The Americas Journal of Plant Science and Biotechnology ©2008 Global Science Books

Sorghum Germplasm Resistance to Anthracnose

John E. Erpelding*

USDA, Agricultural Research Service, Tropical Agriculture Research Station, 2200 Pedro Albizu Campos Ave., Suite 201, Mayaguez, Puerto Rico 00680-5470 USA

Correspondence: * **john.erpelding@ars.usda.gov**

ABSTRACT

Anthracnose is one of the most damaging diseases for sorghum production. The disease can be successfully managed through the use of resistant cultivars, but the development of resistant cultivars is hindered by extensive variation in virulence within the pathogen population. Additional sources of resistance are needed to more effectively manage the disease and sorghum germplasm collections are important sources of genetic variation for anthracnose resistance. A disease inoculation procedure was developed to enhance the evaluation of anthracnose resistance for the sorghum collection maintained by the USDA-ARS National Plant Germplasm System. Germplasm evaluations have suggested that sources of anthracnose resistance could be associated with country of origin. For example, resistant germplasm was more frequently observed for the sorghum collections from Ethiopia, Mali, and Sudan. The anthracnose evaluation of the Mali germplasm collection indicated that resistance was also associated with weather patterns within Mali. Resistant germplasm was more frequently observed in regions associated with higher annual rainfall compared to drier regions. This association of resistance with rainfall patterns has been observed for collections from other African nations. For collections from wetter regions, such as Rwanda and Mozambique, the majority of the accessions were resistant. In comparison, nearly all the accessions in the collections from dry regions, such as Somalia and Algeria, were susceptible to anthracnose. The genetics of host plant resistance is presently being evaluated to determine if the greater frequency of resistance observed in these regions is associated with genetic variation for resistance.

Keywords: *Colletotrichum sublineolum*, disease resistance, disease screening, ecogeographic, genetic resources, *Sorghum bicolor*

Anthracnose is considered one of the most important diseases of sorghum (Leslie 2002) and yield losses greater than 50% have been reported (Harris *et al*. 1964; Thomas *et al*. 1996; Thakur and Mathur 2000). *Colletotrichum sublineolum* P. Henn., Kabát & Bubák is the fungal pathogen responsible for sorghum anthracnose and is only pathogenic to *Sorghum* spp. Morphological, mating, and molecular genetic evaluations have indicated that *C. sublineolum* is a distinct species from *C. graminicola* (Ces.) G.W. Wilson, which is pathogenic on maize, and *C. falcatum* Went that infects sugarcane and numerous grass species (Sutton 1968; Vaillancourt and Hanau 1992; Freeman *et al*. 1993; Sherriff *et al*. 1995; Crouch *et al*. 2006). Morphological characterization of the pathogen was reported by Doggett (1988) and Thakur and Mathur (2000). Conidia spores are nonseptated and have a characteristic sickle shape. Mycelium growth in culture is generally white and fluffy in appearance, but mycelium color, appearance, and growth rate is variable (Pande *et al*. 1991; Marley *et al*. 2001b).

The disease occurs worldwide and was first reported in Togo, West Africa in 1902 and in the United States in 1912 (Harris *et al*. 1964; Thakur and Mathur 2000). Sorghum anthracnose is most prevalent in tropical and subtropical regions where warm, humid climatic conditions enhance the development and spread of the disease (Néya and Le Normand 1998; Thakur and Mathur 2000; Hess *et al*. 2002; Ngugi *et al*. 2002). In the United States, the disease is most frequently observed in the southeastern states (Harris *et al*. 1964) and growing resistant sorghum cultivars is recommended. Infection can be observed on all above-ground tissues of the sorghum plant with disease symptoms generally appearing 30-40 days after seedling emergence, however, infection can occur at every stage of plant development (Thakur and Mathur 2000). Foliar infection is most commonly observed and is referred to as red leaf blight. Anthracnose infection can also occur on the stalk, panicle, and seed. Typical symptoms of foliar infection are circular, elliptical, or elongated lesions on the leaves of susceptible cultivars with purple, red, tan, or black margins depending on host plant pigmentation (**Fig. 1**). Black fruiting bodies (acervuli) appear in the center of the lesions during sporulation of the fungus. Conidia are produced in a mucilaginous substrate and rainfall or prolonged dew periods contribute to the spread and secondary infection. A detailed evaluation of the infection process has been reported by Wharton and Julian (1996) and Wharton *et al*. (2001). The pathogen is classified as hemibiotrophic where living tissue is required to initiate the infection process (biotrophy) followed by the formation of necrotrophic secondary hyphae in compatible interactions (Wharton *et al*. 2001). Red spots or reddening of leaf tissue can be observed for resistant cultivars due to the production of phytoalexins at the site of infection (Snyder *et al*. 1991; Lo *et al*. 1999a). The resistant response can vary from a few small red spots scattered over the leaf surface to complete reddening of the leaf lamina (**Fig. 2**). This red response is clearly visible within 7 days after inoculation for both resistant and susceptible plants followed by acervuli development on susceptible genotypes. Nicholson *et al*. (1987) reported that the response is limited to the site of infection in juvenile leaves; thus, visual symptoms are not observed during the seedling stage for anthracnose resistant or susceptible genotypes. For mature leaves of resistant genotypes, the response is observed as cells surrounding the site of infection show phytoalexin production (Snyder and Nicholson 1990). In greenhouse evaluations, no symptoms were observed when plants were inoculated 15 days after planting; whereas, inoculations conducted 25-35 days after planting resulted in the expression of disease symptoms (Ferreira and Warren 1982). For field evaluations, inoculations conducted approximately 30 days after planting have shown a consistent disease response (Erpelding and Prom 2006) and plants are the most sensitive to infection at this stage of growth (Pande *et al*. 1994). The production of phytoalexins has also been observed in response to inoculation with nonpathogenic fungi (Nicholson *et al*. 1987; Hipskind *et al*. 1990). Numerous studies have repor-

Received: 26 December, 2007. Accepted: 20 January, 2008.

Invited Mini-Review

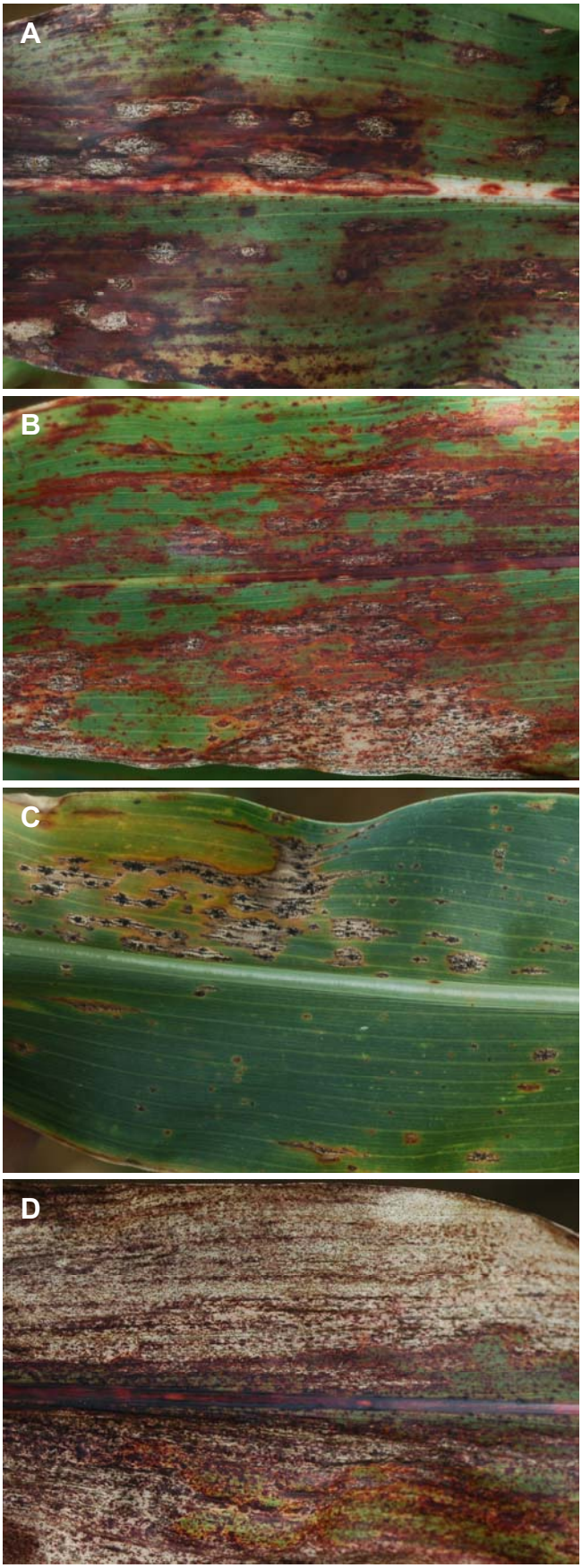

Fig. 1 Examples of the susceptible response from anthracnose inoculation of purple (A), red (B), and tan (C) host plants, and an example of a leaf showing extensive senescence (D) with acervuli present in dead leaf tissue.

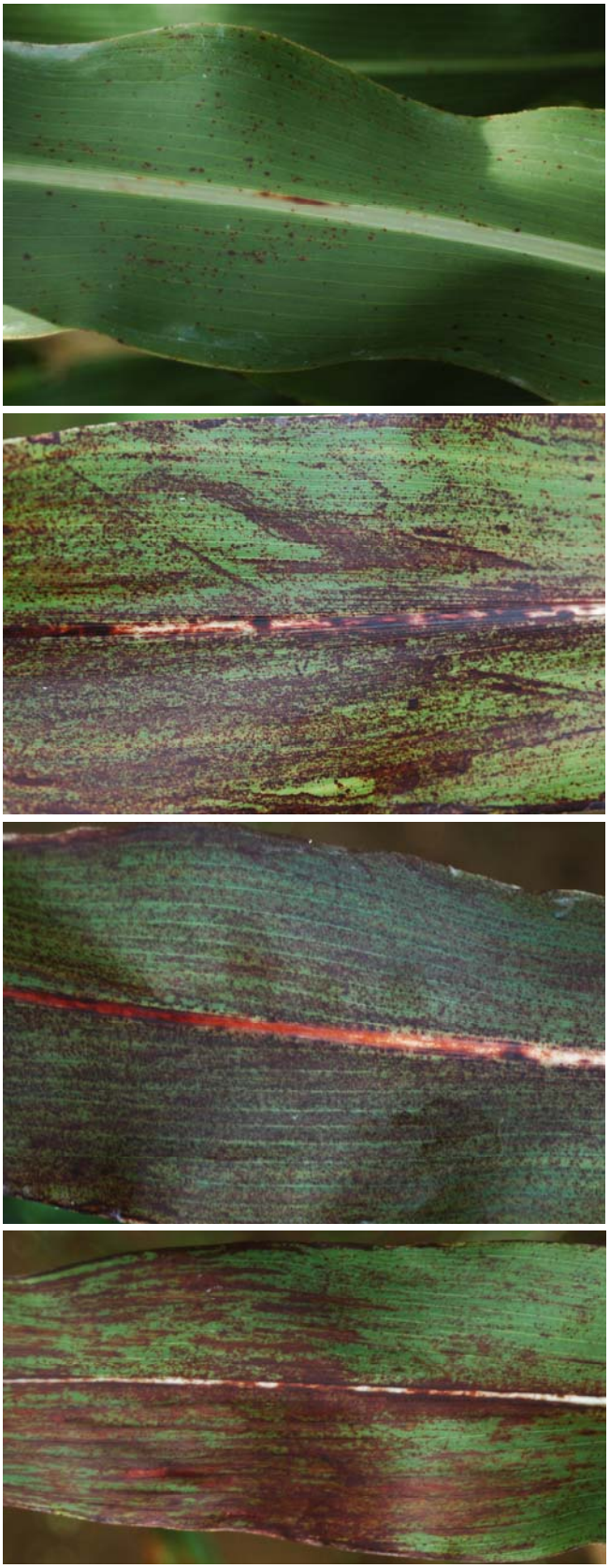

Fig. 2 Examples of the response to anthracnose inoculation for resistant sorghum genotypes ranging from a few red spots to extensive reddening of the leaf lamina.

ted the production of phytoalexins in response to anthracnose infection and have indicated that the response involves a family of flavonoid compounds, including apigeninidin, luteolinidin, apigeninidin acyl ester, 3-deoxyanthocyanidin, and 5-methoxyluteolinidin (Nicholson *et al*. 1988; Hipskind *et al*. 1990; Snyder *et al*. 1991; Lo *et al*. 1999a). For resistant genotypes, the defense response results from a more rapid production and greater accumulation of phytoalexins at the site of infection and the synthesis of phytoalexins

with greater fungitoxicity that prevents further hyphae growth after penetration of host cells and the formation of primary hyphae by the pathogen (Lo *et al*. 1999a). Death of the host cells was also observed at the site of infection (Wharton *et al*. 2001). Senescence of leaf tissue in response to anthracnose inoculation has also been observed in field evaluations of resistant sorghum genotypes. In contrast, infected cells of susceptible genotypes will remain alive until the formation of the biotrophic interaction followed by proliferation of hyphae in cells surrounding the site of infection leading to the development of lesions with acervuli and senescence of host leaf tissue (Wharton *et al*. 2001). Additionally, no restriction of fungal growth was observed due to the production of phytoalexins in susceptible genotypes. However, variation in the colonization of the host plant by the pathogen has been observed for field evaluations (Erpelding and Prom 2006; Erpelding 2007). Chalcone synthase and pathogenesis-related protein (PR-10) transcripts were also shown to accumulate following inoculation, with an earlier induction of these defense-related genes in resistant genotypes (Lo *et al*. 1999a, 1999b). Although these studies have identified components of the defense response, information is lacking on recognition of the pathogen by the host plant, the initial signaling events for expression of resistance, and the role that defense-related proteins play in conferring resistance. For susceptible cultivars, coalescence of lesions can rapidly occur during favorable environmental conditions resulting in leaf senescence, premature defoliation, or plant death. Under field conditions in Puerto Rico, plant death has been observed for highly susceptible genotypes within 30 days after the appearance of visible symptoms.

The disease can be successfully managed through the use of resistant cultivars, fungicide application, or cultural practices. For sustainable agricultural systems, host plant resistance provides the most economical method of stabilizing crop production. Variation within the pathogen population and the advent of new pathotypes has limited the durability of available sources of host plant resistance (Ali and Warren 1987; Cardwell *et al*. 1989; Pande *et al*. 1991; Guthrie *et al*. 1992; Rosewich *et al*. 1998; Marley *et al*. 2001b; Souza-Paccola *et al*. 2003; Valério *et al*. 2005). Thus, additional sources of anthracnose resistance are needed. The availability of multiple sources of resistance will allow pyramiding of resistance genes for more effective disease management. The identification of host resistance requires the development of screening procedures to enhance infection response (Pande *et al*. 1994). Erpelding and Prom (2006) have described an anthracnose inoculation procedure that has been successfully used to identify sources of resistance. This rapid and simple procedure involves culturing the fungus on sorghum seed, with plants inoculated by placing anthracnose colonized seed in the leaf whorl at approximately 30 days after seedling emergence. Germplasm collections provide a valuable resource of disease resistant genes for crop improvement. The objectives of the sorghum research project at the USDA-ARS Tropical Agriculture Research Station, Mayaguez, Puerto Rico are: 1) the evaluation of the USDA-ARS National Plant Germplasm System (NPGS) sorghum collection for resistance to anthracnose; 2) genetic characterization of resistant sources to assess genetic diversity; and 3) the identification of countries or regions associated with genetic diversity for anthracnose resistance to aid in germplasm acquisition to enhance the collection.

Africa is considered the center of origin for sorghum with multiple centers of diversity present in the region (de Wet and Harlan 1971; Harlan 1975). Since anthracnose occurs in Africa (Leslie 2002), genetic diversity for anthracnose resistance may exist in the African sorghum germplasm collections. Approximately 50% of the more than 43,000 sorghum accessions maintained by the NPGS were collected from Africa. However, limited information is available on anthracnose resistance for germplasm in the collection to aid in the selection of resistant sources for sorghum improvement. To identify collections associated with resistance, a disease evaluation was conducted for germplasm from different centers of diversity in Africa (Erpelding and Wang 2007). Sorghum germplasm from 15 African countries was randomly selected and anthracnose resistant accessions were identified from the Benin, Burundi, Ethiopia, Liberia, Malawi, Mali, Nigeria, Sudan, South Africa, and Uganda collections maintained by the NPGS.

Nearly 4,000 sorghum accessions from Sudan are available in the NPGS collection (USDA-ARS National Genetic Resources Program 2007) and germplasm from this country has had a major impact on sorghum improvement in the United States. Thus, 300 accessions with potential breeding value were selected from the Sudan collection and evaluated for anthracnose infection response in Puerto Rico and Texas (Erpelding *et al*. 2005). Approximately 50% of the accessions showed a resistant disease response at both locations. Variation in the anthracnose disease response was observed between Puerto Rico and Texas for 27 accessions indicating genetic diversity for resistance is present in the Sudan collection. In order to provide an estimate of the frequency of anthracnose resistant germplasm in the Sudan collection, a random selection of approximately 5% of the collections was evaluated with approximately 25% of the accessions conferring a resistance response (Erpelding unpublished data). These results would indicate that sorghum germplasm from Sudan is an important source of anthracnose resistance. Passport information is lacking to map the location of individual accessions from the Sudan collection; thus, field evaluation data will be used to select accessions for additional anthracnose evaluations for genetic diversity characterization.

Ethiopia is also an important center of sorghum diversity (Stemler *et al*. 1977) and more than 7,000 accessions are maintained in the NPGS collection from this country (USDA-ARS National Genetic Resources Program 2007). Comprehensive anthracnose evaluations have not been conducted for the Ethiopia sorghum collection. However, approximately 50% of the accessions from a random sample of 42 accessions showed a resistant anthracnose response (Erpelding unpublished data). It is anticipated that anthracnose resistant germplasm would be frequent in the Ethiopia collection as was observed for the Sudan collection. Using available passport information for germplasm selection, additional evaluations will be conducted to estimate the frequency of anthracnose resistance in the collection and to identify resistant germplasm for characterization of genetic diversity.

West Africa is considered a secondary center of sorghum diversity (Harlan 1975), thus 270 accessions from Mali were selected for anthracnose evaluation. Nearly 80% of the accessions showed a resistant response in Puerto Rico and more than 90% of the resistant accessions conferred a resistance response in Texas (Erpelding and Prom 2004). These results indicated that the Mali collection is an important source of anthracnose resistance. Limited passport information was available for these accessions and improved lines were also included in the evaluation, which may have resulted in a higher frequency of resistant accessions as was observed for the Sudan evaluation. Thus, additional disease evaluations were conducted using passport information to select landraces from specific regions of Mali. Mali is composed of eight regions (Gao, Kayes, Kidal, Koulikoro, Mopti, Segou, Sikasso and Tombouctou) and sorghum germplasm was identified from seven regions using latitude, longitude, and passport information. Sorghum germplasm from the Kayes region was evaluated and approximately 43% of the 277 accessions showed a resistant response over multiple growing seasons in Puerto Rico (Erpelding unpublished data). The climatic conditions reported for the Kayes region (Hess *et al*. 2002) also provided an opportunity to compare rainfall with anthracnose resistance. Annual rainfall ranges from less than 400 mm in the north to more than 1,100 mm in the south. The majority of the landraces collected from the wettest regions showed a resistant response

to anthracnose infection in Puerto Rico. In contrast, a high level of anthracnose susceptibility was observed for landraces collected from the dry, northern regions. This ecogeographic association based on rainfall has been observed for sorghum germplasm from the other regions of Mali. Annual rainfall is more than 800 mm for the Sikasso region and nearly all the sorghum landraces from this region were resistant to anthracnose. Additionally, the percentage of infected leaf area was low for the susceptible landraces from this region. Resistant accessions have been selected from the various ecogeographic regions of Mali to evaluate genetic diversity for resistance and to determine if disease selection pressure in the wetter regions contributes to greater genetic diversity for anthracnose resistance.

This ecogeographic association has been successfully used to enhance evaluation of the NPGS sorghum collection to select regions associated with anthracnose resistance. The sorghum collection from Mozambique was evaluated and 12 of the 22 accessions in this collection were resistant to anthracnose (Erpelding and Prom 2006). Preliminary evaluations of the NPGS sorghum collections from Burundi, Gambia, Ghana, Rwanda, Sierra Leone, and Zaire have indicated anthracnose resistant germplasm is also frequent in these collections (Erpelding unpublished data). These African regions are associated with higher annual rainfall and it was expected that anthracnose resistant germplasm would be frequent in these collections. In contrast, regions associated with low annual rainfall, such as Algeria and Somalia, should have a low frequency of anthracnose resistant germplasm. Preliminary anthracnose data from the Algeria and Somalia collections have shown that the majority of the accessions are highly susceptible and for the Algeria collection plant death prior to maturity was frequently observed (Erpelding unpublished data).

The International Crops Research Institute for the Semi-arid Tropics (ICRISAT) maintains the world sorghum collection, which is also a valuable resource for anthracnose resistant germplasm. Nearly 37,000 sorghum accessions are maintained in the collection and approximately 6,800 have been evaluated for anthracnose resistance (ICRISAT 2008). Approximately 1% of the accessions evaluated showed less than 2% leaf damage and would be considered resistant; whereas, nearly 50% of the accessions evaluated would be considered highly susceptible. Geographical mapping information is available for approximately 2,400 accessions evaluated for anthracnose leaf damage and accessions with less 6% leaf damage were collected from Ethiopia, Kenya, Mali, Niger, Nigeria, Sudan, and India. These results are similar to the African countries identified in the evaluation of the NPGS sorghum collection. However, sorghum accessions from India that have been evaluated in Puerto Rico have generally showed a highly susceptible response. Anthracnose evaluations that have been reported from countries in Africa have focused more on evaluation of sorghum germplasm from the ICRISAT collection or breeding lines (Néya and Le Normand 1998; Marley *et al*. 2001a; Hess *et al*. 2002) and resistant sources have been successfully identified for sorghum improvement or for use as inbred cultivars. Marley *et al*. (2001a) evaluated 120 sorghum accessions at two locations in Nigeria and identified 114 accessions resistant to anthracnose. Resistant accessions were identified from Botswana, Cameroon, Ethiopia, Mali, Niger, Nigeria, Rwanda, South Africa, Sudan, Tanzania, and Zimbabwe. The majority of the ICRISAT sorghum accessions are also maintained in the NPGS collection and data from these evaluations can be compared to data collected in Puerto Rico to assess genetic diversity for resistance. Nearly 2,000 accessions from the NPGS sorghum collection have also been evaluated in Brazil and approximately 40% showed a resistant response (USDA-ARS National Genetic Resources Program 2007). As expected, anthracnose resistant accessions were identified for the germplasm collections from Botswana, Burkina Faso, Cameroon, Ethiopia, Kenya, Mali, Nigeria, Uganda, South Africa, Sudan, Tanzania, Togo, and Zambia. However, some accessions from Somalia showed a low percentage of leaf infection in Brazil as compared to the highly susceptible response observed in Puerto Rico. These disease evaluations have focused on identify anthracnose resistant germplasm for sorghum improvement and disease response was generally evaluated from natural infection. Since resistant accessions are available, anthracnose research has focused more on evaluation of the pathogen population (Pande *et al*. 1991; Rosewich *et al*. 1998; Casela *et al*. 2001; Marley *et al*. 2001b; Valério *et al*. 2004, 2005).

For sorghum, a limited number of evaluations have been conducted to determine the genetic control of host plant resistance to anthracnose. Results of these evaluations have indicated that foliar anthracnose resistance is a simply inherited trait conferred by dominant or recessive genes. Le Beau and Coleman (1950) reported that a dominate gene conferred resistant to anthracnose in two sorghum cultivars. In addition, a dominant gene for foliar anthracnose resistance linked to a dominant gene for anthracnose stalk rot resistance was identified by Coleman and Stokes (1954). A recessive gene conferring anthracnose resistance was identified by Boora *et al*. (1998) and RAPD (random amplified polymorphic DNA) markers linked to the resistant allele were identified. Singh *et al*. (2006) also identified a RAPD marker linked to a recessive gene conferring anthracnose resistance. The evaluation of 11 anthracnose resistant germplasm lines identified five distinct sources of anthracnose resistance with a dominant gene conferring resistance for three sources and resistance for two sources conferred by a recessive gene (Mehta *et al*. 2005). Their results indicated that genetic variation exists within and between germplasm collections from individual countries for anthracnose resistance. Also, a genetic evaluation of 41 germplasm lines from the Mali collection indicated anthracnose resistance was conferred by dominant and recessive genes (Erpelding and Prom 2004). Seventy-two anthracnose resistant germplasm lines were selected from the Kayes region of Mali and genetic evaluation indicated that resistance was more frequently controlled by dominant genes with a resistant response observed in the F_1 hybrid for 45 lines (Erpelding unpublished data). Dominant genes conferring anthracnose resistance have also been identified from germplasm lines from the Mozambique, Sudan, and Ethiopia collections (Erpelding unpublished data). The germplasm line, SC748 (PI 533991, USDA-ARS National Genetic Resources Program 2007), was developed from a Sudanese sorghum landrace and is an important source of resistance, since this line is highly resistant to numerous anthracnose pathotypes worldwide (Mehta *et al*. 2005). This line also shows a resistant response in Puerto Rico and is used as an anthracnose resistant control in germplasm evaluations (Erpelding and Prom 2004; Erpelding *et al*. 2005; Erpelding and Prom 2006; Erpelding and Wang 2007). Resistance for SC748 is conferred by a single dominant gene (Mehta *et al*. 2005) and Lo *et al*. (1999a) indicated resistance was associated with the rapid production of 3-deoxyanthocyanidin phytoalexins. Dominant genes conferring anthracnose resistance are more desirable for the hybrid seed industry, since the resistant source can be introgressed into one parental line for expression in the hybrid.

No genetic evaluation has reported the presence of complementary genes for resistance where more than one gene was responsible for eliciting a resistant anthracnose response. The occurrence of single genes for anthracnose resistance should benefit the pyramiding of resistant genes for sorghum improvement. Additionally, allelic variation at a resistant locus has not been reported. However, tissue-specific infection is considered to involve multiple resistant genes as suggested by Coleman and Stokes (1954). Erpelding (2007) also observed genetic variation for tissue specific infection. The disease evaluation of sorghum collections from Africa and the identification of sources of host plant resistance will provide a foundation to enhance genetic characterization of resistance and to determine the genetic diversity for anthracnose resistance. Additionally, an objec-

tive of our research is the development of recombinant inbred line (RIL) populations for genetic characterization with multiple anthracnose pathotypes. These populations will aid in the genetic mapping of disease resistant loci and the identification of linked molecular markers for marker-assisted breeding. The genetic characterization of host plant resistance for germplasm accessions will also aid in the development of a set of sorghum lines to differentiate the anthracnose pathotypes and determine if a gene-for-gene interaction exists for sorghum anthracnose.

ACKNOWLEDGEMENTS

The author is grateful to L.K. Prom, USDA-ARS, College Station, Texas for assistance in establishing the anthracnose research program in Puerto Rico. The author acknowledges R. Goenaga and T. Porch, USDA-ARS, Mayaguez, Puerto Rico and M.L. Wang, USDA-ARS, Griffin, Georgia for helpful suggestion on the manuscript.

REFERENCES

Ali MEK, Warren HL (1987) Physiological races of *Colletotrichum graminicola* on sorghum. *Plant Disease* **71**, 402-404

Boora KS, Frederiksen R, Magill C (1998) DNA-based markers for a recessive gene conferring anthracnose resistance in sorghum. *Crop Science* **38**, 1708-1709

Cardwell KF, Hepperly PR, Frederiksen RA (1989) Pathotypes of *Colletotrichum graminicola* and seed transmission of sorghum anthracnose. *Plant Disease* **73**, 255-257

Casela CR, Ferreira AS, Santos FG (2001) Differences in competitive ability among races of *Colletotrichum graminicola* in mixtures. *Fitopatologia Brasileira* **26**, 217-219

Coleman OH, Stokes IE (1954) The inheritance of resistance to stalk red rot in sorghum. *Agronomy Journal* **46**, 61-63

Crouch JA, Clarke BB, Hillman BI (2006) Unraveling evolutionary relationships among the divergent lineages of *Colletotrichum* causing anthracnose disease in turfgrass and corn. *Phytopathology* **96**, 46-50

de Wet JMJ, Harlan JR (1971) The origin and domestication of *Sorghum bicolor*. *Economic Botany* **25**, 128-135

Dogget, H (1988) *Sorghum* (2nd Edn), John Wiley & Sons, NY, 512 pp

Erpelding JE (2007) Inheritance of anthracnose resistance for the sorghum cultivar Redlan. *Plant Pathology Journal* **6**, 187-190

Erpelding JE, Prom LK (2004) Evaluation of Malian sorghum germplasm for resistance against anthracnose. *Plant Pathology Journal* **3**, 65-71

Erpelding JE, Prom LK (2006) Variation for anthracnose resistance within the sorghum germplasm collection from Mozambique, Africa. *Plant Pathology Journal* **5**, 28-34

Erpelding JE, Prom LK, Rooney WL (2005) Variation in anthracnose resistance within the Sudanese sorghum germplasm collection. *Plant Genetic Resources Newsletter* **141**, 11-14

Erpelding JE, Wang ML (2007) Response to anthracnose infection for a random selection of sorghum germplasm. *Plant Pathology Journal* **6**, 127-133

Ferreira AS, Warren HL (1982) Resistance of sorghum to *Colletotrichum graminicola*. *Plant Disease* **66**, 773-775

Freeman S, Pham M, Rodriguez RJ (1993) Molecular genotyping of *Colletotrichum* species based on arbitrarily primed PCR, A+T-rich DNA, and nuclear DNA analyses. *Experimental Mycology* **17**, 309-322

Guthrie PAI, Magill CW, Frederiksen RA, Odvody GN (1992) Random amplified polymorphic DNA markers: A system for identifying and differentiating isolates of *Colletotrichum graminicola*. *Phytopathology* **82**, 832-835

Harlan JR (1975) Geographic patterns of variation in some cultivated plants. *The Journal of Heredity* **66**, 184-191

Harris HB, Johnson BJ, Dobson Jr. JW, Luttrell ES (1964) Evaluation of anthracnose on grain sorghum. *Crop Science* **4**, 460-462

Hess DE, Bandyopadhyay R, Sissoko I (2002) Pattern analysis of sorghum genotype x environment interaction for leaf, panicle, and grain anthracnose in Mali. *Plant Disease* **86**, 1374-1382

Hipskind JD, Hanau R, Leite B, Nicholson RL (1990) Phytoalexin accumulation in sorghum: Identification of an apigeninidin acyl ester. *Physiological and Molecular Plant Pathology* **36**, 381-396

ICRISAT (2008) International Crops Research Institute for the Semi-arid Tropics, Andhra Pradesh, India. Available online: http://www.icrisat.org/sorghum

Le Beau FJ, Coleman OH (1950) The inheritance of resistance in sorghum to leaf anthracnose. *Agronomy Journal* **42**, 33-34

Leslie JF (Ed) (2002) *Sorghum and millets diseases*, Iowa State Press, Ames, IA, USA, 504 pp

Lo SC, de Verdier K, Nicholson RL (1999a) Accumulation of 3-deoxyanthocyanidin phytoalexins and resistance to *Colletotrichum sublineolum* in sorghum. *Physiological and Molecular Plant Pathology* **55**, 263-273

Lo SC, Hipskind JD, Nicholson RL (1999b) cDNA cloning of a sorghum pathogenesis-related protein (PR-10) and differential expression of defense-related genes following inoculation with *Cochliobolus heterostrophus* or *Colletotrichum sublineolum*. *Molecular Plant-Microbe Interactions* **12**, 479-489

Marley PS, Bandyopadhyay R, Tabo R, Ajayi O (2001a) Sorghum germplasm with multiple resistance to two major diseases in the Nigerian savanna. *Tropical Science* **41**, 44-49

Marley PS, Thakur RP, Ajayi O (2001b) Variation among foliar isolates of *Colletotrichum sublineolum* of sorghum in Nigeria. *Field Crops Research* **69**, 133-142

Mehta PJ, Wiltse CC, Rooney WL, Collins SD, Frederiksen RA, Hess DE, Chisi M, TeBeest DO (2005) Classification and inheritance of genetic resistance to anthracnose in sorghum. *Field Crops Research* **93**, 1-9

Néya A, Le Normand M (1998) Responses of sorghum genotypes to leaf anthracnose (*Colletotrichum graminicola*) under field conditions in Burkina Faso. *Crop Protection* **17**, 47-53

Ngugi HK, King SB, Abayo GO, Reddy YVR (2002) Prevalence, incidence, and severity of sorghum diseases in western Kenya. *Plant Disease* **86**, 65-70

Nicholson RL, Jamil FF, Snyder BA, Lue WL, Hipskind J (1988) Phytoalexin synthesis in juvenile sorghum leaf. *Physiological and Molecular Plant Pathology* **33**, 271-278

Nicholson RL, Kollipara SS, Vincent JR, Lyons PC, Cadena-Gomez G (1987) Phytoalexin synthesis by the sorghum mesocotyl in response to infection by pathogenic and nonpathogenic fungi. *Proceedings of the National Academy of Sciences USA* **84**, 5520-5524

Pande S, Mughogho LK, Bandyopadhyay R, Karunakar RI (1991) Variation in pathogenicity and cultural characteristics of sorghum isolates of *Colletotrichum graminicola* in India. *Plant Disease* **75**, 778-783

Pande S, Thakur RP, Karunakar RI, Bandyopadhyay R, Reddy BVS (1994) Development of screening methods and identification of stable resistance to anthracnose in sorghum. *Field Crops Research* **38**, 157-166

Rosewich UL, Pettway RE, McDonald BA, Duncan RR, Frederiksen RA (1998) Genetic structure and temporal dynamics of a *Colletotrichum graminicola* population in a sorghum disease nursery. *Phytopathology* **88**, 1087-1093

Sherriff C, Whelan MJ, Arnold GM, Bailey JA (1995) rDNA sequence analysis confirms the distinction between *Colletotrichum graminicola* and *C. sublineolum*. *Mycological Research* **99**, 475-478

Singh M, Chaudhary K, Boora KS (2006) RAPD-based SCAR marker SCA 12 linked to recessive gene conferring resistance to anthracnose is sorghum [*Sorghum bicolor* (L.) Moench]. *Theoretical and Applied Genetics* **114**, 187-192

Snyder BA, Leite B, Hipskind J, Butler LG, Nicholson RL (1991) Accumulation of sorghum phytoalexins induced by *Colletotrichum graminicola* at the infection site. *Physiological and Molecular Plant Pathology* **39**, 463-470

Snyder BA, Nicholson RL (1990) Synthesis of phytoalexins in sorghum as a site-specific response to fungal ingress. *Science* **248**, 1637-1639

Souza-Paccola EA, Fávaro LCL, Casela CR, Paccola-Meirelles LD (2003) Genetic recombination in *Colletotrichum sublineolum*. *Journal of Phytopathology* **151**, 329-334

Stemler ABL, Harlan JR, de Wet JMJ (1977) The sorghums of Ethiopia. *Economic Botany* **31**, 446-460

Sutton BC (1968) The appressoria of *Colletotrichum graminicola* and *C. falcatum*. *Canadian Journal of Botany* **46**, 873-876

Thakur RP, Mathur K (2000) Anthracnose. In: Frederiksen RA, Odvody GN (Eds) *Compendium of Sorghum Diseases*, The American Phytopathological Society, St. Paul, MN, USA, pp 10-12

Thomas MD, Sissoko I, Sacko M (1996) Development of leaf anthracnose and its effect on yield and grain weight of sorghum in West Africa. *Plant Disease* **80**, 151-153

USDA-ARS National Genetic Resources Program (2007) Germplasm Resources Information Network (GRIN). Online database. National Germplasm Resources Laboratory, Beltsville, MD, USA. Available online: http://www.ars-grin.gov/

Vaillancourt LJ, Hanau RM (1992) Genetic and morphological comparisons of *Glomerella* (*Collectotrichum*) isolates from maize and from sorghum. *Experimental Mycology* **16**, 219-229

Valério HM, Casela CR, Resende MA, Santos FG (2004) Variability of the anthracnose fungus *Colletotrichum graminicola* in sorghum genotype mixtures. *Fitopatologia Brasileira* **29**, 567-569

Valério HM, Resende MA, Weikert-Oliveira RCB, Casela CR (2005) Virulence and molecular diversity in *Collectotrichum graminicola* from Brazil. *Mycopathologia* **159**, 449-459

Wharton PS, Julian AM (1996) A cytological study of compatible and incompatible interactions between *Sorghum bicolor* and *Colletotrichum sublineolum*. *New Phytologist* **134**, 25-34

Wharton PS, Julian AM, O'Connell RJ (2001) Ultrastructure of the infection of *Sorghum bicolor* by *Colletotrichum sublineolum*. *Phytopathology* **91**, 149-158