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Sorghum Pathology and Biotechnology - A Fungal Disease Perspective: Part II. Anthracnose, Stalk Rot, and Downy Mildew

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ABSTRACT

Foliar diseases and stalk rots are among the most damaging diseases of sorghum in terms of lost production potential, thus commanding considerable research time and expenditure. This review will focus on anthracnose, a fungal disease that causes both foliar symptoms and stalk rots along with the stalk rots caused by *Fusarium* spp. and *Macrophomina phaseolina*. Although the downy mildews are caused by oomycetes rather than true fungi, recent outbreaks have revealed resistance to previously effective chemical seed treatments and the evolution of new pathogenic races, once again pointing out the need for continuous vigilance. Sorghum diseases are described with respect to the causal organism or organisms, infection process, global distribution, pathogen variability and effects on grain production. In addition, screening methods for identifying resistant cultivars and the genetic basis for host resistance including molecular tags for resistance genes are described where possible along with prospects for future advances in more stable disease control.

Keywords: *Colletotrichum sublineolum*, *Colletotrichum graminicola*, *Fusarium*, *Macrophomina phaseolina*, *Peronosclerospora sorghi*

CONTENTS

ANTHRACNOSE

Introduction

Anthracnose, a disease that impacts the health of cereals and grasses worldwide is caused by a monophyletic group of taxa in the genus *Colletotrichum*. During the past decade there have been important changes in our knowledge of how the grass-associated *Colletotrichum* spp. have evolved, and an increased understanding of the mechanisms by which these fungi engage in hemibiotrophic interactions with their host plants (Crouch and Beirn 2009). Anthrac-

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Invited Review

nose of sorghum (*Sorghum bicolor* (L.) Moench) is caused by the fungus *Colletotrichum sublineolum* Henn. (syn. *C. graminicola* (Ces.) G. W. Wilson [Sutton 1980; Sheriff *et al.* 1995]. It is widely prevalent and economically significant in warm and humid regions of Asia, Africa and the Americas (Pastor-Corrales 1980; Ali and Warren 1992; Leslie 2002; Mathur *et al.* 2002; Marley *et al.* 2004; Figueiredo *et al.* 2006; Thakur *et al.* 2007b; Chala *et al.* 2007). The pathogen is capable of infecting the stalk, foliage, panicle, and grain, thereby degrading not only the quantity but also the quality of both grain and stover. Infection of foliar tissues reduces photosynthate accumulation while infection of the stalk leads to stalk rot followed by lodging, a detriment to maximizing harvestable biomass (Waniska *et al.* 2001).

Biology of *Colletotrichum sublineolum*

The causal agent of anthracnose on cereals, including maize and sorghum, has been long regarded as *C. graminicola* (Holliday 1980). However, analyses of rDNA sequences, DNA fingerprints, mating tests, and appressorium morphology have demonstrated that isolates from maize and sorghum belong to distinct species (Sutton 1968; Vaillancourt and Hanau 1992; Sherriff *et al.* 1995). Isolates from maize are now regarded as *C. graminicola*, whereas those from sorghum are designated *C. sublineolum* (Sutton 1980). It was only after rDNA sequences supported the separation from *C. graminicola* that *C. sublineolum* became the generally accepted species for sorghum isolates (Singh and Boora 2008). Conidia are produced terminally and singly on conidiophores among setae and occur as masses immersed in the mucilaginous substrate. They are hyaline, non-septate, uninucleate and sickle or spindle-shaped. Acervuli produced on the infected host tissue are with or without setae, pink or dark brown and oval to cylindrical (**Fig. 1**). Setae in acervuli are long (up to 100 μm), dark and prominent and are intermixed with conidia and conidiophores (Thakur 2007).

Wharton and Julian (1996) conducted cytological studies and showed that *C. sublineolum* has a two-stage, hemibiotrophic infection process on sorghum similar to that of *C. lindemuthianum* on bean (O'Connell *et al.* 1985). The initial biotrophic phase is associated with intracellular infection vesicles and primary hyphae, which colonize many host cells before giving rise to necrotrophic secondary hyphae. In incompatible interactions, host cells die soon after penetration by infection vesicles, and fungal development is restricted to a single epidermal cell (Wharton and Julian 1996). Accumulation of red-pigmented cytoplasmic inclusions, containing 3-deoxyanthocyanidin phytoalexins (Snyder *et al.* 1991), occurs in both compatible and incompatible interactions, but this defense response is expressed much earlier in incompatible interactions (Wharton and Julian 1996; Lo *et al.* 1999). Wharton *et al.* (2001) examined *C. sublineolum* infection of susceptible and resistant sorghum cultivars with transmission and scanning electron microscopy and documented the ultrastructure of the biotrophic interface between intracellular primary hyphae and host cytoplasm to determine the effects of infection on host cells and to examine the ultrastructure of sorghum defense responses.

Disease symptoms and losses

Anthracnose in sorghum was first reported from Togo, West Africa in 1902 (Sutton 1980). Anthracnose symptoms range from grain deterioration to peduncle breakage, to stalk rot and foliar damage. The disease has since been reported in most sorghum growing regions of the world with yield losses as high as 50% in susceptible lines when infection is followed by wet and dry cycles during periods of high temperatures (Harris *et al.* 1964; Warren 1986; Hulluka and Esele 1992; Thomas *et al.* 1995; Waniska *et al.* 2001; Gwary *et al.* 2002; Ngugi *et al.* 2002). Chala *et al.* (2010) surveyed disease severity in different sorghum growing areas in a total of 487 fields in 49 districts for three years (2005 to 2007). The maximum observed severity in the study was 77%.

Although cultural strategies such as rotation may reduce the impact of the disease, the use of resistant genotypes is the best means for anthracnose control. However, the variable nature of the pathogen offers challenges in breeding for durable resistance (Pande *et al.* 1991; Valèrio *et al.* 2005). Three phases of the disease are recognized: foliar anthracnose (**Figs. 1, 2**), anthracnose stalk rot (**Fig. 2**), and panicle and grain anthracnose. Foliar anthracnose, the most common form and the most destructive phase of the disease, usually appears 30-40 days after emergence, during growth stage 4.0 or later (da Costa *et al.* 2003). Symptoms are characterized by circular to elliptical spots with few or numerous fungal fruiting bodies (acervuli) within the leaf lesions (Tarr 1962). Differences in leaf symptoms are common and may be caused by variations in the pathogen, host reaction, or the physiological status of the host (Pastor-Corrales and Frederiksen 1980; Ferreira and Warren 1982). *C. sublineolum* may survive as mycelium, conidia and microsclerotia up to 18 months in crop debris, on or above the soil surface, in alternate hosts, and as mycelium in infected seeds. Microsclerotia are produced in sorghum stalks of susceptible cultivars and survive in crop debris on the soil surface.

Pathogenic races

C. sublineolum is considered to be a very heterogeneous species, primarily based on the large number of pathotypes that have been described based on differential virulence to host lines (da Costa *et al.* 2003). An abundance of pathogenic races of *C. sublineolum* exist in nature. More than 40 races/pathotype have been reported from different geographical areas of the world, using different sets of host differentials (Thakur 2007). Pande *et al.* (1991) noted 9 pathotypes from as many isolates obtained in India. The existence of five pathotypes of *C. sublineolum* among 16 isolates from the major sorghum growing zones of Nigeria was detected based on reactions on 8 host differentials (Marley *et al.* 2001). Mathur *et al.* (2002) used a set of 15 sorghum differentials grown in 16 locations in Africa, Asia and the United States; the interactions showed that different pathotypes prevailed at each location. In Brazil, Casela *et al.* (1992) separated isolates of *C. sublineolum* first into 8 pathotype groups based on resistant or susceptible reactions on 9 common differentials and then added other differentials to further delineate the isolates into 32 pathotypes. Later, Valério *et al.* (2005) reported 22 pathotypes among 37 isolates using an additional differential, 'SC748-5'. In the United States, by using 8 common differentials, Ali and Warren (1987) recorded 3 pathotypes from 9 *C. sublineolum* isolates, Cardwell *et al.* (1989) reported 8 pathotypes from 12 isolates, while Moore *et al.* (2008) established 13 new pathotypes from 87 isolates collected from Arkansas.

Recently, 20 out of 232 isolates (collected from three geographically distinct regions of Texas, and from Arkansas, Georgia, and Puerto Rico between 2002 and 2004) were selected based on amplified fragment length polymorphism (AFLP) genetic diversity and tested for pathogenicity on 14 sorghum lines previously used in Brazil and the United States and 4 from Sudan. Seventeen of the 20 isolates were identified as new pathotypes (Prom *et al.*, unpublished data). Since not all studies have used the same set of host differentials, it is not possible to directly compare the extant pathogen populations.

Disease screening techniques

Pande *et al.* (1991) inoculated both leaf surfaces of the plant with a conidial suspension of *C. sublineolum* using a hand sprayer. One hour after inoculation humidifiers were run continuously for 18 hr to create 100% humidity. To further promote disease development, humidifiers were run continuously for 6 days for 8 hr/day. To enhance infection, the author conducted the experiments five times. Mehta (Mehta 2002; Mehta *et al.* 2005) followed a similar inoculation

Fig. 1 Foliar anthracnose lesions caused by *Colletotrichum sublineolum*. (**A**) Infected leaf with elongated necrotic lesions with dark brown acervuli. (**B**) Acervuli with oval to cylindrical shape. Images courtesy of John Jaster, Pioneer Hybrid Seeds. **Fig. 2** Foliar anthracnose in the field. (**A**) Plant infected with *C. sublineolum*. (**B**) Field-level infection and foliar senescence associated with severe anthracnose. Images courtesy of John Jaster, Pioneer Hybrid Seeds. **Fig. 3** Anthracnose-resistant ('SC748-5') (**A**) and -susceptible ('BTx623') (**B**) lines. Images courtesy of John Jaster, Pioneer Hybrid Seeds. **Fig. 4** Severe lodging due to field-scale stalk rot infection. Image courtesy of John Jaster, Pioneer Hybrid Seeds. **Fig. 5** Examples of artificially inoculated sorghum stalks. (**A**) Sterile water-inoculated sorghum line '99840'; (**B**) *Fusarium andiyazi*-inoculated sorghum hybrid 'Tx2783'; (**C**) *F. thapsinum*-inoculated sorghum line '25056'; (**D**) *M. phaseolina*-inoculated sorghum line 'SC599'. **Fig. 6** Example of internal pith discoloration associated with *Fusarium* stalk rot extending into the peduncle and resulting in head blight. Note reduced caryopsis formation in the split stalk and peduncle at right. **Fig. 7** *Peronosclerospora sorghi*-infected leaf at vegetative stage with sporulating lesions. **Fig. 8** Plant with systemic infection by *P. sorghi*. **Fig. 9** Field view of the healthy and *P. sorghi*-infected plants. Bottom of a mature leaf of the infected plant with abundant sporulation.

method by spraying approximately 3-5 mL of a conidial suspension (10^6 conidia ml^{-1}) onto the leaves and the whorl of each plant, using backpack sprayers or a tractor-mount sprayer at night or in the early morning so as to have low light intensity. However, Mehta (2002) reported that the number of lesions on susceptible plants within susceptible or segregating $F_{2:3}$ lines was very low compared to the infection observed on susceptible checks, which made accurate scoring difficult. An inoculation system (Erpelding and Prom 2006) where colonized sorghum grains are dropped into the whorls of young plants has proven effective in lowering the problem of escapes. In a 2007 field evaluation at College Station, Texas, 95% of the accessions from 100 advanced germplasm lines and 55% from the exotic lines were found to be susceptible using this inoculation method. Thakur (2007) assigned disease ratings on a 1 to 9 scale, where 1 = highly resistant (HR), no lesions or a hypersensitive reaction with mild yellow flecks and ratings 2 to 9 are based on leaf area covered with lesions: 2 (1-5%) and 3 (6-10%) - resistant (R); 4 (11-20%) and 5 (21-30%) - moderately resistant (MR); 6 (31-40%) and 7 (41-50%) - susceptible (S); 8 (51-75%) and 9 (>75%) - highly susceptible (HS). The rating scale is useful for differentiating between lines with minor differences in resistance. However, for large scale field screening Prom *et al.* (2009) introduced a modified 1 to 5 scale, where 1 = no symptoms or chlorotic flecks on leaves; 2 = hypersensitive reaction (reddening or red spots) on inoculated leaves, but no acervulus formation and no spread to other leaves; 3 = lesions with small acervuli in the center of leaves up to one third of plant height from the bottom; 4 = necrotic lesions with acervuli on all leaves except the flag leaf; and 5 = necrotic lesions with abundant acervuli covering the entire plant. Disease assessments are conducted 30 days post-inoculation and thereafter on a weekly basis for four consecutive weeks until flowering, allowing disease progression to be analyzed.

Host resistance

Plants have a wide array of physical (surface features, structural barriers) and chemical strategies (phytoalexins, hydroxyproline-rich glycoproteins, wall papillae) to defend themselves from invasion by pathogens. Young sorghum leaves accumulate phytoalexins in the form of a complex of phenols having fungitoxic activity in response to invasion by both pathogenic and non-pathogenic fungi (Singh and Boora 2008). The fact that many cultivars are susceptible to anthracnose suggests differences in levels or rate of response in these defense mechanisms have a genetic basis. Breeding for stable host plant resistance has been difficult even in regions with endemic anthracnose because of the hypervariable nature of *C. sublineolum* along with strong environmental effects on symptom development and disease spread. Consequently, even though several sources of genetic resistance are known, an understanding of the basis for anthracnose resistance is still lacking. Studies have examined anthracnose resistance in sorghum germplasm from the USDA-TAES sorghum conversion program (Cardwell *et al.* 1989). Coleman and Stokes (1954) reported that resistance to anthracnose in the sorghum line 'Sart' is encoded by two closely linked dominant genes, each conferring resistance to different phases of the disease. Jones (1979) found single gene dominant resistance for leaf anthracnose in one cross but two dominant genes segregated for resistance in a second cross. Tenkouano *et al.* (1993) reported that resistance to anthracnose in 'SC326-6' was controlled by a single genetic locus with multiple allelic forms while in progeny of a cross made by Boora *et al.* (1998) resistance in 'SC326-6' segregated as a simple recessive as was also the case for 'G73' crossed to a highly susceptible cultivar (Singh and Boora 2008). Erpelding and Prom (2004) evaluated 270 Mali accessions to study the mode of inheritance during the dry and wet seasons in 2003 at Puerto Rico and 41 accessions exhibited both dominant and recessive gene action. Mehta *et al.* (2005) identified four converted lines that displayed unique, but simply inherited sources of anthracnose resistance. Resistance from 'SC748-5' (**Fig. 3A**) was the most stable across environments. Resistance to foliar anthracnose in sorghum accession 'Redlan' was found to segregate as a simple dominant trait whereas infection of the leaf midrib was found to be controlled by a single unlinked recessive gene (Erpelding 2007).

Breeding for host plant resistance provides an economical approach for controlling diseases and stabilizing crop production, but pathogen populations are variable and evolving; therefore, the identification of new sources of resistance is essential. Plant germplasm collections have been established by the United States National Plant Germplasm System in Griffin, Georgia to preserve genetic variation for utilization in crop improvement programs. Exotic germplasm materials are continuously being evaluated at the United States of America Department of Agriculture, Agricultural Research Service, National Plant Germplasm System (USDA, ARS, NPGS) Tropical Agriculture Research Station in Isabela, Puerto Rico. Twenty-two Mozambique accessions (Erpelding and Prom 2006), four Chinese accessions ('PI430471', 'PI563905', 'PI563924' and 'PI563960'; Prom *et al.* 2007), 11 Zimbabwe accessions (Erpelding 2008a), 119 Mali accessions (Erpelding 2008b) and six Uganda accessions ('PI534117', 'PI534144', 'PI576337', 'PI297199', 'PI533833', and 'PI297210'; Prom *et al.* 2011) were identified as potential resistance sources over multiple growing seasons, at least to the anthracnose pathotypes at the research site in Puerto Rico. Marley and Ajayi (2002) identified lines 'R 6078', 'IS 14384' and 'CCGM 1/19-1-1' as resistant to anthracnose when evaluated under natural infection at Samaru and Bagauda (Nigeria) in 1996 and 1997. Thakur *et al.* (2007b) tested 15 sorghum lines collected from the Sorghum Anthracnose Virulence Nursery (ISAVN) at 14 anthracnose hotspots in India, Thailand, Ethiopia, Kenya, Zambia, Nigeria and Mali for 4 to 7 years (1992-1998) and identified 'IS 6928', 'IS 18758', and 'IS 12467' as the most resistant across the environments (locations and years). Pereira *et al.* (2011) identified four parental lines 'CMSXS657', 'ATF14', 'ATF08' and 'CMSXS210' as resistant sources against 20 virulent isolates collected from different sorghum producing areas in Brazil. Results of these evaluations suggest that exotic germplasm is an important source of anthracnose resistance and that ecogeographic information could aid in the selection of germplasm and increase the likelihood of identifying additional sources of resistance.

DNA-based diversity and molecular tags

Due to environmental influences on the stability of morphological traits, differentiation between *Colletotrichum* isolates based on conidial morphology or features such as colony color, size, and shape or host origin are not sufficient for assessing genetic diversity. DNA markers including random amplified polymorphic DNA (RAPD) (Guthrie *et al.* 1992) and restriction fragment length polymorphisms (RFLPs) have also been used to examine diversity in the pathogen. Vaillancourt and Hanau (1992) reported significant differences between the restriction fragment patterns of maize and sorghum isolates, while Rosewich *et al.* (1998), using seven low-copy DNA probes, detected nine RFLP haplotypes among 411 *C. sublineolum* isolates collected from one site in Georgia. However, DNA comparisons have revealed less diversity than might have been anticipated. For example, in one study, seven DNA hybridization probes detected multiple RFLP-based haplotypes, but the most common patterns were found in samples collected from Georgia, Honduras, Zambia and Texas (Gale 2002). Intergenic spacer regions of nuclear ribosomal DNA (Latha *et al.* 2003) or isozymes (Horvath and Vargas 2004) used to estimate genetic variation among isolates revealed that host origin plays a more important role than geographic origin in the genetic diversity of anthracnose isolates. (It must be noted, however, that these and similar studies did not clas-

sify *C. sublineolum* separate from *C. graminicola*). Valèrio *et al.* (2005) used RAPD and RFLP-PCR markers to study the molecular diversity of 37 *Colletotrichum* isolates collected from four distinct regions of Brazil and recorded polymorphic differences among isolates belonging to the same race as defined on 10 sorghum differentials. However, no association between virulence phenotypes and molecular profiles was observed. Figueiredo *et al.* (2006) used different molecular markers (SDS-PAGE, RAPD, ARDRA (amplified rDNA restriction analysis) and rDNA sequencing) for identifying C. *sublineolum* pathotypes and concluded RAPD and rDNA sequencing revealed a high degree of polymorphism among the five pathotypes in Brazil. Chala *et al.* (2011) assessed diversity through amplified fragment length polymorphism (AFLP) analysis using 102 isolates of *C. sublineolum* collected from different sorghum-producing regions of Ethiopia. The results of this study confirmed the presence of a highly diverse pathogen, which is in agreement with the existence of diverse host genotypes and widely ranging environmental conditions in sorghum-producing regions in that country.

On the host side, Wang *et al.* (2006) used microsatellite markers for ninety-six accessions randomly selected from the core collection database of the Germplasm Research Information Network (GRIN) to evaluate genetic diversity in relation to rust and anthracnose disease response. The information from genetic classification was used for choosing parents to make crosses in sorghum breeding programs and classifying sorghum accessions in germplasm management.

Molecular markers linked to gene(s) of interest are one possible strategy to permit selection for anthracnose resistance without concern for pathogen pressure. A vast array of genome resources for sorghum has been developed in the past 10 to 15 years. DNA-based molecular markers showing genetic linkage to disease resistance loci in sorghum have been reported for anthracnose by different workers using different marker techniques. Boora *et al.* (1998) identified RAPD markers linked to a recessive gene conditioning anthracnose resistance, butt those markers have not been mapped to a specific sorghum chromosomal location. Panday *et al.* (2002) used bulk segregant analysis and identified two RAPD-based DNA markers (OPI 16 and OPD 12) linked to anthracnose disease resistance in sorghum accession 'SC326-6', found to segregate as a simple recessive trait when crossed with the susceptible cultivar 'BTx623' (**Fig. 3B**). Singh *et al.* (2006) were able to show that an anthracnose resistance gene in sorghum line 'G73' maps to the long arm of chromosome 8 on the basis of linked RAPD (OPJ 01 with 3.26 cM distance from the gene) and SCAR (SCJ 01-1 and SCJ 01-2) markers. Singh and Boora (2008), using bulked segregant analysis, found parental bands from OPA 12, OPJ 01, OPF 07 and OPL 04, OPI 12 and OPD 12 (RAPD markers), *Xtxp 61* and *Xtxp 212* (simple sequence repeat or SSR) markers and SCA 12 and SCJ 01 (SCAR markers) from two mapping populations ['HC136' (susceptible to anthracnose) × 'G73' (anthracnose resistant) and 'SC326-6' (anthracnose resistant) × 'BTx623' (anthracnose susceptible)] provide closely linked markers to the anthracnose resistance gene. Perumal *et al.* (2009) identified AFLP marker *Xtxa6227*, previously mapped to the end of sorghum linkage group LG-05, which mapped within 1.8 cM of the anthracnose resistance locus *Cg1*, a dominant gene for resistance originally identified in cultivar 'SC748-5'. BAC clones spanning this chromosome led to the discovery that *Xtxp549*, a polymorphic (SSR) marker, mapped within 3.6 cM of the anthracnose resistance locus. The efficacy of *Xtxa6227* and *Xtxp549* were examined for marker-assisted selection and 13 breeding lines derived from crosses with sorghum line 'SC748-5' were genotyped. In 12 of the 13 lines the *Xtxa6227* and *Xtxp549* polymorphism associated with the *Cg1* locus was still present, suggesting that *Xtxp549* and *Xtxa6227* could be useful for marker-assisted selection and for pyramiding *Cg1* with other genes conferring resistance to *C. sublineolum* in sorghum for more stable disease resistance. These markers could also facilitate marker-assisted selection in breeding for anthracnose resistance gene and map-based cloning of resistance gene(s).

Future prospects

Variation in the natural populations of *C. sublineolum* and the powerful selective advantage of a natural or mutant strain that is able to reproduce on a previously resistant host means that screening and identification of resistant cultivars must persist. The use of molecular tools to combine different sources of genetic resistance may prolong the useful life of a cultivar, but there can be no guarantee of permanence. Genetic transformation provides another avenue for increasing tolerance to plant diseases such as anthracnose. Introducing genes encoding proteins such as chitinases and chitosanases that hydrolyze fungal cell walls is a potential strategy. For example, Kosambo-Ayoo *et al.* (2011) used particle bombardment genetic transformation in sorghum by introducing genes encoding proteins such as chitinases (*harchit*) and chitosanases (*harcho*) that hydrolyze fungal cell walls. Chitinases endolytically hydrolyze the beta-1,4-linkages of chitin whereas, chitosanases hydrolyze the beta-1,4-linkages between N-acetyl-D-glucosamine and D-glucosamine residues in a partially acetylated chitin polymer. The two antifungal genes introduced into sorghum genome could be introgressed into other sorghum lines for fungal diseases resistance.

STALK ROT

Introduction

Stalk rots are among the most prevalent diseases of sorghum in most places where the crop is cultivated (Zummo 1984). They are also common in other crop species including maize, millet, soybean, and sunflower (Pappelis and BeMiller 1984; Rane *et al.* 1997). This disease complex occurs in wide geographic regions both in tropical and temperate environments (Tarr 1962). It is a widespread disease in the west and central Africa region extending from Chad to Senegal (Frowd 1980; Zummo 1980). The disease is also prevalent in North and East African countries including Egypt, Sudan, Uganda, Kenya, Somalia (Gray *et al.* 1991; Hulluka and Esele 1992) and the semi-arid Rift Valley region of Ethiopia (Gebrekidan and Kebede 1979). It is one of the major diseases of sorghum in India, particularly during the dry rabi season (Khune *et al.* 1984; Seetharama *et al.* 1987), and Australia mainly in New South Wales (Trimboli and Burgess 1982). In the United States, stalk rot is a common problem in the southern states and in the central Great Plains extending from Texas to Kansas (Edmunds 1964; Edmunds and Zummo 1975). There are also reports of stalk rot incidence as far north as Nebraska (Reed *et al.* 1983; Duncan 1983) and the Southeastern United States (Duncan 1983). It is also an important disease of sorghum in Brazil and most of Latin American countries (Foster and Frederiksen 1979; Frederiksen 1984).

The disease usually develops at later growth stages during grain filling period and is characterized by degradation of the pith tissue at or near the base of the stalk causing death of stalk pith cells (Edmunds 1964). This premature death of stalk cells may result in reduced transportation of nutrients and water, thus disrupting photosynthetic activity and may also cause breakage of the stalk at the zone of infection leading to lodging (Mughogho and Pande 1984; Hundekar and Anahahusor 1994; Maranville and Clegg 1984). These phenomena may slow down or inhibit the grain-filling process and thus result in shriveled seeds (Zummo 1980). Growing interest in the use of the crop as an alternative cellulosic feedstock for bio-fuel production requires cultivation of taller and high biomass cultivars; the apparent relationship between stalk rot incidence and crop lodging is a major source of concern to exploitation of the crop as bio-fuel feedstock.

Biology of stalk rot

Several genera and species of fungi are known to cause stalk rot diseases in sorghum. Only one species of bacteria, *Erwina chrysanthemi* pv. *zeae* (Sabet) Victoria, has been reported to cause stalk rot in sorghum (Saxena *et al.* 1991). It is a very aggressive pathogen that can cause complete death of the plant within two to three days after the appearance of initial symptoms, a brownish lesion in the infected area.

Most of the common stalk rotting fungal organisms belong to the genera *Macrophomina*, *Fusarium*, *Alternaria*, *Colletotrichum*, *Nigrospora*, *Trichoderma*, *Periconia* and *Cephalosporium* (Reed *et al.* 1983; Frederiksen 1984; Hassan *et al.* 1996). The mechanism by which the pathogens infect and establish in the host tissue may vary between species. However, most of them survive unfavorable condition as conidia or sclerotia in infected plant debris at or near the soil surface. Infection usually starts in the roots and gradually advances to aboveground stalks. Two or more pathogen species usually attack the same plant forming a disease complex making it difficult to distinguish the primary invader (Reed *et al.* 1983; Trimboli and Burgess 1983; Mughogho and Pande 1984). Reed *et al.* (1983) identified more than eight *Fusarium* spp. from the same infected sorghum plant tissue. Very few of these organisms, on only a few genotypes, cause significant rot on sorghum prior to flowering. One of these, *Periconia circinata* (M. Mangin) Sacc. has been reported to cause early rot ("Milo disease") on susceptible genotypes in the southwestern United States, but this disease has been overcome by incorporating a simply inherited gene that is now available in most available elite germplasm (Dodd 1980). *Colletotrichum graminicola* has also been reported to cause early rot in susceptible sorghum genotypes, although the species that attacks sorghum has been renamed *C. sublineolum* (see *Anthracnose* section). Some strains of *F. moniliforme* sensu lato (also now separated into a number of species) cause infection in certain hybrids as early as 30 days after planting (Khune *et al.* 1984). However, the majority of stalk rotting organisms colonize the stalk and cause the disease during the post-flowering period (Reed *et al.* 1983; Jardine and Leslie 1992).

Despite the complexities caused by the number of pathogens involved, stalk rots of sorghum are broadly categorized into two classes based on the major causal organisms responsible for the diseases. These are charcoal rot caused by *Macrophomina phaseolina* (Tassi) Goid., and Fusarium stalk rot, also called 'soft rot', caused by *Fusarium* spp. Although several other pathogens are involved in causing stalk rot disease these are reported as the most widespread types in ecologically diverse areas in the tropics, subtropics as well as temperate regions (Tarr 1962).

Charcoal rot, known to be the most widespread and destructive stalk rot disease of sorghum (Mughogho and Pande 1984) is caused by *M. phaseolina*. Charcoal rot is also common in India, especially during the dry rabi season (Khune *et al.* 1984; Seetharama *et al.* 1987), Australia (Trimboli and Burgess 1982) and the United States, particularly in the southern states of Texas, Georgia and Arizona (Edmunds 1964; Edmunds and Zummo 1975; Duncan 1983). *M. phaseolina* is a plurivorus pathogen attacking over 75 different plant families and about 400 plant species (Mughogho and Pande 1984). It is a root inhabiting fungus with little or no saprophytic growth in either soil or host cells of infected plants (Edmunds 1964). In the absence of the host, the pathogen survives predominantly as small black microsclerotia in debris from diseased plants or in soil after decay of the plant material (Smith 1969). The process and mechanisms by which *M. phaseolina* penetrates and colonizes roots are not clearly known, but it is reported that the growing hyphae can infect the roots only when the plants are subjected to both moisture and temperature stresses (Odvody and Dunkle 1979). Once the roots are invaded, the pathogen quickly moves to above ground basal stalk portions, attacking the lower internodes and eventually resulting in poor grain filling or premature death of the plant. The distinctive charcoal rot symptom can be revealed by splitting the infected stalks. Doing so reveals characteristic grey to black pigmentation of the entire infected tissue, with bundles of sclerotia covering the invaded area.

Fusarium stalk rot develops under moisture stress after flowering so is associated with a reduction in photo-assimilate translocated to the roots an event either triggered by stress induced metabolic changes in the plant or as a result of competition for photo-assimilate by the developing grain, or both (Dodd 1980). Though the pathogen is not virulent enough to cause infection on young vigorous plants, it is capable of spreading rapidly and can destroy the whole sorghum fields in 2-3 days if the plants are subjected to predisposing factors or when they are in senescence due to maturation (Zummo 1980). Edmunds (1964) pointed out that plants must be in early milk to dough stages of seed development for the pathogen to attack.

Although *Fusarium* species are generally regarded weak pathogens or secondary invaders (Clark and Miller 1980), they may become aggressive and spread quickly when environmental conditions are favorable for disease development. At least eight different *Fusarium* spp. have been reported to cause stalk rot in sorghum (Leslie *et al.* 1990). Isolation and characterization of the pathogens identified *F. moniliforme* sensu lato as the most important causative agent of *Fusarium* stalk rot, while other species occur at lower frequency (Reed *et al.* 1983). Several other virulent strains previously recognized as different mating populations of *F. moniliforme sensu lato* (*Gibberella fujikuroi* species complex) are recently recognized as separate species including *F. andiyazi* Marasas, Rheeder, Lamprecht, Zeller & Leslie, *F. brevicatenulatum* Nirenberg, O'Donnell, Kroschel & Andrianaivo, *F. nygamai* Klaasen & Nelson, *F. proliferatum* (Matsushima) Nirenberg, *F. pseudoanthophilum* Nirenberg, O'Donnell & Mubatanhema, *F. thapsinum* Klittich, Leslie, Nelson & Marasas, and *F. verticilloides* (Saccardo) Nirenberg, all of which are commonly found in sorghum though some prefer other species as major host (Leslie 1991).

Fusarium stalk rot also occurs in maize and millet. In the United States, the disease is generally found in the same area where charcoal rot occurs. It is particularly important on the high plains from Texas to Kansas (Edmunds and Zummo 1975). *F. moniliforme sensu lato* is the primary pathogen associated with sorghum stalk rot in Kansas (Jardine and Leslie 1992).

Similar to *M. phaseolina*, infection by *Fusarium* spp. usually occurs when the host is weakened by predisposing factors (Dodd 1980). The pathogens persist in soil, on crop residue and on weed hosts as mycelium or conidiomata, such as sporodochia. The infection starts on the cortical tissues of the roots and eventually invades the vascular tissues as the pathogen progresses towards the stalk (Zummo 1980). The rate of invasion by *Fusarium* is less rapid compared to charcoal rot (Zummo 1980). However, in certain susceptible hybrids, *Fusarium* can spread very fast, beginning as early as 30 days after planting (Khune *et al.* 1984). Unlike *M. phaseolina*, *Fusarium* spp. do not produce microsclerotia. But upon sudden occurrence of dry conditions, the fungus appears to form a sclerotia-like structure for its survival (Khune *et al.* 1984). Symptoms of the disease are prevalent in tissues that are injured or damaged by insects. The infected plant parts contain large areas of reddish pith and the upper internodes have discolored vascular bundles. Premature plant death, poor grain development and crop lodging are some of the characteristic symptoms of *Fusarium* stalk rot (**Fig. 6**).

Disease symptoms and losses

Initial symptoms of *M. phaseolina* develop on roots appearing as water-soaked lesions that turn brown or black with age. The fungus continues to invade the host starting

from the crown and causes watersoaking and discoloration in the pith. Infected tissue eventually disintegrates leaving only the vascular strands intact. Numerous, small, black microsclerotia form on the vascular strands and are easily visible when stalks are split open. The most characteristic outward symptom of the disease is stalk lodging, which often occurs in the driest portions of the field (**Fig. 4**). Other yield reducing physiological events associated with stalk rotting are poor grain filling and premature ripening. In addition to lodging, bleaching of outer stalk tissue may be evident (Jardine 2006).

One of the earliest signs of *Fusarium* root and stalk rot diseases is the premature death of scattered individual plants or adjacent plants in small patches within a given field. Such plants often exhibit leaves that appear to be frost damaged or suffering from desiccation stress. The most characteristic symptom of *Fusarium* stalk rot, however, is the shredding of the internal tissue of the lower internodes. This shredded region may be pink or reddish-brown depending on the plant pigment type. As with charcoal rot, the decay of interior stalk tissue results in stalk lodging (Jardine 2006).

Though little or no quantitative crop loss assessment results have been reported in the recent past, the widely recognized effect of the disease on standability and grain weight indicate that yield losses as a result of the disease may be economically significant. In mechanized agriculture, yield loss due to stalk rot is directly proportional to percentage lodging since lodged plants cannot be harvested (Mughogho and Pande 1984). However, the magnitude of yield loss may vary from region to region depending on the severity of the disease and the type of cultivars grown. In Kansas, although the average yield loss is estimated at 4%, the loss is believed to reach 50% in areas of high disease incidence (Jardine and Leslie 1992).

Charcoal rot has been reported to cause significant yield reduction in major sorghum growing areas of Africa (Gebrekidan and Kebede 1979; Frowed 1980; Hulluka and Esele 1992). Garud and Changule (1984) reported an average yield loss of 48 to 67% due to charcoal rot infestation in India. In India and Sudan, charcoal rot infection has been reported to result in up to 100% lodging in susceptible hybrids (Mughogho and Pande 1984). In India, yield losses of 56 to 65% and 19 to 30% have been reported in fields infected with *M. phaseolina* and *F. moniliforme sensu lato*, respectively (Hundekar and Anahusor 1994).

Factors associated with stalk rot incidence in grain sorghum

External abiotic factors and overall crop management are recognized as elements linked with development and spread of stalk rot diseases (Jordan *et al.* 1984; Flett 1996). Stalk rotting pathogens are often considered weak parasites, since they are capable of invading host and causing a disease only when environmental conditions are favorable for disease development. Conditions that are adverse to growth of plants such as water deficit, high temperature, and unbalanced mineral nutrition are reported to be the major predisposing factors to stalk rot disease (Dodd 1980; Seetharama *et al.* 1987). These stresses, especially when encountered during later stages of growth, create favorable conditions for disease spread (Odvody and Dunkle 1979). In the case of charcoal rot, moisture deficit accompanied by hot, dry weather during the grain filling period has been the major factor that aggravates the disease (Rao *et al.* 1980). Water stress after flowering has been reported to lead to increased charcoal rot incidence by up to 90% (Edmunds 1964). Odvody and Dunkle (1979) showed that *M. phaseolina* only penetrated host cells after application of stress. Seetharama *et al.* (1987) demonstrated the impact of water stress on charcoal rot incidence using a line source sprinkler irrigation technique that produced a gradient of water deficit by decreasing the water supply with increasing distance from the sprinkler line. These authors recorded high incidence of charcoal rot and low grain yield from plants grown farther from the water source (i.e., exposed to more severe stress). Edmunds (1964) reported total death of sorghum plants five days after inoculation at soil temperatures of 35°C and less than 25% available soil moisture.

Fusarium stalk rot, unlike charcoal rot, has been particularly important during wet conditions following hot, dry weather (Zummo, 1980), but exposure to stress is essential for the spread of *Fusarium* spp. Trimboli and Burgess (1983) reproduced basal stalk rot in greenhouse studies by growing sorghum plants on *F. moniliforme sensu lato*-infested soil at optimal soil moisture until flowering, then subjected to a gradual development of severe moisture stress between flowering and mid-dough stage, followed by rewetting. Stalk rot did not develop in plants grown to maturity at optimal soil moisture, although many of these plants were infected by *F. moniliforme sensu lato*. Stalk rot developed on the majority of stressed plants, including those grown on soils initially non-infested but contaminated by *F. moniliforme sensu lato* after planting.

Studies have also indicated that fertilizer application has a direct relationship with charcoal rot and *Fusarium* stalk rot incidence (Edmunds and Zummo 1975). Mote and Ramshe (1980) have shown the impact of high levels of nitrogen fertilizer on increasing charcoal rot incidence, regardless host genotype differences. They observed an increase in disease incidence from 8 to 41% when the nitrogen level was increased from 0 to 50 kg ha^{-1}. Similarly, Patil *et al.* (1982) observed a marked increase in charcoal rot infestation from 34% in unfertilized plots to 58% in plots that received 90 kg ha^{-1} nitrogen. Avadehany and Mallanagouda (1979) found a similar result in earlier work.

Certain plant biochemical compounds (sugars, phenols and proteins) are associated with the incidence of stalk rot in both sorghum and maize (Craig and Hooker 1961). Stalk rot resistant maize and sorghum genotypes have been reported to contain higher total sugar and phenols in the internodes than the susceptible genotypes. This was at first attributed to preferential use of these compounds by the fungus. However, reports by Clark and Miller (1980) confirmed that stalk sugar content is positively correlated with stalk rot resistance. An earlier study by Odvody and Dunkle (1979) where male-fertile sorghum plants subjected to post-flowering drought stress exhibited more rapid spread of *M. phaseolina* than male-sterile plants agrees with this observation. Removal of the panicle from grain sorghum plants is also reported to reduce the rate of senescence of stalk pith tissue and the spread of *C. graminicola* (Frederiksen 1984). Both male sterility and panicle removal reduce sink size and consequently enable the plant to maintain higher sugar content in the stalk. The increased incidence of stalk rot in plants where leaves are removed (Rajewiski and Francis 1991) also indicates the possible role of stalk sugar on stalk rot development. Hence, these findings generally support the hypothesis that stalk rot is more severe on high yielding cultivars that mobilize the reserve carbohydrates in the stem for grain development than low yielding types that maintain high stem carbohydrate at the expense of low grain yield (Seetharama *et al.* 1991). Interest in the use of sorghum stover as a source of bioenergy led Funnell-Harris *et al.* (2010) to test disease response to plants with reduced lignin as a result of 'brown midrib' mutations *bmr6* and *bmr12*. The results showed that the low-lignin plants differed in the array of colonizing *Fusarium* spp. and that lesion size was significantly reduced following toothpick inoculations compared to the near-isogenic normal plants.

Green leaf area retention, also called "staygreen" is another physiological character related to stalk rot resistance (Duncan 1983). Described as a reduced progressive senescence resulting in increased functional leaf area during grain filling and an extension of photosynthetic capability after grain maturity (Oosterum *et al.* 1996), staygreen helps plants reduce the need for translocation of stored assimilates and enables them to maintain high concentrations of soluble sugar in the stem. Senescence is associated with the

loss of RNA, DNA, total nitrogen, chlorophyll, protein and dry weight in plants (Potter 1971; Hoffman 1972). This condition may dictate plant response to both abiotic and biotic stresses and thus resistance to stalk rot diseases. Non-senescent genotypes such as 'SC599' and 'B-35' have been shown to express excellent levels of resistance to *Fusarium*, *Colletotrichum* and *Macrophomina* spp. (Duncan 1983; Tesso *et al.* 2005). The trait has also been reported as having high and positive correlation with lodging resistance in sorghum Woodfin *et al.* (1988).

Pathogen races

The two major stalk rotting organisms *M. phaseolina* and *Fusarium* spp. and other causal agents are known to harbor wide ranges of pathogenic races. While the recent re-classification of the *F. monliforme* species complex identified several independent species that were originally recognized as different isolates of the same species, similar effort in *Macrophomina* did not lead to a conclusive re-grouping of the pathogens. Although a wide variety subdivision with markedly variable virulence is available among *Macrophomina* isolates, most isolates attack multiple hosts. RAPD AFLP marker genotyping of different *M. phaseolina* isolates have shown significant genetic variability (Rajkumar and Kuruvinashetti 2007). Further, using AFLPs and ribosomal gene internal transcribed spacer (ITS) sequences, Saleh *et al.* (2010) found that a group of 143 *M. phaseolina* isolates from both natural and agroecosystems in Kansas could be divided into four clusters based upon host origin, where one cluster contained isolates from sorghum, which were distinguishable from the clusters containing isolates from maize and soybean, wild plants (including *Ambrosia* sp., *Asclepias* sp., *Cornus drummondii*, *Helianthus* sp., *Lespdeza capitata*, *Panicum virgatum*, and *Physalis* sp.), and goldenrod (*Solidago canadensis* L.). Similarly, characterization of large number of isolates within *Fusarium* species causing stalk rot diseases in both sorghum and maize have shown marked variability (Leslie *et al.* 1996; Chulze *et al.* 2000). It is possible that some of these variations may represent inherent genetic differences in pathogenicity among isolates.

Disease screening techniques

Quantitative description of diseases requires techniques for accurate scoring of disease symptoms and methods for artificial inoculation. Over years, various techniques have been developed and used to quantify stalk rot diseases in sorghum. Direct measurement of pathogen effects such as percent plants infected (*disease incidence*) and length of lesion in the stalk and numbers of diseased (*disease severity*) nodes have been used as disease scoring parameters (Deshmane *et al.* 1979; Bramel-Cox and Claflin 1989).

Earlier studies classified stalk rot disease reactions into five categories based on the percentage of plants infected: *highly resistant* (plants are completely free of disease), *resistant* - less than 10% infection, *moderately resistant* - 10 to 30% infection, *susceptible* - 30 to 50% infection and *highly susceptible* - greater than 50% infection (Deshmane *et al.* 1979; Patil *et al.* 1980). Similarly, Bramel-Cox and Claflin (1989) rated genotypic reaction based on stalk disintegration using a numeric score of 1 to 6 with 1 being resistant and 6 highly susceptible. Scores of 1 to 4 are based on discoloration within the first internode; scores from 4 to 5 indicate discoloration of the first internode and a certain proportion of the second internode. Scores of 5 to 6 depend on the number of internodes greater than two discolored, with a score of 6 being premature death of the main stalk. One or a combination of these techniques may be applied for scoring stalk rot disease under natural or artificial infection condition.

In addition to disease scoring, it is also important to develop inoculation techniques that allow uniform and consistent exposure of sorghum genotypes to pathogens in germplasm evaluation. Since natural infections normally involve more than one pathogen species and lack of uniformity in inoculum dose, it is difficult to rely on natural infection to rate genotypic reactions. For this reason efforts have been made to develop effective inoculation procedures for germplasm screening. The techniques were primarily applied for the most common pathogen species *M. phaseolina* and *Fusarium* spp., but with slight modifications they can be adopted for other pathogens as well.

One of the methods used for *M. phaseolina* is soil inoculation. This method involves the use of dried microsclerotia to inoculate sterilized soils. The procedure for inoculum preparation and inoculation is described by Abawi and Pastor-Corrales (1989). A similar method was used for *F. moniliforme sensu lato* as well (Trimboli and Burgess 1983. Because of the amount of soil that can be sterilized, this technique is limited to small-scale screening in greenhouses. The other widely practiced inoculation method is the use of infected toothpicks. This method works well for both *Macrophomina* and *Fusarium* spp. Sterilized toothpicks are incubated with pathogens in potato dextrose agar until they are fully colonized by the growing mycelia. The infected toothpicks are then inserted in the stalks of target plants. The method is used for large-scale germplasm screening in the field as well as for small-scale greenhouse studies (**Fig. 5**). This method has been widely utilized for screening genotypes for charcoal rot and *Fusarium* stalk rot resistance both in maize and sorghum (Bramel-Cox and Claflin 1989; Afolabi *et al.* 2008; Tesso and Ejeta 2011).

A more recent procedure is the liquid inoculation method. This method is particularly effective for conidia-producing *Fusarium* spp. Here pure cultures of the pathogen are incubated in potato dextrose broth and the conidia are separated from the mycelia by straining the suspension through cheesecloth. The inoculum concentration is determined by counting the number of conidia using a hemacytometer and the inoculum concentration adjusted to the required dose. The pathogens are then injected into the stalk using a modified syringe and needle that is calibrated to deliver a uniform inoculum suspension. Detailed procedure for inoculum preparation and inoculation is described in Tesso *et al.* (2009). This method has also been modified to suit *M. phaseolina*. Though *Macrophomina* does not produce conidia, the mycelial mass can be broken into small fragments that can be plated on nutrient agar to determine colony forming unit numbers.

Each method has advantages and disadvantages. Soil inoculation under controlled conditions seems appropriate in that it mimics natural infection that occurs through the root. However, soil inoculation does not allow large-scale screening of germplasm due to the limits of greenhouse space and the method cannot be applied in the field with same level of environmental control. Toothpick inoculation allows screening of relatively large number of genotypes both in the field and greenhouse. But controlling inoculum delivery for each plant is difficult resulting in variable results. The liquid inoculation procedure seems to overcome some of these difficulties in that fairly uniform amounts of inoculums can be delivered to each plant.

Host resistance

Earlier studies have shown lodging resistance as closely related to stalk characteristics in both grain sorghum and corn (Craig and Hooker 1961; Thompson 1963; Esechie *et al.* 1977; Dodd 1980). Some of these characters include basal internodes and peduncles with larger diameters, shorter peduncle length, shorter plant height, higher weight of 5 cm basal and peduncle stalk sections, and a thicker stalk rind (Thompson 1963; Esechie *et al.* 1977). Sorghum plants with these characteristics have been reported to have strong stalks and are classified as resistant to lodging. Crushing strength and pith density have also been found to significantly (negatively) correlate with lodging resistance in corn (Craig and Hooker 1961; Thompson 1963). Resistant sorg-

hum types also appeared to be more perennial in habit and thus were resistant to senescence, particularly in tall accessions. Recent studies indicate that lodging in the semi dwarf modern cultivars and hybrids is mainly associated with the incidence of stalk rot diseases (Seetharama *et al.* 1987). Genotypes susceptible to the disease, regardless of their morphological attributes have shown to have higher degree of lodging than those that are resistant to the disease. Most genotypes that have been identified as resistant to stalk rot including 'SC33', 'SC35', and 'SC599' have been reported to be staygreen types (Bramel-Cox *et al.* 1988; Woodfin *et al.* 1988). As a result many researchers suggest that indirect selection for staygreen characteristics can be used to identify genotypes with superior stalk rot resistance. Several attempts have been made to determine the mode of inheritance of this character (Tuinstra *et al.* 1997; Tenkuano *et al.* 1993; Walulu *et al.* 1994; Oosterom *et al.* 1996).

As for all other biotic and abiotic stresses, host plant resistance is the most effective way for reducing losses incurred by stalk rot diseases. However, a disease caused by a complex of pathogens and confounded by environmental stresses has complicated the development of resistant cultivars. These complications and the lack of effective inoculation procedures that mimic naturals condition have presented challenges to genotype screening under field conditions. Nevertheless, progress has been made in identifying genotypes resistant to the predominant causal agent of stalk rot, *M. phaseolina* and some genotypes against *Fusarium* species under common predisposing environmental factors (Dodd 1980; Reed *et al.* 1983).

Using one or a combination of the screening tools described above, several genotype-screening experiments have been conducted. The results reveal the existence of genetic variability for stalk rot resistance and the potential for developing resistant varieties or hybrids (Tesso *et al.* 2005). Because of the strong relationship between drought stress and onset of stalk rot infection, indirect selection for drought tolerance has contributed to improvement of stalk rot resistance. Most of the public releases that are tolerant to post-flowering drought stress have been shown to contribute resistance (or tolerance *per se*) to stalk rot diseases caused both by *Macrophomina* and *Fusarium* spp.

Efforts to understand the mode of inheritance of resistance traits have generated different results. Most of the studies indicate that stalk rot resistance is controlled by dominant and additive gene action for both *Fusarium* stalk rot and charcoal rot (Thakur *et al.* 1996). Bramel-Cox *et al.* (1988) reported significant general combining ability (GCA) effects for both *M. phaseolina* and *F. moniliforme* in some selected lines. The works by Tesso *et al.* (2004, 2005) also showed additive genes with dominant inheritance as being more important in determining resistance to both *Fusarium* and charcoal rot. Most resistance sources, including 'SC599', 'SC134', 'SC1039' and 'SC35', have significant GCA for resistance under both greenhouse and field conditions, while resistance in a few other sources such as 'SC564' appears to be recessive (Tesso *et al.* 2005). Most of the sources resistant to *Fusarium* appear to have consistent expression across pathogen species, but resistance to *Fusarium* and *Macrophomina* seem to be under different genetic control even though both diseases are aggravated by similar predisposing conditions. Many of the known sources of resistance to *Fusarium* are not resistant to charcoal rot. But there are exceptions: some genotypes, such as 'SC599', express a high level of resistance to both pathogen groups, but genes associated with resistance to the different pathogen species may differ.

DNA-based diversity tags

Perhaps due to the difficulty associated with evaluating large genotype cohorts, little has been done to characterize and tag genomic regions associated with stalk rot resistance in sorghum. As a result, few, if any marker tools are available to use in breeding programs to improve this trait. The only recent attempt is the one made by a group in India where a set of F9 recombinant inbred lines initiated from a cross between 'IS22380' (susceptible) and 'E-36-1' (resistant) were used to map charcoal rot resistance QTLs (Reddy *et al.* 2008). An attempt to genetically characterize potential stalk rot resistance sources using unlinked SSR markers not only revealed marked variation among the genotypes, but also resulted in a fascinating genetic structure where the different sources expressed distinct patterns of association. Most of the resistance sources from the *durra* race of east Africa were grouped together showing a clear geographic pattern of variability (Tesso *et al.* 2005). This and the fact that most of the post-flowering drought tolerant staygreen sources are among the gene pool from the east African region indicates that this area harbors germplasm sources that may be of interest for stalk rot resistance and post-flowering drought tolerance.

DOWNY MILDEW

Introduction

The disease sorghum downy mildew (SDM) is caused by the oomycete *Peronosclerospora sorghi*. As for all host-pathogen interactions, environment has an important role in the occurrence and spread of disease. In the case of downy mildews, dew formation is critical for the production of sporulating lesions on susceptible host varieties (**Fig. 7**). It is the resulting white fluffy appearance of the lesions on the underside of leaves that accounts for the "downy mildew" name. Other than for Australia (Ryley *et al.* 2002) where measures have been taken to prevent the introduction of *P. sorghi*, SDM occurs wherever sorghum is grown, but the disease is especially damaging in more tropical climates. *P. sorghi* can also cause SDM on maize, although oospores are typically not produced on this alternate host. Similarly, there are related *Peronosclerospora* species that can attack sorghum. Examples include *P. maydis* (Ryley *et al.* 2002), *P. sacchari* (Bonde and Peterson 1983), *P. heteropogoni* (Nair *et al.* 2004) and *P. philippinensis* (Bonde and Peterson 1983). Because the latter species is especially virulent on a wide range of important host crops, including maize, it has been subject to strict quarantine and was added to the U.S. list of select agents as a potential bioterrorism threat.

Biology of *Peronosclerospora sorghi*

Because downy mildews have a growth pattern typical of fungi, they have always been classified as fungal diseases. Even now that molecular data show they are more closely related to alga and have been assigned to a separate classification, for practical reasons, downy mildews are still typically grouped with fungal diseases. *P. sorghi* produces conidial spores asexually and produces oospores from the union of oogonia ("female" gametangia) and antheridia ("male" gametangia) born on the same vegetative hyphae in infected leaf tissues. The conidia, produced abundantly in lesions on the underside of the leaf, are very fragile but serve to spread the infection when environmental conditions are appropriate. Oospores by contrast are relatively long-lived and are able to overseason in leaf debris to initiate infections through roots in the next season. Failure to find different mating types while still finding both antheridia and female structures led Pawar (1986) to conclude that *P. sorghi* is homothallic, which is unusual among obligate biotrophs. The fact that *P. sorghi* can only be grown on living susceptible hosts greatly increases the work required to identify pathotypes that differ in the ability to cause disease on host differentials.

Disease symptoms and losses

Plants infected through the roots via oospores develop a systemic infection (**Fig. 8**). Infected plants that survive through the seedling stage are stunted, chlorotic and typic-

ally fully or partially sterile (**Fig. 9**). As the plant matures, lesions characterized by fluffy white mycelia and conidia develop on the underside of leaves. By maturity, the leaves of severely affected plants become shredded, releasing fresh inoculum into the soil. Local lesion infections generally arise as secondary infection from conidia, which are formed only at night and require a wet leaf surface to infect. Cool nights that lead to dew formation greatly increase the level of infection. Symptoms in this case seldom turn systemic, but are limited to the "downy mildew" seen at the site of infection; they do not produce oospores or the associated degree of leaf shredding (Frederiksen and Odvody 2000).

Although high planting rates can minimize yield loss even when up to 30% of the plants are systemically infected, losses to SDM can be severe. High losses are typically seen when susceptible sorghum varieties are planted in oospore-contaminated soils and under conditions favorable to disease development. The highest loss noted in the literature is 78% in cv. 'DMS 652' in India (Thakur and Mathur 2002). Yield losses of up to 11.7% have been reported in Africa (Bock *et al.* 1998). Within Brazil, SDM was initially restricted to the Southern region but has now also spread to the Southeast and Central-West regions, causing significant losses specifically in areas of seed production (Barbosa *et al.* 2006). In the United States, epidemic outbreaks were first seen in Kansas and Texas in the 1960s with subsequent outbreaks of economic significance reported in Kansas (1978-9) and Nebraska (1987) (Jensen *et al.* 1989). For the most part, SDM has been controlled through the use of resistant sorghum varieties and seed treatment with metalaxyl (methyl N-(2,6-dimethylphenyl)-N-(methoxyacetyl)-DL-alaninate). However in 2001 and 2002, the disease reappeared at high levels in sorghum fields in the upper gulf coast of Texas that had been planted with treated seed. It was subsequently determined that not only was a new strain of metalaxyl-resistant *P. sorghi* present, but that a new pathotype had arisen, likely as a result of growers who had not used resistant varieties, which allowed rampant reproduction via the spread of conidia from local lesions (Isakeit *et al.* 2003; Isakeit and Jaster 2005).

Pathogen races

The discovery of a new race, subsequently labeled "pathotype 6" (P6), led to screening programs to identify new resistance sources. Prom *et al.* (2010) found that 6 of 20 accessions in the U.S. sorghum germplasm that originated from Chad were resistant when grown in the area where the new race is prevalent and that 6 of 40 accessions originating from China were also resistant to P6 (Prom *et al.* 2007). A larger greenhouse screening was undertaken with a set of 245 accessions assembled from the nearly 40,000 lines in the ICRISAT germplasm collection to provide a 'minicore' collection that includes much of the genetic diversity of sorghum (Upadhyaya *et al.* 2009). The tests also included breeding lines from the Kansas State and Texas A&M sorghum breeding programs as well as commercial hybrids. Overall, only 52 of the minicore accessions were resistant to the new pathotype, but nearly half of the elite breeding lines and commercial hybrids from both states showed resistance (Radwan *et al.* 2011).

Disease screening techniques

Several methods have been developed to screen for resistance, including inoculation of soil with oospore-laden leaf fragments and the use of 'downy mildew nurseries' (Thakur *et al.* 2007a). However, due to the exacting conditions under which sporulation and infection will occur, greenhouse based tests allow for the most controlled experiments and the lowest frequency of escapes. Even if a dew chamber is not available, relatively simple adaptations can be made to allow inoculation and screening of flats of germinated seedlings. The "sandwich procedure" (Thakur *et al.* 2007a) as illustrated in Radwan *et al.* (2011) proved to be very effective in the screening tests for P6 described previously.

Disease control and host resistance

Sorghum downy mildew can be effectively controlled by a combination of methods, which include cultural, chemical, and host resistance. Cultural practices such as crop rotation, deep plowing, early planting, and the use of trap crops have been shown to significantly reduce downy mildew incidence (Frederiksen 1980; Schuh *et al.* 1987; Pande *et al.* 1997). Other than in areas where the previously mentioned fungicide-resistant strain has developed, metalaxyl or fosetyl-Al applied as a seed treatment can provide effective chemical control of SDM (Odvody and Frederiksen 1984). The recent severe epiphytotic of SDM observed in Texas and the emergence of a new race that is pathogenic to previously resistant cultivars has increased efforts to identify alternate sources of host plant resistance for managing the disease.

Navi *et al.* (2003) evaluated 1671 *S. bicolor* accessions (945 elite landraces and 726 breeding lines) for multiple disease reaction under field conditions and natural inoculation during the 2001 rainy season at ICRISAT. Of the 945 landraces from 46 countries, 82 accessions from India, Lesotho, Botswana, Zimbabwe, Russia, Swaziland, Cameroon, Sudan, Ethiopia, Malawi and Nigeria were free from all diseases including downy mildew, ergot, head smut and anthracnose.

In Brazil, sources of resistance for downy mildew among 42 sorghum genotypes were identified through field evaluation under natural infection. Cultivar 'SC283' served as a susceptible check. The tests were conducted in two separate nurseries in Sete Lagoas and Minas Gerais expected to have different pathtypes of *P. sorghi*. While 15 cultivars were classified as highly resistant in both nurseries, only 'SC170-6-17' and '9910296' showed 0% systemic infection (Barbosa *et al.* 2005).

Forty Chinese sorghum accessions maintained by the USDA-ARS, Plant Genetic Resources Conservation Unit, Griffin, Georgia were evaluated by Prom *et al.* (2007) for multiple disease resistance. The level of sorghum downy mildew (SDM) infection with systemic infection and local lesion development for infected plants was low to very high. Accessions 'PI511832', 'PI563519', 'PI563521', 'PI563850', 'PI610677' and 'P610724' were the most resistant to sorghum downy mildew, whereas 'PI610692' and 'PI610720' were the most susceptible to SDM.

In another study by Prom *et al.* (2010), 78 accessions from Chad, Central Africa and 20 photoperiod insensitive accessions from Uganda were evaluated for downy mildew resistance in Ocotlan, Mexico in 2004 and 2005. Several sources of downy mildew resistance were identified. Three accessions 'PI282860', 'PI282864', and 'PI563505' from Chad were shown to possess high levels of downy mildew resistance in Mexico and Texas, whereas 'PI282843', 'PI282877', 'PI549196', and 'PI563438' also from Chad exhibited high levels of resistance to the disease in Texas. Accessions 'PI297210', 'PI576386' and 'PI576395' from Uganda also showed downy mildew resistance in Mexico and Texas. These sorghum accessions from Chad and Uganda can be utilized in breeding for downy mildew resistance in Mexico and Texas.

Sharma *et al.* (2010) in India and Radwan *et al.* (2011) in the United States screened the sorghum "minicore" germplasm collection, a subset of the sorghum germplasm collection maintained at ICRISAT. The minicore collection includes 245 representatives assembled to include much of the diversity of the overall collection of over 37,000 accessions (Upadhyaya *et al.* 2009). In these tests, the accessions were screened in the greenhouse following a sandwich inoculation technique, with '296B' and 'QL3' respectively, serving as susceptible and resistant checks. Downy mildew resistance (mean incidence $\leq$ 10%) was observed only in six accessions in India ('IS 28747', 'IS 31714', 'IS 23992', 'IS 27697', 'IS 28449', and 'IS 30400'), whereas 52 resistant

accessions (28 were photoinsensitive) were identified by Radwan *et al.* (2011) in the United States against the P6 pathotype.

DNA-based diversity and molecular tags

DNA based analysis techniques have been applied both to attempt to tag genes for resistance in the host and to identify species and races of the downy mildews that can attack sorghum. Yao *et al.* (1991b) were able to use DNA extracted from *P. sorghi* spores to show that the pathogen could be seedborne in maize, but this is not considered a problem if the seed is dried below 20% moisture. They subsequently showed that repeated sequence clones readily detect RFLP fingerprint differences among *P. sorghi*, *P. maydis* and *P. sacchari*, which were not differentiated from *P. philippinensis*. A Thailand isolate previously presumed to be a derivative of *P. sorghi* was different and thus was renamed *P. zeae* (Yao 1991). He also found differences in the base sequences of the internal transcribed spacer (ITS2) of the ribosomal DNA of the different species (Yao *et al.* 1992) and developed PCR primers that specifically amplify DNA from all the *Peronosclerospora* spp. and from *P. sorghi* only (Yao *et al.* 1991a).

In a study by Mathiyazhagan *et al.* (2008), DNA was extracted from *P. sorghi* isolates from sorghum and maize collected from different locations of Tamil Nadu, India, and their genetic variability was investigated using restriction fragment length polymorphism (RFLP) analysis of the PCR-amplified internal transcribed spacer (ITS) region of ribosomal DNA. PCR amplification of the ITS regions of the *P. sorghi* isolates from sorghum and maize gave products of approximately 550 bp in length with slight variations among isolates. RFLP analysis of the ITS regions of nuclear rDNA of *P. sorghi* with *Hha*I, *EcoR*I and *Msp*I revealed differences among *P. sorghi* isolates sampled from sorghum and those from maize. Subsequently, Ladhalakshmi *et al.* (2009) developed SCAR primers based on a sequence identified via PCR amplification with a RAPD primer that amplified an 800 bp fragment only from isolates from maize. Because inoculum from one host can generally be used to infect the other, such host specific unique sequences suggest that *P. maydis* or *P. zeae* may have been present, although the possibility exists that host-specific *formae speciales* have evolved within *P. sorghi*. In an attempt to discover the origin of pathotype 6 and to develop DNA-based markers to distinguish among pathotypes, Perumal *et al.* (2008) used AFLP and SSR to characterize a number of isolates of pathotypes 1, 3 and 6. Although the results strongly suggest that pathoype 6 arose from pathotype 3, unfortunately none of the individual markers showed total concordance with pathotype (Perumal *et al.* 2006, 2008).

Kamala *et al.* (2006) used 10 simple sequence repeat (SSR) marker loci and 20 phenotypic traits to assess diversity among 36 downy-mildew-resistant sorghum accessions. The lines were chosen at random from 130 (out of ~16,000 screened) that showed a high level of resistance (0 to 5% mean disease incidence of the total plants systemically infected) to the ICRISAT isolate of *P. sorghi*. High gene diversity and allelic richness were observed in sorghum races durra caudatum and guinea caudatum and in accessions from east Africa. The pattern of SSR-based clustering of accessions was more in accordance with their geographic proximity than with race designations based on phenotypic traits. Eleven pairs of resistant accessions had a SSR genetic distance of more than 0.70 suggesting they will likely provide different sources of resistance and be useful as parents in sorghum downy mildew resistance-breeding programs.

With respect to tagging host genes for resistance, the first success came from the use of RFLPs and RAPD primers. Because the two parents used in creating an RFLP map for sorghum differed in susceptibility to SDM ('BTx623' is resistant to P1 while 'IS3620C' is susceptible), Gowda *et al.* (1995) were able to show that clone *txs552* revealed an RFLP linked to resistance. Also using the same clones developed to create an RFLP map for sorghum, Oh *et al.* (1996) identified two RFLPs that segregated with a resistance gene in F_2 progeny of a cross between 'SC325' (resistant to P1, P2 and P3) in a cross to 'RTx7078' (susceptible to all pathotypes). Although the markers were 5 to 7 map units from the gene for resistance they were useful in determining that the two sources of resistance were not the same gene. In a third cross segregating for resistance ('SC414', resistant and 'RTx7078', susceptible) a screen of 674 primers revealed two that amplified bands that segregated relatively closely with resistance. The same differences did not show up in amplification products of the mapping parents, suggesting that this represents a third independent source of SDM resistance, at least to P1. In each of these cases resistance has been associated with a single gene. However, there are also cases in maize where SDM resistance has been shown to segregate as a quantitative (polygenic) trait (Agrama *et al.* 1999).

Future directions

Further research in the area of race determination in *P. sorghi* perhaps taking advantage of the advances in low-cost DNA sequencing should help to identify differences between races that could be used to greatly simplify pathotype identification and resolve the question of the origins of new pathotypes. In addition, DNA markers should help to identify those sources of resistance that differ among sorghum cultivars, and enable subsequent gene stacking that is predicted to enhance the durability of resistance.

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