Journal of Food Protection, Vol. 72, No. 9, 2009, Pages 2006–2016

Review

Biological Strategies To Counteract the Effects of Mycotoxins

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MS 09-003: Received 3 January 2009/Accepted 8 April 2009

ABSTRACT

Mycotoxins are fungal secondary metabolites that if ingested can cause a variety of adverse effects on both humans and animals, ranging from allergic responses to death. Therefore, exposure to mycotoxins should be minimized. A variety of physical, chemical, and biological methods have been developed for decontamination and/or detoxification of mycotoxins from contaminated foods and feeds. This overview details the latest developments in the biological control of both fungal infection and mycotoxin formation and describes the detoxification of many of the most important mycotoxins by microorganisms. This review also addresses the potential for use of microorganisms as mycotoxin binders in the gastrointestinal tract of both humans and animals, thereby reducing the potential deleterious effects of exposure to these toxins.

Mycotoxins are secondary metabolites produced by fungi belonging predominantly to the *Aspergillus, Penicillium,* and *Fusarium* genera. These toxins can contaminate foods and animal feeds and cause several toxic effects in vertebrates. Although *Aspergillus* and *Penicillium* species are generally found as contaminants in food during drying and storage, *Fusarium* species can produce mycotoxins before or after harvesting *(125)*. Mycotoxin contamination of agricultural products is a global problem but is most severe in tropical and subtropical regions. The Food and Agriculture Organization *(53)* has estimated that up to 25% of the world's food crops are significantly contaminated with mycotoxins. The mycotoxins of most significance from both a public health and agronomic perspective are the aflatoxins, ochratoxin A (OTA), fumonisins, deoxynivalenol (DON or sometimes called vomitoxin), and zearalenone (ZEA). However, there is recent growing interest among researchers in other mycotoxins such as moniliformin, patulin, T-2 toxin, and HT-2 toxin *(55)*. In 1993, the World Health Organization International Agency for Research on Cancer *(68)* evaluated mycotoxins with respect to their carcinogenic potential. Naturally occurring aflatoxins were classified as being carcinogenic to humans (group 1), whereas aflatoxin M_1 (AFM_1), OTA, and fumonisins were considered as possible carcinogens (group 2B). ZEA, DON, nivalenol (NIV), fusarenone X, and T-2 toxin were not classified as human carcinogens (group 3).

Mycotoxins can cause acute and chronic intoxications in both humans and animals depending on various factors including intake levels, duration of exposure, toxin type, mechanisms of action, metabolism, and defense mechanisms *(65)*. Mycotoxins exhibit four basic kinds of toxicity: acute, chronic, mutagenic, and teratogenic. The most commonly described effect of acute mycotoxin poisoning is deterioration of liver or kidney function, which in extreme cases may lead to death. However, some mycotoxins act primarily by interfering with protein synthesis and produce effects ranging from skin sensitivity or necrosis to extreme immunodeficiency *(104)*. For this reason, many of the developed countries have adopted regulations stipulating the maximum permissible levels for many important mycotoxins in foods and feeds. Although clear regulatory limits exist for European Union countries, the limits differ among other countries.

Three main strategies can be used to help prevent the toxic effects of mycotoxin-contaminated food and animal feeds *(7)*:

- prevention of mycotoxin contamination during the preharvest and postharvest periods,
- detoxification of mycotoxins present in foods and feeds, and
- inhibition of mycotoxin absorption in the gastrointestinal tract.

Preventive measures aimed at the inhibition of mycotoxin formation in agricultural products are the most effective approach for avoiding consumer exposure. However, when contamination is not prevented during the preharvest and postharvest periods, several approaches can be employed to help remove mycotoxins from the subsequently contaminated commodities, including physical, chemical, and biological techniques. Several comprehensive reviews on the detoxification of mycotoxins by physical and chemical means have been published in the last three decades. However, the use of many of the available physical and chemical methods for the detoxification of

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food products contaminated with mycotoxins can sometimes be restricted primarily because of problems concerning safety issues, possible losses in the nutritional quality of treated commodities, limited efficacy, and cost implications *(69)*. These issues have prompted a search for alternative strategies such as the use of biological agents for decontamination, and many microorganisms have been tested for their ability to degrade many of the most important mycotoxins. Among these organisms, *Flavobacterium aurantiacum* is the most effective for detoxifying AFB_1 in various food products such as milk, peanuts, maize, and paprika without producing toxic end products. There also has been increased interest in the use of mycotoxin-binding agents that can be added to the diet to bind mycotoxins in the gastrointestinal tract. In recent years, much interest has been directed at the addition of yeasts, yeast-derived products, and probiotic bacteria to the diet (instead of chemical sorbents).

In this review, recent developments in prevention of mycotoxin formation by microorganisms during preharvest and postharvest, biological detoxification strategies, and the potential use of microorganisms and their derived products as mycotoxin binders in the diet are discussed.

PREVENTION OF CONTAMINATION BY MYCOTOXIGENIC FUNGI AND MYCOTOXINS

Preharvest. The use of nontoxigenic biocompetitive microorganisms is one of the most recent approaches being employed to reduce mycotoxin contamination in crops. Nonaflatoxigenic strains of *Aspergillus flavus* and *Aspergillus parasiticus* have been applied to reduce the levels of aflatoxin contamination in agricultural products such as peanuts *(33),* maize *(34),* and cottonseed *(25)*. The effect is believed to be mediated mainly through competition for substrate and through the production of inhibitory metabolites. A patent has been granted for the use of nonaflatoxigenic strains of *A. parasiticus* for control of aflatoxin contamination *(22)*. When late-season drought conditions make peanuts susceptible to invasion and growth by aflatoxigenic fungi, nonaflatoxigenic biocontrol fungal strains can competitively exclude the toxigenic strains present in the soil and thereby reduce subsequent aflatoxin levels *(33)*. In a 2-year study, Dorner et al. *(32)* found that the application of nonaflatoxigenic strains of *A. flavus* and *A. parasiticus* at different rates to peanut soil reduced aflatoxin contamination of peanuts by 74.3 to 99.9%. Similarly, Cotty *(25)* found that the application of nonaflatoxigenic strains of *A. flavus* to soils around developing cotton plants was effective for controlling aflatoxin production in cottonseed. A patent also has been issued for the use of nonaflatoxigenic *A. flavus* strains to prevent aflatoxin contamination *(24)*. In another study, an atoxigenic strain of *A. flavus* reduced aflatoxin contamination by 80 to 95% in maize *(14)*.

Two strains of nonaflatoxigenic *A. flavus,* AF36 and NRRL 21882, are currently being used to reduce aflatoxin contamination in crops. *A. flavus* AF36 is considered a biopesticide by the U.S. Environmental Protection Agency as has been approved for the management of aflatoxin-producing fungi during cotton production. *A. flavus* NRRL 21882 has been used to prevent aflatoxin contamination in peanuts *(18)*. The biopesticide Afla-guard was used commercially for the first time on the 2004 peanut crop in the southeastern United States *(35)*.

For atoxigenic strains of *A. flavus* and *A. parasiticus* to be effective for protecting crops by reducing the levels of mycotoxin contamination, they must be applied at a time and in a manner that allows successful competition with aflatoxin producers. In theory, the nonaflatoxigenic strain should be applied when the overall *A. flavus* levels are low, thereby providing the applied strain with preferential exposure to the crop and a subsequent advantage when competing for crop resources. Atoxigenic strains are routinely applied once per growing season at levels of 11.2 to 22.4 kg ha^{-1} *(20)*.

Transgenic approaches also are being developed as an additional strategy aimed at either preventing mycotoxin biosynthesis or detoxifying mycotoxins in the crops themselves. Such approaches could provide further protection for the grower in environments in which fumonisins present a risk to the crop even when the maize is relatively resistant to *Fusarium* infection *(39)*. Two fungal species, *Exophiala spinifera* (a ''black yeast'') and *Rhinocladiella atrovirens,* with a bacterium believed to be either a *Xanthomonas* or a *Sphingomonas* species have been isolated from field-grown maize kernels in an effort to identify a biological means to facilitate the detoxification of fumonisins. These organisms were isolated based on their ability to use fumonisin B_1 (FB_1) as a sole carbon source and thus are a potentially valuable source of fumonisin detoxifying enzymes. The genes encoding the deesterification and oxidative deamination enzymes that degrade fumonisins have been cloned and expressed in transgenic maize *(40)*. Other potentially useful genes associated with FB_1 degradation also may be present in the recently isolated bacterium belonging to the *Delftia-Comamonas* group, which was isolated from soil samples. The bacterium completely degraded FB_1 after incubation at 25°C for 1 day, even when the mycotoxin was present as the sole carbon source *(10)*.

Antagonistic microorganisms can reduce growth in *Fusarium* species and thus reduce the levels of DON production. In glasshouse studies involving the preinoculation of wheat ears at anthesis with the two nonpathogens *Phoma betea* and *Pythium ultimum,* reductions in disease development and severity from infection with *Fusarium culmorum, Fusarium avenacum, Fusarium poae,* and *Microdochium nivale* have been reported *(30)*. A strain of *Fusarium equiseti* (G9) has been reported to be consistently effective in controlling *Fusarium* head blight (FHB) and reducing DON formation (by more than 70%) on wheat *(27)*.

Yeasts of the genus *Cryptococcus* also are effective against FHB, with *Cryptococcus* sp. OH 71.4, OH 181.1, and OH 182.9 having the highest potential for reduction of FHB (up to 59%) on durum wheat in the field *(76)*. Important factors for the successful application of FHB antagonists in the field include the potential deleterious effect of UV light, variable and sporadic arrival of pathogen inoculum on wheat heads over an extended period of head

susceptibility, and the phylloplane environment with marked fluctuates in temperature, moisture, and available nutrients *(114)*. Several saprophytic yeast strains isolated from the fruits of almond, pistachio, and walnut trees also inhibit aflatoxin biosynthesis at an earlier step in the polyketide pathway *(64)*. Four of 54 microbial strains that were obtained from wheat anthers and that utilized tartaric acid in vitro were effective against *Gibberella zeae,* whereas only 3 of 170 isolates tested that did not utilize tartaric acid were effective against this pathogen, showing the potential benefit of prescreening candidate FHB antagonists for their ability to utilize tartaric acid *(76)*. In addition, four yeasts (NRRL Y-30213, NRRL Y-30214, NRRL Y-30215, and NRRL Y-30216) and a bacterium (*Bacillus* sp. NRRL B-30212) have been identified as excellent antagonists that are capable of suppressing FHB in cereals, particularly wheat and barley *(112)*. In another report, three bacterial isolates *Bacillus subtilis* H-08-02, *Bacillus cereus* L-07-01, and *Bacillus mycoides* S-07-01 inhibited *Fusarium graminearum* by 60, 52, and 55%, respectively *(50),* whereas *Lysobacter enzymogenes* strain C3 significantly reduced FHB severity in the Russ wheat cultivar but had little or no effect on disease development in the other wheat cultivars such as Alsen, Ingot, and Norm *(134)*. Treatment of heads of FHB-susceptible wheat with a *Streptomyces* sp. reduced both FHB disease severity and associated loss in grain weight by approximately 50% under glasshouse conditions *(92)*. Khan and Doohan *(75)* found that the use of *Pseudomonas fluorescens* strains MKB 158 and MKB 249 and *Pseudomonas frederiksbergensis* strain 202 reduced the severity of FHB disease symptoms caused by *F. culmorum* in wheat and barley grown under both glasshouse and field conditions, and a pseudomonad isolated from wheat anthers (*Pseudomonas* sp. AS 64.4) was as effective as the fungicide tebuconazole for controlling FHB disease severity under field conditions *(113)*. Treatment with either of the two *P. fluorescens* strains (MKB 158 and MKB 249) also resulted in a 74 to 78% reduction in DON levels in wheat and barley grains in the *F. culmorum*-inoculated field trails *(75)*. In another study, bacterial strains isolated from wheat anthers in Argentina reduced the growth of *F. graminearum* and decreased the production of DON on irradiated wheat grains by 60 to 100% *(94)*.

Postharvest. Some studies have highlighted the capacity of antagonistic bacteria, fungi, and yeasts to counteract mycotoxin formation postharvest. Antagonistic yeasts can reduce the growth of spoilage molds both in vitro and under simulated full-scale storage conditions *(101)*. *Pichia anomala* and *Saccharomyces cerevisiae* were effective for reducing OTA accumulation in vitro in two isolates of *Penicillium verrucosum,* and *P. anomala* reduced OTA synthesized by *P. verrucosum* from 100,000 ng g^{-1} to <10 ng g^{-1} in wheat at 25°C after 21 days *(100)*. *Kluyveromyces* isolates Y_{14}, Y_{16}, Y_{22}, and Y_{25} also can inhibit both growth of *Aspergillus* species in section Flavi and AFB_1 accumulation *(79)*. Yeast species isolated from grape berries (*Issatchenkia orientalis, Metschnikowia pulcherrima, Issatchenkia terricola,* and *Candida incommunis*) reduced colonization of grape berries by *Aspergillus carbonarius* and *Aspergillus niger,* the main species responsible for the production of OTA in grapes *(11)*.

Shantha *(118)* reported that several fungal cultures including a *Phoma* species, a *Mucor* species, *Trichoderma harzianum, Trichoderma* sp. 639, *Rhizopus* sp. 663, *Rhizopus* sp. 710, *Rhizopus* sp. 668, an *Alternaria* species, and some strains belonging to the *Sporotrichum* group can inhibit AFB_1 biosynthesis by $\geq$90%. AFB_1 production by *A. flavus* also can be inhibited by *Fusarium proliferatum* when both fungi are cocultured under optimal growth conditions *(102)*. Two antifungal compounds, fusapyrone and deoxyfusapyrone, isolated from rice cultures of *Fusarium semitectum* exhibited toxic activity against some pathogenic and mycotoxigenic fungi, including *Alternaria altarnata, Aspergillus fumigatus, Penicillium* spp., *Phoma tracheiphila, Ascochyta rabiei, Cladosporium* spp., and *Botrytis cinerea (48)*. Similarly, aflastatin isolated from *Streptomyces* sp. MRI 142 at a concentration of 0.5 μg ml^{-1} completely inhibited aflatoxin production by *A. parasiticus (111)*. In another study, the culture filtrate from *Rhodococcus erythropolis* at a concentration of 25 ml kg^{-1} completely inhibited AFB_1 production by *A. flavus* in stored rice, and biocontrol agents *P. fluorescens, Trichoderma virens,* and *B. subtilis* at a concentration of 200 ml kg^{-1} produced a 93, 80, and 68% reduction in *A. flavus* growth and a 83.7, 72.2, and 58% reduction in AFB_1 levels, respectively *(108)*. Because enzymes present in the extracellular extracts of *R. erythropolis* are involved in the degradation of AFB_1 *(2),* the inhibitory activity observed in the extracellular extracts of these strains is likely to be enzymatically mediated. In another study, Pereira and coworkers *(97)* reported that treatment of seeds with *Bacillus amyloliquefaciens* and *Microbacterium oleovorans* reduced *Fusarium verticillioides* counts in maize grains and significantly decreased the amounts of FB_1 and FB_2 in grains from plants grown from the treated seeds. Gwiazdowska et al. *(61)* reported that *Propionibacterium freudenreichii* subsp. *freudenreichii* strain 111 completely inhibited both DON and NIV production by *F. graminearum,* and supernatants from the bacterium caused 37.5 and 34.7% reductions in DON and NIV formation, respectively. A *Propionibacterium* culture and a cell-free supernatant fraction from the culture also decreased fumonisin production by *F. verticillioides* by 90 and 88.4%, respectively.

Numerous recent reports have dealt with the antifungal properties of various lactic acid bacteria (LAB) (Table 1), which can exhibit activities against a broad range of mycotoxigenic fungi. These LAB are of special interest as biopreservation organisms because they have a long history of use in food and have been designated as generally regarded as safe *(86)*. Biopreservation refers to extended shelf life and enhanced safety of foods obtained by using natural or added microflora and their antimicrobial products *(115)*. These LAB produce antimicrobial compounds, e.g., the pH-reducing fermentation products lactic acid and acetic acids, hydrogen peroxide, bacteriocins, and low-molecular-weight proteinaceous compounds, during carbon source metabolism, and they compete with other species by acidifying the environment and rapidly depleting nutrients *(29)*.

TABLE 1. *Antifungal activity of lactic acid bacteria*

LAB isolate	Activity against	Inhibitory metabolites	Reference
Lactobacillus casei ATCC 393	*A. parasiticus*	ND[a]	El-Gendy and Marth *(41)*
Lactococcus lactis C10	*A. parasiticus*	ND	Wiseman and Marth *(131)*
L. lactis ATCC 11454	*A. flavus*	Heat-stable low-molecular-weight compounds	Coallier-Ascah and Idziak *(21)*
L. acidophilus, *L. delbrueckii* subsp. *bulgaricus*, *L. plantarum*	*A. parasiticus*	ND	Karunaratne et al. *(74)*
L. casei subsp. *pseudoplantarum*	*A. flavus*	Possibly proteinaceous	Gourama and Bullerman *(58)*
Lactococcus lactis subsp. *lactis* CHD 28.3	*A. flavus*, *A. parasiticus*, *Fusarium* spp.	Proteinaceous compounds	Roy et al. *(110)*
L. casei subsp. *pseudoplantarum* 371	*A. parasiticus*	Possibly proteinaceous	Gourama and Bullerman *(59)*
L. casei	*Penicillium citrinum*, *P. expansum*	Possibly proteinaceous	Gourama *(57)*
L. acidophilus CH5	*Penicillum* sp., *Mucor* sp., *Fusarium* sp.	Acidocin CH5 and fermentation products	Plocková et al. *(105)*
L. sanfrancisco CB1	*Fusarium* spp., *Penicillium* spp., *Aspergillus* spp., *Monilia* spp.	Caproic, acetic, formic, propionic, buytric, n-valeric acids	Corsetti et al. *(23)*
L. plantarum VTT E78076	*F. avenaceum*	Benzoic acid, methylhydantoin, mevalonolactone, cyclo (Gly-L-leucyl)	Niku-Paavola et al. *(91)*
L. plantarum 21B	Broad spectrum	Phenyllactic acid, 4-hydroxy-phenyllactic acid	Lavermicocca et al. *(80)*
L. casei, *L. delbrueckii* subsp. *bulgaricus*, *Lactococcus lactis* subsp. *cremoris*	*P. expansum*	ND	Florianowicz *(51)*
L. plantarum E76, *L. plantarum* E98	*Fusarium* spp.	ND	Laitila et al. *(78)*
L. rhamnosus VT1	*Fusarium* spp., *Penicillium* spp., *Aspergillus* spp., *Rhizopus* spp., *Alternaria* spp., *Cladosporium* spp.	ND	Stiles et al. *(122)*
L. plantarum MiLAB 393	Broad spectrum	3-Phenyllactic acid, cyclo(L-Phe-L-Pro), cyclo(L-Phe-*trans*-4-OH-L-Pro), 3-phenyllactic acid	Ström et al. *(124)*
L. coryniformis Si3	Broad spectrum	Phenyllactic acid, cyclo(Phe-Pro), cyclo(Phe-4-OH-Pro)	Magnusson et al. *(86)*
L. acidophilus LMG 9196, *L. amylovarus* DSM 20532, *L. brevis* LMG 6906, *L. coryniformis* LMG 9196, *L. plantarum* LMG 6907	Broad spectrum	Organic acids, proteinaceous compounds	De Muynck et al. *(29)*
L. delbrueckii subsp. *bulgaricus* NCC 855, *L. delbrueckii* subsp. *bulgaricus* NCC 861	*P. expansum*	ND	Erginkaya et al. *(47)*
Propionibacterium acidipropionici, *P. jensenii*, *P. thoenii*, *P. freudenreichii* subsp. *freudenreichii*, *P. freudenreichii* subsp. *shermanii*	Broad spectrum	Organic acids	Lind et al. *(81)*
L. plantarum CM8, *Weissella confuse* I5, *Pediococcus pentosaceus* R47, *W. cibaria* R16	Broad spectrum	Possibly proteinaceous	Rouse et al. *(109)*

[a] ND, not determined.

DETOXIFICATION OF MYCOTOXINS BY MICROORGANISMS

Biological detoxification involves enzymatic degradation or modification of toxins, resulting in a decrease in potential toxicity. Recent advances in molecular microbial genomics coupled with the discovery of the diverse catabolic capabilities of many different types of microbial populations has led to an increased interest in this area. Many different types of bacterial species have been reported

to possess the ability to degrade mycotoxins. Earlier work by Ciegler and coworkers *(19)* revealed the ability of *F. aurantiacum* NRRL B-184 to irreversibly remove AFB_1 from a variety of food products, including milk, oil, peanut butter, peanuts, and maize, without leaving any toxic by-products. Recent investigations have focused on elucidating the possible mechanisms of degradation by *F. aurantiacum (36, 38)*. This work has established that factors such as temperature, pH, and the concentration of viable cell populations influence the uptake of the toxin by the cells. Line and Brackett *(82)* found that older (72-h) cultures of *F. aurantiacum* were more effective for removing AFB_1 from solution than were younger (24- and 48-h) cultures. In a study focused on the influence of divalent cations and chelators on AFB_1 degradation by *F. aurantiacum,* the divalent cations Mg^{2+} and Ca^{2+} stimulated AFB_1 degradation by *F. aurantiacum,* and binding of these cations by chelators such as EDTA and 1,10-phenanthroline made them unavailable to the cell, thereby decreasing AFB_1 degradation *(37)*. The increase in AFB_1 degradation caused by the addition of divalent cations is believed to be attributable to the fact that these divalent cations are important cofactors of various dehydrogenases and decarboxylases involved in both glycolysis and the trichloroacetic acid cycle. Unfortunately, the bright orange pigmentation associated with *F. aurantiacum* restricts its use in food and feed fermentations *(83)*.

Apart from *F. aurantiacum,* the yoghurt bacteria (*Streptococcus thermophilus* and *Lactobacillus delbrueckii* subsp. *bulgaricus*) and *Bifidobacterium bifidum* possess the ability to degrade OTA in milk by fermentation *(120)*. A variety of LAB originating from fermented products also can inhibit the mutagenic activity of AFB_1 *(63)*. The antimutagenic activity of *Lactobacillus* sp. LA2 against AFB_1 was 77.4% on *Salmonella* Typhimurium strain TA 100 *(63)*. *Acinetobacter calcoaceticus* totally degraded OTA in an ethanol minimal salts medium containing 10 μg ml^{-1} OTA after 120 h at 30°C *(67)*. Similarly, a mixed culture of bacteria consisting of *Pseudomonas, Alcaligenes, Bacillus, Achromobacter,* and *Flavobacterium* species isolated from soil achieved complete degradation of ZEA (50 mg $liter^{-1}$) without leaving any toxic substances *(87)*. Fodor and coworkers *(52)* found that the microbiota from healthy pigs was able to degrade FB_1 to hydrolyzed FB_1 by 46% within 48 h in buffered solution. In another report, the fermentation of maize samples with a mixture culture of *Streptococcus lactis* and *L. delbrueckii* resulted in 78.2% reductions in AFB_1 levels, and an approximately 40% reduction in toxin occurred in the spontaneously fermenting maize meal *(88)*. *Bacillus licheniformis* isolated from Thai fermented soybean detoxified both AFB_1 and OTA by 74 and 92.5%, respectively *(98)*.

Many groups have reported on the fate of mycotoxins following yeast-mediated fermentation processes. In earlier work, more than 99% of the patulin (50 μg $liter^{-1}$) was removed during alcoholic fermentation of apple juice *(123)*. In later work on three commercial cider strains of *S. cerevisiae,* patulin was degraded during active fermentative growth of the yeasts but not during anaerobic growth, and the patulin was converted to two major products, E-ascladiol and its isomer Z-ascladiol *(89)*. With respect to other mycotoxins, fermentation by *S. cerevisiae* of wort containing ZEA resulted in the conversion of 69% of the toxin to β-zearalenol and 8.1% to α-zearalenol *(116)*. Similarly, cultures of *Candida tropicalis, Torulaspora delbrueckii, Zygosaccharomyces rouxii,* and seven *Saccharomyces* strains converted ZEA to α- and β-zearalenol *(13)*. However, this transformation of ZEA is not regarded as detoxification, because both α- and β-zearalenol are still estrogenic *(73)*. In another study, OTA, FB_1, and FB_2 at 0.19, 0.95, and 0.95 μg ml^{-1}, respectively, were degraded by 87 to 91% by three strains of *S. cerevisiae* during the fermentation of wort at 25°C for 8 days *(117)*. In a later study, small losses in OTA (<40%) occurred during fermentations *(8),* but in another study average decreases of 53% of both DON and T-2 toxin were reported after alcoholic fermentation of malt by *S. cerevisiae (56)*.

Many fungal cultures, including *Trichoderma* sp. 639, *Rhizopus* sp. 668, *Rhizopus* sp. 720, *Alternaria* sp., and a few species belonging to the *Sporotrichum* group, have degraded AFB_1 by up to 65 to 99% after growth for 5 days at 28 ± 2°C *(118)*. The detoxification of AFB_1 by cell-free enzyme isolated from mycelium pellets of *Armillariella tabascens*, a nontoxic edible fungus used in Chinese traditional medicine, has been reported *(84)*. The Ames test subsequently revealed that the mutagenicity of the treated AFB_1 was markedly affected, whereas infrared spectrum analysis indicated an opening of the difuran ring of AFB_1 following exposure to the enzyme preparation *(84)*. The biodegradation of AFB_1 when treated with extracellular fractions from *R. erythropolis* coincided with a total loss in the mutagenicity of the AFB_1 and its breakdown products, as evaluated by the Ames test *(2)*. *Rhizopus* strains have efficiently detoxified patulin, ZEA, and OTA *(129)*. *A. fumigatus, Aspergillus japonicus,* and *A. niger* eliminated OTA from a liquid medium, and *A. niger* in particular degraded OTA to the less toxic molecule ochratoxin-α *(130)*. Similarly, some strains of *A. carbonarius, A. niger* aggregate, and *A. japonicus* isolated from French grapes degraded more than 80% of OTA to ochratoxin-α in liquid medium *(9)*. Abrunhosa et al. *(1)* observed that 51 of the 76 predominantly *Aspergillus* strains isolated from Portuguese grapes degraded more than 80% of the OTA added to the test culture medium. The most potent degrading species were the black aspergilli, *A. clavatus, A. ochraceus, A. versicolor,* and *A. wentii*. *Rhizopus stolonifer* degraded 96.5% of the OTA in wheat spiked with OTA at 7.5 μg g^{-1} *(129)*.

INHIBITION OF MYCOTOXIN ABSORPTION BY MICROORGANISMS

Another approach to reducing the bioavailability of mycotoxins in animals is the addition of nonnutritional inert sorbents in the diet. These sorbents sequester the mycotoxins in the gastrointestinal tract, thereby reducing their bioavailability. Although various adsorbents such as activated carbon, hydrated sodium calcium aluminosilicate,

zeolite, bentonite, cholestyramine, and certain clays have been tested and produced promising results with respect to mycotoxin binding *(66),* the use of microorganisms has become increasingly attractive as a reliable alternative to chemical sorbents in the gastrointestinal tract.

Since the early 1900s, live yeast cells (*S. cerevisiae*) have been used as general performance promoters in the poultry industry and have had beneficial effects on both weight gains and immunological responses in broiler chickens exposed to aflatoxins *(121)*. Dietary *Trichosporon mycotoxinivorans* completely blocked OTA-induced immunosuppression in broiler chicks *(106)*. In a recent study in mice, Madrigal-Santillán and coworkers *(85)* reported that *S. cerevisiae* improved weight gain and reduced genotoxicity produced by AFB_1 during a 6-week period. Similarly, Baptista et al. *(5)* observed that yeasts added to rat diet containing aflatoxins at 0.4 mg kg^{-1} were effective for reducing aflatoxin toxicity. Yeast cell wall components, as opposed to whole cells, also have been evaluated as adsorbents *(66)*. Esterified glucomannan (EG) displayed a very high capacity (97%) to adsorb AFB_1 from aqueous solutions *(31)*. However, EG was ineffective against DON and NIV *(4)*. The addition of EG (0.1%) to a diet containing aflatoxin (2 mg kg^{-1}) significantly improved the potential adverse effects of the aflatoxins on hematological parameters, total protein, albumin values, and aspartate aminotransferase activity in broiler chickens. EG also partially improved both body weight gains (59%) and biochemical parameters *(6)* and decreased the number and severity of pathological changes caused by aflatoxin treatment *(72)*. EG has a protective effect against OTA and T-2 toxin *(107)* but not against DON *(26),* and EG diminished the growth depression caused by a naturally contaminated diet (168 μg kg^{-1} aflatoxin, 8.4 μg kg^{-1} OTA, 5.4 μg kg^{-1} ZEA, and 32 μg kg^{-1} T-2 toxin) in broilers *(3)*. Researchers observed that the β-D-glucans isolated from *S. cerevisiae* cell walls are directly involved in the ZEA binding process *(133),* and both hydrogen bonds and van der Walls interactions were identified as important in these ZEA-glucan complexes *(132)*.

Some strains of *S. cerevisiae* isolated from fermented foods of West African origin also can bind up to 75% of AFB_1 present in buffered solution *(119)*. Yeasts can bind OTA in wine, and reductions of 46.8 to 52.16% and 53.2 to 70.1% have been reported in white wine and red wine, respectively *(17)*. Caridi et al. *(16)* reported the removal of OTA in wines by 20 different *Saccharomyces* sensu stricto strains using a grape must with naturally occurring and spiked OTA (1.58 and 7.63 ng ml^{-1}, respectively); 39.9 to 92.1% and 67.9 to 83.4% of the OTA, respectively, was removed after 90 days of fermentation. Heat treatment may enhance the OTA adsorbing capacity of yeast cells *(99, 119)*. Viable yeast strains can remove 4.75 to 21.40% of OTA present in white wine, whereas 8 to 30.5% of the OTA was removed by heat-killed cells within 4 h *(127)*. Yeast mannoproteins are important in OTA removal, and the mycotoxin-binding capacity has been clearly demonstrated for modified mannanoligosaccharide derived from the cell wall of *S. cerevisiae (15)*.

Some dairy strains of LAB and bifidobacteria also are known to be effect binders of aflatoxins and other toxins. The percentages of mycotoxins removed by viable probiotic bacteria from liquid solutions are listed in Table 2. Although the mechanisms of aflatoxin removal are still unknown, some researchers have suggested that the aflatoxin molecules bind onto the cell wall components of the bacteria *(12)*. Cell surface hydrophobicity also may play an important role in the binding mechanisms *(93)*. Cell wall polysaccharide and peptidoglycan may be the most important elements responsible for the binding of toxins by LAB *(77)*. Heat treatment of the bacterial cells improves their ability to remove AFB_1, possibly via protein denaturation or due to the formation of Maillard reaction products between polysaccharides, peptides, and proteins *(42)*. Pierides and coworkers *(103)* found that heat inactivation significantly enhanced the removal of AFM_1 by eight *Lactobacillus* strains except for *Lactococcus lactis* subsp. *cremoris* ARH74. In contrast, heat-treated dairy strains of LAB seem to have the same ability to remove AFB_1 as do viable bacteria, indicating that viability may not be an essential prerequisite for the removal of aflatoxins and possibly other toxins by dairy strains of LAB and bifidobacteria *(44)*. Similarly, viable LAB were equally as effective as nonviable LAB for removal of both DON and NIV *(90)*.

The polarity of the toxins also plays an important role in the binding mechanisms. The percentage of aflatoxin removed by dairy strains of LAB and bifidobacteria decreases in the order $AFB_1 > AFB_2 > AFG_1 > AFG_2$, which is correlated with the decreasing polarity of these toxins and is consistent with hydrophobic interactions, which probably also play a role in the binding mechanism *(62)*. AFM_1 was removed less effectively than was AFB_1, possibly because of the additional -OH group in AFM_1 that increases the polarity of this molecule *(103, 128)*. El-Nezami et al. *(43)* clearly demonstrated that the size of the bacterial population is one of the most critical factors in the binding of aflatoxins by lactobacilli and bifidobacteria and reported that a minimum of approximately 2×10^9 CFU ml^{-1} was required for significant AFB_1 removal. This population is similar to that need for binding of trichothecenes *(42)* and OTA and patulin *(54)* by strains of *Lactobacillus, Propionibacterium,* and *Bifidobacterium*. For DON, FB_1, and FB_2, small reductions also have been reported with bacterial levels of $\leq 10^8$ CFU ml^{-1} *(90)*.

Many researchers have reported on probiotic bacteria that have removed various mycotoxins from PBS solutions, but to date reductions in the bioavailability of mycotoxins to probiotic bacteria under gastrointestinal conditions have not been fully investigated. Limited in vivo experiments with LAB have suggested that the addition of some dairy strains of LAB in the diet can reduce the toxicity of some mycotoxins, indicating possible stability of the bacterial complex through the gastrointestinal tract in healthy volunteers *(46)*. This same research group also found a 74% reduction in the uptake of AFB_1 by the intestinal tissue in chickens in the presence of *L. rhamnosus* GG, and this effect was mediated within 60 min *(45)*. Gratz and

TABLE 2. *Mycotoxin-binding abilities of probiotic bacteria*

Bacteria[a]	Bacteria counts (CFU ml^{-1})	Toxin concn ($\mu g\ ml^{-1}$)	% bound	Reference
Lactobacillus rhamnosus GG	10^{10}	5 AFB_1	86.0	Haskard et al. *(62)*
L. rhamnosus GG	10^{10}	5 AFB_1	80.9	Turbic et al. *(126)*
L. rhamnosus LC705	10^{10}	5 AFB_1	76.1	El-Nezami et al. *(43)*
L. rhamnosus LC705	10^{10}	5 AFB_1	77.2	Turbic et al. *(126)*
L. acidophilus	10^{10}	5 AFB_1	55.4	El-Nezami et al. *(43)*
L. acidophilus NCC 12	10^{8}	1 AFB_1	37.0	Var and Kabak *(128)*
L. acidophilus NCC 36	10^{8}	1 AFB_1	34.0	Var and Kabak *(128)*
L. acidophilus E-94507	10^{10}	5 AFB_1	18.2	Peltonen et al. *(95)*
L. gasseri	10^{10}	5 AFB_1	48.4	El-Nezami et al. *(43)*
L. crispatus M247	10^{10}	5 AFB_1	5.8	Peltonen et al. *(96)*
L. fermentum	10^{10}	AFB_1	61.0	Fazeli et al. *(49)*
L. plantarum	10^{10}	AFB_1	56.0	Fazeli et al. *(49)*
L. plantarum E-79098	10^{10}	5 AFB_1	28.4	Peltonen et al. *(95)*
L. johnsonii LJ-1	10^{10}	5 AFB_1	31.3	Peltonen et al. *(96)*
L. casei Shirota	10^{10}	5 AFB_1	43.8	El-Nezami et al. *(43)*
L. delbrueckii MK9	10^{10}	5 AFB_1	17.3	Peltonen et al. *(95)*
Bifidobacterium longum Bl 24	10^{8}	1 AFB_1	31.0	Var and Kabak *(128)*
B. bifidum Bb13	10^{8}	1 AFB_1	46.5	Var and Kabak *(128)*
B. longum CSCC 5304	10^{10}	5 AFB_1	37.5	Peltonen et al. *(95)*
B. lactis CSCC 5094	10^{10}	5 AFB_1	34.7	Peltonen et al. *(95)*
PFS	10^{10}	5 AFB_1	34.1	El-Nezami et al. *(43)*
L. rhamnosus LBGG	10^{8}	0.15 AFM_1	50.7	Pierides et al. *(103)*
L. rhamnosus LBGG	10^{10}	5 AFM_1	53.8	Turbic et al. *(126)*
L. rhamnosus LC705	10^{8}	0.15 AFM_1	46.3	Pierides et al. *(103)*
L. rhamnosus LC705	10^{10}	5 AFM_1	45.7	Turbic et al. *(126)*
L. rhamnosus 1/3	10^{8}	0.15 AFM_1	18.1	Pierides et al. *(103)*
L. acidophilus LA1	10^{9}	0.15 AFM_1	18.3	Pierides et al. *(103)*
L. acidophilus NCC 12	10^{8}	0.1 AFM_1	30.5	Kabak and Var *(70)*
L. acidophilus NCC 36	10^{8}	0.1 AFM_1	28.0	Kabak and Var *(70)*
L. acidophilus NCC 68	10^{8}	0.1 AFM_1	25.7	Kabak and Var *(70)*
L. gasseri	10^{8}	0.15 AFM_1	30.8	Pierides et al. *(103)*
B. longum Bl 24	10^{8}	0.1 AFM_1	26.7	Kabak and Var *(70)*
B. bifidum Bb13	10^{8}	0.02 AFM_1	24.7–26.3	Kabak and Var *(71)*
L. rhamnosus	10^{8}	0.02 AFM_1	21.6–22.9	Kabak and Var *(71)*
L. rhamnosus LBGG	10^{10}	5 OTA	47.1	Turbic et al. *(126)*
L. rhamnosus LC705	10^{10}	5 OTA	36.4	Turbic et al. *(126)*
L. acidophilus VM20	10^{9}	1 OTA	96.0	Fuchs et al. *(54)*
B. longum VM14	10^{9}	1 OTA	58.0	Fuchs et al. *(54)*
L. rhamnosus VM04	10^{9}	1 OTA	2.0	Fuchs et al. *(54)*
L. brevis RM273	NR[b]	5 OTA	42.5	Del Prete et al. *(28)*
L. plantarum RM28	NR	5 OTA	55.0	Del Prete et al. *(28)*
L. mesenteroides RM54	NR	5 OTA	48.6	Del Prete et al. *(28)*
Oenococcus oeni RM8	NR	5 OTA	47.8	Del Prete et al. *(28)*
L. rhamnosus LBGG	10^{10}	20 DON	87.9	El-Nezami et al. *(42)*
L. rhamnosus LC705	10^{10}	20 DON	80.0	El-Nezami et al. *(42)*
PFS	10^{10}	20 DON	92.2	El-Nezami et al. *(42)*
L. reuteri R0365	10^{10}	10 DON	22.0	Niderkorn et al. *(90)*
L. rhamnosus GG	10^{10}	10 DON	54.0	Niderkorn et al. *(90)*
L. rhamnosus LBGG	10^{10}	20 T-2	85.4	El-Nezami et al. *(42)*
L. rhamnosus LC705	10^{10}	20 T-2	82.5	El-Nezami et al. *(42)*
PFS	10^{10}	20 T-2	80.0	El-Nezami et al. *(42)*
L. rhamnosus GG	10^{10}	5 NIV	13.0–16.0	Niderkorn et al. *(90)*
Leuconostoc mesenteroides	10^{10}	5 NIV	5.0–6.0	Niderkorn et al. *(90)*
L. rhamnosus GG	10^{10}	20 NIV	20.0	El-Nezami et al. *(42)*
L. acidophilus R0052	10^{10}	10 FB_1	54.0	Niderkorn et al. *(90)*
L. plantarum R1012	10^{10}	10 FB_1	74.0	Niderkorn et al. *(90)*
L. helveticus	10^{10}	10 FB_1	51.0	Niderkorn et al. *(90)*
L. rhamnosus GG	10^{10}	2 ZEA	61.0–75.0	Niderkorn et al. *(90)*
Leuconostoc mesenteroides	10^{10}	2 ZEA	64.0–68.0	Niderkorn et al. *(90)*

TABLE 2. *Continued*

Bacteria[a]	Bacteria counts (CFU ml^{-1})	Toxin concn ($\mu g\ ml^{-1}$)	% bound	Reference
PFF 111	10^{11}	10 ZEA	2.5–6.7	Gwiazdowska et al. *(61)*
PFF 111	10^{11}	20 ZEA	9.0–17.6	Gwiazdowska et al. *(61)*
B. animalis VM12	10^{9}	1 Patulin	78.0	Fuchs et al. *(54)*
B. breve LA14	10^{9}	1 Patulin	3.0	Fuchs et al. *(54)*
L. plantarum VM37	10^{9}	1 Patulin	39.0	Fuchs et al. *(54)*

[a] PFS, *Propionibacterium freudenreichii* subsp. *shermanii*; PFF, *Propionibacterium freudenreichii* subsp. *freudenreichii*.
[b] NR, not reported.

coworkers *(60)* found that preexposure of probiotic cultures of *L. rhamnosus* GG to AFB_1 reduced the subsequent binding of the toxin to the intestinal mucus, resulting in faster removal.

CONCLUSIONS

The preferred strategy for reducing the concentrations of mycotoxins in foods and animal feeds is prevention of mycotoxin formation during preharvest and postharvest of the various susceptible crops. In this respect, atoxigenic fungal strains have been used to prevent preharvest aflatoxin contamination of crops such as peanuts, maize, and cottonseed. Recent advancements in the use of biocontrol strategies involving mycotoxigenic fungi should soon lead to increased practical applications for the benefit of growers. However, genetic and molecular approaches aimed at preventing mycotoxin biosynthesis have not yet reached commercial application in the field and require substantial further development. More studies are clearly required to evaluate the potential efficacy of various biological agents, including studies focusing on the dose and timing of the applications.

Although encouraging results have been obtained for the use of some microorganisms, much less information is available concerning the effects of changing microbial loads on crops. Some filamentous fungi such as *R. stolonifer* and *A. fumigatus,* which have been used in mycotoxin removal experiments, are unlikely to be used in the field because their use may result in fungal infection of the plants. However, these fungi could be used to provide enzymes or enzyme systems as part of treatment strategies for prevention of mycotoxin formation. Alternatively, the genes encoding these enzymes could be incorporated directly into the host plant genome to produce transgenic crops that possess the ability to degrade various mycotoxins.

The use of antagonistic microorganisms such as LAB or of their metabolites as natural biopreservative agents to control undesirable molds has received much attention in recent years. Although the use of LAB has produced promising results under controlled conditions with in vitro experiments, this work needs to be extended in situ to systems involving foods or feeds. More work is required to further characterize the antifungal and antimycotoxigenic mechanisms involved.

Although most studies of mycotoxin detoxification by microorganisms have been undertaken under laboratory conditions, some data have been generated on the effective use of *F. aurantiacum* for detoxifying AFB_1 from various food products, including milk, peanuts, maize, and paprika, without the formation of toxic end products. However, the bright orange pigment of this bacterium restricts its use in both food and feed formulations.

Some of the most promising interventions studied to date involve the use of microorganism to reduce the absorption of mycotoxins from consumed foods in the gastrointestinal tract. Although clear evidence exists regarding the ability of probiotic bacteria to decrease the potential bioavailability of certain mycotoxins in humans, much work is needed to elucidate the mechanisms involved in mycotoxin removal, particularly under conditions likely to be prevalent in the human gastrointestinal tract. Successful application of this approach will contribute to overall improvements in human health and may result in increased productivity of farm animals.

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