African Journal of Biotechnology Vol. 2 (12), pp. 570-579, December 2003
Available online at http://www.academicjournals.org/AJB
ISSN 1684–5315

Review

Infection of maize by *Fusarium* species and contamination with fumonisin in africa

P. Fandohan[1*], K. Hell[2], W.F.O. Marasas[3], M.J. Wingfield[4]

[1]Programme on Agricultural and Food Technology, National Institute of Agricultural Research of Benin, P. O. Box 128, Porto-Novo, Benin.
[2]International Institute of Tropical Agriculture (IITA), P. O. Box: 08-0932 Tri Postal, Cotonou, Benin.
[3]Programme on Mycotoxins and Experimental Carcinogenesis (PROMEC), Medical Research Council, P. O. Box 19070, Tygerberg 7505, South Africa.
[4]Forestry and Agricultural Biotechnology Institute (FABI), Faculty of Biological and Agricultural Sciences, University of Pretoria, Pretoria 0002, South Africa.

Accepted 24 November 2003

***Fusarium* is one of the major fungal genera associated with maize in Africa. This genus comprises several toxigenic species including *F. verticillioides* and *F. proliferatum*, which are the most prolific producers of fumonisins. The fumonisins are a group of economically important mycotoxins and very common contaminants of maize-based foods and feeds throughout the world. They have been found to be associated with several animal diseases such as leukoencephalomalacia in horses and pulmonary oedema in pigs. Effects of fumonisins on humans are not yet well understood. However, their occurrence in maize has been associated with high incidences of oesophageal and liver cancer. Infection of maize by *Fusarium* species and contamination with fumonisins are generally influenced by many factors including environmental conditions (climate, temperature, humidity), insect infestation and pre- and postharvest handling. Attempts to control *F. verticillioides* and to detoxify or reduce fumonisin levels in maize have been undertaken. However, more research studies are urgently needed in order to understand more about this toxin. Fumonisins are less documented because they are recently discovered mycotoxins compared to aflatoxins. To date in Africa, apart from South Africa, very little information is available on *Fusarium* infection and fumonisin contamination in maize. It is a matter of great concern that on this continent, millions of people are consuming contaminated maize and maize-based foods daily without being aware of the danger.**

Key words: *Fusarium*, fumonisins, maize, Africa.

INTRODUCTION

Maize (*Zea mays* L.) is a cereal crop grown throughout the word. Maize plays an important role in the diet of millions of African people due to its high yields per hectare, its ease of cultivation and adaptability to different agro-ecological zones, versatile food uses and storage characteristics (Asiedu, 1989). The total production of Africa in 2001 was estimated to be about 42 millions tons (FAO, 2002).

In the field as well as in the store, many pests and parasites attack maize and during the storage period. Insects are most often considered as the principal cause of grain losses (Gwinner et al., 1996). However, fungi are also important and rank second as the cause of deterioration and loss of maize (Ominski et al., 1994). Kossou and Aho (1993) reported that fungi could cause about 50 – 80 % of damage on farmers' maize during the

*Corresponding author. Tel: +229 21 41 60. E-mail: lta@intnet.bj.

storage period if conditions are favourable for their development. The major genera commonly encountered on maize in tropical regions are *Fusarium*, *Aspergillus* and *Penicillium* (Samson, 1991; Orsi et al., 2000).

FUSARIUM SPECIES AND THEIR IMPORTANCE IN MAIZE

Fusarium species are ubiquitous in soils. They are commonly considered as field fungi invading more than 50% of maize grains before harvest (Robledo-Robledo, 1991). Several phytopathogenic species of *Fusarium* are found to be associated with maize including *F. verticillioides* (Sacc.) Nirenberg, *F. proliferatum* (Matsushina) Nirenberg, *F. graminearum* Schwabe and *F. anthophilum* (A. Braun) Wollenweber (Lawrence et al., 1981; Scott, 1993; Munkvold and Desjardins, 1997). Among them, *F. verticillioides* is likely to be the most common species isolated worldwide from diseased maize (Munkvold and Desjardins, 1997). Doko et al. (1996) reported *F. verticillioides* as the most frequently isolated fungus from maize and maize-based commodities in France, Spain and Italy. Likewise, Orsi et al. (2000) found in Brazil that *F. verticillioides* was the predominant *Fusarium* species on maize. In general in Africa, very little information is available on *F. verticillioides* occurrence on maize. Reports of surveys conducted in some African countries however showed it as the most prevalent fungus on maize (Marasas et al., 1988; Allah Fadl, 1998; Baba-Moussa, 1998; Kedera et al., 1999).

F. verticillioides is an endophyte of maize establishing long-term associations with the plant (Baba-Moussa, 1998; Pitt and Hocking, 1999). Symptomless infection can exist throughout the plant in leaves, stems, roots, grains, and the presence of the fungus is in many cases ignored because it does not cause visible damage to the plant (Munkvold and Desjardins, 1997). This suggests that some strains of *F. verticillioides* produce disease in maize and others do not (Bacon and Williamson, 1992).

F. verticillioides infects maize at all stages of plant development, either via infected seeds, the silk channel or wounds, causing grain rot during both the pre- and postharvest periods (Munkvold and Desjardins, 1997). Figure 1 shows *Fusarium* spp. damage on maize cob. A diagrammatic illustration of the disease cycle of *F. verticillioides* in maize is proposed on Figure 2 showing the following possible infection pathways:

- Infection from seed to cob and further to grain through systemic movement in stalk,
- Infection from root to grain through stalk and cob,
- Infection from airborne or water-splashed conidia to silk and further to grain,
- Infection through wounds caused by insects that can also act as vectors of inoculum (Munkvold and Desjardins, 1997).

Figure 1. Apparently healthy maize cob (left) and *Fusarium*-infected maize cob (right).

FUMONISINS AND THEIR TOXICOLOGICAL EFFECTS

Maize contamination by fungi not only renders grains unfit for human consumption by discoloration and reduction of nutritional value, but can also lead to mycotoxin production. Mycotoxins are poisonous secondary metabolites produced by some fungi in staple foods and foodstuffs. Many of them are considered to be important worldwide, but the five most often reported and well documented are deoxynivalenol/nivalenol, zearalenone, ochratoxin, aflatoxins and fumonisins (Pittet, 1998; Pitt, 2000). There is ample evidence that mycotoxin problems affect the agricultural economies of many countries in the world, mainly the African countries. The FAO estimated that each year, between 25% and 50% of the world's food crops are contaminated by mycotoxins (Mannon and Johnson, 1985). The direct impact of mycotoxins on the staple product quality constitutes an important danger for human health and among them fumonisins produced by some toxigenic *Fusarium* species on maize and maize-based foods and feeds increase the risk.

Fumonisins are recently discovered mycotoxins. In 1988, their chemical structure and biological activity were elucidated in South Africa (Gelderblom et al., 1988; Marasas, 2001). Since the discovery of these toxins, numerous research works have been undertaken to investigate further about them. The interesting results found so far have been thoroughly reviewed (Norred, 1993; Riley et al., 1993; Cardwell and Miller, 1996; Gelderblom et al., 1996; Shephard et al., 1996; Marasas, 1996; IPCS, 2000; Bolger et al., 2001; Marasas, 2001; WHO, 2002). These reviews mainly highlighted:

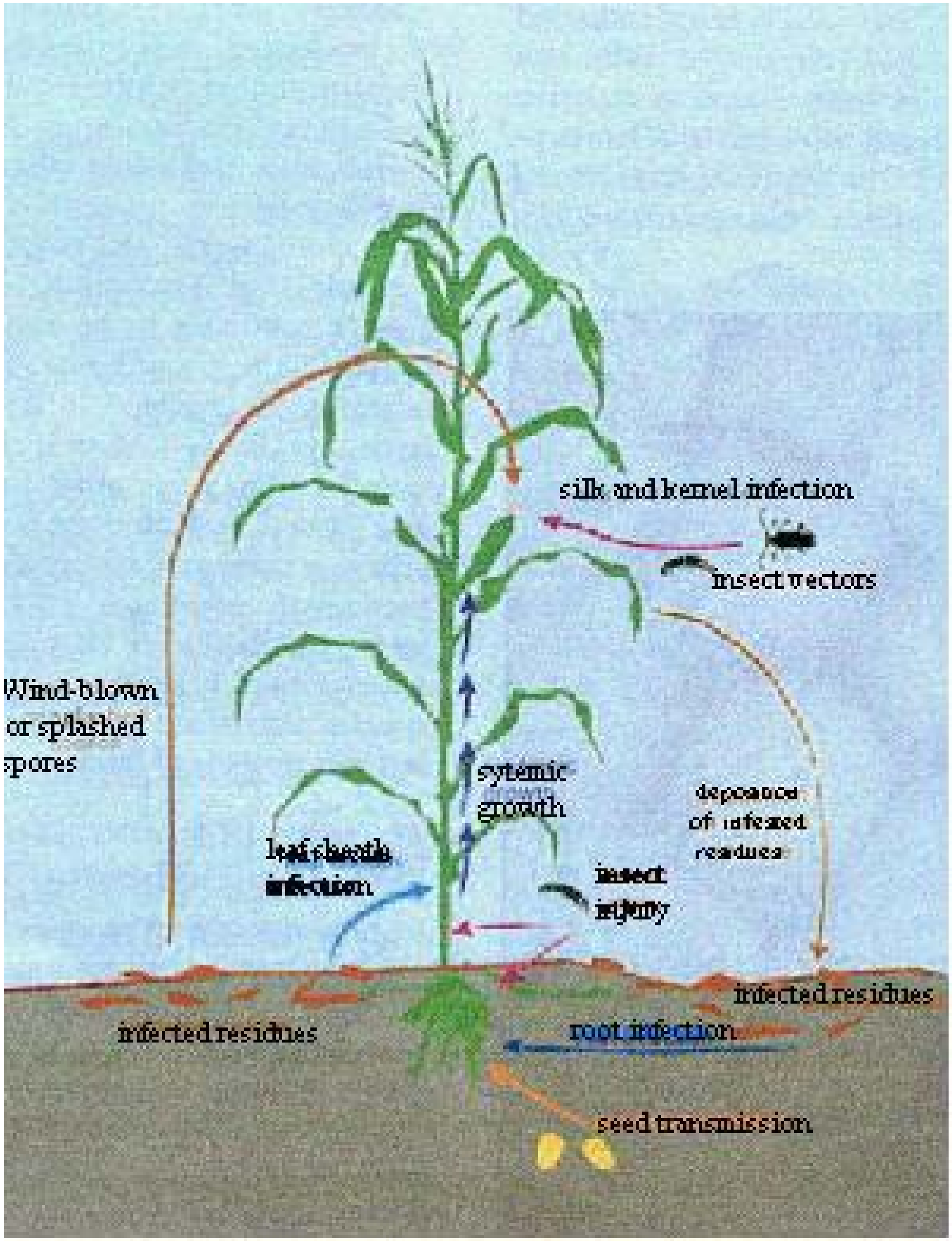

Figure 2. Disease cycle of *F. verticillioides* on maize showing various infection pathways. Source: Munkvold and Desjardins (1997).

- Events leading to the discovery of the fumonisins,
- The toxicological effects of these toxins,
- Their worldwide occurrence in maize and maize-based foods and feeds,
- Their association with animal and human diseases,
- Their impact on animal and human health.

Fumonisins have been found as very common contaminants of maize-based foods and feeds in the United States of America, China, Europe, South America and Africa (Sydenham et al., 1991; Thiel et al., 1992, Visconti and Doko, 1994; Shephard et al., 1996). To date, a total of 28 fumonisin analogs have been identified and characterised (Rheeder et al., 2002). But the most abundantly found in naturally contaminated foods and feeds are FB_1, FB_2, FB_3 (Shephard et al., 1996; Rheeder et al., 2002).

Fumonisins are produced by several *Fusarium* species (Marasas, 2001) including:

- *F. verticillioides* (Sacc.) Nirenberg,
- *F. proliferatum* (Matsushina) Nirenberg,

- *F. nygamai* Burgess & Trimboli,
- *F. anthophilum* (A. Braun) Wollenweber,
- *F. dlamini* Marasas, Nelson & Toussoun,
- *F. napiforme* Marasas, Nelson & Rabie,
- *F. thapsinum* Klittich, Leslie, Nelson & Marasas,
- *F. globosum* Rheeder, Marasas & Nelson.

Amongst these, *F. verticillioides* and *F. proliferatum* are by far the most prolific fumonisin producers (Shephard et al., 1996). They produce the highest amounts of toxins: up to 17900 µg/g of FB_1 have been recorded in cultures for the former, and 31000 µg/g FB_1 for the latter (Rheeder et al., 2002). Maize is the product in which fumonisins are most abundant (Shephard et al., 1996). Fumonisins have been also detected but at lower levels in sorghum (Shetty and Bhat, 1997; Leslie and Marasas, 2001), rice (Abbas et al., 1998) and spices (Pittet, 1998). Fumonisins can contaminate maize foods and feeds as a result of the *Fusarium* invasion before and after harvest (Doko et al., 1995).

Fumonisins have emerged as a highly visible animal and human health safety concern since they have been associated with many animal diseases such as leukoencephalomalacia (LEM) in horses (Marasas, 1996), pulmonary oedema syndrome (PES) in pigs (Harrison et al., 1990; Colvin and Harrison, 1992) and hepatocarcinogenesis in rats (Gelderblom et al., 2001). With respect to humans, studies on the prevalence of oesophageal cancer in regions of South Africa, China, Italy and Iran, revealed an association between this disease and the consumption of maize contaminated by *Fusarium* spp (Franceschi et al., 1990; Rheeder et al., 1992; Chu and Li, 1994; Marasas, 1996; Ueno et al., 1997; Shephard et al., 2000; Wang et al., 2000). The International Agency for Research on Cancer (IARC) evaluated in 1992 the toxins derived from *F. verticillioides* as possibly carcinogenic to humans (IARC, 1993). More recently, based on the research results obtained so far, FB_1 has been evaluated as possibly carcinogenic to humans (class 2B) (IARC, 2002).

Although the effects of fumonisins on humans are not yet well understood, legislation is being put in place to regulate commercial exchanges of fumonisin-contaminated maize and maize-based foods. The US Food and Drug Administration (FDA) recommended that the fumonisin levels should not be higher than 4 µg/g in human foods (FDA, 2000a; FDA, 2000b). In Switzerland, tolerance levels for fumonisins of 1 µg/g in dry maize products intended for human consumption were proposed (Marasas et al., 2001). The Joint FAO/WHO Expert Committee on Food Additives (JECFA) allocated a group provisional maximum tolerable daily intake (PMTDI) of 2 µg/g for FB_1, FB_2 and FB_3, alone or in combination (WHO, 2002). With respect to animals, the total fumonisin maximal tolerable levels recommended by FDA in maize-based feeds are 5 µg/g for horses, 5 µg/g for rabbit, 60 µg/g for ruminants (cattle, sheep, goat) and 100 µg/g for poultry (chicken, turkey, duckling) (FDA, 2000c).

The mechanism of action of fumonisins in animal diseases is quite complex, but it appears that the toxins mainly cause disruption of lipid metabolism in cells, an event that can lead to cellular deregulation or toxic cell injury and finally to cell death (Wang et al., 1991; Riley, 1998; Marasas et al., 2001).

Fumonisins are found to be phytotoxic. FB_1 can indeed damage a wide variety of plants including maize (Scott, 1993; Lamprecht et al., 1994). Doehlert et al. (1994) showed that the presence of high levels of fumonisins in maize seeds might have deleterious effects on seedling emergence. Elongation of maize radicles was inhibited by about 75% after 48 h of imbibition in 100 µg/g of fumonisins and amylase activities in seeds significantly decreased as well.

Fumonisins are also found to be relatively heat stable (Alberts et al., 1990; Howard et al., 1998) and light stable (IARC, 1993). They are also stable in stored products when these are kept in airtight at very low temperatures (Gelderblom et al., PROMEC Unit, Medical Research Council, Tygerberg, South Africa, 2002, unpublished data), or γ-irradiated (Visconti et al., 1996). However, instability of fumonisins in contaminated products over time has been shown (Scott et al., 1999; Kim et al., 2002). Fumonisins are also water soluble (IPCS, 2000).

FACTORS INFLUENCING INFECTION OF MAIZE WITH *FUSARIUM* SPECIES AND FUMONISIN DEVELOPMENT

Infection of maize with *Fusarium* species and its contamination by fumonisins are generally influenced by many factors including environmental conditions (climate, temperature, humidity), insect infestation and pre- and postharvest handling. These factors do not influence infection independently but most often there are complex interactions.

Influence of abiotic factors

A. Environmental factors

Worldwide surveys showed high levels of fumonisins associated with warmer and drier climates (Shephard et al., 1996) and when weather conditions are favourable for *Fusarium* infection (Marasas et al., 2001). At the same location, fumonisin contamination is not necessarily the same from one year to another. Hennigen et al. (2000) found in Argentina a marked difference in terms of fumonisin contamination for the same maize varieties during two consecutive growing seasons, due to the fact that environmental conditions may differ from one growing season to another.

Studying the effect of climatic conditions on fumonisin occurrence in freshly harvested maize in different regions of the State of Parana in Brazil, Ono et al. (1999) detected higher fumonisins levels in maize samples from the Northern and Central-Western regions compared to that from the South. The authors suggested that it could be due to the differences in rainfall levels during the month preceding harvest (92.8 mm in South, 202 mm in North).

Physiological stress during the period just preceding maize harvest, due to drastic oscillations in rainfall and relative humidity, is likely to create favourable conditions for fumonisin production (Visconti, 1996). Shelby et al. (1994) suggested that dry weather at or just prior to pollination of maize might be an important factor for fumonisin production in maize. All this leads to the conclusion that some climatic events such as changes in rainfall patterns or stress during the last stages of maize plant development in the field are likely to have a great influence on fumonisin production in maize before harvest.

Furthermore, temperature and moisture conditions during the growing season as well as during storage are often pointed out to affect maize infection by *Fusarium* spp. and fumonisin synthesis. In this connection, water activity (a_w), the water available for fungal growth, plays a key role. Velluti et al. (2000) working *in vitro* on fungal competition on maize found that the growth rate of *F. verticillioides* was higher at a temperature of 25°C, whereas at 15°C, growth was much lower. These researchers also found that at a constant temperature, the growth rate of *F. verticillioides* increased with water activity. Scott (1993) suggested that the best temperature for production of fumonisin B_1 in maize is 20°C. Marin et al. (1999) rather found that the toxin was optimally produced at 30°C and 0.98 a_w. However, Alberts et al. (1990) showed that the mean FB_1 yield obtained at 25°C (9.5 g/kg) was significantly higher than that at 20°C (8.7 g/kg) and 30°C (0.6 g/kg). Munkvold and Desjardins (1997) reported that *F. verticillioides* generally grows in grain when moisture content is more than 18 – 20%.

B. Agricultural practices

It has been reported that late planting of maize with harvesting in wet conditions favours disease caused by *F. verticillioides* (Bilgrami and Choudhary, 1998), and the prevalence of this fungus is considerably increased with wet weather later in the season (Al-Heeti, 1987). Moreover, repeated planting of maize and other cereal crops in the same or in nearby fields favours fungal infection by increasing the fungal inoculum and insect population that attack maize plants (Bilgrami and Choudhary, 1998). Lipps and Deep (1991) found that the rotation maize/nonhost crop of *Fusarium* was better than maize/maize, as the former was less favourable to *Fusarium* disease outbreak than the latter. Weed control also affects fungal infection in maize fields because it helps to eliminate nonhost weeds on which *Fusarium* can also be found (Bilgrami and Choudhary, 1998).

C. Maize characteristics

The type of maize cultivar and grain characteristics such as colour, endosperm type, chemical composition and stage of development may also influence fungal infection and subsequent fumonisin production. Late-maturing maize cultivars in which grain moisture content decreases slowly below 30% are most susceptible to *Fusarium* disease (Manninger, 1979). It is thought that maize cultivars with upright cobs, tight husks (Emerson and Hunter, 1980), thin grain pericarp (Riley and Norred, 1999), and an increased propensity for grain splitting (Odvody et al., 1990) are likely to be more susceptible to *Fusarium* infection. Tight-husked varieties favour *Fusarium* problems because of slow drying (Dowd, 1998).

Fumonisins are found more concentrated in the pericarp and germ of the grain than in the endosperm, so that removal of those outer parts by mechanical processes such as dehulling can significantly reduce the toxin in maize (Charmley and Prelusky, 1995; Sydenham et al., 1995; FDA, 2000b). However, influence of maize grain colour on fumonisin contamination does not seem to be clear. Shephard et al. (1996) reported that in some years, fumonisin levels were significantly lower in yellow than in white maize, but the reverse situation was observed in other years.

Hennigen et al. (2000) compared contamination of maize varieties of flint endosperm to that of dent type and did not find significant differences. Shelby et al. (1994) tested fifteen maize hybrids and found no significant correlation between starch, lipid, fibre, and protein contents and fumonisin production in maize.

Grain age may also influence fumonisin production in maize. Warfield and Gilchrist (1999) found higher levels of fumonisins in maize grains at the dent stage and significantly lower levels in grains at the immature stage, suggesting that production of the toxin may begin early in cob development and increase as the grains reach physiological maturity. Likewise, Chulze et al. (1996) reported that contamination of maize by fumonisins was greater after physiological maturity.

D. Postharvest operations

Postharvest handling and processing (sorting, washing, dehulling, milling, fermentation, cooking) favourably or unfavourably affect fungal infection and fumonisin production in maize. Mechanical damage during and after harvest may offer entry to the fungal spores either in maize cobs or grains. Dharmaputra et al. (1994) found that motorised shellers can cause mechanical damage on grains providing entry points to fungal spores. Substantial amounts of fumonisins (up to 74%) can be removed by simply washing maize grains, immersing them in water and by removing the upper floating fraction, as contaminated grains generally have a low density (Shetty and Bhat, 1999). These authors also found that removal of the toxin is more significant (about 86%) if salt is added to the water during that process. Likewise, sorting and removal of small, broken and visibly contaminated grains during processing can significantly reduce toxin levels (Charmley and Prelusky, 1995). Steeping maize grains in water has also been found effective in reducing fumonisin content (Canela et al., 1996). In contrast, fermentation of maize does not seem to reduce fumonisin levels (Shephard et al., 1996; Desjardins et al., 2000).

As for milling, Bennett et al. (1996) found that by wet-milling fumonisin-contaminated maize, the toxin distribution in the different fractions is as follows: very little or no fumonisin in the starch fraction, but detectable fumonisins in fibre, germ and steep water fractions. This indicates that maize-based foods derived from the starch fraction are likely to contain less fumonisins than that derived from the other fractions. After dry-milling contaminated maize, fumonisins levels were found lower in grits and higher in germ, bran and fines (Bolger et al., 2001). It has also been shown that fumonisin levels decrease as the level of refinement of maize meal during milling increases (Shephard et al., 1996).

Regarding cooking, it has been observed that fumonisins are fairly heat-stable and that ordinary cooking does not substantially reduce the toxin (Alberts et al., 1990; Scott, 1993). Significant removal of fumonisins is more likely to occur only when temperature during cooking is more than 150°C (Bolger et al., 2001).

Although some processing methods potentially can be selected as favourable ways to reduce fumonisin levels in maize-based products, it is important to keep in mind that their success would depend on many factors including the moisture content of the product, the degree of contamination and distribution of the toxin in the product, and the presence of additives (Charmley and Prelusky, 1995; Bolger et al., 2001).

Influence of biotic factors

A. Storage insects

Insects also play an important role in infection of maize by *Fusarium* spp. They can act as wounding agents or as vectors spreading the fungus from origin of inoculum to plants (Dowd, 1998). Wounding by insects may provide an opportunity for the fungus to circumvent the natural protection of the integument and establish infection sites in the vulnerable interior (Bilgrami and Choudhary, 1998). Borers and insects of the family Nitidulidae are most often cited as favouring maize infection by *Fusarium* spp. They include among others the lepidopteran stem and cob borers (*Ostrinia nubilalis*, *Sesamia calamistis*, *Eldana saccharina*, *Mussidia nigrivenella* and *Busseola fusca*), thrips and sap beetles (family Nitidulidae) (Flett and Van Rensburg, 1992; Munkvold and Desjardins, 1997; Cardwell et al., 2000; Ako et al., 2003). Sobek and Munkvold (1995) found in the USA that damage caused by the European maize borer *Ostrinia nubilalis* increased infection by *F. verticillioides* by three- to ninefold over those with simple mechanical damage. Moreover, larvae of *O. nubilalis* can also act as vectors of *F. verticillioides* by carrying inoculum from plant surfaces into maize cobs (Munkvold et al., 1997). In South Africa, Flett and Van Rensburg (1992) showed that *Busseola fusca* infestation significantly increased the incidence of *F. verticillioides*-infected maize cobs, irrespective of whether the cobs are artificially inoculated with the fungus or not. In a recent study in Benin, it has been observed that cob/stem infection by *F. verticillioides* positively correlated with infestation of *Eldana saccharina*, *Cryptophlebia leucotreta*, *Mussidia nigrivenella* and *Sesamia calamistis* (Schulthess et al., 2002).

Regarding the beetles, it has been shown that not only nitidulid beetles are strongly implicated in *F. verticillioides* infection, but also cucurlionid and silvanid species positively correlated with fungal infection (Cardwell et al., 2000). All these findings pose the problem of cause and effect relationships between fungal infection and insect infestation on maize plants. It is likely that the presence of *F. verticillioides* promotes insect attacks (Schulthess et al., 2002) and insect infestation favours fungal infection (Dowd, 1998). *F. verticillioides* may be introduced into the stem and cob via insects (Munkvold and Carlton, 1997). Likewise, incidence of infection by *F. verticillioides* in maize stems is a source for cob infection by the fungus, not only through movement of the fungus, but also through increased activity of stem borers (Baba-Moussa, 1998). On the other hand, *F. verticillioides* produced volatiles that are quite attractive to nitidulid beetles (Bartelt and Wicklow, 1999). It has been shown that fecundity, laying of eggs and survival of larvae of *Eldana saccharina* were significantly higher on inoculated maize plants (Ako et al., 2003). The authors also found that development time of *Carpophilus dimidiatus* was lower and its fecundity higher on infected grain than on non-infected grain. Schulthess et al. (2002) suggested that keeping the plant free of the fungus could be an effective way to reduce insect damage to both stem and grain. On the other hand, any action also to avoid insect infestation is useful for reducing infection of maize by *F. verticillioides* (Riley and Norred, 1999).

B. Fungal interactions

Interactions among fungi in maize also constitute an important factor influencing fungal infection and subsequent mycotoxin production. Harvested maize grains in the tropical zones contain mycelium and spores of several fungal species including mainly *Fusarium*, *Aspergillus* and *Penicillium* that can come into contact, grow and compete for food if environmental conditions are favourable. As far as *Fusarium* species are concerned, many research reports highlighted their interaction with other fungi. Velluti et al. (2000) showed that populations of *F. verticillioides* and *F. proliferatum*, the most important fumonisin producers, are markedly reduced by the presence of *F. graminearum*, and that fumonisin B_1 (FB_1) production by them can be significantly inhibited as well in the presence of *F. graminearum*. On the other hand, Marin et al. (1998) found that *F. verticillioides* and *F. proliferatum* are generally very competitive and dominant against *Aspergillus flavus* and *Penicillium* spp., especially at a_w more than 0.96. This inhibition can lead to significantly reduced aflatoxin contamination in infected grains (Zummo and Scott, 1992).

ATTEMPTS TO CONTROL *F. VERTICILLIOIDES* AND TO DETOXIFY OR REDUCE FUMONISIN LEVELS IN MAIZE

There is strong evidence that due to its endophytic habit, control of *F. verticillioides* in the field is very difficult. Novel control strategies are being investigated and some reported technologies include:

- The use of an endophytic bacterium (e.g. *Bacillus mojavensis*) as a biological control agent on maize seed (Bacon and Hinton, 2000).
- The use of an iodine-based product called Plantpro 45™ as a biocompatible control of the fungus. The active ingredient of that product has been used as a disinfectant in human and animal health care products (Yates et al., 2000).
- The use of non-producing strains of *F. verticillioides* aiming to minimise fumonisin levels in maize (Plattner et al., 2000).

Additional investigations are however needed to render some of those technologies more applicable.

Decontamination of fumonisins in maize and maize-based products by means of chemical reactions is the object of many research studies. Fumonisins are quite stable molecules and their destruction is likely to be also quite difficult. Ammoniation, initially used for detoxify products from aflatoxins has been investigated for fumonisin reduction but does not always give satisfactory results. Scott (1993) reported that treatment of *F. verticillioides* culture material with 2% of ammonium hydroxide at 50°C decreased fumonisin concentration by 89%, but only 32% of toxin reduction were later measured after four days air-drying. In contrast, nixtamalization, the alkaline cooking of maize for tortilla production in Central America, significantly reduces fumonisin concentration in maize (Dombrink-Kurtzman et al., 2000). However, Voss et al. (1996) found that nixtamalized *F. verticillioides* culture remained toxic. This indicates that reduction in detectable fumonisins does not necessarily result in reduced toxicity.

It is therefore clear that detoxification of mycotoxins in foods is not so easy. Sinha (1998) suggested that it must be economical, simple, easy to be applied by unskilled person, not too time-consuming, capable of removing all traces of the active toxin without hazardous chemical residues in the decontaminated food, and does not impair the nutritional quality of the food.

Considering the above-mentioned review of existing findings on fumonisin contamination, several points arise and need to be emphasised.

1. Contamination of food commodities by fumonisins has become a serious food safety problem throughout the world. People are more and more aware that the fumonisins, in addition to aflatoxins, constitute a real threat to human and animal health. However, in contrast to aflatoxins, fumonisins are less documented. Indeed, they are recently discovered mycotoxins and more research studies are urgently needed in order to understand more about them.
2. Some information is available on factors contributing to fumonisin production and on those able to reduce fumonisin levels in foods. However, research results on some factors remain uncertain, or are not applicable to a developing country situation. The need for more information about environmental and agroecological influences, fumonisin toxicology in respect to human and animal health, prevention methods against fungal infection and fumonisin contamination, methods to use for reducing the toxin in foods and other aspects of fumonisins, is great enough to challenge scientists to undertake many research studies.
3. To date in Africa, apart from South Africa, very little information is available on the natural occurrence of both *Fusarium* and fumonisins, although this part of the world is most often suspected of having potentially higher levels of fumonisins due to its position in tropical and subtropical zones. Work undertaken so far in a few African countries basically consisted of sporadic surveys of farmers' stores and retail markets, mostly basing data measurements on a relatively small number of samples (Shephard et al., 1996). It is a matter of great concern that in Africa, millions of people are consuming contaminated maize and maize-based foods daily without being aware of the danger. Efforts are, however, to be saluted in investigating fumonisins contamination in maize and maize-based foods in some African countries other than South Africa such

as Benin, Cameroon, Ghana, Kenya, Zambia and Zimbabwe (Shephard et al., 1996; Doko et al., 1995; Hell et al., 1995; Kedera et al., 1999; Kpodo et al., 2000; Gamanya and Sibanda, 2001; Ngoko et al., 2001). Consequently, there is great need for more investigations on the continent, mainly in the maize production and consumption zones.

REFERENCES

Abbas HK, Cartwright RD, Shier WT, Abouzied MM, Bird CB, Rice LG, Ross PF, Sciumbato GL, Meredith FI (1998). Natural occurrence of fumonisins in rice with *Fusarium* sheaf rot disease. Plant Dis. 82: 22-25.

Ako M, Schulthess F, Gumedzoe MYD, Cardwell KF (2003). The effect of *Fusarium verticillioides* on oviposition behaviour and bionomics of lepidopteran and coleopteran pests attacking the stem and cobs of maize in West Africa. Entomol. Exper. Appl. 106: 201-210.

Alberts JF, Gelderblom WCA, Thiel PG, Marasas WFO, Van Schalkwyk DJ, Behrend Y (1990). Effects of temperature and incubation period on production of fumonisin B_1 by *Fusarium moniliforme*. Appl. Environ. Microbiol. 56: 1729-1733.

Al-Heeti AA (1987). Pathological, toxicological and biological evaluations of *Fusarium* species associated with ear rot of maize. PhD thesis. Wisconsin, Madison University.

Allah Fadl ME (1998). Occurrence and toxigenicity of *Fusarium moniliforme* from freshly harvested maize ears with special references to fumonisin production in Egypt. Mycopathologia 140: 99–103.

Asiedu JJ (1989). Processing tropical crops. A technological approach. The Macmillan Press, London and Basingstoke. 266 p.

Baba-Moussa AAMT (1998). La microflore associée aux dégâts des lépidoptères foreurs de tiges et mineurs d'épis de maïs (*Zea mays*) dans la région Sud du Bénin avec référence spéciale à *Fusarium moniliforme* Sheld. Mémoire d'Ingénieur Agronome. Université du Bénin, Lomé, Togo.

Bacon CW, Hinton DM (2000). Biological control of *Fusarium moniliforme* in corn by competitive exclusion using *Bacillus mojavensis*. Aflatoxin/Fumonisin Workshop. October 25–27, 2000. Tenaya Lodge, Fish Camp, Yosemite, California, USA. p 35–37.

Bacon CW, Williamson JW (1992). Interactions of *Fusarium moniliforme*, its metabolites and bacteria with corn. Mycopathologia 117: 65–71.

Bartelt RJ, Wicklow DT (1999). Volatiles from *Fusarium verticillioides* (Sacc.) Nirenb. and their attractiveness to Nitidulid beetles. J. Agric. Food Chem. 47: 2447-2454.

Bennett GA, Richard JL, Eckhoff SR (1996). Distribution of fumonisins in food and feed products prepared from contaminated corn. In: Jackson LS, Devries JW, Bullerman LB. eds. Fumonisins in Food. Plenum Press, New York. p 317–322.

Bilgrami KS, Choudhary AK (1998). Mycotoxins in preharvest contamination of agricultural crops. In: Sinha KK, Bhatnagar D. eds. Mycotoxins in agriculture and food safety. Marcel Dekker, New York. p 01-43.

Bolger M, Coker RD, DiNovi M, Gaylor D, Gelderblom W, Olsen M, Paster N, Riley RT, Shephard G, Speijers GJA (2001). Fumonisins. In: Safety Evaluation of Certain Mycotoxins in Food, WHO Food Additives Series 47, FAO Food and Nutrition Paper 74, Prepared by the 56th Meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA), WHO, Geneva. P 103-279.

Canela R, Pujol R, Sala N, Sanchis V (1996). Fate of fumonisin B_1 and B_2 in steeped corn kernels. Food Addit. Contam. 13: 511-517.

Cardwell KF, Kling JG, Maziya-Dixon B, Bosque-Perez NA (2000). Interactions between *Fusarium verticillioides*, *Aspergillus flavus*, and insect infestation in four maize genotypes in lowland Africa. Phytopathology 90: 276–284.

Cardwell KF, Miller JD (1996). Mycotoxins in Foods in Africa. Nat. Toxins. 4: 103-107.

Charmley LL, Prelusky DB (1995). Decontamination of *Fusarium* mycotoxins. Appl. Environ. Microbiol. 1: 421–435.

Chulze SN, Ramirez ML, Farnochi MC, Pascale M, Visconti A, March G (1996). *Fusarium* and fumonisin occurrence in Argentina corn at different ear maturity stages. J. Agric. Food Chem. 44: 2797–2801.

Colvin BM, Harrison LR (1992). Fumonisin-induced pulmonary edema and hydrothorax in swine. Mycopathologia 117: 79–82.

Chu FS, Li GO (1994). Simultaneous occurrence of fumonisin B_1 and other mycotoxins in mouldy corn collected from the People's Republic of China in regions with high incidence of oesophageal cancer. Appl. Environ. Microbiol. 60: 847–852.

Desjardins AE, Manandhar G, Plattner R.D, Maragos C.M, Shrestha K, McCormick SP (2000). Occurrence of *Fusarium* species and mycotoxins in Nepalese maize and wheat and the effect of traditional processing methods on mycotoxin levels. J. Agric. Food Chem. 48: 1377–1383.

Dharmaputra OS, Purwadaria HK, Susilo H, Ambarwati S (1994). The effects of drying and shelling on *Fusarium* spp. infection and *Fusarium* toxins production in maize. URL: http://library.biotop.org/administrative_report.htm.

Doehlert DC, Knutson CA, Vesonder RF (1994). Phytotoxic effects of fumonisin B_1 on maize seedling growth. Mycopathologia 127: 117-121.

Doko MB, Canet C, Brown N, Sydenham EW, Mpuchane S, Siame BA (1996). Natural co-occurrence of fumonisins and zearalenone in cereals and cereal-based foods from eastern and southern Africa. J. Agric. Food Chem. 44: 3240–3243.

Doko MB, Rapior S, Visconti A, Schjoth JE (1995). Incidence and levels of fumonisin contamination in maize genotypes grown in Europe and Africa. J. Agric. Food Chem. 43: 429–434.

Dombrink-Kurtzman M.A, Dvorak TJ, Barron M.E, Rooney LW (2000). Effect of nixtamalization (alkaline cooking) on fumonisin-contaminated corn for production of masa and tortillas. J. Agric. Food Chem. 48: 5781–5786.

Dowd PF (1998). Involvement of arthropods in the establishment of mycotoxigenic fungi under field conditions. In: Sinha KK, Bhatnagar D. eds. Mycotoxins in agriculture and food safety. Marcel Dekker, New York. p. 307-350.

Emerson PM, Hunter RB (1980). Response of maize hybrids to artificially inoculated ear mould incited by *Gibberella zeae*. Can. J. Plant Sci. 60: 1463.

FAO (1996). Combattre la faim et la malnutrition. Journée mondiale de l'alimentation. FAO Press Releases. Food and Agriculture Organisation, Rome, Italy. URL: http://www.fao.org.

FAO (2002). FAOSTAT Database, Food and Agriculture Organisation, Roma, Italy. URL: http://apps.fao.org/lim500/nph-wrap.pl.

FDA (2000a). Guidance for Industry: Fumonisin levels in human food and animal feed. Draft Guidance. USA Food and Drug Administration, Centre for Food Safety and Applied Nutrition, Centre for Veterinary Medicine. URL: http://vm.cfsan.fda.gov/~dms/fumongui.html.

FDA (2000b). Background Paper in Support of Fumonisin Levels in Corn and Corn Products Intended for Human Consumption. USA Food and Drug Administration, Centre for Food Safety and Applied Nutrition. URL: http://vm.cfsan.fda.gov/~dms/fumonbg1.html.

FDA (2000c). Background Paper in Support of Fumonisin Levels in Animal Feeds. (Draft) Guidance for Industry: Fumonisin Levels in Human Foods and Animal Feeds. URL: http://vm.cfsan.fda.gov/~dms/fumonbg2.html.

Flett BC, Van Rensburg JBJ (1992). Effect of *Busseola fusca* on the incidence of maize ear rot caused by *Fusarium moniliforme* and *Stenocarpella maydis*. S. Afr. J. Plant Soil 9: 177–179.

Franceschi S, Bidoli E, Baron AE, La Vechhia C (1990). Maize and the risk of cancers of the oral cavity, pharynx, and oesophagus in North-eastern Italy. J. Nat. Cancer Inst. 82: 1407–1410

Gamanya R, Sibanda L (2001). Survey of *Fusarium moniliforme* (*F. verticillioides*) and production of fumonisin B_1 in cereal grains and oilseeds in Zimbabwe. Int. J. Food Microbiol. 71: 145-149.

Gelderblom WCA, Abel S, Smuts CM, Marnewick J, Marasas WFO, Lemmer ER, Ramljak D (2001). Fumonisin-induced hepatocarcinogenesis: mechanisms related to cancer initiation and promotion. Environ. Health Perspect. 109: 291–299.

Gelderblom WCA, Jaskiewicz K, Marasas WFO, Thiel PG, Horak R.M, Vleggaar R, Kriek NPJ (1988). Fumonisins-Novel mycotoxins with cancer-promoting activity produced by *Fusarium moniliforme*. Appl. Environ. Microbiol. 54: 1806–1811.

Gelderblom WCA, Snyman SD, Abel S, Lebepe-Mazur S, Smuts CM, Van der Westhuizen L, Marasas WFO, Victor TC, Knasmuller S, Huber W (1996). Hepatotoxicity and carcinogenicity of the fumonisins in rats. A review regarding mechanistic implications for establishing risk in humans. In: Jackson LS, Devries JW, Bullerman LB. eds. Fumonisins in Food. Plenum Press, New York. p 279-295.

Gwinner J, Harnisch R, Müch O (1996). Manuel sur la manutention et la conservation des grains après-récolte. GTZ, Eschborn, Germany. 368 p. URL: http://www.fao.org/inpho/vlibrary/move_rep/x0298f/x0298F31.htm-34k.

Harrison LR, Colvin B.M, Green JT, Newman LE, Cole JR (1990). Pulmonary edema and hydrothorax in swine produced by fumonisin B_1, a toxic metabolite of *Fusarium moniliforme*. J. Vet. Diagn. Invest. 2: 217–221.

Hell K, Udoh J, Setamou M, Cardwell KF, Visconti A (1995). Fungal infection and mycotoxins in maize in the different agroecological zones of Benin and Nigeria, West Africa. In: Cardwell KF. ed. Workshop on mycotoxins in food in Africa. November 6–10, 1995. Cotonou, Republic of Benin. International Institute of Tropical Agriculture. p 31.

Hennigen MR, Valente Soares LM, Sanchez S, Di Benedetto NM, Longhi A, Eyhérabide G, Torroba J, Zanelli M (2000). Fumonisin in corn hybrids grown in Argentina for two consecutive seasons. In: De Koe WJ, Samson RA, van Egmond HP, Gilbert J, Sabino M. eds. Proceeding of the X^{th} international IUPAC symposium on mycotoxins and phycotoxins. 21–25, May 2000. Guaruja, Brazil. p 331–339.

Howard PC, Churchwell MI, Cough LH, Marques MM, Doerge DR (1998). Formation of N-(carboxymethyl) fumonisin B_1, following the reaction of fumonisin B_1 with reducing sugars. J. Agric. Food Chem. 46: 3546-3557.

IARC (1993). IARC monographs on the evaluation of carcinogenic risks to humans Vol. 56. Some naturally occurring substances: food items and constituents, heterocyclic aromatic amines and mycotoxins. International Agency for Research on Cancer, Lyon, France. 599 p.

IARC (2002). IARC monographs on the evaluation of carcinogenic risks to humans Vol. 82. Some traditional herbal medicines, mycotoxins, naphthalene and styrene. International Agency for Research on Cancer, Lyon, France. 590 p.

IPCS (2000). Environmental health criteria 219. Fumonisin B_1. International Programme on Chemical Safety. World Health Organisation, Geneva. 150 p.

Kedera CJ, Plattner RD, Desjardins AE (1999). Incidence of *Fusarium* spp. and levels of fumonisin B_1 in maize in Western Kenya. Appl. Environ. Microbiol. 65: 41-44.

Kim EK, Scott PM, Lau BPY, Lewis DA (2002). Extraction of fumonisins B_1 and B_2 from white rice flour and their stability in white rice flour, cornstarch, cornmeal, and glucose. J. Agric. Food Chem. 50: 3614-3620.

Kossou DK, Aho N (1993). Stockage et conservation des grains alimentaires tropicaux : principes et pratiques. Les Editions du Flamboyant, Benin. 125 p.

Kpodo K, Thrane U, Hald B (2000). *Fusaria* and fumonisins in maize from Ghana and their co-occurrence with aflatoxins. Int. J. Food Microbiol. 61: 147–157.

Lamprecht SC, Marasas WFO, Alberts JF, Cawood ME, Gelderblom WCA, Shephard GS, Thiel PG, Calitz FJ (1994). Phytotoxicity of fumonisins and TA-toxin to corn and tomato. Phytopathology 84: 383-391.

Lawrence EB, Nelson PE, Ayers JE (1981). Histopathology of sweet corn seeds and plants infected with *F. moniliforme* and *F. oxysporum*. Phytopathology 71: 379–386.

Leslie JF, Marasas WFO (2001). *Fusarium* from sorghum: life in interesting times. Proceeding of the 22^{nd} biennial grain sorghum research and utilisation conference, 2001 sorghum industry conference. 18–20 February 2001. Nashville, Tennessee, USA. p 76–83.

Lipps PE, Deep IW (1991). Influence of tillage and crop rotation in yield, stalk rot and recovery of *Fusarium* and *Trichoderma* spp. from corn. Plant Dis. 75: 828-833.

Manninger I (1979). Resistance of maize to ear rot on the basis of natural infection and inoculation. In: Proceeding 10^{th} Meeting, Eucarpia, Maize, Sorghum Sec. Varna, Bulgaria. p 181–184.

Mannon. J and Johnson, E (1985). Fungi down on the farm. New Sci.: 12–16.

Marasas WFO (1996). Fumonisins: history, worldwide occurrence and impact. In: Jackson LS, Devries JW, Bullerman LB. eds. Fumonisins in Food. Plenum Press, New York. p 1-17.

Marasas WFO (2001). Discovery and occurrence of the fumonisins: a historical perspective. Environ. Health Perspect. 109: 239-243.

Marasas WFO, Jaskiewicz K, Venter FS, Van Schalkwyk DJ (1988). *Fusarium moniliforme* contamination of maize in oesophageal cancer areas in Transkei. S. Afr. Med. J. 74: 110-114.

Marasas WFO, Miller JD, Riley RT, Visconti A (2001). Fumonisins – occurrence, toxicology, metabolism and risk assessment. In: Summerell BA, Leslie JF, Backhouse D, Bryden WL, Burgess LW. eds. *Fusarium*. Paul E. Nelson Memorial Symposium. APS PRESS. The American Phytopathological Society. St. Paul, Minnesota. p 332-359.

Marin S, Homedes V, Sanchis V, Ramos A.J, Magan N (1999). Impact of *Fusarium moniliforme* and *F. proliferatum* colonisation of maize on calorific losses and fumonisin production under different environmental conditions. J. Stor. Prod. Res. 35: 15–26.

Marin S, Sanchis V, Ramos AJ, Vinas I, Magan N (1998). Environmental factors, *in vitro* interactions, and niche overlap between *Fusarium moniliforme*, *F. proliferatum*, and *F. graminearum*, *Aspergillus* and *Penicillium* species from maize grain. Mycol. Res. 102: 831–837.

Munkvold GP, Carlton WM (1997). Influence of inoculation method on systemic *Fusarium moniliforme* infection of maize plants grown from infected seeds. Plant Dis. 81: 211–216.

Munkvold GP, Desjardins AE (1997). Fumonisins in maize. Can we reduce their occurrence? Plant Dis. 81: 556–564.

Munkvold G.P, Hellmich RL, Showers WB (1997). Reduced *Fusarium* ear rot and symptomless infection in kernels of maize genetically engineered for European Corn Borer resistance. Phytopathology 87: 1071-1077.

Ngoko Z, Marasas WFO, Rheeder JP, Shephard GS, Wingfield MJ, Cardwell KF (2001). Fungal infection and mycotoxin contamination of maize in the humid forest and the western highlands of Cameroon. Phytoparasitica 29: 352-360.

Norred WP (1993). Fumonisins-Mycotoxins produced by *Fusarium moniliforme*. J. Toxicol. Environ. Health 38: 309-328.

Odvody GN, Remmers JC, Spencer NM (1990). Association of kernel splitting with kernel and ear rots of corn in a commercial hybrid grown in the coastal bend of Texas. Phytopathology 80 : 1045.

Ominski KH, Marquardt RR, Sinha RN, Abramson D (1994). Ecological aspects of growth and mycotoxin production by storage fungi. In: Miller JD, Trenholm HL. eds. Mycotoxins in Grains. Compounds other than Aflatoxin. Eagen Press, USA. p 287–305.

Ono EYS, Sugiura Y, Homechin M, Kamogae M, Vizzoni E, Ueno Y, Hirooka EY (1999). Effect of climatic conditions on natural mycoflora and fumonisins in freshly harvested corn of the State of Parana, Brazil. Mycopathologia 147: 139–148.

Orsi RB, Corrêa B, Possi CR, Schammass EA, Nogueira JR, Dias SMC, Malozzi MAB (2000). Mycoflora and occurrence of fumonisins in freshly harvested and stored hybrid maize. J. Stor. Prod. Res. 36: 75-87.

Pitt JI (2000). Toxigenic fungi: which are important? Med. Mycol. 38: 17–22.

Pitt JI, Hocking AD (1999). Fungi and food spoilage. Second edition. Aspen Publishers, Inc. Gaithersburg, Maryland.

Pittet A (1998). Natural occurrence of mycotoxins in foods and feeds – an updated review. Rev. Méd. Vét. 149: 479–492.

Plattner R, Proctor RH, Brown DW, Desjardins AE (2000). Potential for minimisation of fumonisin levels in corn using fumonisin non-producing strains. Aflatoxin/Fumonisin Workshop. October 25–27, 2000. Tenaya Lodge, Fish Camp, Yosemite, CA, USA. p 35-37.

Rheeder JP, Marasas WFO, Thiel PG, Sydenham EW, Shephard GS, van Schalkwyk DJ (1992). *Fusarium moniliforme* and fumonisins in corn in relation to human oesophageal cancer in Transkei. Phytopathology 82: 353–357.

Rheeder JP, Marasas WFO, Vismer HF (2002). Production of fumonisin analogs by *Fusarium* species. Appl. Environ. Microbiol. 68: 2101–2105.

Riley RT (1998). Mechanistic interactions of mycotoxins: theoretical considerations. In: Sinha KK, Bhatnagar D. eds. Mycotoxins in agriculture and food safety. Marcel Dekker, New York. p 227-253.

Riley RT, Norred WP (1999). Mycotoxin prevention and decontamination. Corn – a case study. Third Joint FAO/WHO/UNEP International Conference on Mycotoxins, Tunis, Tunisia, 3–6 March 1999, 11 p.

Riley RT, Norred WP, Bacon CW (1993). Fungal toxins in foods: Recent concerns. Ann. Rev. Nutr. 13: 167-189.

Robledo-Robledo E (1991). Strategies for the prevention and control of fungi and mycotoxins in Central and South America. In: Champ BR, Highley E, Hocking AD, Pitt JI. eds. Fungi and mycotoxins in stored products. Proceedings of an international conference, Bangkok, Thailand, 23–26 April 1991. p 39–46.

Samson RA (1991). Identification of food-borne *Penicillium*, *Aspergillus*, and *Fusarium* species. In: Champ BR, Highley E, Hocking AD, Pitt JI. eds. Fungi and mycotoxins in stored products. Proceedings of an international conference, Bangkok, Thailand, 23–26 April 1991.

Schulthess F, Cardwell KF, Gounou S (2002). The effect of endophytic *Fusarium verticillioides* on infestation of two maize varieties by lepidopterous stemborers and coleopteran grain feeders. Phytopathology 92: 120–128.

Scott PM (1993). Fumonisins. Int. J. Food Microbiol. 18: 257–270.

Scott PM, Lawrence GA, Lombaert GA (1999). Studies on extraction of fumonisins from rice, corn-based foods and beans. Mycotoxin Res. 15: 50-60.

Shelby RA, White DG, Bauske EM (1994). Differential fumonisin production in maize hybrids. Plant Dis. 78: 582–584.

Shephard GS, Marasas WFO, Leggott NL, Yazdanpanah H, Rahimian H, Safavi N (2000). Natural occurrence of fumonisins in corn from Iran. J. Agric. Food Chem. 48: 1860–1864.

Shephard GS, Thiel PG, Stockenstrom S, Sydenham EW (1996). Worldwide survey of fumonisin contamination of corn and corn-based products. J. AOAC Int. 79: 671–687.

Shetty PH, Bhat RV (1997). Natural occurrence of fumonisin B_1 and its co-occurrence with aflatoxin B_1 in Indian sorghum, maize and poultry feeds. J. Agric. Food Chem. 45: 2170–2173.

Shetty PH, Bhat RV (1999). A physical method for segregation of fumonisin-contaminated maize. Food Chem. 66: 371–374.

Sinha KK (1998). Detoxification of mycotoxins and food safety. In: Sinha KK, Bhatnagar D. eds. Mycotoxins in agriculture and food safety. Marcel Dekker, New York. p 381-405.

Sobek EA, Munkvold GP (1995). European corn borer as a vector of *Fusarium moniliforme* in symptomatic and asymptomatic infection of corn kernels. Phytopathology 85: 1180.

Sydenham EW, Shephard GS, Thiel PG, Marasas WFO, Stockenstrom S (1991). Fumonisin contamination of commercial corn-based human foodstuffs. J. Agric. Food Chem. 39: 2914-2018

Sydenham EW, Stockenstrom S, Thiel PG, Shephard GS, Koch KR, Marasas WFO (1995). Potential of alkaline hydrolysis for the removal of fumonisins from contaminated corn. J. Agric. Food Chem. 43: 1198-1201.

Thiel PG, Marasas WFO, Sydenham EW, Shephard GS, Gelderblom WCA (1992). The implications of naturally occurring levels of fumonisins in corn for human and animal health. Mycopathologia 117: 3–9.

Ueno Y, Iijima K, Wang SD, Sugiura Y, Sekijima M, Tanaka T, Chen C, Yu SZ (1997). Fumonisins as a possible contributory risk factor for primary liver cancer: a 3-year study of corn harvested in Haimen, China, by HPLC and ELISA. Food Chem. Toxicol. 35: 1143–1150.

Velluti A, Marin S, Bettucci L, Ramos AJ, Sanchis V (2000). The effect of fungal competition of maize grain by *Fusarium moniliforme*, *F. proliferatum* and *F. graminearum* and on fumonisin B_1 and zearalenone formation. Int. J. Food Microbiol. 59: 59-66.

Visconti A (1996). Fumonisins in maize genotypes grown in various geographic areas. In: Jackson LS, de Vries JW, Bullerman LB. eds. Fumonisins in food. Plenum Press, New York. p 193–204.

Visconti A, Doko MB (1994). Survey of fumonisin production by *Fusarium* isolated from cereals in Europe. J. AOAC Int. 77: 546–550.

Visconti A, Solfrizzo M, Doko MB, Boenke A, Pascale M (1996). Stability of fumonisins at different storage periods and temperatures in γ-irradiated maize. Food Addit. Contam. 13: 929-938.

Voss KA, Bacon CW, Meredith FI, Norred WP (1996). Comparative subchronic toxicity studies of nixtamalized and water-extracted Fusarium moniliforme culture material. Food Chem. Toxicol. 34: 623-632.

Wang E, Norred WP, Bacon CW, Riley RT, Merrill AH (1991). Inhibition of sphingolipid biosynthesis by fumonisins: implications for diseases associated with *Fusarium moniliforme*. J. Biol. Chem. 266: 14486–14490.

Wang H, Wei H, Ma J, Luo X (2000). The fumonisin B_1 content in corn from North China, a high-risk area of esophageal cancer. J. Environ. Pathol. Toxicol. Oncol. 19: 139–141.

Warfield CY, Gilchrist DG (1999). Influence of kernel age on fumonisin B1 production in corn by *Fusarium moniliforme*. Appl. Environ. Microbiol. 65: 2853–2856.

WHO (2002). Evaluation of certain mycotoxins in food. Fifty-sixth report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series 906. World Health Organisation, Geneva.

Yates I, Arnold J, Hinton DM, Basinger W, Walcott R (2000). The use of an iodine-based product as a biocompatible control of *Fusarium moniliforme*. Aflatoxin/Fumonisin Workshop, October 25–27, 2000. Tenaya Lodge, Fish Camp, Yosemite, California, USA, pp. 35–37.

Zummo N, Scott GE (1992). Interaction of *Fusarium moniliforme* and *Aspergillus flavus* on kernel infection and aflatoxin contamination in maize ears. Plant Dis. 76: 771–773.