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Trends in Food Science & Technology 10 (1999) 223–228

TRENDS IN FOOD SCIENCE & TECHNOLOGY

Review

Detoxification of mycotoxin-contaminated food and feed by microorganisms

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Different physical and chemical methods have been recommended for detoxification of mycotoxin-contaminated food and feed. Nevertheless, only a few of them (e.g. destruction of aflatoxin by ammonia treatment) have been accepted for practical use. Many specialists are of the opinion that the best approach for decontamination should be degradation by selected microorganisms, giving a possibility of removal of mycotoxins under mild conditions, without using harmful chemicals and without significant losses in nutritive value and the palatibility of detoxified food or feed. The present state of research in this field and the perspectives of such procedures are reviewed.

Introduction

According to current estimates, about 100,000 moulds have been identified, from which over 400 can be considered potentially toxic and of which about 5% are known to produce toxic compounds or classes of compounds causing problems in one or more parts of the world [1]. The worldwide distribution of contamination of cereals, oilseeds and other crops by mycotoxin-producing moulds has been amply documented in the literature [2–5]. Toxic metabolites were found to occur naturally on foods and feeds from cereals (corn, barley, grain sorghum, oats, rice, triticale, rye, wheat), pulses, legumes, soybean, peanut, etc.

Feeds contaminated with fungal toxins (mycotoxins) pose a health risk to animals and, as a consequence, may cause big economical losses due to the lower efficacy of animal husbandry. In addition, directly or indirectly (carry through to animal products), contaminated foods may also pose a health risk to humans. So it is understandable that much research has been conducted in an attempt to salvage mycotoxin-contaminated commodities and to avert health risks associated with the toxins.

Whichever decontamination strategy is used, it must meet some basic criteria [6–8]:

- The mycotoxin must be inactivated (destroyed) by transformation to non-toxic compounds;
- fungal spores and mycelia should be destroyed, so that new toxins are not formed;
- the food or feed material should retain its nutritive value and remain palatable;
- the physical properties of raw material should not change significantly;
- and it must be economically feasible (the cost of decontamination should be less than the value of contaminated commodity).

Principally there are three possibilities to avoid harmful effects of contamination of food and feed caused by mycotoxins:

- prevention of contamination,
- decontamination of mycotoxin-containing food and feed
- and inhibition of absorption of mycotoxin in consumed food in the digestive tract.

The theoretically soundest approach of prevention is doubtless the breeding of cereals and other feed plants for resistance to mould infection and, consequently, mycotoxin production. Particularly in breeding wheat and corn, significant improvement of resistance has been achieved [9,10]. According to data reported in a recent review paper [11] in Germany, about 25% of the area under wheat is occupied by resistant varieties. Despite progress in breeding, full resistance has not been achieved.

0924-2244/99/$ - see front matter
PII: S0924-2244(99)00050-3

Another applied way of prevention is the inhibition of the growth of moulds and their production of mycotoxins. First of all, well-selected harvesting, storage- and processing methods may be a successful tool in prevention of mould growth. A practical guide to the prevention of mycotoxin contamination of grain and animal feedstuffs by Fusaria was developed and published by Trenholm [12].

Treatment of grains by some chemicals is also possible; e.g. approximately 100 compounds have been found to inhibit aflatoxin production [13]. Most of them appear to do so by inhibiting fungal growth. Two extensively studied inhibitors of aflatoxin synthesis are dichlorvos (an organophosphate insecticide) and caffeine. As reported by Rodriguez and Mahoney [14] some surfactants have suppressed the growth of *Aspergillus flavus* and aflatoxin synthesis.

When contamination can not be prevented, decontamination is needed before the use of such raw materials for food or feed purposes. The simplest way may be the physical removal of contaminated grains by manual selection. This is a time-consuming procedure and, in many cases, is impossible. A density method to separate zearalenone-deoxynivalenol-infected wheat, corn and sorghum grains has been studied [15,16]. A partial removal of mycotoxin may be achieved by dry cleaning of the grain and also in the milling process. Milling leads to a fractionation with increased level of mycotoxin in bran and a decreased level in flour.

The majority of mycotoxins are heat-stable, so heat treatment, usually applied in food technology, does not have a significant effect on mycotoxin level.

Efforts were made in several countries to find an economically acceptable way of destruction of mycotoxins to non-toxic products using different chemicals. Alkaline compouds, such as ammonia, sodium- and calcium hydroxide etc, were used particularly for destruction of aflatoxin (for a review, see Samaraeva *et al*., [17]). Although such treatment reduces nearly completely the mycotoxin concentration, these chemicals cause losses of some nutrients.

The effect of oxidizing agents was also studied. Bleaching of flour with chlorine in a commercial mill resulted in a 10% reduction of deoxynivalenol content, treatment with ozone caused significant reduction [18]. Aqueous sodium bisulfite effected the greatest reduction in mycotoxin levels [19]. Such treatment is too drastic for grain destined for food uses.

Finally, it should be mentioned that some indigestible adsorbents may adsorb the mycotoxins and so that they are not absorbable in the digestive tract. Such adsorbents are used in some feed supplements; e.g. a successful adsorption of aflatoxin B1 on hydrated -sodium-calcium-aluminosilicate [20], patulin [21] and ochratoxin-A [22] on activated carbon has been reported. The possibility of adsorption of important micronutrients is the potential disadvantage of such methods of detoxification. Nevertheless, some adsorbents are commercially used in feed supplements.

Although the different methods used at present are to some extent successful, they have big disadvantages with, limited efficacy and possible losses of important nutrients and normally with high costs. Many workers in the field are of opinion that the best solution for decontamination should be detoxification by biodegradation, giving a possibility for removal of mycotoxins under mild conditions. without using harmful chemicals without significant losses in nutritive value and palatability of decontaminated food and feed.

Biodegradation of mycotoxins—present situation and future trends

Decontamination by fermentation

The idea to decontaminate cereals by fermentation was born in the early eighties. The use of zearalenone-contaminated corn to produce ethanol by fermentation was described by Bennet *et al*. [23]. The ethanol produced was free of mycotoxin. However, the residual solids and solubles, which are used in animal feed, were still contaminated.

Studies of Sawinski-Acsadi [24] have shown that corn contaminated with F-2 toxin could be used as a substrate for *Candida intermedia* fermentation. The initial toxin activity was reduced 10-fold and was present almost entirely in the fermentation liquor, whereas no toxin could be detected in the feed protein.

A lot of papers have been published relating the fate of mycotoxins during fermentation of beer and wine. From recent reports, the papers of Scott *et al*. [25], Scott and Lawrence [26,27], Flesch and Voight-Scheuerman [28], and Torres *et al*. [29] may be mentioned. According to the data published, zearalenone and alpha- or beta-zearalenol do not appear in Canadian and European beers. Aflatoxins have not been detected in European beers and ochratoxin-A has been rarely detected in beers at levels above 1 ng/ml. Scott and Lawrence [26] analysing all commercial beer types in USA and Mexico reported that only one sample contained a low level (under the limit) of aflatoxin. The same researchers [27] analysed 41 Canadian beers for fumonisin. All LC analyses were carried out using the o-phtalaldehyde (2-mercaptoethanol) OPA/MCA or 4-fluoro-7-nitrobenzofurazan (NBD-F) derivatization process. Detection limits were 0.4 to 1.0 ng of fumonisin B1 per ml (OPA-MCA method) resp. 0.7–3.0 ng per ml (NBD-F method). Recoveries were 47–97% and 67–114%, respectively. Four samples contained more than 2 ng per ml fumonisin B1 and 7.6 ng per ml fumonisin B2. Majority of samples contained very low quantities of fumonisins. Occurrence of fumonisins in Spanish beers was studied by Torres *et al*. [28]. Fumonisin was detected in 43.8% of analysed samples at a low level (4.76–85.53 ng per ml) which is evaluated as non toxic.

Among the reports dealing with potential effect of ethanol fermentation by yeasts on mycotoxins, the paper by Flesch and Voight-Scheuerman [28] is interesting. These researchers investigated the decomposition of trichotecin and iso-trichotecin during alcoholic fermentation of grape juice. As a result, it was established that isotrichotecin, trichotecolone and several unidentified substances originate from trichotecin. Two of those previously unidentified compounds (termed FA o.6 and TSS 4) have been identified (Figs. 1 and 2). These are derivatives of trichotecin and also do not contain epoxide groups. Concerning the mechanism of decomposition, it is suggested that a yeast epihydroxylase may be present. So far, a similar reaction in the case of trichotecene has been not published. The transformation of trichotecin to its iso-form is also enzymatically catalyzed. It is also suggested that the yeast probably produces ligases and also a keto-enol tautomerase. It is also interesting that the addition of bisulfite increases the rate of decomposition of trichotecin and its isomers.

Iso-trichotecin is partially converted to trichotecin by yeast from which trichotecolone can originate by decomposition of the side chain. The toxicological activities of the named compounds remained nearly constant after alcoholic fermentation. The originating products are slightly more or less toxic than the initial compounds. It was further established that a significant amount of mycotoxins (about 20%) is taken up by yeast.

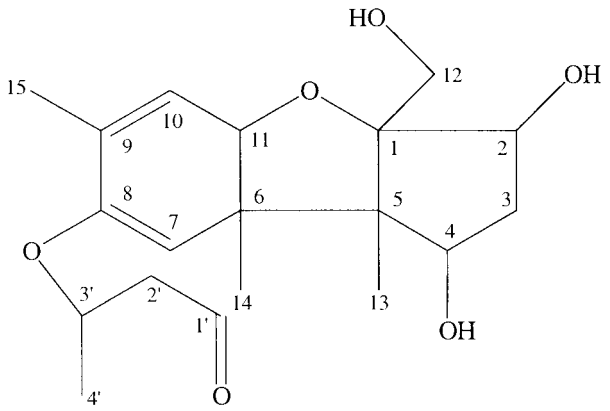

Fig. 1. Suggested structure for the compound FA 0.6.

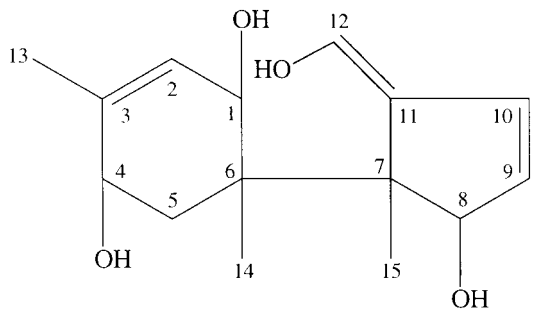

Fig. 2. Suggested structure for the compound TSS as enol.

In another experiment Scott *et al.* [25] fermented a worth containing added ochratoxin-A and fumonisin B1 and fumonisin B2. After 8 days fermentation using three different yeast strains (Saccharomyces), a maximal decrease was observed in the case of ochratoxin-A 13%, and 28% resp. 17% for fumonisin B1 and fumonisin B2. The uptake of ochratoxin by yeast was 21%; no uptake of fumonisins was detected.

Decontamination of mycotoxin-contaminated cereals and other commodities by bacteria

In the 1960s Ciegler *et al.* [30] screened over 1000 microorganisms for the ability to degrade aflatoxins. Only one bacterium, *Flavobacterium aurantiacum* B-184, was able to irreversibly remove aflatoxin from solutions [31]. The early investigations showed that pH and temperature influenced the uptake of the toxin by the cells. High populations of the cells (10^{11} per ml) permanently removed greater percentages of the aflatoxin from solutions than did lower populations. Large populations of heat-inactivated cells was also shown to bind some aflatoxin, which was easily recovered by washing with water [32].

Ability of this microorganism to remove aflatoxins from foods was demonstrated in milk, vegetable oil, corn, peanut, peanut butter and peanut milk [30,33]. More recently, Hao and Bracket [34] detoxified peanut milk using this microorganism. In both cases, these authors suggested that this microorganism might be of potential value in the biological detoxification of these or other foods and feeds. The importance of biological methods of aflatoxin degradation will likely increase if consumer resistance to chemical treatments continue to grow. However, the bright orange pigmentation associated with this bacterium would likely limit its applicability for food and feed fermentations.

The recent investigations in this field [32, 35, 36] concentrate on the study of possible mechanisms of degradation. The first important question which must be answered is: whether this bacterium (*Flavobacterium aurantiacum*) actually degrades the aflatoxin or whether the disapperance of the toxin resulted from adsorption to the cells. Without this knowledge, no real advantage can be taken of this microbial method of removing aflatoxin. To determine the exact fate of aflatoxin B1 exposed to the cells of *Flavobacterium aurantiacum*, Line *et al.* [36] used labeled ($^{14}C-$) aflatoxin B1, which could be accurately traced and detected using a scintillation counting procedure. The carbon-dioxide production, the quantity of aflatoxin B1 (soluble in chloroform), the amount of water-soluble degradation products, and the aflatoxin adsorbed by the cells was controlled. Control experiments were also included in the program with cell-free blanks and heat-inactivated cells. Analysis of radioactivity revealed that the chloroform- soluble aflatoxin B1, was rapidly converted to

water-soluble products, when incubated with five cells of *F. aurantiacum*. After 6 hours in the presence of the live cells, only 24.1% of the initial radioactivity remained in the chloroform phase. Cell-free samples did not produce water-soluble compounds. In the control samples, 99.7% of the radioactivity remained in the chloroform phase after 72 hours. Dead cells were likewise unable to produce water-soluble aflatoxin B1 degradation products.

The radioactivity of water-soluble fractions following exposure of radioactively labeled aflatoxin B1 to live, dead or no *F. aurantiacum* cells is shown in Fig. 3. Loss of aflatoxin-associated radioactivity from the chloroform phase is demonstrated in Fig. 4.

Both live and dead cells adsorb a quantity of the aflatoxin. This removal of aflatoxin B1 by dead cells appeared unchanged after the initial sampling, confirming the physical nature of the binding of the toxin. During the experiment, labelled carbon dioxide is released by the live cells. Dead cells or neither control released radioactive carbon-dioxide. This is compelling evidence that at least part of the labelled aflatoxin B1 is being metabolized by the living flavobacteria. The fact

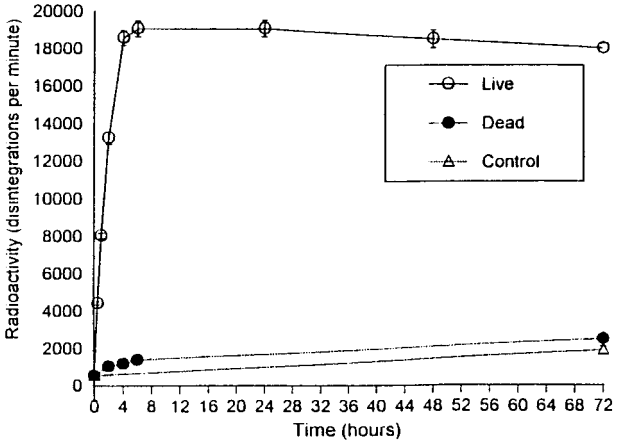

Fig. 3. Radioactivity of water-soluble fractions following exposure of radioactively-labeled aflatoxin B1 to live, dead or no *F. aurantiacum* cells.

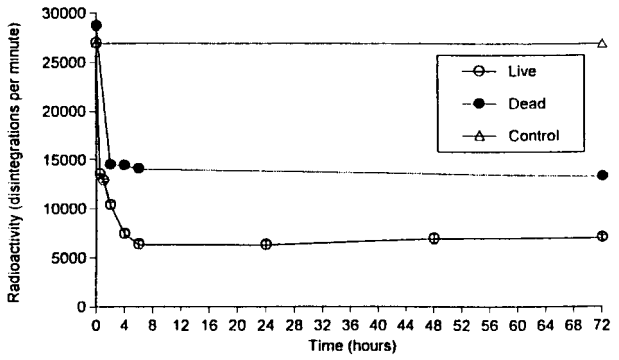

Fig. 4. Loss of aflatoxin associated radioactivity from the chloroform phase.

that dead cells and control samples did not produce labelled carbon-dioxide reveal that aflatoxin B1 was metabolized by living cells and not adsorbed by cell walls. The elucidation of the structure of water-soluble products of decomposition of aflatoxin B1 and also their toxicity needs further investigation. Lillehoj *et al.* [31] found that detoxification with *F. aurantiacum* does not produce acutely toxic residual compounds in duckling assays. In every case, an identification of these compounds is needed. Positive identification of these compounds may give insight in the possible pathway of degradation.

Investigations on the role of toxin concentration and secondary carbon source in microbial transformation of aflatoxin B1 revealed that neither added nutrients (e.g. glucose), nor added nonlabeled toxin had a significant effect on the microbial transformation of aflatoxin B1. These results suggest that the microbial degradation of aflatoxin B1 by *F. aurantiacum* is probably a mineralization phenomenon and not co-metabolism. The ability of this microorganism to detoxify aflatoxin without the need for exogeneous energy sources could be important to future endeavors attempting to use the organism, or the mechanism responsible, in fermentation reactions.

The role of trace-metal ions in microbial aflatoxin B1 degradation was recently studied by D. Souza *et al.* [37]. It was found that copper and zinc ions may inhibit the degradation of aflatoxin B1 by *Flavobacterium aurantiacum* This effect is probably connected with an influence on the enzyme system involved in the degradation process.

Another relatively frequently occurring mycotoxin, ochratoxin-A, was reported to be degradable by rumen bacteria [38,39]. However, the responsible bacteria have not been isolated and identified. Cheng-An Hwang and Draughon [40] screened bacteria and yeasts in order to detect microorganisms able to degrade ochratoxin-A. Thirty seven bacteria, 10 strains of yeasts, and 12 moulds were screened. *Acinetobacter calcoaceticus* was able to degrade ochratoxin-A in ethanol-minimal salts medium with an initial concentration of 10 μg per ml at both 25 and 30°C. It was suggested that the endproduct of degradation of ochratoxin-A by *Acinetobacter calcoaceticus* is a less-toxic compound named alpha-ochratoxin by authors. This compound has been shown to be much less toxic on chicken embryos than ochratoxin-A. Therefore, the degradation of ochratoxin-A by this microorganism may be considered a form of microbial detoxification of ochratoxin-A. The results of this study look promising. Nevertheless, they don't imply the degradation of ochratoxin-A in an *in situ* system using food or feed. Further studies are needed to characterize the products of degradation and to investigate the activity of this bacteria in food and feedstuffs.

Recently, a soil bacterium was isolated that metabolizes some trichothecene toxins [41]. A mixed microbial

culture capable of metabolizing deoxynivalenol was obtained from soil samples by an enrichment culture procedure. A bacterium isolated from the enrichment culture completely removed exogeneously supplied deoxynivalenol from the culture medium after incubation for 1 day. On the basis of morphological and phylogenetical studies, strain E3-39 was classified as a bacterium belonging to the *Agrobacterium Rhizobium* group. The main metabolite was identified as 3-keto-4-deoxynivalenol. This compound exhibited a remarkably decreased (to less than one tenth) toxicity relative to deoxynivalenol. Strain E3 -39 *was also able to transform 3-acetyldeoxynivalenol but not nivalenol and fusarenone-X*.

Future trends

It seems that, according to results of experiments realized till present time, microorganisms are the main living organisms applicable for mycotoxin biodegradation. Further screening of microorganisms may lead to detection of more efficient and better applicable bacteria. First of all, the microorganisms resistant to mycotoxins, should be investigated. Investigations of several bacteria and yeasts showed that differences may be found both in sensitivity and selectivity of these microorganisms against mycotoxins [42], e.g. *Bacillus brevis* was sensitive to eight mycotoxins including zearalenone and ochratoxin-A, but was not affected by high concentrations of trichothecene toxins. *Kluyveromyces marxianus* was sensitive to all trichothecene toxins, but this yeast was not inhibited by other mycotoxins.

The study of the mechanisms involved in resistance (e.g. selective uptake through membranes, decomposition by selective enzymes, blocking by complex formation etc.) may be helpful in finding applicable microbes.

The control of degradation products, the effect of detoxification, and on nutritive and organoleptic properties is, in every case, a decisive part of research and potential applications. Finally, a technology must be developed that is suitable for economically feasible treatment of cereals and legumes.

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