentration (<1%) and
25 an augmentation of carbon dioxide concentration are efficient to prevent mould development
26 (Driehuis and Oude-Elferink, 2000). Physical integrity of grains should be preserved. Mixing
27 grains and a long-time storage should be avoided.
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31 Despite all precautions, it may happen that stored grain will become damaged by
32 mould. It must then be assumed that the grain could also be contaminated with mycotoxins.
33 If the farmer has plenty of grain in store, he can afford to lose a small quantity that has turned
34 mouldy. Ideally, the farmer must discard mouldy grain and any that is suspected of being
35 contaminated with mycotoxins; this will include apparently clean grain that is in the vicinity of
36 the mouldy produce. This grain should be burnt or buried.
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45 *Sorting*

46 Contaminated grain does not have the same color or density as safe grain. Thus, grain can
47 be sorted according to appearance or density (Murphy *et al.*, 1993; Guerre, 2000; Afolabi *et*
48 *al.*, 2006; Kabak *et al.*, 2006). These methods are not very specific and in general not
49 exhaustive. When mycotoxin contamination is heterogeneous the removal of the
50 contaminated part could reduce the level of mycotoxin in the final product (Barnett *et al.*,
51 1978; Osborne *et al.*, 1996). For example, apple dissection before making apple juice led to
52 a 95% reduction of patulin (Lovett *et al.*, 1975). Washing of food or grain can also reduce
53 mycotoxin levels. For example, the first step in spaghetti production with wheat is a washing
54 which remove of 23% of DON (Visconti *et al.*, 2004).
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Eliminating mycotoxins from food and feed

Different methods must be used to decontaminate food and feed before ingestion. For the moment, regulations do not permit the decontamination of food that exceeds the limit concentration thresholds. Mycotoxin reduction could be carried out during food industry processes (Bullerman and Bianchini, 2007) or by using additives which eliminate or deactivate mycotoxins in the organism. 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 the contamination but will also reduce at the same time the nutritional value of the feed.

The most commonly-used technique for reducing exposure to mycotoxins is to decrease their bio-availability by the inclusion of various mycotoxin binding agents or adsorbents, which leads to a diminishing of mycotoxin uptake and subsequent distribution to the blood and target organs. These adsorbants are efficient only if the complex is stable in an animal digestive tract so that bound mycotoxins are channelled to its urine and feces.

An important criterion for the evaluation of mycotoxin adsorbants is their effectiveness at different levels of pH (acidic and neutral) since the adsorbant must be efficient throughout the entire gastro-intestinal tract and that the complex mycotoxin-adsorbant remains stable, in order 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 are efficient to bind aflatoxin but few of them 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 charcoal may not be as effective in binding aflatoxin as clay-based binders (Edrington *et al.*, 1996, 1997). Activated charcoal may be interesting 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 availability of ZEA, DON and nivalenol (Avantaggiato *et al.*, 2003, 2004). However, adsorption of ochratoxin A (OTA) does not lead to a positive effect on chicken body weight (Rotter *et al.*, 1989). On 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 animals. 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. Perhaps the most extensively studied of these materials is termed hydrated sodium calcium aluminosilicates (HSCASs). Several reviews are available (Ramos *et al.*, 1996; Döll and Dänicke, 2004; Avantaggiato *et al.*, 2005). 80% of AFB1 could be adsorbed by HSCASs *in vitro* (Phillips *et al.* 1988) and could prevent aflatoxicosis. Positive effects of other HSCASs on drosophile 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).

1 In general, HSCAS have low affinity with OTA (Huff *et al.*, 1992; Galvano *et al.*, 1998).
2 Adsorption results for ZEA are variable: some studies show a decrease in chromosomic
3 aberrations for mice (Abbes *et al.*, 2007) and a positive effect on the length of mink gestation
4 (Bursian *et al.*, 1992). Patterson and Young (1993), however, failed to see any benefit in the
5 addition of HSCAS to pig diets containing DON. This result was confirmed *in vitro* by
6 Galvano *et al.* (1998) and Sabater-Vilar *et al.* (2007). Garcia *et al.* (2003), using a silicate
7 material, demonstrated reduced T-2 toxicity while Kubena *et al.* (1990) did not see any effect
8 from the addition of HSCAS. Aly *et al.* (2004) showed an adsorption efficiency for a mix of
9 AFB1 and FB1 of, respectively, 95% and 85%. However, Watts *et al.* (2003) showed that 1%
10 HSCAS did not protect chicks and poults receiving diets containing 1 mg DON, 5 mg
11 moniliformin, 5 mg FB1, 100 µg AFB1, 1 mg ZEA and 0.5 mg OTA per kg of diet.

12 Other silicates that have been studied include bentonites, zeolites, clinoptilolites and
13 various others that often have not been completely characterized. Bentonite is a general clay
14 material originating from volcanic ash and containing primarily montmorillonite as the main
15 constituent. Montmorillonite clay is a hydrated sodium calcium aluminum magnesium silicate
16 hydroxide. Clays are silica sheets that are similar to other phyllosilicates but contain a high
17 concentration of water. The zeolite structure provides vacant spaces that form channels of
18 various sizes allowing movement of molecules into and out of the structure. Thanks to this
19 structure, zeolites present a very big specific surface (about 1000 m² per gramme of
20 zeolite).

21 Bentonite and montmorillonite could adsorb from 40% to 100% of OTA in wine
22 (Kurtbay *et al.*, 2008). However, Lemke *et al.* (2001a) failed to show a positive *in vivo* effect
23 on rats. Indeed, rat uterus weight increased with montmorillonite treatments suggesting an
24 increase of oestrogenic toxicity with treatment. A slight positive effect was observed by
25 Carson and Smith (1983a) for the addition of bentonite in T-2 toxin-contaminated food since
26 8% of body weight was recovered after treatment. A 9% body weight gain was observed for
27 chicken treated with bentonite when their feed contained a mix of AFB1 and FB1 (Miazzo *et al.*,
28 2005). Another study performed by Kubena *et al.* (1998) demonstrated that an HSCAS
29 could provide protection against AFB1 but not against T-2 toxin in young broiler chicks.

30 Zeolites are very efficient in bovine rumen juice since 100% of AFB1 was adsorbed
31 (Spotti *et al.*, 2005). Zeolites have not proven to reduce the toxicity of T-2 toxin (Dvorska and
32 Surai, 2001). However, some types of clinoptilolite could not be used as a binder since its
33 adding in presence of AFB1 caused severe lesions. This could be explained by a non-
34 specific adsorption of important compounds involved in aflatoxicosis regulation (Mayura *et al.*,
35 1998).

36 A number of studies have examined chemically-modified silicates. Döll *et al.* (2004)
37 examined a chemically-modified aluminosilicate that showed good binding with ZEA *in vitro*,
38 confirming previous work (Lemke *et al.*, 1998; Tomasevic-Canovic *et al.*, 2003). Others have

1 shown that chemical modifications have increased the binding of HSCAS with ZEA
2 (Pimpukdee *et al.*, 2004). Non-modified zeolithes are used to adsorb AFB1 while
3 hydrophobic zeolithes (octadecyldimethylbenzyl, an ammonium-treated zeolite) are mostly
4 used to adsorb OTA and ZEA (Dakovic *et al.*, 2003, 2005, 2007). The adsorption efficiency of
5 hydrophobic zeolithes could be explained by hydrophobic interactions between OTA and
6 ZEA and the binder.
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12 *Other mineral adsorbants.* Some synthetic polymers such as cholestyramine and
13 polyvinylpyrrolidone can adsorb mycotoxins. Undigestible dietary fiber has adsorbance
14 potential for mycotoxins. Fibers have reduced the effects of ZEA (Tangni *et al.*, 2006) in rats
15 and swine and T-2 toxin in rats (Carson and Smith, 1983b). Cholestyramine resin is used in
16 human medicine for the reduction of cholesterol and functions through adsorption of bile
17 acids. Cholestyramine has been shown to adsorb ZEA (Ramos *et al.*, 1996; Döll *et al.*, 2004;
18 Avantaggiato *et al.*, 2005) and FB1 (Solfrizzo *et al.*, 2001). In rats consuming OTA,
19 cholestyramine increased fecal OTA excretion (Kerkadi *et al.*, 1998). In another *in vivo* study,
20 cholestyramine did not bind OTA (Bauer, 1994). Only 5% of DON could be adsorbed by
21 cholestyramine in a dynamic digestive tract (Avantaggiato *et al.*, 2005).
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28 A synthetic water-soluble polymer, polyvinylpyrrolidone (PVP), has been investigated
29 as a binder for mycotoxins. PVP is reported to bind with AFB1 and ZEA *in vitro* (Alegakis *et al.*
30 *et al.*, 1999) but did not alleviate the toxicity of DON seen in pigs (Friend *et al.*, 1984).
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33 Some commercial adsorbants consist of a mixture of different adsorbants. For example, the
34 product Standard Q/FIS allies charcoal and HSCAS. These products are in general tested for
35 their capacity to adsorb various mycotoxins at the same time (Avantaggiato *et al.*, 2007).
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40 *Limits of mineral adsorbants.* Mineral adsorbants are in general efficacious against AFB1 but
41 their efficacy varies for other mycotoxins. Moreover, in some cases, the addition of clay
42 increases the effects of mycotoxicosis (Carson and Smith, 1983a; Mayura *et al.*, 1998;
43 Lemke *et al.*, 2001a). Furthermore, these adsorbants are not specific to mycotoxins and
44 could adsorb other types of molecules, some of which essential for fighting mycotoxicosis or
45 in nutrition (Mayura *et al.*, 1998).
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51 *Organic adsorbants.* Humic acids are complex organic substances, a component of humus.
52 They also have the capacity to adsorb mycotoxins, especially AFB1 and ZEA (Jansen van
53 Rensburg *et al.*, 2006) but not DON (Sabater-Vilar *et al.*, 2007).
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57 *Biological adsorbants*

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59 Because of the limitations of mineral adsorption, many studies have been conducted
60 over the last decade on biological adsorbents, trying to obtain greater efficacy and specificity

1 and at the same time reducing the impact on nutritional quality compared to mineral
2 adsorbents.
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5 *Yeast or yeast extract.* One example is *Saccharomyces cerevisiae* which has been shown to
6 bind with AFB1 (Shetty and Jespersen, 2006) and reduce the detrimental effects of AFB1 in
7 broiler diets (Stanley *et al.*, 1993) or on rats (Madrigal-Santillán *et al.*, 2006). The protective
8 effect of live yeast against aflatoxin was confirmed in rats but thermolysed yeast was shown
9 to be ineffective (Babista *et al.*, 2002). In contrast, some thermolysed yeast cell walls were
10 more efficient in adsorbing ZEA (Yiannikouris, 2004). Esterified glucomannan polymer
11 extracted from the yeast cell wall was shown to bind with AFB1, OTA (Bejaoui *et al.*, 2004;
12 Cecchini *et al.*, 2007; Angioni *et al.*, 2007) and T-2 toxin (Freimund *et al.*, 2003), individually
13 and in combination (Raju and Devegowda, 2000; Yiannikouris, 2004; Aravind *et al.*, 2003;
14 Karaman *et al.*, 2005). Additions of esterified glucomannan at 0.5 or 1.0 g/kg to diets
15 containig 2 mg of total aflatoxin resulted in dose-dependent responses in broiler chicks
16 (Basmacioglu *et al.*, 2005). The addition of esterified glucan polymer to aflatoxin-
17 contaminated diets of dairy cows has significantly reduced milk aflatoxin residues (Diaz *et al.*,
18 2004). The body weight and biochemical parameters were recovered in horses after adding
19 glucomannan to their diet (Raymond *et al.*, 2003).
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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). The glucan polymer product gave protective against depression in antioxidant
activities resulting from T-2 toxin consumed by growing quail (Dvorska and Surai, 2001). A
glucan polymer product has protected swine, broilers (Swamy *et al.*, 2002a, b) 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. A glucan polymer product did
not alleviate the toxic effects on 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.*, 2002a). 183 mg of T-2/HT-
2 toxin was adsorbed per gramme of yeast-modified glucan *in vitro* (Freimund *et al.*, 2003).
FB1 adsorption by yeast or yeast extract is limited (Yiannikouris, 2004). T-2 and HT-2
binding responses are 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

1 the bacterial surface (Haskard *et al.*, 2000; El-Nezami *et al.*, 2004). Adsorption efficiency
2 depends on strains (Peltonen *et al.*, 2001). Adsorption of AFB1 and AFM1 was reversible
3 and could be performed with living or dead bacteria. Acid or heat inactivation of lactic acid
4 bacteria increased adsorption efficiency (Haskard *et al.*, 2001; Pierides *et al.*, 2000; El-
5 Nezami *et al.*, 1998, 2000, 2002b; Bueno *et al.*, 2007; Fazeli *et al.*, 2009). OTA could also be
6 adsorbed by lactic acid bacteria (Fuchs *et al.*, 2002) and led to a diminution of toxicity on
7 human hepatic cells (Del Prete *et al.*, 2007). ZEA could be adsorbed between pH 4 and pH 8
8 (El-Nezami *et al.*, 2002b) and heat or acid inactivation increased adsorption efficiency
9 (Niderkorn *et al.*, 2006; El-Nezami *et al.*, 1998, 2002b). Binding on FB1 and DON by
10 *L.rhamnosus* was not a very efficient solution (Niderkorn *et al.*, 2006). Hydrophobic
11 interactions were suspected to be involved in the binding (Haskard *et al.*, 2001).

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21 *Other biological materials.* Fungal conidia are able to bind mycotoxins and especially ZEA
22 and OTA, separately or together (Jard *et al.*, 2009). From 29% to 60% of ZEA were eliminated
23 depending on the isolate and incubation time. Jard *et al.* (2009) suggested a hydrophobic
24 interaction based on conidia wall characteristics. It has been suggested that conidia could be
25 used to decontaminate wine affected by OTA (Bejaoui *et al.*, 2005).

26 27 28 29 30 *Binder efficiency*

31 Many solutions are available for binding mycotoxins but few of them are actually used
32 in an industrial context. Decreasing the bioavailability of AFB1 by the inclusion of binding
33 agents is particularly effective as this group of toxins has a chemical structure which favors
34 adsorption, especially by materials of mineral origin such as clay and zeolites. OTA, ZEA and
35 FB1 adsorption has been more extensively studied with biological adsorbents on account of
36 the lack of efficacy of mineral adsorbents. DON and T-2 or HT-2 toxin do not bind easily on
37 every kind of binding agents.

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

5 *Transformation by physical and chemical treatment*

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

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16 *Thermal treatment.* Mycotoxins are generally very stable and can hardly be eliminated by
17 thermic treatment (Kabak, 2009). Little or no reduction in mycotoxin levels occurs as a result
18 of normal cooking conditions such as boiling, frying. For example, DON is stable at 120°C,
19 moderately stable at 180°C and partially stable at 210°C (WHO, 2001). Fumonisin are
20 completely destroyed at 220°C (Dupuy *et al.*, 1993). Torrefaction process could eliminate
21 between 45 and 83% of aflatoxin content while panification or pasteurization is not efficient to
22 remove aflatoxin (CAST, 2003). Thermal treatment on ZEA contaminated food is not efficient
23 (Ryu *et al.*, 2003). The initial level of contamination, type of mycotoxin and its concentration,
24 heating temperature and time, the degree of heat penetration, the moisture content, pH, ionic
25 strength of food play a significant role in the achievement of toxin degradation (Rustom,
26 1997).

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34 *Degradation by extrusion.* Extrusion enables aflatoxins, DON, ZEA and FB1 to be removed
35 from maize (Cazzaniga *et al.*, 2001; Rustom, 1997; Voss *et al.*, 2008). Extrusion implies
36 molecular modifications such as protein denaturation and the inhibition of the enzymatic
37 activity.
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50 *Radiation.* Most mycotoxins have complex molecular structures and are not often affected by
51 irradiation. Radiolysis of water produces free radicals that would react with mycotoxins
52 (Stepanik *et al.*, 2007). AFB1 is sensitive to UV, X and gamma rays. Radiation of AFB1 is
53 able to reduce its level of contamination (Rustom, 1997; Afifi *et al.*, 2003). Gamma radiation
54 can also reduce microbial flora. Using micro-waves permit the reduction of aflatoxin content
55 inpeanuts (Farag *et al.*, 1996) and trichotecenes in corn (Scott, 1998).

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Oxydation. Some oxidizing agents like ozone and hydrogen peroxyde have been used to
render mycotoxin-contaminated feed harmless. Chemical oxidizing agents can react with
numerous functional groups. McKenzie *et al.* (1998) showed that a treatment of
contaminated corn with electrochemically produced O₃ 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 permitted the reduction of
microbiological and mycotoxin contamination but it seems to induce some side effects such
as ADN adducts in wheat. Abd Alla (1997) revealed that ZEA was degraded by 83.9% when

1 using 10% H₂O₂ at 80°C for 16h. It was confirmed by Lemke *et al.* (1999) showing ozone
2 could prevent the estrogenic effects of ZEA in mice. Biological activities of trichothecenes are
3 also changed by oxidation. Ozone is most likely to attack the double bond in trichothecenes
4 (McKenzie *et al.*, 1997).

7 *Reduction.* Reducing agents (ascorbic acid, NaHSO₃, Na₂S₂O₅) permitting the reduction of
8 such mycotoxins as AFB1 and DON (Kabak *et al.*, 2006). Sodium bisulfite transforming DON
9 to DON-sulfonate, which is less toxic than DON, was reported to be an effective tool for
10 overcoming the depressive effects of Don on feed-intake in piglets (Dänicke *et al.*, 2005).

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

18 *Ammoniation.* Maize ammoniation, largely used to diminish the level of aflatoxins in feed is
19 an efficient method for detoxifying feed that has been in use for several years (Park *et al.*,
20 1988). This process is particularly effective against AFB1 when carried out at high
21 temperature and pressure. One of the degradation products is AFD1, less toxic than AFB1.
22 However, this costly method is not effective against other mycotoxins and can damage food
23 quality because of an excessive level of ammonia in the food involved (Huwig *et al.*, 2001).

26 *Alkalization.* Under alkaline conditions, mycotoxins structure can change. For example, under
27 alkaline conditions, the 12-13 epoxy group of DON could be opened (Bretz *et al.*, 2006).

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

35 *Deamination.* Adding NaNO₂ to an aqueous medium deaminates FB1 and so reduces its
36 toxicity (Lemke *et al.*, 2001b).

41 *Transformation by microorganisms and enzymes*

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

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

55 *AFB1 transformation.* Removal and detoxification of AFB1 by transformation have been
56 investigated for many years. Unfortunately, few studies have led to the identification of the
57 transformed product. One of the first bacteria studied for its capacity to remove AFB1 was
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1 *Flavobacterium aurantiacum* (also known as *Nocardia corynebacterioides*). Its crude extract
2 was shown to remove AFB1 (Ciegler *et al.*, 1966; Hao and Brackett, 1989). Further studies
3 had shown that the transformation did not lead to a toxic product (Lillehoj *et al.*, 1967) and
4 that an intracellular enzyme was involved (Smiley and Draughon, 2000). Guan *et al.* (2008)
5 succeeded in obtaining *Stenotrophomonas maltophilia* isolates on selective medium
6 containing only coumarin (chemical component of AFB1 nucleus) as the carbon source and
7 showed that it is capable of transforming AFB1. *Bacillus subtilis* was also able to detoxify
8 AFB1-contaminated feed and thus facilitate animal growth rate (Kubo, 1996; Petchkongkaew
9 *et al.*, 2008). Actinomyceta like *Mycobacterium fluoranthenivorans* was shown to remove
10 AFB1 from contaminated media (Hormisch *et al.*, 2004; Teniola *et al.*, 2005). Teniola *et al.*
11 (2005) succeeded in isolating extracellular enzymes from *Rhodococcus erythropolis*,
12 responsible for the transformation of AFB1. Moreover, Alberts *et al.* (2006) observed the
13 elimination of toxicity using the Ames test after transformation. Nakazato *et al.* (1990) and
14 later Shantha (1999) observed the removal of AFB1 by moulds but no transformation product
15 was observed. Extracellular enzymes from the macroscopic fungus *Pleurotus ostreatus* were
16 shown to be able to cleave the AFB1 lactone ring Motomura *et al.* (2003) leading to a loss of
17 toxicity. *Armillariella tabescens* is able to open the AFB1 difuran cycle (Liu *et al.*, 1998,
18 2001).

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32 *OTA transformation.* Certain bacteria, moulds, yeasts and plants are able to transform OTA.
33 Among these microorganisms, many transform OTA into OT α (Galtier and Alvinerie, 1976;
34 Kiessling *et al.*, 1984; Hwang and Draughon, 1994; Wegst and Lingens, 1983 ; Skrinjar *et al.*,
35 1996; Abrunhosa *et al.*, 2002; Péteri *et al.*, 2007), a less toxic compound. This transformation
36 leads to a formation of phenylalanine (Figure 1). Moulds like *Aspergillus*, *Rhizopus*,
37 *Penicillium* species are particularly effective at removing OTA (Abrunhosa *et al.*, 2002;
38 Bejaoui *et al.*, 2006; Varga *et al.*, 2000). *Aureobasidium pullulans* was used as a biocontrol
39 agent in wine, preventing OTA accumulation in grapes and decreasing the aspergillosis
40 symptom (De Felice *et al.*, 2008). Plants like wheat and maize (Ruhland *et al.*, 1996) or fungi
41 like *P. ostreatus* (Engelhardt, 2002) are able to remove OTA but no transformation products
42 have been identified. *Trichosporon mycotoxinivorans* is a microorganism that has been
43 developed into a commercial product for detoxifying OTA in animal feed (Molnar *et al.*, 2004).
44 Total transformation of OTA to OT α occurred in 2.5 h. Moreover, Schatzmayr *et al.* (2003,
45 2006) showed that the toxic effects of OTA could be alleviated by the addition of this yeast to
46 chicken diet. The main microorganisms which transform OTA into Ota use
47 carboxypeptidases (EC 3.4.17.1). Purified enzymes are able to cleave OTA and have been
48 characterized (Stander *et al.*, 2000; Abrunhosa *et al.*, 2006).

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ZEA transformation. ZEA can be transformed into an oxydised compound such as
zearalanone, hydroxyl such as α and β ZEA, methyl compounds, gluco- or sulfo-conjugates

1 and hydrolysed compounds (Table 1) by bacteria, yeasts, moulds or plants (El-Sharkawy and
2 Abul-Hajj; 1987a, b, 1988). The transformation of ZEA to α -zéaralénol (McMullen, 1977;
3 Kiessling *et al.*, 1984; Kamimura, 1986; Böswald *et al.*, 1995) does not lead to actual
4 detoxification since this product has an oestrogenic activity even higher than that of ZEA
5 (Minervini *et al.*, 2005, Fitzpatrick *et al.*, 1989). A proteic sequence of a ZEA esterase
6 transforming ZEA into decarboxylated ZEA from a *Rhodococcus* or *Nocardia* species has
7 been patented for insertion in transgenic plants (Duvick and Rood, 2000; Karlovsky *et al.*,
8 2003). *Trichosporon mycotoxinivorans* is also able to decarboxylate ZEA (Molnar *et al.*,
9 2004; Vekiru *et al.*, 2010). Some conjugates can also be formed from ZEA, such as ZEA-
10 glucoside (El-Sharkawy and Abul-Hajj, 1987a; Kamimura, 1986) and ZEA-sulfate (El
11 Sharkawy *et al.*, 1991; Plasencia and Mirocha, 1991). Recently, Jard *et al.* (2010), using a
12 MCF-7 cell line, showed that ZEA sulfonation leads to a reduction in oestrogenic toxicity.
13 This confirms the study done by Plasencia and Mirocha (1991), using the uterus weight test,
14 showing a decline in toxicity by ZEA sulfonation. However, it has not been proven that
15 sulfonation or glycosylation leads effectively to a detoxification insofar as hydrolysis of this
16 conjugate could occur in the digestive tract. Moreover, plants are able to transform ZEA into
17 ZEA-glycoside (Schneweis *et al.*, 2002) leading to masked mycotoxins which could not be
18 measured but could be released by feeding with contaminated plants. Other degradation
19 products have been observed, including decarboxylated and hydrolysed compounds (El-
20 Sharkawy and Abul-Hajj, 1988; Kakeya *et al.*, 2002; Takahashi-Ando *et al.*, 2002; Igawa *et al.*,
21 2007; Duvick and Rood, 2000; Karlovsky *et al.*, 2003). The corresponding gene of this
22 enzymatic activity was cloned and transferred to different organisms like yeast (Takahashi-
23 Ando *et al.*, 2005) and plants (Kakeya *et al.*, 2002; Takahashi-Ando *et al.*, 2002; Igawa *et al.*,
24 2007). Utermark and Karlovsky (2007) showed that ZEA lactonohydrolase from
25 *Clonostachys rosea* (El-Sharkawy and Abul-Hajj, 1988) prevents the growth inhibition
26 observed for others types of fungi growing on ZEA-contaminated media. Gromadzka *et al.*
27 (2009) showed that this fungus could be used as a biocontrol agent for mycotoxin production
28 in cereals. *Pseudomonas sp.*, bacteria from soil are able to remove ZEA (El-Deeb, 2005).
29 The product was not identified but was assumed to be less toxic than ZEA. Megharaj *et al.*
30 (1997) showed that a mixed culture from soil was able to remove ZEA. No transformation
31 product was observed. An unidentified bacterium from pig intestine was also able to remove
32 ZEA (Kollarczik *et al.*, 1994). Some microorganisms have the capacity to remove ZEA
33 producing different derived compounds. For example, El Sharkawy *et al.* (1991) identified
34 26% of ZEA-sulfate and 18% of α -ZOL after a ZEA transformation by *Rhizopus*. An enzyme
35 from *Pseudomonas sp.* responsible for the transformation into a less toxic product is
36 encoded by a plasmid (Skrinjar *et al.*, 1996). Cheng *et al.* (2006) studied an enzyme able to
37 transform ZEA but its origin and transformation product(s) are unknown.
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1 *Trichothecenes transformation.* Trichothecenes transformation by microorganisms was
2 reviewed by Zhou *et al.* (2008) and He *et al.* (2010). The 12,13-epoxy cycle of DON and of T-
3 2 toxin seems to be the part of the molecule which is responsible for the toxicity. By opening
4 this cycle, the mycotoxin become less toxic (Swanson *et al.*, 1988; Zhou *et al.*, 2008).
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7 Some studies report a transformation of DON by mixed cultures of microorganisms
8 but few of these have been identified. The two main metabolites are de-epoxydised DON
9 (Kollarczik *et al.*, 1994; Young *et al.*, 2007; Yoshizawa *et al.*, 1983; Côté *et al.*, 1986; He *et*
10 *al.*, 1992; Swanson *et al.*, 1988; King *et al.*, 1984) and 3-keto-DON (Young *et al.*, 2007;
11 Binder and Binder, 2004; Shima *et al.*, 1997). The chemical structures of these DON
12 metabolites are presented on Table 2. Both are less toxic than DON. For example, Shima *et*
13 *al.* (1997) observed a weaker immunosuppressive activity for 3-keto-4-DON produced by
14 *Agrobacterium-Rhizobium E 3-39* than for DON.
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16 The *Eubacterium sp.* strain BBSH 797 has been developed into a commercial product
17 Mycofix plus (Biomin®) for detoxifying trichothecenes in animal feed (Schatzmayr *et al.*,
18 2006). An enzyme named MDE (mycotoxin-degrading enzyme), whose origin is not specified
19 is able to degrade DON with ZEA (Cheng *et al.*, 2006). Some transformation products have
20 not yet been identified (Binder and Binder, 2004; Völkl *et al.*, 2004; Guan *et al.*, 2009).
21 Among these transformations, one was done by a bacterium from fish intestine, achieving
22 100% removal in 96h.
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24 Few studies exist concerning the transformation of T-2 and HT-2 toxins. Some
25 metabolites were identified to be de-acetylate and de-epoxydised T-2 (Table 3). Some
26 microorganisms were able to transform T-2 toxin into HT-2 toxin but this did not lead to
27 detoxification (Fuchs *et al.*, 2002; Swanson *et al.*, 1988). Bacteria isolated by enrichment
28 from contaminated soil or water can transform T-2 toxin into T-2 triol and T-2 tetraol (Beeton
29 and Bull, 1989). A similar transformation was observed for carboxyl esterase isolated from
30 rat liver (Johnsen *et al.*, 1986). Some moulds can transform T-2 toxin but the transformation
31 products were not identified (Jesenska and Sajbidorova, 1991). The metabolism of T-2 toxin
32 was elucidated by Swanson *et al.* (1988) and Beeton and Bull (1989). It includes a sequence
33 of different steps: the molecule is first transformed into HT-2 toxin by deacetylation. A second
34 deacetylation was observed to transform HT-2 toxin into T-2 triol, 20 times less toxic than the
35 T-2 toxin (Ueno *et al.*, 1983). Finally, this molecule is transformed into T-2 tetraol. Other
36 metabolites observed are the de-epoxyed metabolites cited previously (Table 3).
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53 *Fumonisin B1 transformation.* The primary amine of FB1 confers its toxicity. The deamination
54 of this molecule greatly reduces its toxicity. Very few studies have been done on the
55 biological degradation of FB1. The main microorganism able to degrade FB1 is the black
56 yeast *Exophiala spinifera*. The different metabolites produced by this yeast are presented in
57 Table 4. The transformation of FB1 into AP1 is performed by an extracellular
58 carboxylesterase. This enzyme has been cloned and was shown to be efficient in transgenic
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1 maize, as the plant became resistant to fumonisin (Duvick *et al.*, 2003). Other enzymes are
2 involved in FB1 degradation and some derived products have been characterised (Blackwell
3 *et al.*, 1999). A patent was taken out in 2003 (Duvick *et al.*, 2003) for esterase used to
4 detoxify fumonisins.
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8 **Conclusion**

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10 Although there are many publications on the removal of mycotoxins by adsorption and
11 transformation, their applications in detoxification have been limited.

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13 Chemical treatment for the detoxification of aflatoxins using ammonia is the only application
14 currently licensed in the United States. Chemical treatment is not allowed within the
15 European Union for products used by humans (EU Commission Regulation, 2001). Recourse
16 to chemical transformation may lead to toxic derivatives in the treated products. These side-
17 effects limit their use in the human and animal food chains (Kabak *et al.*, 2006) with the
18 decreased interest in chemical transformations, interest has been increasingly focused on
19 adsorption or transformation of mycotoxins. Adsorption by yeast cell walls added to feed is
20 used most commonly in the industrial context. Legislation is changing to allow the marketing
21 of mycotoxin binders. Up to now, these products have been sold for other applications such
22 as increasing antioxidant activity. It has proved difficult for governments to legislate on this
23 topic without encouraging suppliers to sell contaminated commodities. Mycotoxin adsorption
24 is quite well documented and was tested *in vitro* in a gastrointestinal tract which has enabled
25 us to better determine the stability of the binder-mycotoxin complex (Avantaggiato *et al.*,
26 2004, 2007).
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36 Transformation of mycotoxins for detoxification is not much used in industry. This may be
37 due to the lack of information about the transformation mechanisms, the toxicity of products
38 derived from transformation, and the effect of transformation reactions on the nutritional
39 value of food and feed. In some cases, transformation products have not been identified and
40 so cannot be used in industrial processes. The stability of transformations and their potential
41 side-effects should be investigated.
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46 Indeed, the use of a microorganism for detoxifying mycotoxins in feed requires the
47 observance of certain conditions:
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- 49 - The first step is identification of a microorganism's degradation potential;
- 50 - Each product must be identified and its toxicity must be tested, using different
51 methods if possible;
- 52 - Derived products must be non-toxic;
- 53 - Biotransformation must be fast and microorganisms must be efficient at different
54 levels of oxygen or pH, especially when transformation takes place during digestion;
- 55 - Microorganisms must be non-pathogenic;
- 56 - Microorganisms must be active in a complex environment and not inhibited by
57 nutrients;
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- 1 - The efficiency of the transformation must be assessed *in vivo*.

2 The use of microbial detoxification agents (living cells) in the human food and animal feed
3 industries seems to be diminishing on account of regulatory, toxicological and consumer
4 considerations. Thus, applications of detoxification enzymes and genes can represent
5 alternative detoxification methods. Some interesting studies have been carried out using
6 molecular engineering techniques to obtain genes (Takahashi-Ando *et al.*, 2004). Such
7 genes were obtained, cloned and incorporated into microorganisms to produce recombinant
8 enzymes that are suitable for industrial-scale enzyme production and purification (Althali and
9 Deeb, 2009).

10 Today, chemical transformation is no longer suitable for detoxifying mycotoxin-contaminated
11 food and feed. Mycotoxin binding may be a feasible industrial solution because of its relative
12 low cost compared to biotransformation. However, this technique can lead to negative side-
13 effects. Indeed, adsorption could be reversible as a mycotoxin could desorb in the digestive
14 tract, and adsorption can be non-specific thus decreasing overall nutritional value (Phillips *et*
15 *al.*, 2008). Mycotoxin transformation appears to offer great interest as a technique for
16 increasing the quality of food and feed. Studies involving biotransformation have led to
17 encouraging results. But, further studies must be done to ensure the safety of this method.
18 The transformation of mycotoxins in food and feed promises to be a very reliable technique
19 for enhancing food safety and eliminating all negative effects of the mycotoxins present.

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Tables

Table 1: Chemical structure of zearalenone metabolites

ZEA and its metabolites	Chemical structure
ZEA	
Oxydised Compounds	
Zearalanone	
Hydroxylated and methyl compounds	
α - β zearalenol	
α - β zearalanol	
Methoxy-ZEA	
Hydroxy-ZEA	
Gluco- and sulfo-conjugates	
ZEA-4- β -glucopyranoside	
ZEA-4-sulfate	
Hydrolysed compounds	
Decarboxylated ZEA	
Hydroxylated ZEA	

Table 2: Chemical structure of deoxynivalenol metabolites

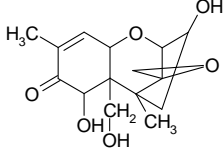
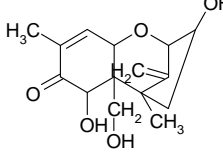
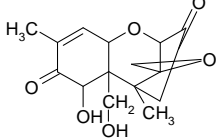
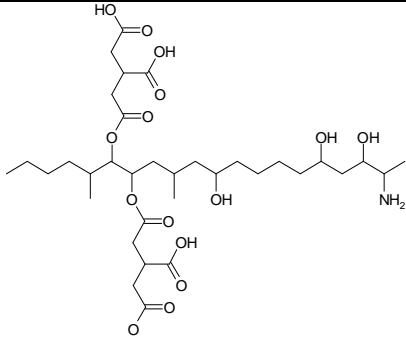
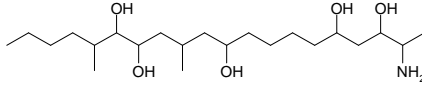
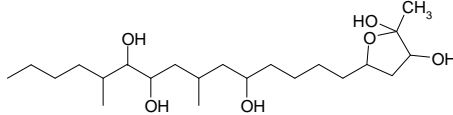
DON and its metabolites	Chemical structure
DON	
De-epoxy DON	
Ketonic compound	

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-2 triol	
T-2 tetraol	
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	
Polyol amine (AP1)	
2-OP1	

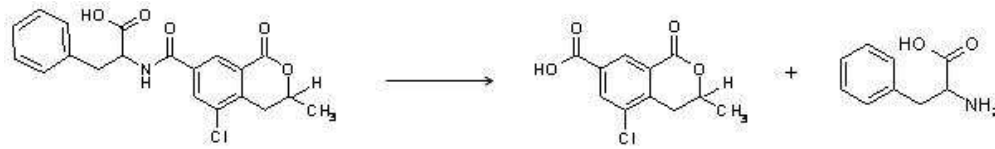


Figure 1: Transformation of OTA to Ota and phenylalanine
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