ABSTRACT. Anthracnose caused by Colletotrichum graminicola remains the most important foliar disease of sorghum in West and Central Africa. This paper describes the advances made in sorghum anthracnose research that has led to a better understanding of pathogenic and genotypic diversity, epidemiology and important disease management strategies. We further highlight how understanding pathogen diversity interplays with the major sustainable anthracnose management strategies such as the use of host plant resistance and crop residue management within the region. [Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <docdelivery@haworthpress.com> Website: <http://www.HaworthPress.com> © 2004 by The Haworth Press, Inc. All rights reserved.]
INTRODUCTION

Sorghum (*Sorghum bicolor* (L.) Moench) is a very important food crop in West and Central Africa (WCA). It is well adapted to the semi-arid tropics, with production concentrated in three agro-ecological zones—Sahel (400-600 mm), Sudan (600-1000 mm) and Guinea (1000-1300 mm) annual rainfall that cut across the region. The total area sown to sorghum in WCA is estimated at 12.5-14.9 million hectares with average yields of 0.78 tons/ha, respectively. The total production of this crop is reported to be 10.2 million tons of which Nigeria is the major producer with an estimated total production of 8.5 million tons (ICRISAT/FAO, 1996; Marley and Ogunbile, 2002). This crop is primarily produced for human consumption, however, in Nigeria and to some extent Ghana, over 600,000 tonnes is utilized in the industrial production of livestock feed, production of beer and other malted beverages, and for the production of sweeteners (Ogunbile and Marley, 2001).

Sustainable production of sorghum in West and Central Africa is limited by many constraints including biotic factors, e.g., Marley (2003) reports the occurrence of over 32 diseases of sorghum in Nigeria (Table 1). Some of the more common diseases are caused by fungi, bacteria, viruses, and nematodes of which anthracnose caused by *Colletotrichum graminicola* (syn. *Colletotrichum sublineolum*) is the most important foliar disease on both local and improved sorghum varieties in West and Central Africa (Thomas et al., 1996; Marley et al., 2001a, 2002a,b). Although sorghum anthracnose was first reported in Togo in 1902 (Stoop et al., 1982), information available on the disease and its management for most countries within the sub-region is limited. This paper reviews information on anthracnose available within WCA and highlights areas of sustainable anthracnose disease management that could contribute to increased production within the sub-region.

GEOGRAPHICAL DISTRIBUTION AND ECONOMIC IMPORTANCE

Sorghum anthracnose is prevalent whenever sorghum is grown in a warm and humid environment. It is widely prevalent and considered of primary importance in all parts of West Central Africa (Marley et al., 2002a,b). Many local land races and improved varieties are susceptible to the foliar stage of the disease with serious epidemics occurring in farmers’ and research fields (Thomas, 1995; Marley, 2002b). It is most prevalent and destructive in the northern Guinea and Sudan zones (Pande et al., 1993).
The disease causes both direct and indirect yield losses in West Africa (Thomas, 1993). The extent of direct losses varies with location, cultivar and prevalent climatic conditions. The reduction in 1000-seed mass and seed density, and early abortion of seeds are the most important factors in yield reduction. The premature drying of leaves and defoliation due to foliar anthracnose can reduce the yield of sorghum grain and fodder by 30-50%, or more, in susceptible cultivars during severe epidemics (Mathur et al., 2002). In Burkina Faso, secondary infection of the stalk by red rot phase may cause yield losses proportional to the severity of leaf anthracnose and stalk rot (Neya and Kabore, 1987). They recorded a yield loss of 8-46% in a local variety Gnofing. In Mali, yield losses due to foliar anthracnose ranged between 44 and 67% in a susceptible variety, IS 18696, while grain abortion was observed in varieties IS 18696 and IS 18442 (Thomas, 1995; Thomas et al., 1996). At Samaru, Nigeria, Marley (1996) reported a yield loss of 47% on susceptible variety BES (KSV 4) caused by foliar anthracnose. Yield loss due to panicle anthracnose has not been determined within the sub-region. Indirect losses also are due to grain anthracnose, which results in reduced seed germination, and the transmission of the disease to new geographic locations (Marley et al., 2003). Further, sorghum anthracnose often occurs as a mixed infection with zonate leaf spot in Mali and Nigeria (Marley and Bandyopadhyay, 1996; Bandyopadhyay

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**TABLE 1.** Cultural characteristics of five representative isolates\(^1\) of *Colletotrichum graminicola* on potato dextrose agar with streptomycin\(^2\)

<table>
<thead>
<tr>
<th>Plant part</th>
<th>Isolate</th>
<th>Type of growth</th>
<th>Colony color</th>
<th>Acervuli production(^3)</th>
<th>Setae presence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grain</td>
<td>G1-G3</td>
<td>Cottony and dense, slow growing</td>
<td>Brownish black</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Leaf</td>
<td>L1-L3</td>
<td>Appraised with cottony areas, moderately fast growing</td>
<td>Grey</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Rachis</td>
<td>R1-R3</td>
<td>Felty and dense, slightly elevated, moderately fast growing</td>
<td>White</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Peduncle</td>
<td>P1-P3</td>
<td>Cottony and dense appraised at the center and slightly raised towards periphery, fast growing</td>
<td>Greenish grey</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Stalk</td>
<td>S1-S3</td>
<td>Cottony and loose, moderately fast growing</td>
<td>Dull-white</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

\(^1\)Each of the five isolates is a mean representation of a total of nine isolates per each plant part (3 each obtained from three locations viz: northern Guinea, Sudan and Sahel savanna zones).

\(^2\)Characteristics observed 7 days after planting on the medium.

\(^3\)(+) = Present.

Adapted from Anas et al. (2001).
et al., 1996). There have been no studies to assess the extent of damage due to mixed infection by two or more diseases.

**THE PATHOGEN, ETIOLOGY AND SYMPTOMATOLOGY**

The taxonomic status of the sorghum anthracnose pathogen *Colletotrichum graminicola* (Ces.) Wils (= *C. sublineolum* Henn. Kabat & Bub.) has been controversial, probably due to the wide host range and the morphological and pathogenic variability associated with this fungus (Pastor-Corrales and Frederiksen, 1980). Isolates recovered from one host will not necessarily infect other hosts. In Nigeria, Alawode et al. (1983) reported that pinpoint isolates of *C. graminicola* from sorghum caused mid-rib infection in millet. However, in a follow-up study, Bindawa (1987) found that isolates from sorghum were specific to sorghum and will not infect maize or millet. This study confirmed the midrib infection in millet was caused by other pathogens (*Cercospora* spp.) rather than *C. graminicola*. In a related study, Zarafi et al. (1995) confirmed the identity of the causal agents of the millet midrib spot disease to be *Curvularia pennisetii*, *C. eragrostidis*, *C. intermedia* and *C. verrucosa*. All *Curvularia* spp. induced typical midrib spot symptoms on maize, sorghum, *Pennisetum* sp., *Eragrostis* sp. and pearl millet in host range studies. This study clearly confirmed that strains of *C. graminicola* infecting sorghum did not infect millet and were different from strains infecting maize, particularly in Nigeria and possibly in the whole of West and Central Africa. This supports the designation of strains infecting sorghum as *C. sublineolum* on the basis of their appressorial morphology while strains infecting maize were designated *C. graminicola* (Sutton, 1968; Sutton, 1992) and rDNA analysis (Bailey et al., 1995; Sheriff et al., 1995).

*C. graminicola* can infect all above-ground plant parts, including stem, leaves, peduncle, inflorescence and grain (Zummo, 1984). The most common form of this disease is leaf or foliar anthracnose, characterized by circular to elliptical, red spots up to 0.5 mm diameter with few to numerous acervuli on lamina. Differences in foliage and grain symptoms may be due to host reaction, physiological status of the host, or the environment (Ferreira and Warren, 1982; Pastor-Corrales and Frederiksen, 1980), and pathogenic variability (Frederiksen, 1984; Pande et al., 1991). The large range in symptoms (patchy, midrib and pinpoint), cultural characters and isolate morphology led to the description of a forma speciales *C. graminicola var. isolatum* from Nigeria based on morphological characters and pathogenicity within West and Central Africa (Alawode et al., 1983).

There are conflicting reports about host specificity and host range of *C. graminicola* isolates from different hosts. *Digitaria exilis* and *Dactyloctenium aegyptium* are all putative collateral hosts of *C. graminicola* infecting sorghum (Mathur et al., 2002). *C. graminicola* can survive in infected leaf debris buried
in the soil. In the sandy loam soil of Bobo-Dioulasso, Burkina-Faso, infective conidia could be recovered from soil nine months after harvest (Traore and Kabore, 1992). They showed that accumulation of *C. graminicola* spores begin near the last week of July and survive until the end of the rainy season. As conidia are thin-walled and prone to desiccation and fungistasis in the soil, the origin of these infective conidia is thought to be sporogenic sclerotia in the soil. Use of specific techniques to identify sclerotia in soil may provide more information on the survival of *C. graminicola*. Infected seeds are also potential sources of infection in fields first planted to sorghum, and may give rise to infected seedlings (Marley et al., 2003). Midrib infection may occur on cultivars that have little or no lamina infection, and this response may be independent of leaf infection (Alawode et al., 1983; Zummo, 1984).

Panicle infection could occur through direct infection by germinating conidia from leaves (foliar phase) due to rain splash or may also be an extension of the stalk rot phase. Infection first appear as elliptical pockets or bar immediately beneath the epidermis which may look water-soaked and discolored lesions that later become tan to blackish purple. Acervuli appear as small, black streaks that may extend to the seed. Sporulation occurs on the central rachis, on primary, secondary and tertiary branches, on glumes and on seed. Early infection of panicle causes production of small-sized seeds while severely infected grains may be completely discolored. In the stalk phase of the disease, it develops from inoculum produced during the foliar phase. Conidia are disseminated by splashing rain, germinate and infect the stalk directly. The rate of stalk colonization by *C. graminicola* is related to varietal reaction, environment and level of inoculum (Frederiksen and Odvody, 2000; Marley and Ajayi, 2002).

**CULTURAL, MORPHOLOGICAL AND PATHOGENIC VARIABILITY**

Isolates of *C. graminicola* from different locations, lesions, or even the single-conidial derivatives from single-lesion cultures can vary considerably in morphology when grown in culture (Marley et al., 2001a, Ozolua et al., 1986a). Acervuli may range from not well differentiated with occasional setae (indistinct) to well developed (distinct) with 2 to > 20 setae per acervulus. Other variable characters include: size of acervuli, and production of appressoria, chlamydospores and sclerotia (Alawode et al., 1983, Ozolua et al., 1986a, Anas et al., 2001). Thomas (1995) reported the presence of two isolates of *C. graminicola* in the Samanko area of Bamako, Mali. Separation of isolates was based on symptom type, broad culture and morphological characteristics. Earlier, Alawode et al. (1983) had identified three races of *C. graminicola* based on symptom types. Based on morphological and cultural characteristics, Marley et al. (2001a) identified nine morphological groups from 50 isolates collected from major sorghum growing areas of Nigeria. Further, Anas et al.
(2001) in a complimentary study reported the existence of five morphological groups within five isolates collected from various plant parts infected with disease. Significant intra-population variation in colony growth, pigmentation, and conidial size occurred within isolates sub-cultured from a colony initiated from a single conidium. Morphological variation, however, is independent of pathogenic or genetic variation (Rao et al., 1998).

There is genetic variation within the foliar population of *C. graminicola* in West and Central Africa (Ozolua et al., 1986b; Thomas and Frederiksen 1995; Thomas et al., 1995; Neya and Normand, 1998) although variation in Burkina Faso is limited (Neya and Normand, 1998).

The use of virulence to assess genetic variability provides direct information on the effect of host selection. Within *C. graminicola*, variation is known for virulence (disease reaction) and for aggressiveness (disease severity). This variation usually is detected on the basis of differential foliage reaction amongst a set of sorghum cultivars (Ozolua et al., 1986b, Marley et al., 2001a, Anas et al., 2001). In sorghum anthracnose up to nine races/pathotypes have been identified amongst diverse strains that have been tested under both screen house and field conditions in Nigeria (Ozolua 1986b). However, recent work at Samaru, Nigeria identified five physiological races from 50 foliar isolates collected from all major sorghum growing areas (Marley et al., 2001a).

Current evidence shows that fungal isolates from foliage, grain, and stalks are different within the region. Sorghum lines resistant to foliar anthracnose can develop heavy grain infection, e.g., Nagawhite indicating that foliar and grain anthracnose are independent to each other (Hess et al., 2001; Marley and Ajayi 2002). A study carried out at Samaru, Nigeria show that there is pathogenic variability between isolates from the same plant. Anas et al. (2001) reported that isolates from grain, rachis, peduncle and stalk differed pathogenically from the foliar isolate, all from the same plant.

A single International Sorghum Anthracnose Virulence Nursery (ISAVN) coordinated by ICRISAT was established in 1992 to help standardize pathotypes of *C. graminicola* (Thakur, 1995). The nursery (a set of sorghum lines, which are tested at different sites against the local fungal population) consists of a local resistant and a local susceptible check and 15-18 sorghum lines, with diverse geographic origins and differential reactions to know pathogen populations identified in earlier nurseries. Until 1998 the ISAVN was evaluated at 9-19 locations in 12 countries in Asia, Latin America, and southeastern, western and central Africa (Mathur et al., 2002). Anthracnose severity was scored at the soft dough stage, on a standard disease rating scale of 1-9, based on percent leaf area covered with necrotic lesions (Thakur, 1995). The basic assumption underlying the nursery is that each environment/location represents a unique pathogen population that is being evaluated on a set of differential sorghum lines. There is an apparent need to continue the use of the ISAVN as an international set of differentials for use by future workers to enable evalu-
tion of populations and establishment of race groups (Marley et al., 2001a) as in other pathogens, e.g., *Sporisorium sorghi* (Frowd, 1980). If a pathogen caused a susceptible reaction (necrotic lesions with acervuli), it was considered ‘virulent,’ otherwise ‘avirulent.’ Virulence was determined from the mean severity score on a host differential line over years. Isolates (or locations) with a mean severity score ≤ 5 as virulent. Correspondingly, host differential lines with a mean score ≥ 5 were considered resistant, and those with mean severity score > 5, susceptible. Disease severity scores of sorghum lines varied by location and by year. Based on the mean severity score across the entries, the population at Bagauda (Nigeria), Samaru (Nigeria) and Maiduguri (Nigeria) were among the aggressive within WCA.

**SUSTAINABLE ANTHRACNOSE MANAGEMENT APPROACHES**

**Host-Plant Resistance**

This is the most sustainable, reliable and economical method of anthracnose management under farmer’s conditions in West and Central Africa. Although breeding for anthracnose resistance appears simple, the numerous physiological races of the pathogen often result in breakdown of resistance. Identification of durable resistance requires the screening of sorghum genotypes against multiple races of the pathogen at multiple locations (Mathur et al., 2002).

Using standardized field screening techniques, anthracnose severity is best scored at the soft-dough stage for field evaluation, and 14 days after foliar inoculations or 30 days after grain inoculations for greenhouse tests. Various disease-scoring scales for foliar anthracnose severity, usually based on the % leaf area covered with necrotic lesions have been used. A 1-9 scale is now used with the ISAVN where, 1 = no lesions, 2 = 1-5%, 3 = 6-10%, 4 = 11-20%, 5 = 21-30%, 6 = 31-40%, 7 = 41-50%, 8 = 51-75%, and 9 = > 75% of the leaf area covered with lesions (Thakur, 1995). In greenhouse inoculations it is possible to record latent period (time in days to development of first necrotic/chlorotic lesion), disease severity on the 1-9 scale, and disease reaction, R = resistant (no symptoms or chlorotic flecking); MR = moderately resistant (hypersensitive lesions, red spots or necrotic spots without acervuli); and S = susceptible (lesions with acervuli). Disease severity on grain anthracnose can be recorded 30 days after inoculation by using a similar 1-9 scale and based on the percent head area infected (Frederiksen et al., 1982). Disease ratings can be converted to AUDPC, or to a virulence index: (1+ virulence \times aggressiveness \times latent period\(^{-1}\)) (Mathur et al., 1997; Rao et al., 1998).

Stable sources of resistance to anthracnose have been identified in West and Central Africa. In Mali, Thomas (1995) reported 16 local and improved varieties to have resistance to foliar anthracnose. These include CSM 388, IS 8283,
ICSB 38, ICSB 39, 84 S 82, BOPER r3, BOPER r4, BOPER r5, KAMPTI GLM, BLANC-KARIMANA, NSV 1, MANKANGARA, LOCAL-29, NSV 68, NSV 74-1 and NSV 83. Recently, Hess et al. (2001) observed nine genotypes with good resistance to both foliar and panicle anthracnose. These are: ICSB 88019, IS 2834, IS 14384, IS 21629, ICSV 901, ICSV 902, CEM 328/1-1-1-2, CEM 330/1-1-2-1 and NAGAWHITE. In Nigeria, Marley et al. (2001b) reported the genotypes CSM 417, MALISOR 84-5, NAGAWHITE, YAR’RURUKA, ICSV 901 NG amongst the early maturing, and CGM 19/9-1-1, KASSOUROKA, SABAKOUN, SARIASSO 9 and GAYA EARLY among the medium maturing to be resistant to foliar anthracnose. Further, Marley et al. (2001c) reported 74 out of 120 genotypes to be highly resistant to foliar anthracnose. Some of these genotypes include IS 359, IS 5360, MG 114, IS 4509, IS 20302, IS 24844, IS 24829, IS 24873, IS 24733, IS 34767, IS 33255, IS 32588, IS 25547, IS 3025, IS 3384, IS 1212 amongst others. Gwary et al. (2001) reported KSV 4, IS 8758, IS 6958, ICSV 247, IS 6928 and IRAT 204 to be resistant to foliar anthracnose at Maiduguri, Nigeria. Some of these genotypes—e.g., KSV 4 (BES) and IRAT 204—are highly susceptible to anthracnose at Samaru, Nigeria. This underscores the role of pathogen variability in the sustainable management of the disease using host plant resistance. Recently, Marley et al. (2002c) reported 42 lines among 64 lines tested to be resistant to foliar anthracnose at Samaru. These include ICSV 424, ICSV 1049, ICSV 93027, ICSV 95072, ICSV 95043, ICSV 95044, ICSV 95045, ICSV 95046 and ICSV 95957. Others are PB 15833-1-1, PB 148441-1, PB 155020-2-2, PB 15828-2-1-1, IS 854, IS 8354, IS 3758, IS 3552, IS 1006, IS 12447 SAMSORG 14 (KSV 8), SAMSORG 17 (SK 5912), NR 71198, NR 71176 and NR 71137. Local varieties BAGAUDA FARAFARA, YARDU, JAWO SANDA, KAURA and MORI showed high resistance. Marley and Ajayi (2002) assessed resistance to foliar, peduncle, rachis, grain and panicle anthracnose in 21 genotypes at two locations, Bagauda and Samaru. Three genotypes—R 6078, IS 14384 and CCGM 1/19-1-1—were completely resistant to the disease while NAGAWHITE was resistant to foliar, peduncle and rachis but susceptible to grain anthracnose. In Burkina Faso, Neya and Kabore (1992) identified 13 local varieties resistant to foliar, stalk and grain infection. However, in another study using 14 local and improved sorghum genotypes in a multi-locational trial, Neya and Normand (1998) found that none of the genotypes showed resistant reactions. However, four genotypes—Gnofing, Sariasso 10, Siripe 1 and ICSV 1002 BF—were moderately resistant. The reports clearly show the availability of sorghum germplasm with moderate to resistant resistance to anthracnose within the region. However, despite these achievements, very few of these genotypes have been actually developed and are available as cultivars to farmers in WCA. This indicates the need for the development of anthracnose resistant sorghum cultivars within the region. Where resistant cultivars are available to farmers, these should be exploited
where possible by encouraging farmers to use those that have been released, e.g., Nagawhite in Ghana and SAMSORG 14 (KSV 8) and SAMSORG 17 (SK 5912) in Nigeria. Also, sorghum varieties Sariasso 10 and Siripe 1 are currently being cultivated in Burkina Faso, while MALISOR 84-5 (moderately resistant to anthracnose) is currently widely grown in the Kolokani area (> 800 mm rainfall) while the cultivation of Ntenimissa (moderately resistant to anthracnose) is also been promoted in Mali. Cultivation of high yielding local cultivars that are found to also have moderate to high resistance to anthracnose should be encouraged in locations that they are suitable—e.g., YAR’RURUKA, and BAGAUDA FARAFARA in the Sudan savanna and MORI in the northern Guinea savanna zones of Nigeria. Efforts targeted towards the identification and incorporation of foliar, stalk and panicle anthracnose resistance into new varieties should continue with high priority if continuous sustainable management of the disease within WCA is to be achieved.

**Crop-Residue Management**

A 4-year trial conducted at three locations within WCA (Samaru, Bamako and Bobbo-Dioulasso) on the use of crop residue management for control of anthracnose indicates that the farm practice of sanitation is best. Cleaning fields at the end of harvest for feeding livestock and again at the beginning of the season significantly reduces the incidence and severity of anthracnose compared to the practices of incorporating crop residue into the soil for enhancement of soil fertility or leaving the residue on fields for animal grazing and cleaning the fields before land preparation (Table 2). The results indicate clearly that the management of anthracnose using proper field sanitation as a cultural control measure remains highly sustainable within WCA.

**Other-Management Methods**

Seed treatment with Apron-plus (methalaxyl + carboxin + furathiocarp) alongside foliar applied fungicides such as carbendazin + manebe and mancozeb has been reported to be effective in the control of anthracnose in Nigeria (Akpa et al., 1992). The use of 2-3 foliar sprays of benomyl have also been found to be effective in controlling anthracnose in sorghum in experimental and seed production plots at Samaru, Nigeria (Marley, 1996, 1997). However, the use of fungicides to control anthracnose is not economical and sustainable. Based on the economic returns farmers in the WCA receive for cultivating sorghum, the use of fungical control as a method for anthracnose control is not recommended for use by resource poor farmers of West and Central Africa.
CONCLUSION

Anthracnose of sorghum causes economic losses to farmers in WCA. Research within WCA has identified various sorghum germplasm with moderate to high resistance to the disease. Crop residue management in the form of cleaning of fields after harvest is found to significantly reduce the level of anthracnose infection in fields. The fulcrum of sustainable anthracnose management in WCA will rely on the continuous use of host plant resistance where available and good field sanitation. Current priority research focus remains the identification, incorporation and promotion of anthracnose resistant varieties. Further, more epidemiological studies will broaden the role of cultural control measures aimed at anthracnose. The integration of these control measures where possible is highly desirable and should be promoted.

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