

Fusarium Grain Mold: The Major Component of Grain Mold Disease Complex in Sorghum (*Sorghum bicolor* L. Moench)

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ABSTRACT

Sorghum grain mold, particularly Fusarium and Curvularia grain mold, are important on improved, short- and medium-duration sorghum cultivars worldwide. There is hardly any literature that deals with Fusarium grain mold as a component of sorghum grain mold disease complex in detail. This review summarizes many aspects related to Fusarium grain mold: typical symptoms, causal organisms in relation to mold development phases, colonization processes, nature of damage and its significance on yield and quality and management options. The review also attempts to shortlist probable mechanisms that might be useful for developing resistance against early infection events. There are three major and proven pathogenic species of *Fusarium* (*F. andiyazi*, *F. proliferatum* and *F. thapsinum*) that are capable of infecting sorghum flower. Others are predominantly saprophytes. Some strains of these species are highly toxigenic and responsible for Fusarial-toxicoses in human beings, animals and poultry birds. There is necessity to identify resistance against Fusarium grain mold in general and toxigenic strains in particular and incorporate the resistance in new varieties and hybrids of sorghum.

Keywords: grain mold, Fusarium grain mold, sorghum, grain quality, Fusarial-toxins

Abbreviations: ELEM, equine leucoencephalomalacia; FB1, fumonisin B1; FGM, Fusarium grain mold; GM, grain mold; GMDC, grain mold disease complex; GW, grain weathering; PGM, panicle grain mold rating; PM, physiological maturity; QTL, quantitative trait locus; RIP, ribosome inactivating protein; TGM, threshed grain mold rating

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INTRODUCTION

Grain mold is a major biotic constraint in the way of production, marketing and utilization of sorghum grain. It is one of the most important diseases of sorghum in many countries in Asia, Africa, North America and South America (Williams and Rao 1981; Frederiksen *et al.* 1982; Louvel and Arnoud 1984). The disease is particularly important on improved, short- and medium-duration sorghum cultivars that mature during the rainy season in humid, tropical

and subtropical climates. Usually the term ‘grain mold’, in literature, is used to describe the diseased appearance of sorghum grains resulting from infection by one or more pathogenic or saprophytic fungi. Infection may take place on developing as well as mature grain in the field. The term ‘grain mold disease complex’ (GMDC) has been used in a few instances to describe the above disease conditions (Prom *et al.* 2003). In a few literature grain mold development or grain deterioration stages have been divided into two phases: (i) ‘grain mold’ (GM) that occurs before phy-

Table 1 *Fusarium*-induced diseases in sorghum.

Growth stages	Disease	Causal organism	Reference
Pre- or post-emergence seedling	Damping-off	<i>Pythium</i> , <i>Fusarium</i> , <i>Aspergillus</i> , <i>Rhizoctonia</i> , <i>Phoma</i> spp.	Frederiksen 1986
Vegetative stage	Root and stalk rot	<i>F. moniliforme</i>	Reed <i>et al.</i> 1983
	Pokkah boeng or twisted top	<i>F. moniliforme</i> var <i>subglutinans</i>	Frederiksen and Duncan 1992
Reproductive stage	Grain mold	<i>F. moniliforme</i> (<i>F. andiyazi</i> , <i>F. proliferatum</i> , <i>F. thapsinum</i>)*	Zummo 1984; Onyike and Nelson 1992; Navi <i>et al.</i> 1999; Summerell <i>et al.</i> 2003
	Head blight	<i>F. moniliforme</i>	Frederiksen <i>et al.</i> 1973

*only proven pathogens that cause grain mold disease before physiological maturity are given here. Grain mold disease complex is caused by many *Fusarium* spp. (for details please refer to the text).

siological maturity (PM) and (ii) 'grain weathering' (GW) that takes place after PM (Forbes *et al.* 1992; Bandyopadhyay *et al.* 2000). In sorghum, PM is denoted by the deposition of black layer at the hilar end (Castor 1981). GM is caused by pathogenic fungi and so can be called 'pathogenic mold'. GW, on the other hand, is caused by both pathogenic (which infect immature grain, remain there and push through the pericarp when environmental conditions are appropriate) and saprophytic (which invade dead grain tissue) fungi and is entirely influenced by environment (Forbes *et al.* 1992; Audilakshmi *et al.* 2011). For purpose of this review, the terminologies namely, GM (refers to grain mold occurring before PM), GW (refers to grain mold occurring after PM), and GMDC (includes both GM and GW) have been used.

Several fungi (more than 40 genera) are associated with sorghum grain (Williams and Rao 1981). Most of these fungi are generally restricted to the pericarp, but penetration into the endosperm can occur if the mature grain is exposed to high humidity for extended period (Glueck and Rooney 1980). Depending upon the timing and degrees of penetration, these fungi are considered to be saprophytes or apathogenic weak parasites (Neergaard 1977). The frequently encountered genera are *Fusarium*, *Curvularia*, *Phoma*, *Alternaria*, *Drechslera*, *Cladosporium*, *Aspergillus* and *Olpitrichum*. However, only a few species infect sorghum flower during the early stages of grain development and are considered as pathogens. On approximate order of importance these are *Fusarium moniliforme*, *Curvularia lunata*, *Fusarium semitectum*, and *Phoma sorghina*. The work of Rao and Williams (1977) and of Castor and Frederiksen (1977) clearly showed that the principal grain mold fungi were pathogen and the problem of mold was not due to saprophytic fungi invading a source of carbohydrate under moisture conditions; rather it was a problem of pathogenic fungi. Notwithstanding the focus of most of the studies on resistance breeding had been related to GW rather than pathogenic mold *per se*. However, success in breeding for resistance to GW has been slow because of many mechanisms governing resistance, complex genetics and large environmental influence (Hall *et al.* 2000). Recently, the focus has been directed towards development of resistance against mold components, particularly the pathogenic components, rather than GMDC (Nutsugah and Wilson 2007; Das *et al.* 2010; Prom *et al.* 2011; Sharma *et al.* 2011). Once resistance genes against pathogenic components are identified subsequently, the resistance can be pyramided into a single sorghum cultivar using an appropriate breeding programme. Huge amount of literature is available on GMDC including some reviews (Williams and Rao 1981; Williams and McDonald 1983; Forbes *et al.* 1992; Chandrashekar *et al.* 2000). Focal areas of most of these literatures have been the development of resistance against GW while little information is available on works exclusive to pathogenic grain mold. Recently, Leslie and Marasas (2002) have given a detailed account of *F. moniliforme* with respect to sorghum and millets. But there is hardly any review in literature that deals with *Fusarium* as a component of GMDC. This paper reviews the role of *Fusarium* (Fusarium grain mold) in GMDC and its significance in sorghum production, marketing and utilization.

FUSARIUM AND SORGHUM

The genus *Fusarium* was introduced by Link in 1809 (Link 1809). Members of this genus are among the most widespread and important plant pathogens in the world. *F. moniliforme* is the name that has traditionally been used for various isolates of *Fusarium* from the Liseola section of the genus recovered from sorghum stalks and grain (Wollenweber and Reinking 1935). Starting from seedling to harvest followed by storage of the grain several *Fusarium* species are associated with sorghum. *F. moniliforme*, is the pathogen of several diseases of sorghum including seedling blight, root and stalk rot, pokkah boeng or twisted top, grain mold and head blight (Table 1) worldwide.

SYMPTOMS OF FUSARIUM GRAIN MOLD

Fusarium grain mold (FGM) is a component of GMDC and is caused by *Fusarium* spp. Symptoms of FGM vary with the severity of infection and grain development stages. The first visible symptom of *Fusarium* infection is pigmentation of the spikelet tissues including sterile lemma, palea, lodicules and glumes. Anthers and filaments can also be infected depending on severity of infection. Early infection of sorghum floret at anthesis results in loss of caryopsis formation (Little and Magill 2009), florets blasting, poor seed setting and development of small and shriveled grains (Castor and Frederiksen 1980) (Fig. 1). Under humid conditions severely infected grains become fully covered by fungal growth even before PM and such grains disintegrate under slight pressure. Disintegration of molded grain before PM is termed as 'pre-mature kernel rot' or 'kernel rot' (DSR 2010). *Fusarium* species generally produce pinkish white mycelium; powdery in appearance during early stages which later becomes pinkish fluffy and fluffy white (Fig. 2). Internal colonization of grain often leads to sprouting of grains in the field under wet conditions. Such sprouted grains become soft due to the digestion of parts of the endosperm by α -amylase and are predisposed to extensive colonization by mold fungi, primarily species of *Fusarium* and *Curvularia*. Pre-harvest sprouting can occur as early as 15 days after pollination (Maiti *et al.* 1985; Steinbach *et al.* 1995). Often the infected grains are discolored. Discoloration of grain is more prominent in white-grain than in brown/red grain sorghums. Some apparently normal grain may not show external symptoms but produce fungal growth on incubation. Fungal growth first occurs at the hilar end of the grain, and subsequently extends on the pericarp surface. Sometimes the fungus damages large areas of panicle including peduncle and rachis branches and spikelets, resulting in blighted panicle or head blight. Moisture content in grains of such blighted panicles becomes significantly less (10.4%) than that of normal panicles (13.0%) at harvest (Castor and Frederiksen 1980).

THE CAUSAL ORGANISM

Present understanding of *Fusarium* classification based on morphology and sexual cross-fertility (mating type), divides *F. moniliforme* into 14 recognized species (Leslie and Marasas 2002). Of these, *F. andiyazi*, *F. nygamai*, *F. proliferatum*, *F. thapsinum* and *F. verticillioides* have been reported to be

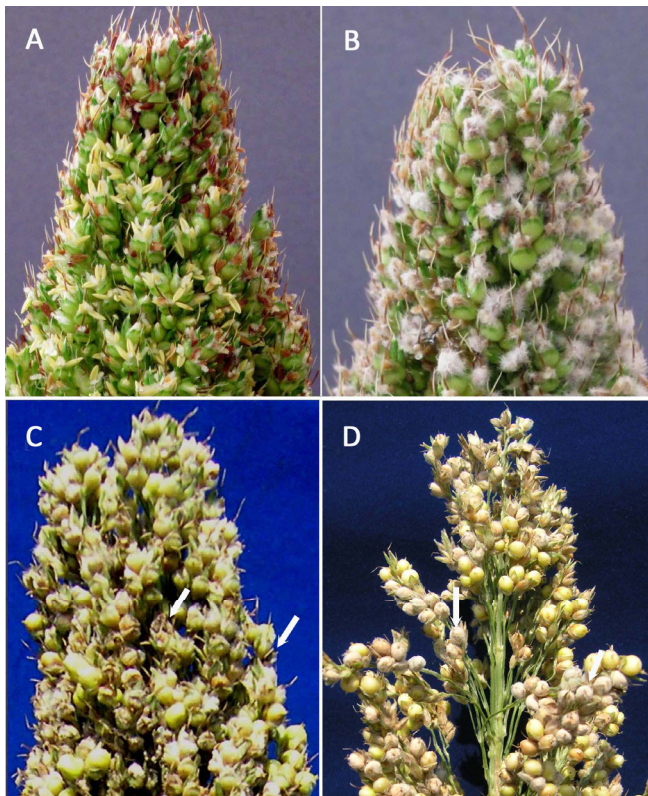


Fig. 1 Symptoms of *Fusarium* grain mold at pre-maturity stage. (A) Disease-free panicle at flowering; (B) symptoms (fungal growth) on spikelets, anthers and filaments; (C) blasted florets and shriveled grains (arrow) and (D) colonization on immature grain (arrow).

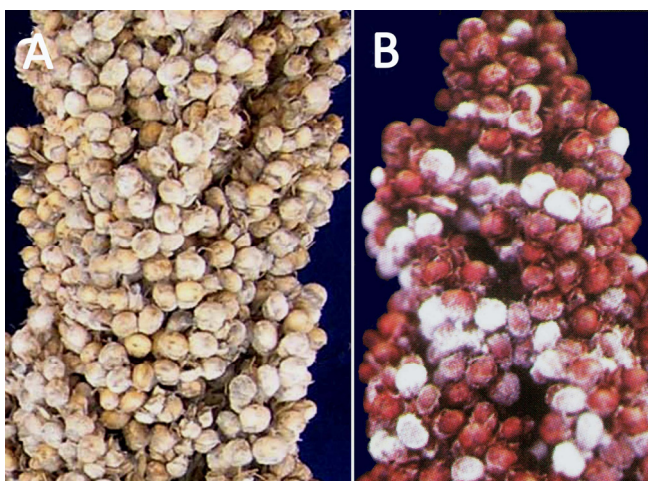


Fig. 2 Symptoms of *Fusarium* grain mold at post-maturity stage. (A) White and (B) red grain cultivar.

associated with sorghum grain and *F. napiforme* with sorghum field debris (Table 2). Other *Fusaria* that are not *F. moniliforme* but have been frequently isolated from sorghum grain in different countries include *F. anthophilum*, *F. chlamydosporum*, *F. culmorum*, *F. equiseti*, *F. graminearum*, *F. oxysporum*, *F. pallidoroseum*, *F. sacchari*, *F. semitectum*, *F. solani* and *F. sporotrichioides*. It is important to note that the above mentioned species were isolated from mature sorghum grain (weathered grain) either collected from field, farmer's storage or from the market. Therefore, all of them may not be actual pathogen that can infect sorghum floret and cause GM. However, some of these saprophytes may be important for weathering of sorghum grain in the field. Till now there are only a few pathogenic *Fusarium* species (e.g., *F. andiyazi*, *F. proliferatum* and *F. thapsinum*) with proven capacity to cause GM in sorghum (Summerell *et al.* 2003;

Little and Magill 2009; Prom *et al.* 2011). It would be interesting to know whether *F. graminearum*, a serious pathogen causing diseases on panicles of many important cereals (wheat, barley, maize, etc.) (Table 3), can infect sorghum floret and cause GM. To date pathogenicity of *F. graminearum* to sorghum is not clear, though there is report to include this in the list of fungi associated with early infection events of sorghum floret along with *F. thapsinum* (Little 2000).

SOURCES OF INOCULUM

The *Fusarium* species causing grain mold of sorghum can be soil-borne, airborne, or carried in plant residue, and can be recovered from any part of an infected plant from the root to the flower (Burgess and Trimboli 1986; Marasas *et al.* 1987; Leslie *et al.* 1990; Klittich *et al.* 1997). Plant residues and soil debris containing fungal hyphae and conidia seem to be the primary sources of inocula in the field. Special fungal structures (e.g., chlamydospores) may not be essential for winter survival of *F. moniliforme*. Manzo and Claflin (1984) have demonstrated that conidia and hyphae of this fungus in sorghum stalks could survive two winters in Kansas without any loss of viability or pathogenicity. Liddell and Burgess (1985) further demonstrated that *F. moniliforme* microconidia can survive up to 900 days at different levels of humidity and temperature under laboratory conditions. Crop residue buried deep (30 cm) generally survives longer than the surface residue (Nelson *et al.* 1983). The natural inocula present over sorghum field during rainy season also suggested being sufficient for development of GMDC without any artificial inoculation (Bandyopadhyay *et al.* 1991).

INFECTION AND COLONIZATION

Compared to huge amount of literature that is available on various aspects of GMDC, limited information is available on infection and colonization processes of sorghum floret by mold pathogen *vis-à-vis* histopathology. Presently available information is based on three independent studies conducted during mid-eighties (Castor 1981; Bandyopadhyay 1986; Forbes 1986). Comparatively less focus on this area might have been resulted from bestowing major emphasis on identification of resistance to GW that involves colonization of grain by saprophytes during post-maturity stage. All three studies indicate similar patterns of initial infection and subsequent colonization of sorghum spikelet tissues. Later, Forbes *et al.* (1992) reviewed colonization events in mold resistant and susceptible cultivars in detail. Early events can be summarized as follows: initial infection by *F. moniliforme* occurs on the apical ends on the spikelet tissues including lemma, palea, glumes, filaments, and senescing styles. Fungal mycelium advances basipetally, either by colonizing spikelet tissues or by growing in voids between these tissues. Early colonization of glumes was found to be very heavy and caused little cellular disruption or pigmentation in the host (Forbes 1986). Within 5 days of inoculation, mycelium can be seen in all parts of the spikelet, with a denser growth around the ovary base. Lodicules appear to serve as an important energy source, and are always surrounded by dense fungal growth. From this energy source, near the point of attachment to the pedicel, infection of the ovary wall occurs. In the next stages of invasion, a dense mycelial mat progresses acropetally, between the aleurone layer and the pericarp. Subsequent invasion of the endosperm, embryonic tissues, and pericarp originates from this peripheral mat. Glueck and Rooney (1980) observed colonies of fungi in the starchy mesocarp and the cross and tube cells of the pericarp in grain at PM. When environmental conditions are appropriate, mycelial growth pushes through the pericarp, producing a white or pink fungal mass which can completely cover the grain. There are differences in early invasion processes and responses in resistant and susceptible cultivars. Resistant cultivar shows

Table 2 *Fusarium* species associated with sorghum.

Sample type	<i>Fusarium</i> spp.	Reported country	References
Sorghum grain	<i>F. andiyazi</i> [#]	India, South Africa, Nigeria, USA	Thakur <i>et al.</i> 2006; Sharma <i>et al.</i> 2011; Marasas <i>et al.</i> 2001
	<i>F. anthophilum</i>	India, Tanzania, USA	Sreenivasa <i>et al.</i> 2008; Mansuetus <i>et al.</i> 1997; Leslie and Plattner 1991
	<i>F. chlamydosporum</i>	Zimbabwe, Nigeria	Onyike and Nelson 1992
	<i>F. culmorum</i>	Ghana, Mali, Niger	Zummo 1984
	<i>F. equiseti</i>	India, Nigeria	Sharma <i>et al.</i> 2011; Tyagi 1980; Onyike and Nelson 1992
	<i>F. graminearum</i>	Zimbabwe, Nigeria	Onyike and Nelson 1992
	<i>F. moniliforme</i>	India, Zimbabwe, Ghana, Mali, Nigeria, Argentina, Colombia, Venezuela	Gopinath and Shetty 1986; Navi <i>et al.</i> 1999 ; Onyike and Nelson 1992; Zummo 1984; Tyagi 1980; Teyssandier 1992
	<i>F. nygamai</i> [#]	Zimbabwe, Nigeria	Onyike and Nelson 1992
	<i>F. oxysporum</i>	India, Ghana, Mali, Niger	Gopinath and Shetty 1986; Sreenivasa <i>et al.</i> 2008; Zummo 1984
	<i>F. pallidoroseum</i>	India	Sreenivasa <i>et al.</i> 2008
	<i>F. proliferatum</i> [#]	India, Tanzania, USA	Thakur <i>et al.</i> 2006; Sharma <i>et al.</i> 2011; Sreenivasa <i>et al.</i> 2008; Mansuetus <i>et al.</i> 1997; Leslie and Plattner 1991
	<i>F. sacchari</i>	India	Sharma <i>et al.</i> 2011
	<i>F. semitectum</i>	India, Zimbabwe, Ghana, Mali, Niger, Nigeria	Gopinath and Shetty 1986; Navi <i>et al.</i> 1999; Onyike and Nelson 1992; Zummo 1984
	<i>F. solani</i>	India	Gopinath and Shetty 1986
	<i>F. sporotrichioides</i>	India	Sreenivasa <i>et al.</i> 2008
	<i>F. thapsinum</i> [#]	India, Thailand, Australia, South Africa, Egypt	Thakur <i>et al.</i> 2006; Sharma <i>et al.</i> 2011; Klittich <i>et al.</i> 1997; Huang and Backhouse 2006
	<i>F. verticillioides</i> [#]	India, Tanzania, USA	Thakur <i>et al.</i> 2006; Sreenivasa <i>et al.</i> 2008; Mansuetus <i>et al.</i> 1997; Leslie and Plattner 1991
	<i>Fusarium</i> spp.	Pakistan, Philippines, Thailand, Mexico	Hamid 1980; Dalmacio 1980; Pupipat 1980; Narro <i>et al.</i> 1992
Sorghum plant tissue	<i>F. moniliforme</i>	USA	Leslie <i>et al.</i> 1990
	<i>F. nygamai</i> [#]	Australia, USA	Burgess and Trimboli 1986
	<i>F. thapsinum</i> [#]	South Africa, USA	Klittich <i>et al.</i> 1997
Sorghum field debris	<i>F. acuminatum</i>	USA	Leslie <i>et al.</i> 1990
	<i>F. chlamydosporum</i>	USA	Leslie <i>et al.</i> 1990
	<i>F. equiseti</i>	USA	Leslie <i>et al.</i> 1990
	<i>F. graminearum</i>	USA	Leslie <i>et al.</i> 1990
	<i>F. napiforme</i> [#]	Australia, South Africa	Marasas <i>et al.</i> 1987
	<i>F. solani</i>	USA	Leslie <i>et al.</i> 1990

[#] *Fusarium* species that form microconidia in chains and might be identified as *F. moniliforme* at some point of time (Leslie and Marasas 2002).

Table 3 *Fusarium*-induced panicle diseases of cereals other than sorghum.

Cereal	Disease	<i>Fusarium</i> species	Reference
Maize (<i>Zea mays</i>)	Ear rot	<i>F. graminearum</i>	Sutton 1982
Pearl millet (<i>Pennisetum glaucum</i>)	Grain mold	<i>F. semitectum</i> , <i>F. chlamydosporum</i> , <i>F. verticillioides</i>	Nutsugah and Wilson 2007
Wheat (<i>Triticum aestivum</i>)	Head blight or head scab	<i>F. graminearum</i> , <i>F. avenaceum</i> , <i>F. culmorum</i> , <i>F. nivale</i> , <i>F. poae</i>	Sutton 1982; Parry <i>et al.</i> 1995; Kolb <i>et al.</i> 2001; Schlang <i>et al.</i> 2008
Barley (<i>Hordeum vulgare</i>)	Head blight or scab	<i>F. graminearum</i>	Kolb <i>et al.</i> 2001

much mycelial growth in the voids between spikelet structures than the susceptible one. Unlike the susceptible cultivar the resistant one shows rapid localized pigmentation in areas where host and fungal tissues are in close association.

Few recent studies corroborated the above findings of the earlier researchers. Inoculation of sorghum panicles at anthesis with *F. thapsinum* resulted in reduced caryopsis formation (Little and Magill 2009) suggesting its deleterious effect on the ovary and peripheral tissues resulting in seed abortion, especially in grain mold susceptible cultivars. Butler *et al.* (2008) reported that *Fusarium* spp. colonized the lodicule and ovary base and then progressed in an acropetal fashion as the caryopsis matures. While isolating mold fungi from different tissues of sorghum grain (black layer, pericarp, endosperm, and germ), they observed that black layer and pericarp yielded the highest levels of total fungi, than the endosperm and germ, irrespective of mold response of a genotype. Frequency of isolation of *Fusarium* spp., including *F. thapsinum*, was significantly greater ($P \leq 0.05$) from mold susceptible genotype than from the resistant one. According to above early events and colonization processes, most of the infection takes place at anthesis and whatever fungal bloom seen on matured grain is mainly the outer growth of the fungi that infected living kernel tissues before PM. Strong positive correlation between mold scores at physiological and harvest maturity ($P \leq 0.01$) partly support the above colonization processes (Audilakshmi *et al.* 2011). Post-maturity grain infection and colonization, on the other hand, involves many genera of field fungi that colonize pri-

marily the non-living tissues. Information on factors affecting germination of conidia on the floral tissue, biochemical pathways leading to infection, fungal interactions and varietal response on these functions will give more insight in the process of pathogenicity.

NATURE OF DAMAGE

Fusarium species causes wide range of losses to sorghum. It is the dominant component among the three major genera of grain mold pathogens (*Fusarium* spp., *Curvularia* spp., and *Phoma* spp.). Frequency of occurrence and severity of disease caused by these fungi vary with the geographic location (country) and environmental conditions. Therefore, losses caused by these pathogens and their importance vary among sorghum growing countries. As a pathogen each of these fungi has certain effects on yield and quality of sorghum that can be analyzed to assess their relative importance in sorghum. Result of such an assessment based on information available in the literature has been presented in **Table 4** that shows *Fusarium* spp. as the most damaging and economically important mold fungi in sorghum grain mold complex. Apart from the pathogens, there are saprophytic *Fusarium* species which infect and colonize mature sorghum grains. The saprophytes may not be the actual yield reducers but have immense effect on grain quality particularly grain discoloration and appearance that determines market price of the produce. A few other fungi in saprophytic group (e.g., *Aspergillus* spp.) also produce mycotoxins and have

Table 4 Relative importance of *Fusarium* and other genera of grain mold fungi for yield and quality of sorghum.

Parameters	Components	Relative importance (1-5 scale) ^a				Related references ^b
		<i>Fusarium</i> spp.	<i>Curvularia</i> spp.	<i>Phoma</i> spp.	Other molds	
Yield	Kernel formation	4	3	1	1	Castor 1981; Forbes 1986; Little and Magill 2009
	Kernel mass and density	4	3	1	1	Castor and Frederiksen 1977; Ibrahim <i>et al.</i> 1985
Seed and grain quality	Pre-harvest sprouting	4	2	1	1	Maiti <i>et al.</i> 1985; Steinbach <i>et al.</i> 1995
	Seed germination and viability	3	2	1	2	Castor 1981; Garud <i>et al.</i> 2000; Little and Magill 2003; Prom <i>et al.</i> 2011; Rodriguez-Herrera <i>et al.</i> 2006a
Marketability	Seedling vigor	2	2	2	2	Kannababu <i>et al.</i> 2009
	Appearance	3	4	3	3	Indira and Rana 1997; Audilakshmi <i>et al.</i> 2007
	Grain size	4	3	1	1	Ibrahim <i>et al.</i> 1985; Little and Magill 2003
Food safety	Mycotoxins contamination	5	1	1	3	Flannigan 1991; Bhat <i>et al.</i> 1997; Leslie <i>et al.</i> 2005; Das <i>et al.</i> 2010

^aRelative importance of the fungal genera on a 1 - 5 scale (1 = minimum and 5 = maximum importance) on the yield, quality and safety parameters related to sorghum.

^bIncludes references that give some indications or provide direct or indirect information for scoring relative importance of the component.

adverse effect on food and feed value of the sorghum-based products. Overall *Fusarium* is responsible for reduction in grain yield, seed and grain quality, market acceptability and causing food and feed related toxicity to human, animal and poultry bird.

ECONOMIC SIGNIFICANCE

Effect on grain yield

Fusarium species are one of the major constraints for sorghum production. Infection of sorghum flowers by *Fusarium* spp. not only reduces grain yield but also sets adverse effect on marketing and consumption of the infected grains. It is difficult to estimate accurate losses caused by GMDC or its component *Fusarium*, since it involves the assessment of losses from production to marketing and finally utilization of the grain or seed. Total annual loss due to GMDC in the semi-arid tropics is about US\$ 130 million (ICRISAT 1992) and major share of this loss is possibly due to *Fusarium*. Production loss ranges from 30-100% depending on cultivar, and prevailing weather conditions during flowering to harvesting (Singh and Bandyopadhyay 2000). Damages resulting from the early infection of sorghum flowers by *Fusarium* spp. include reduction in caryopsis formation (Little and Magill 2009), arrest of kernel development, and decrease in grain mass and grain density. All these are directly related to grain yield. Infection by *F. moniliforme* and *C. lunata* has been reported to interfere with carbohydrate translocation to developing kernels, and thus causing reduction in size and weight of seed (Mathur *et al.* 1975; Castor and Frederiksen 1977). In such a case, grain yield is reduced without visible mold development. Mold fungi are reported to cause significant loss in grain weight in sorghum (40-70%) (Gray *et al.* 1971; Glueck and Rooney 1976; Singh and Agrawal 1989).

Effect on seed quality

Seed quality is very important for the grower of the crop. Various physiological and biochemical tests, viz., seed germination, vigour index, field emergence, speed of germination, electrical conductivity of seed leachate, dehydrogenase and α -amylase activity in seeds give an insight about the biological quality of the grain and its value as seed. Certain grain mold pathogens have been repeatedly associated with losses in seed mass, grain density (Ibrahim *et al.* 1985), germination (Maiti *et al.* 1985; Little and Magill 2003) and seed viability (Castor 1981). Seed quality parameters in sorghum significantly decline with increasing temperature and relative humidity that support sporulation by the mold fungi and grain colonization (Tonapi *et al.* 2007). *Fusarium* species have relatively more adverse effect on seed germination of sorghum than other fungi (Garud *et al.* 2000;

Prom *et al.* 2011). Effects of mold infection on seed quality vary among genotypes with white or colored grain. Decrease in test weight and seed germination in white cultivars is more than the red or brown ones (Martinez *et al.* 1994). However, mere discoloration of grain does not mean that it is always internally infected or there is loss in total seed quality.

Grain quality and marketability

FGM reduces quality of sorghum grain and its acceptability in the market. During the process of post-maturity weathering, sorghum grains are infected and heavily colonized by mold fungi including *Fusarium*. This causes discoloration and resultant reduction in market price of the grain (Ibrahim *et al.* 1985; Indira and Rana 1997; Little 2000). Molded grains (grain with visible mold sign) fetch in much lower market price (around 20% less) than that of normal grain (Indira and Rana 1997). Audilakshmi *et al.* (2007) reported that the market price of the produce was reduced by 10 and 30% when it showed grain mold score of 3 and 4, respectively (on a 1 to 5 scale where 1 indicates no mold and 5 indicates more than 50% grain surface molded). On visibly molded grain, *F. moniliforme* and *C. lunata* secrete enzymes that can degrade endosperm (starch) and germ tissues (Wajde and Deshpande 1976). In addition, *F. moniliforme* may stimulate plant enzymes causing the initiation of germination and the subsequent-breakdown of endosperm tissue. Regardless of the source, the enzymes reduce feed or food value per kernel. Other damages that arise from FGM are related to storage quality (Hodges *et al.* 1999), food and feed processing quality, and market value.

Fusarial-toxins

Fungi causing FGMs often contaminate sorghum grain with mycotoxins (Fusarial-toxins). Fusarial-toxicoses are a global problem, occurring in Africa, the Americas, Asia, and Australia (Placinta *et al.* 1999). The change of the staple diet of Black South Africans from sorghum to maize was because of contamination of sorghum with mycotoxins (Isaacson 2005). *Fusarium* species associated with sorghum synthesize a wide range of mycotoxins including fumonisin, moniliformin, fusarenon-X, T-2 and nivalenol (Flannigan 1991; Leslie *et al.* 2005). Often minor infections in healthy looking grains are invisible. *Fusarium* in such grains produces Fusarial-toxins under inappropriate storage conditions (Hodges *et al.* 1999). Toxicogenicity varies among the species or strains within a species. *Fusarium* spp. from sorghum is known for variation in toxigenicity (Leslie *et al.* 2005; Ratnavathi and Das 2008). In India, *F. proliferatum* is reported to have maximum frequency of toxigenic strains for fumonisin than other *Fusaria* (Sharma *et al.* 2011). Fumonisin B1 (FB1) has many adverse effects on humans,

animal and poultry bird including porcine pulmonary edema (Harrison *et al.* 1990), liver toxicity and liver cancer in rats, atherosclerosis in monkeys, immunosuppression in poultry (Norred 1993) oesophageal cancer in human (D'Mello *et al.* 1997). In India, fumonisin levels between 300 and 600 mg/kg grain have been reported in corn and 0.1-2.7 mg/kg in sorghum grain infected with *F. moniliforme* (Chatterjee and Mukherjee 1994). Fumonisin has been implicated as a possible cause of an acute disease outbreak in human beings in several areas of India (Anonymous 1998). An outbreak of food poisoning characterized by abdominal pain and diarrhea, attributed to the ingestion of fumonisin-contaminated moldy sorghum and maize had been reported from several villages in South India (Bhat *et al.* 1997). The fungus, *F. moniliforme* was found to be the causative agent of equine leucoencephalomalacia (ELEM), a fatal disease of horses (Marasas *et al.* 1976). On the basis of available data on the toxicity and carcinogenicity of FB1, the International Agency for Research on Cancer classified the *F. moniliforme* toxin as a group 2B carcinogen (IARC 2002).

FGM thus comes in the way of utilization of sorghum grain as food and feed. This holds special significance for countries like India and African nations where sorghum is generally used as human food as well as in Americas and Australia where grains are used as animal feed. Mycotoxins produced by *Aspergillus* spp. (e.g., aflatoxins, ochratoxins) are also equally important in sorghum because of their deleterious effects on human and livestock health as well as trade. However, time and duration of association of these two fungi with sorghum are different. *Fusarium* species infects early at the initiation of flowering and may continue to be in the grain while in storage. Varietal resistance would, therefore, be more effective against *Fusarium* management (Ratnavathi and Das 2008) than *Aspergillus* that infects mature grains either in the field or during transit and storage. Of late, non-food uses of moldy sorghum grains find application as raw material in industrial sectors. Brewing industries use moldy and germinating sorghum for making beer and whisky (Sheorain *et al.* 2000) thus offer new market opportunities for molded sorghum. Mold infection during germination can exhibit slightly higher α -amylase activity compared to healthy grain suggesting that moldy grain may be suited for malting (Satish Kumar *et al.* 1992). The research process to enhance the efficiency of industrial use of grain and to encourage adoption of other postharvest methods to reduce grain mold should, therefore, consider the socioeconomic, operational, and institutional framework of the target group in addition to the technical aspect (Thakur *et al.* 2006).

MANAGEMENT OF FUSARIUM GRAIN MOLD

Strategies followed to manage GMDC can be used for management of FGM which is the dominant component in the complex. Adjusting sowing time to avoid high humidity conditions during grain maturity, harvesting of panicles at PM followed by drying in community drier to avoid GW in the field, application of fungicides, botanicals and bio-control agents are some of the control measures followed for management of sorghum grain mold. Because of the host-pathogen-environment interaction is highly complex and variable for this disease, no single control method has been found effective. Host-plant resistance forms the major component of grain mold management, and this should be complemented with other practices to help reduce the disease severity.

Host-plant resistance

Use of host-plant resistance has been the major focus for management of GMDC. Identification of sources of resistance through appropriate screening techniques and transferring resistance to desired cultivar has been the mainstay of resistance breeding programme for sorghum GMDC worldwide.

Screening for resistance

Screening for resistance to GMDC in sorghum has been done through field, greenhouse and laboratory methods. Most of these methods focus on identifying resistance caused by air-borne natural inoculums. The methods have been re-evaluated (Bandyopadhyay and Mughogho 1988) and modified over time. Screening for resistance to individual mold fungus by artificial inoculation has been infrequent and only recently has got some attention (Prom *et al.* 2003; Nutsugah and Wilson 2007; Das *et al.* 2010). Thakur *et al.* (2006) have given detail procedures of screening techniques for identification of resistance to individual mold pathogen (e.g., *Fusarium*) by artificial inoculation. Water suspension of spores (1×10^6 spores/ml) of *Fusarium* species is spray inoculated on the sorghum panicle at 80% anthesis stage. High humidity is maintained (>95%) by overhead fogger for 48 h to facilitate infection. Grains in the panicles or threshed grains after harvest are scored for mold incidence. In general, evaluation of resistance is done based on visual mold score recorded either by observing panicles in the field at PM (panicle grain mold rating or PGMR) or by observing threshed grain after harvest (threshed grain mold rating or TGMGR). Mold score is recorded on a progressive 1 to 5 scale (where 1, no mold, and 5, > 50% grains molded) (Bandyopadhyay and Mughogho 1988; Thakur *et al.* 2009) or 1 to 9 scale (where 1, no mold, and 9, > 75% grains molded (Audilakshmi *et al.* 2011). PGMR is aimed at evaluating resistance to GM, while TGMGR is to GMDC. In addition to visual mold score, other variables like incidence of GM pathogen, grain quality, germination and seedling viability have also been used for identification of resistance (Louvel and Arnoud 1984). Recently, inclusion of a few important characters like 'caryopsis formation frequency' (Little and Magill 2009) and 'kernel rot' (disintegration of molded grain before PM) (DSR 2010) have been suggested to further increase the precision of evaluation for genetic resistance against *Fusarium*. Kernel rot shows a significant positive relationship with PGMR for FGM and seed-borne *Fusarium*, and a negative relationship with seed germination and kernel weight (IK Das, unpublished data).

Sources of resistance

Variation in resistance to GMDC exists in sorghum germplasm. Bandyopadhyay *et al.* (1988) screened 7132 germplasm lines from world collection of 26564 accessions, and identified 156 genetically diverse colored-grain lines with high level of resistance (TGMGR ≤ 3.0 on a 1 to 5 scale) to GMDC. Of these, 14 lines were without testa layer and were low in tannin content. This indicates that resistance in colored-grain sorghum is not always associated with high level of tannin in testa and there are possibilities to get high level of resistance in red-grain sorghum which lacks testa. They, however, could not identify any white-grain germplasm line, with such a high level of resistance. Later, Singh *et al.* (1995) identified four white-grain *guinea* sorghum lines as resistant out of 66 accessions screened. But *guinea* sorghum has poor agronomic traits and low grain yield. The factors associated with mold resistance in *guinea* sorghum are difficult to be transferred to an agronomically desirable white-grain cultivar (Mukuru 1992). Recently, moderate to high level of mold resistance has been identified in elite white-grain sorghum lines (Audilakshmi *et al.* 1999; Thakur *et al.* 2003; Ambekar *et al.* 2011) based on field level performance at multi-location testing in sorghum growing areas in India. Evaluation of these lines with assured inoculums pressure under artificial inoculation and high humidity will provide more insight into the resistance and mechanisms in white-grain elite lines. Color-grain lines 'IS25070' and 'IS25100' have been identified as highly resistant to FGM (TGMGR ≤ 2.3 on a 1 to 9 scale) under artificial inoculation (Table 5) (DSR 2009). These lines are being used in a resistant breeding programme for development of FGM resistant hybrid parents (IK Das, pers. obs.).

Table 5 *Fusarium* grain mold resistance of some color-grain lines.

Lines	TGMR ^a	<i>Fusarium</i> infection (%) ^b	Healthy grain (%) ^c	Grain size (g/100)
IS4131	6.0	30 (28) ^d	6 (1) ^d	2.6
IS8525	4.3	21 (14)	30 (28)	1.7
IS20831	4.7	23 (16)	28 (25)	1.8
IS25100	2.3	21 (15)	31 (31)	2.6
IS25104	4.0	18 (10)	42 (55)	2.3
IS25070	2.3	21 (14)	26 (20)	2.6
296B (SC)	7.5	44 (58)	6 (1)	2.1
CD ($p = 0.05$)	2.6	7.8	5.7	0.6

^a Threshold grain mold ratings on a 1-9 scale (where 1 = no mold, and 9 = >75% grains molded);

^b Percentage of surface sterilized grains that developed *Fusarium* growth on incubation (at 28 ± 1°C for 7 days) in blotter test;

^c Percentage of surface sterilized grains that did not develop any fungal growth on incubation (at 28 ± 1°C for 7 days) in blotter test;

^d Figures in the parenthesis are real values from which angular transformed values were derived.

SC = grain mold susceptible check.

Genetics of resistance

Success in breeding for resistance to GMDC had been slow partly due to incomplete understanding of the genetics of resistance and the complex interaction of traits that influence resistance (Thakur *et al.* 1997; Stenhouse *et al.* 1998; Reddy *et al.* 2000). The resistance has been reported to be complex, governed by major and minor genes, additive and epistatic effects with significant genotype by environment ($G \times E$) interactions (Stenhouse *et al.* 1998; Rodriguez-Herrera *et al.* 2000). Recent studies report that resistance in colored grain sorghum, is governed by 2-3 dominant major genes (Audilakshmi *et al.* 2000), while that in white-grain is polygenic, and with significant additive \times additive gene interaction (Audilakshmi *et al.* 2005). The complex genetics of mold resistance is due to the presence of different mechanisms of inheritance from various sources, involvement of many fungi and large environment and genotype by environment ($G \times E$) interactions (Rodriguez-Herrera *et al.* 1999; Audilakshmi *et al.* 2011).

Molecular biology tools have been used to know the genetic composition of resistance to GMDC. Klein *et al.* (2001) worked on 125 F₅ RILs of 'Sureño' (resistant) \times 'RTx430' (susceptible) and reported 5 QTLs (quantitative trait loci) for grain mold incidence which were located on 5 different linkage groups D, E, F, G and I. Each QTL (quantitative trait locus) accounted for between 10 and 23% of the phenotypic variance. They observed that detection of QTLs for mold incidence was dependent on the environment, which was consistent with heritability estimates that show strong environmental and genotype \times environment effects. Further, Douglas (2004) reported that revalidation

of QTLs for mold incidence was possible only in the initial cross of 'RTx430' \times 'Sureño', and not in any other crosses (four crosses) where 'Sureño' was used with other susceptible lines. He further reported that the results could be due to profound environmental influence on mold incidence post-PM. To reduce these environmental effects, later Audilakshmi *et al.* (2011) suggested that identification of QTLs for resistance to the pathogenic grain mold fungi that cause disease at pre-PM stage would be more fruitful than for saprophytic molds that were responsible for post-maturity GW. This was based on their observations that the grain mold occurring before PM was influenced by genotype and to some extent by environment while that occurring after PM was influenced entirely by environment.

Mechanisms of resistance

Fusarium species are involved in both GM and GW. Mechanisms of resistance are different for these two stages of mold development. Available information on mechanisms of resistance against pathogenic mold (GM) fungi is comparatively less than the vast amount of literature available on mechanisms of resistance to GW. Basic mechanisms are related to flower and panicle structure, grain characters (hardness, endosperm texture), association of plant phenolics, flavonoids, hydrolytic enzymes and antifungal proteins. The relative importance of these mechanisms in relation to grain type and mold development stages has been summarized in Table 6. Glume coverage and lax panicles have been shown to contribute to reduction in mold severity (Mansuetus *et al.* 1988). A strong association is found between glume color and resistance to GMDC (Audilakshmi *et al.* 1999). Incorporation of this trait may help to enhance resistance in white-grain sorghum (Reddy *et al.* 2005). There is direct relationship between grain hardness and resistance (Aruna and Audilakshmi 2004). However, high degree of grain hardness is not compatible with traits required for food quality. A pigmented testa, where condensed tannins are present, is the most important trait conferring grain mold resistance (Esele *et al.* 1993). Red pericarp, where flavan-4-ols are located, also confers resistance to GMDC, but not as strongly as pigmented testa. Pigmented testa and red pericarp when combined provide additive effects on resistance. However, not all sorghums with red pericarp are resistant to GMDC. The associations of flavan-4-ols and tannins with resistance have been demonstrated in cultivars with color pericarp and with pigmented testa (Melake-Berhan *et al.* 1996).

Preformed secondary metabolites like flavan-4-ols and plant defense proteins like chitinase, β -glucanase, sormatin and ribosome-inactivating proteins (RIPs) may play a greater role in defense against early infection. Hydrolytic enzymes such as chitinase and β -glucanase have been shown

Table 6 Role of plant, panicle, flower and grain characters in imparting resistance to *Fusarium* grain mold in different grain types.

Mechanisms of resistance	Brown and red grain sorghum		White grain sorghum		Related references ^d
	Early infection ^a	Weathering ^b	Early infection ^a	Weathering ^b	
Panicle compactness	nr	+ ^c	nr	+ ^c	Glueck <i>et al.</i> 1977; Mansuetus <i>et al.</i> 1988; Menkir <i>et al.</i> 1996; Audilakshmi <i>et al.</i> 1999
Glume cover	nr	+ ^c	nr	+ ^c	Glueck <i>et al.</i> 1977; Mansuetus <i>et al.</i> 1988; Menkir <i>et al.</i> 1996; Audilakshmi <i>et al.</i> 1999
Glume pigmentation	nr	+	nr	+	Audilakshmi <i>et al.</i> 1999
Grain hardness	nr	+++	nr	+++	Jambunathan <i>et al.</i> 1992; Audilakshmi <i>et al.</i> 1999
Polyphenols (tannins)	+	+++	nr	nr	Waniska <i>et al.</i> 1989; Esele <i>et al.</i> 1993; Menkir <i>et al.</i> 1996
Flavonoids (flavan-4-ols)	+	++	nr	nr	Jambunathan <i>et al.</i> 1990
AFP (chitinases, glucanases, sormatin, PR-10, RIPs)	++	+	++	+	Roberts and Selitrennikoff 1990; Lin <i>et al.</i> 1996; Seetharaman <i>et al.</i> 1997; Rodriguez-Herrera <i>et al.</i> 2006b; Katile <i>et al.</i> 2010

^a Indicates defense against early interactions between host and pathogen that include floral infection followed by colonization on immature grain.

^b Defense against weathering of matured grain; Relative importance of the mechanism: low (+), medium (++) and high (+++).

^c Conflicting reports regarding role in resistance.

^d 'nr' indicates that the role is unknown or insignificant.

AFP = antifungal proteins.

^d Includes references that give some indications or provide direct or indirect information for determining role.

to be 'upregulated' after plants were treated with fungal elicitor or when they were infected (Seetharaman *et al.* 1997). Antifungal proteins, 'permatins' (such as sormatins) are closely related to the thaumatin-like proteins which can turn-on in incompatible interactions (Lin *et al.* 1996). The permatins act by permeabilizing fungal membranes and may work in concert with the hydrolytic enzymes (Roberts and Selitrennikoff 1990). The RIPs acts as *N*-glycosidases and cleave adenine *N*-glycosidic bonds in rRNA. Levels of some antifungal proteins (chitinases, β -1,3-glucanases, sormatins, and PR-10) are reported to be more in resistant cultivar than in susceptible when challenged with pathogenic mold fungi (Rodriguez-Herrera *et al.* 2006b; Katile *et al.* 2010) indicating their importance in development of mold resistant sorghum line. Rodriguez-Herrera *et al.* (2006b) observed that levels of antifungal proteins like sormatin, and hydrolytic enzymes like chitinases and glucanases increased during seed development till PM and then reduced substantially and these proteins were in higher levels in resistant variety and its F₁s in comparison to susceptible one. Engineering plants to produce antifungal proteins is a possible approach in enhancing resistance to fungi. This further supports the existence of interrelationships among these defense proteins with resistance. Identification of active defense genes that respond quickly at high level of expression on challenge with *Fusarium* will facilitate in strategizing management of FGMs.

Other management practices

Apart from host-plant resistance there are many other practices for management of FGM. These practices are mainly to complement host-plant resistance to help reduce the disease severity. Adjusting sowing dates (Castor 1981; Williams and Rao 1981) to avoid warm and humid conditions during flowering to grain maturity does reduce mold severity, but it is not realistic in most environments due to the constraint of limited growing season. Moreover, under changing climatic situations the rainfall pattern has been highly erratic and unpredictable and this may further reduce the scope of adjusting sowing time for mold reduction. Specific fungicides are effective in reducing grain mold incidence. Somani *et al.* (1995) used captan, aureofungin, mancozeb, carbendazim, thiram, ziram and various mixtures of these fungicides and found that all these were effective in controlling the GM caused by *C. lunata* and *F. moniliforme* in field experiments. But chemical control does not seem to be cost effective in sorghum mainly because of low return from the crop. Bio-control agents have been shown to provide some degree of protection under experimental conditions, but their effectiveness and economic feasibility in on-farm situations have not been well demonstrated. Among the bio-agents tested for antagonistic property against the GM pathogens, *Trichoderma viride*, *T. harzianum* and *Pseudomonas* spp. showed promising results both at laboratory and at field level. Other bio-agents such as *T. hamatum* and *T. koenigii* performed fairly well in checking the growth of the major mold pathogens (Indira and Muthusubramanian 2004). Spraying of panicles with fluorescent *Pseudomonas* species at grain filling stage significantly increased seed germination, improved seedling vigor index and reduced mold severity on tolerant and susceptible genotypes (Kannababu *et al.* 2009). Karthikeyan *et al.* (2007) reported that formulated zimmu extract (50 EC at 3 ml/L, v/v) was significantly effective in reducing the mold incidence under field conditions. Recently, Audilakshmi *et al.* (2005) demonstrated that harvesting panicles at PM followed by drying in community drier significantly reduced GM infestation and grain deterioration and such grains fetched 55% more market price than that harvested at normal maturity. However, it requires a minimum 15 ha of sorghum crop area to cover the cost of the artificial dryer in a season and can be used by sorghum farmers in a village. This method, though useful to avoid GW in the field, is not effective against FGM occurring before PM.

MANAGEMENT OF FUSARIAL-TOXINS

Management practices that can reduce concentrations of Fusarial-toxins in sorghum grain can be broadly categorized into two groups: methods that reduce *Fusarium* infection and subsequent colonization in grain and practices that detoxify toxins in contaminated grains. Incorporation of *Fusarium* resistance into hybrids and selection of hybrids less susceptible to the accumulation of fumonisins can be attempted in sorghum. There may be significant influence of environmental conditions on infection and toxins accumulation, both in naturally contaminated and in artificially inoculated sorghum panicles in the field. To reduce these effects, experimentation under controlled conditions might be useful. Biological control of *Fusarium* by competitive exclusion is another approach which is untested in sorghum. The most feasible approach may be to reduce infection by fumonisin-producing *Fusarium* strains through competition with nonproducing *Fusarium* strains. This approach has been used successfully with *Aspergillus flavus* to reduce aflatoxin concentrations in cotton seed in small field experiments (Cotty 1994). Appropriate handling and storage of grain is another important aspect of mycotoxins management. Damaged and broken grains that are easily colonized by fungi can be removed from grain. This can significantly reduce the fumonisin concentration in grain. Storing grains at recommended level of grain moisture (~13%) for long-term storage will prevent fungal growth and resultant mycotoxins contamination. *F. moniliforme* has not been reported to grow in grain at moisture content below 18 to 20% (Kommedahl and Windels 1981).

Detoxification of contaminated grains has been attempted mainly in maize. Fumonisin detoxifying enzymes could be introduced in plant *via* genetic engineering to prevent the accumulation of fumonisins in *Fusarium*-infected sorghum grain. Two species of saprophytic fungi isolated from moldy corn ears were shown to be capable of utilizing FB1 as a sole source of carbon and energy. These fungi were shown to possess enzymes capable of hydrolyzing and further metabolizing fumonisins (Duvick *et al.* 1994). Another approach is non-enzymatic browning reaction that can help reducing fumonisin from sorghum grain. During such reaction (that occurs in the presence of a primary amine, a reducing sugar, and water at alkaline pH), the primary amine group is removed from the fumonisin molecule. Lu *et al.* (1997) reported that treatment of FB1 with fructose under these conditions resulted in a significant reduction in detectable FB1. Attempts to detoxify fumonisins by chemical methods have met with limited success. Several commercially available enzymes have been tested for their ability to detoxify fumonisins in maize (Murphy *et al.* 1996). But none of these products significantly reduced recovery of FB1. Ammoniation may successfully detoxify the fumonisins when combined with high temperature (Park *et al.* 1992).

CONCLUSION

Among the fungal genera causing grain mold in sorghum *Fusarium* species are economically the most important fungi worldwide followed by *Curvularia* species. The FGM is responsible for reduction in grain yield, seed and grain quality and market acceptability. Fusarial-toxicoses are matters of concern especially in Africa and some parts of India for those poor farmers who do not have other food options than sorghum. It causes sorghum food and feed related toxicity to human, animal and poultry bird and thus affects their health. Altogether three major and proven pathogenic species of *Fusarium* (in alphabetical order they are: *F. andiyazi*, *F. proliferatum* and *F. thapsinum*) that are capable of infecting sorghum flower. They vary in toxigenic properties. There are differences in the infection patterns of *Fusarium* and *Curvularia* spp. that explains why resistance to the two fungi occasionally differs. Among the known mechanisms of resistance antifungal proteins like permeations, PR-10,

chitinases, and glucanases hold promise for developing resistance to early infection of *Fusarium* especially for white grain cultivars where tannins and flavan-4-ols do not have any role in resistance. Identification of active defense genes that respond quickly at high level of expression on challenge with *Fusarium* may be helpful for strategizing management of pathogenic grain mold fungi. Considering the significance of losses due to FGM (seed and grain quality and food and feed safety) it is necessary to identify resistance against *Fusarium* grain mold in general and toxigenic strains in particular and incorporates the resistance in new varieties and hybrids of sorghum.

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