Mycotoxin prevention and decontamination – a case study on maize

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Numerous reviews and publications have summarized the approaches already in use and being developed to minimize mycotoxin contamination of maize (Agricultural Research Service, 1997a and 1997b; CAST, 1989; Robens, 1990; Jackson, DeVries and Bullerman, 1996; Sinha and Bhatnagar, 1998; Scott, 1998). Many of the strategies used for other commodities can generally also be applied to maize (McMullen, Jones and Gallenberg, 1997). In this article, "prevention of mycotoxin production in maize" encompasses prevention of toxin biosynthesis and metabolism in the field (pre-harvest) or in storage (post-harvest). "Mycotoxin detoxification" refers only to post-harvest treatments designed to remove, destroy (decontaminate) and ultimately reduce the toxic effects of mycotoxins (detoxify). The potential ability of a plant to detoxify mycotoxins *in situ* is treated as a prevention strategy.

HEALTH AND TRADE EFFECTS

Clearly the pre- or post-harvest prevention of mycotoxin contamination is the preferred strategy for minimizing mycotoxins in foods and feeds. Failure to prevent fungal invasion and toxin formation in the field or in storage will inevitably lead to an increased risk of adverse health effects and economic loss. However, if chemical monitoring is successful, maize consumption should not be a significant source of increased health risk from mycotoxins. Early identification of mycotoxin-contaminated grains provides the opportunity to direct the most highly contaminated grains into uses that minimize consumption by sensitive species. Nonetheless, the lower economic value of contaminated maize can result in large monetary losses for the farmer. In addition, regulatory limits that are inappropriately stringent can effectively remove large amounts of maize from the market. For example, in a year of high fumonisin contamination in maize-producing countries (in the absence of careful monitoring), an action limit set too low could result in a significant loss of food from the market. Thiel *et al*. (1992) reported that 35 percent of the commercial maize products that they sampled from the United States contained more than 1 part per million (ppm) of total fumonisins. Worldwide, total fumonisins in maize meal range from < 50 parts per billion (ppb) to several ppm (Shephard *et al*., 1996). Thus, an action limit set at 1 ppm could result in a significant loss of maize meal from the world market. In a time of dwindling food supplies, this is clearly an unacceptable loss.

PREVENTION STRATEGIES

Some prevention strategies that are currently in use or under development are summarized in Box 1. An integrated multipronged approach is the preferred strategy (CAST, 1989). However, some specific strategies are more practical and timely than others. For example, the strategies listed as "known to be effective" are labour-intensive and relatively low-cost. The specifics of these approaches vary from fungus to fungus and mycotoxin to mycotoxin. The pre- or post-harvest strategy that should be emphasized in a particular year will depend on the climatic conditions of that year. Unfortunately, humans cannot usually avoid weather that favours fungal infection, and the combination of high temperature and drought often precludes increased irrigation and, consequently, adequate mineral nutrition. Conversely, reducing the moisture level in the field at critical periods is equally impossible. Nonetheless, understanding the environmental factors that promote infection, growth and toxin production is the first step in developing an effective plan to minimize mycotoxins in foods and feeds.

Prevention or reduction in the incidence of pre- and post-harvest infection is clearly a critical factor in reducing mycotoxin accumulation, since the concentrations of aflatoxins, deoxynivalenol or fumonisins have been shown to be greater in symptomatic than in non-symptomatic maize ears or kernels (Desjardin *et al*., 1998; Reid, Mather and Hamilton, 1996; Scott and Zummo, 1995). Environmental factors that favour *Aspergillus flavus* infection include high soil or air temperature, drought stress, nitrogen stress, crowding of plants and conditions that aid dispersal of conidia during silking (CAST, 1989;

BOX 1
STRATEGIES FOR PREVENTION OF MYCOTOXINS IN MAIZE

Pre-harvest strategies

Known to be effective

- Reduction in plant stress through irrigation, mineral nutrition, protection from insect damage.
- Avoidance of environmental conditions that favour infection in the field.
- Minimization of crop residues and other point sources of inoculum.

Potentially effective

- Breeding for maize cultivars resistant to fungal infection.
- Use of crop protection chemicals that are antifungal agents.

Developmental

- Development of transgenic maize plants resistant to fungal infection.
- Development of transgenic maize cultivars capable of catabolism/interference with toxin production.
- Development of maize genetically engineered to resist insect damage.
- Development of maize seeds containing endophytic bacteria that exclude toxigenic fungi.
- Exclusion of toxigenic fungi by pre-infection of plants with biocompetitive-non-toxigenic fungal strains.

Post-harvest strategies

Known to be effective

- Harvesting when water content is optimal to prevent saprophytic development of toxigenic fungi.
- Removal of damaged maize and drying of kernels to the optimal moisture content before storage.
- Control of insect and rodent activity and maintenance of appropriate moisture levels and temperature.
- Frequent cleaning of feed delivery systems and short-term storage areas.

Potentially effective

- Use of antifungal agents such as propionic and acetic acids.

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Robens, 1990). The growth of *A. flavus* and *A. parasiticus*, and subsequent aflatoxin production in storage, are favoured by high humidity (> 85 percent), high temperature (> 25° C) and insect or rodent activity (CAST, 1989).

Typically, infections with *Fusarium graminearum* are localized, sporadic, favoured by cool, moist conditions during silking (Munkvold and Yang, 1995; Park, Smalley and Chu, 1996) and most common in wetter summers (Sutton, 1982; Vigier *et al*., 1997). Epidemics are usually associated with wet conditions late in the growing season. Storage of maize that is wet and at cool to moderate temperatures has been associated with outbreaks of oestrogenic syndrome and feed refusal (CAST, 1989) owing to the presence of zearalenone and deoxynivalenol, respectively.

Fusarium moniliforme is the most common pathogen of maize (Munkvold and Desjardin, 1997). Because *F. moniliforme* is a seed-borne, symptomless endophyte in maize, its elimination is very difficult (Agricultural Research Service, 1997a). Maize ear rot associated with *F. moniliforme* and *F. proliferatum* is accompanied by significant fumonisin levels. In the United States, warm, dry years have resulted in greater fumonisin accumulation than cooler years (Shephard *et al*., 1996), whereas maize grown in cooler areas usually contains low amounts of fumonisin (Doko *et al*., 1995; Miller, 1994). However, dry weather early in the growing season followed by wet weather during silking and later has been associated with severe ear rot (Munkvold and Desjardin, 1997). Moisture on the silks clearly promotes infection (Munkvold, McGee and Carlton, 1997). *F. moniliforme* and *F. proliferatum* kernel rot of maize is also an important ear disease in hot maize-growing areas (De Leon and Pandey, 1989; King and Scott, 1981; Ochor, Trevathan and King, 1987) and is associated with warm, dry years and / or insect damage (Shurtleff, 1980). High incidence of European corn borer damage increases *F. moniliforme* disease and fumonisin concentrations (Lew, Adler and Edinger, 1991), while high levels of thrip (*Frankliniella occidentalis*) infestation are correlated with increased disease incidence (Farrar and Davis, 1991). Ear rot is reduced in maize that has been genetically engineered for resistance to the European corn borer (Munkvold, Hellmich and Showers, 1997). Hybrids with thin kernel pericarp and increased incidence of kernel splitting (aggravated by drought) are more susceptible to kernel rot (Hoenisch and Davis, 1994; Odvody, Spencer and Remmers, 1997). As with aflatoxins, fumonisin production in the field is promoted by environmental stress, as evidenced by the fact that maize hybrids grown outside their range of adaptation or under cool but drought-stressed conditions, had higher fumonisin concentrations (Shelby, White and Burke, 1994; Doko *et al*., 1995; Miller, 1994; Visconti, 1996).

Tillage practices, crop rotation, weed control, late seasonal rainfall, wind and pest vectors can all affect the amount and source of fungal inoculum that maintains the disease cycle in maize (Munkvold and Desjardin, 1997). Any action that interrupts the cycle should reduce the probability of silk and kernel infection. For example, elimination of waste maize deposits will reduce the

incidence of *A. flavus* in kernels and soil beneath the piles (Olanya *et al.*, 1997). Not surprisingly, nitidulid beetles (vectors for *A. flavus*) living under or near waste maize deposits were found to be highly contaminated with *A. flavus*. In the same study, it was reported that soil-borne *A. flavus* was greatly increased when soil temperatures were 35° to 40° C. So, in drought years with high temperatures, when increased irrigation is not practical, steps to reduce inoculum may be one of the few practical strategies for reducing the probability of an aflatoxin (or other mycotoxin) epidemic.

Breeding for resistance to toxin production has met with limited success (McMullen, Jones and Gallenberg, 1997; Munkvold and Desjardin, 1997; Robens, 1990). Maize cultivars that are resistant to aflatoxin production have been reported (Windham and Williams, 1998; Campbell, Hamblin and White, 1997; Russin *et al.*, 1997). However, breeding to control aflatoxins, fumonisins, deoxynivalenol and zearalenone production *in planta* has yet to meet with any real success since the occurrence of these, the most common mycotoxins in maize, is clearly quite unpredictable and often quite high. If productive, resistant varieties of maize were readily available, they would preclude concern about detoxification and animal and human disease. However, they are clearly not available.

The use of chemicals is a very attractive strategy to prevent mycotoxin production. Some chemical treatments will prevent mould growth and, potentially, reduce mycotoxin production in the field and in storage (Wegulo *et al.*, 1997). However, with regard to maize and mycotoxins, the economic and ecological hurdles seem to be quite high (McMullen, Jones and Gallenberg, 1997), as evidenced by the fact that few of these approaches are being marketed widely.

Many new and exciting pre-harvest prevention strategies that involve new biotechnologies are being explored. These new approaches involve the design and production of maize plants that reduce the incidence of fungal infection, restrict the growth of toxigenic fungi or prevent toxin accumulation. Biocontrols using non-toxigenic biocompetitive agents are another potentially useful strategy in maize (Agricultural Research Service, 1997a and 1997b; Robens, 1990). However, the possibility of recombination with toxigenic strains is a concern (Geiser, Pitt and Taylor, 1998). In the case of *F. moniliforme* in maize, the use of bacterial biocompetitive agents and non-toxigenic *F. moniliforme* isolates is under development (Agricultural Research Service, 1997a). One interesting approach is the engineering of maize plants to catabolize fumonisins *in situ* (Munkvold and Desjardin, 1997). Typically, these approaches require considerable research and development but offer the potential of ultimately producing low-cost and effective solutions to the mycotoxin problem in maize.

Post-harvest prevention of mycotoxin production is primarily dependent on good management practices before and after harvest (Box 1). Aflatoxins are potentially serious post-harvest problems, unlike fumonisins, since *F. moniliforme*, like other Fusaria, does not grow in maize at less than 18 to 20 percent moisture (Munkvold and Desjardin, 1997). Nonetheless, animal health problems associated with the consumption of poorly preserved (mouldy) maize contaminated with fumonisins are not uncommon. As a last resort, chemical treatment with antifungal agents is a possibility. However, the cost-effectiveness of this approach is questionable. Chemical treatments that prevent mould growth and/or toxin production include the treatment of stored maize with propionic or acetic acid (CAST, 1989).

DETOXIFICATION STRATEGIES

Strategies currently in use and under development for detoxification of mycotoxin-contaminated maize are summarized in Box 2. Not surprisingly, the mycotoxins in maize that are of the greatest concern (aflatoxins, deoxynivalenol, zearalenone, ochratoxin A, fumonisins) are also quite stable and, therefore, difficult to degrade. Fumonisins, for example, are relatively stable molecules that do not react readily with macromolecules (although they bind quite specifically to the enzyme ceramide synthase) and are not mutagenic. Their lack of reactivity creates a challenge to anybody aiming at detoxifying maize. Conversely, fusarin C (a *Fusarium* mycotoxin detected in mouldy maize) is very unstable, reacts readily with macromolecules and is mutagenic. Because of its reactivity, it breaks down rapidly and would therefore be easy to detoxify if it caused food safety problems.

Detoxification and decontamination are not always the same thing. The fact that a toxin cannot be detected in a food or feed by analytical chemistry does not mean that the product has been detoxified. In Box 2, detoxification strategies have been arbitrarily divided into those that are primarily dependent on physical, chemical or microbiological processes that detoxify by destroying, modifying or absorbing the mycotoxin so as to reduce or eliminate the toxic effects. One promising method is the use of selective high-affinity hydrated sodium calcium aluminosilicates to bind aflatoxin in feeds and foods. However, some commonly used non-selective silico-aluminates do not completely protect, and may enhance toxicity of aflatoxin (Mayura *et al.*, 1998). Dietary

BOX 2

STRATEGIES FOR DETOXIFICATION OF MYCOTOXINS IN MAIZE

Physical methods

Cleaning: screening out fine materials reduces fumonisins and other mycotoxins – simple but incomplete.

Segregation and sorting: "black light" test for aflatoxins – simple but misleading; colour sorting technology – unproven with maize, but promising.

Density segregation and washing: of fumonisins, deoxynivalenol, zearalenone – non-specific and incomplete, but suitable for wet milling and alkaline processing of maize.

Thermal degradation: incomplete for most mycotoxins.

Microwave treatment: high levels destroy trichothecenes.

Solar degradation: of aflatoxins – results in maize oil encouraging.

Extrusion cooking: of fumonisins – temperature- and screw speed-dependent destruction – very promising (Katta *et al.,* 1998).

Wet milling: produces starch free, or almost free, of zearalenone, fumonisins and aflatoxins, but T-2 toxin is increased in maize germ.

Chemical methods

Thermal treatment plus reducing sugars: of fumonisins – promising but toxicology and stability uncertain.

Nixtamalization/alkaline hydrolysis: reversible degradation of aflatoxins and partial degradation of fumonisins, but toxicity remains – not an effective method for detoxification of fumonisins or aflatoxin; reduced zearalenone and deoxynivalenol.

Bisulphite: destroys aflatoxin B1, reduces deoxynivalenol in maize – bisulphite is a common food additive (the DON sulphonate is unstable in alkali).

Ammoniation: approved method for aflatoxin in maize in Mexico, South Africa and several states in the United States – may not be effective in detoxifying fumonisins in maize.

Hydrogen peroxide/sodium bicarbonate: destroys fumonisin in maize.

Ozonation: degrades and detoxifies aflatoxins in naturally contaminated maize – promising.

Hydrated sodium calcium aluminosilicates: bind aflatoxins with high affinity and capacity – demonstrated efficacy *in vivo* when added to diets; non-selective aluminosilicates may pose significant risks and should be avoided (Mayura *et al.,* 1998).

Activated charcoal: reduces dietary conversion of aflatoxin B_1 to aflatoxin M_1 in cows.

Microbiological methods

Ethanol fermentation: does not break down aflatoxin B_1, zearalenone or fumonisin B_1; toxins may actually be increased in spent grain used in animal feeds.

Probiotic mixtures: Lactobacillus and *Propionibacterium* may reduce bio-availability of dietary aflatoxin (Ahokas *et al.,* 1998).

Dietary interventions

Choline, methionine, vitamins, protein, dietary fat, antioxidants and inducers of metabolizing enzymes: addition to animal feeds can lower toxicity caused by mycotoxins in maize.

Sources: Except where indicated the information is summarized from CAST, 1989; Jackson, DeVries and Bullerman, 1996; Scott, 1998; T.D. Phillips, Texas A&M University, personal communication.

interventions intended to reduce toxicity after a mycotoxin has been absorbed are another detoxification strategy.

RECOMMENDATIONS

In view of the research carried out to date, the following recommendations can be made:

- Maize grown for human consumption in areas where conditions are frequently favourable to fungal invasion and mycotoxin production in the field should be tested for mycotoxin contamination before use.
- Additional research is needed to develop the ability to predict when and where environmental conditions may make mycotoxin contamination probable and to develop the means to disseminate warnings to farmers and processors.
- If invasion in the field is probable and environmental conditions are favourable for mycotoxin production in the field, care should be taken to reduce sources of inoculum and to minimize plant stress and insect damage.
- Once it is determined that field contamination is likely, care should be taken to minimize the growth of the fungus after harvest and while in storage. Planning should begin for decontamination and include the diversion of contaminated maize away from human consumption or consumption by sensitive species.
- Maize cultivars that are resistant to drought stress, insect damage and fungal infection need to be developed.
- Easy and economical decontamination procedures need to be developed. ♦

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Mycotoxin prevention and decontamination – a case study on maize

Prevention of mycotoxins in maize encompasses prevention of toxin biosynthesis and metabolism in the field or in storage. Mycotoxin detoxification refers to post-harvest treatments to remove, destroy or reduce the toxic effects. Failure to prevent mycotoxin formation in the field or in storage will inevitably lead to increased health risks and economic loss. However, successful monitoring should prevent mycotoxins from becoming a significant source of increased health risks. An integrated multi-pronged approach is the preferred strategy for controlling mycotoxin contamination. The pre- or post-harvest strategy that is most appropriate will depend on the climatic conditions of that particular year. Understanding the environmental factors that promote infection, growth and toxin production is the first step towards an effective plan to minimize mycotoxins in foods and feeds.

Fusarium moniliforme is the most common pathogen of maize. In maize, *F. moniliforme* is a seed-borne endophyte without symptoms, so its elimination is difficult. Tillage practices, crop rotation, weed control, late-season rainfall, wind and pest vectors can all affect the amount and source of the fungal inoculum that maintains the disease cycle in maize. Many of the new and exciting pre-harvest prevention strategies that are being explored involve production of genetically engineered resistant maize. Biocontrols using non-toxigenic biocompetitive agents are also a potentially useful strategy in maize. Post-harvest prevention of mycotoxin production is primarily dependent on good management practices before and after harvest. Detoxification strategies that are physical, chemical or microbiological can detoxify maize by destroying, modifying or absorbing the mycotoxins so as to reduce or eliminate the toxic effects.

Prévention des mycotoxines et décontamination – une étude de cas sur le maïs

La prévention des mycotoxines dans le maïs englobe la prévention de la biosynthèse et du métabolisme des toxines dans le maïs sur pied ou stocké. Par détoxication des mycotoxines, on entend les traitements après récolte visant à supprimer, détruire ou réduire les effets toxiques des mycotoxines. L'échec des tentatives faites pour prévenir la formation de mycotoxines dans le maïs sur pied ou stocké conduira inévitablement à une augmentation des risques sanitaires et à des pertes économiques. Toutefois, un suivi attentif devrait empêcher les mycotoxines de devenir une source importante de risques sanitaires. Une approche intégrée à multiples facettes semble la meilleure stratégie pour lutter contre la contamination par les mycotoxines. La stratégie, préalable ou postérieure à la récolte la plus appropriée à appliquer, dépendra des conditions climatiques de l'année considérée. Une bonne compréhension des facteurs écologiques qui favorisent l'infection ainsi que la croissance et la production de toxines est un préalable indispensable à la mise au point d'un plan efficace de réduction des mycotoxines dans les aliments, qu'ils soient destinés à l'alimentation humaine ou animale.

Fusarium moniliforme est le pathogène le plus commun du maïs. Dans la mesure où il s'agit d'un endophyte du maïs transmis par les semences et non accompagné de symptômes, son élimination sera difficile. Les façons culturales, la rotation des cultures, la lutte contre les adventices, les précipitations de fin de campagne, le vent et les vecteurs sont autant de facteurs déterminant la quantité et la source de l'inoculum fongique qui entretient le cycle de la maladie dans le maïs. Bon nombre des nouvelles stratégies de prévention préalables à la récolte qui sont actuellement à l'étude impliquent la production de maïs résistant génétiquement modifié. Les contrôles biologiques utilisant des agents biocompétitifs non toxicogènes constituent une autre stratégie potentiellement utile pour le maïs. La prévention après récolte de la production de mycotoxines dépend essentiellement de bonnes pratiques de gestion avant et après la récolte. Les stratégies de détoxication de nature physique, chimique ou microbiologique peuvent détoxiquer le maïs en détruisant, modifiant ou absorbant les mycotoxines de façon à en réduire ou à en supprimer les effets toxiques.

Prevención y descontaminación de micotoxinas: estudio monográfico sobre el maíz

La prevención de las micotoxinas en el maíz comprende la prevención de la biosíntesis de toxinas y su metabolismo sobre el terreno o en almacén. La descontaminación de micotoxinas se refiere a los tratamientos poscosecha para eliminar o reducir los efectos tóxicos. El no evitar la formación de micotoxinas en el campo o en el almacén conducirá inevitablemente a un aumento del riesgo para la salud y a una pérdida económica. Sin embargo, un buen seguimiento impedirá que las micotoxinas se conviertan en causa importante de aumento del riesgo para la salud. La estrategia preferida para controlar la contaminación por micotoxinas es un enfoque polifacético integrado. La estrategia de precosecha o poscosecha más apropiada dependerá de las condiciones climáticas de ese determinado año. El conocer los factores medioambientales que fomentan la infección, el desarrollo y la producción de toxinas es el primer paso para un plan eficaz encaminado a reducir al mínimo las micotoxinas en los alimentos y los piensos. El *Fusarium moniliforme* es el patógeno más conocido del maíz. Dado que es un endofito asintomático transportado por la semilla, será difícil su eliminación. Las prácticas de labranza, la rotación de cultivos, el control de malezas, la pluviosidad en la temporada tardía, el viento y los vectores de plagas son factores que influyen en la cantidad y origen del inóculo fúngico que mantiene el ciclo de la enfermedad en el maíz. Son muchas las estrategias nuevas y prometedoras de precosecha para la prevención que se están explorando y que consisten en la producción de maíz resistente sobre la base de la ingeniería genética. Otra estrategia que puede resultar útil es la del control mediante el empleo de agentes no toxígenos y competitivos biológicamente. La prevención poscosecha de la producción de micotoxinas depende fundamentalmente de unas buenas prácticas de gestión antes de la cosecha y después de ella. Con las estrategias de descontaminación, que pueden ser físicas, químicas o microbiológicas, puede descontaminarse el maíz destruyendo, modificando o absorbiendo la micotoxina de suerte que se reduzcan o se eliminen sus efectos tóxicos. ♦