

# Plant Pathology Journal

ISSN 1812-5387





## Variation for Anthracnose Resistance within the Sorghum Germplasm Collection from Mozambique, Africa

<sup>1</sup>John E. Erpelding and <sup>2</sup>Louis K. Prom <sup>1</sup>USDA Agricultural Research Service, Tropical Agriculture Research Station, 2200 Pedro Albizu Campos Ave., Suite 201, Mayaguez, Puerto Rico 00680-5470 USA <sup>2</sup>USDA Agricultural Research Service, Southern Plains Agriculture Research Center, 2765 F and B Road, College Station, Texas 77845 USA

Abstract: Plant germplasm collections have been established to preserve genetic variation for utilization in crop improvement programs. Breeding for host plant resistant provides an economical approach to controlling diseases and stabilizing crop production but pathogen populations are variable and evolving; therefore, the identification of new sources of resistance are essential. The Mozambique sorghum collection maintained by the US National Plant Germplasm System in Griffin, Georgia was inoculated with Collectrichum sublineolum and evaluated for anthracnose resistance in 2004 during the dry and wet growing seasons in Puerto Rico. Twelve of the 22 sorghum accessions showed a resistant response in both seasons. Four resistant accessions were evaluated in an anthracnose disease nursery in Georgia and found to be resistant, suggesting possible host plant resistance to different pathotypes of the disease. A susceptible disease response was observed for four accessions during both seasons. Six accessions varied in disease response within and between experiments suggesting environmental conditions influenced infection response. The anthracnose resistant germplasm identified from the Mozambique germplasm collection could be a valuable source of disease resistance for sorghum improvement programs.

Key words: Mozambique, Sorghum bicolor, anthracnose, Colletotrichum sublineolum, germplasm

### INTRODUCTION

Sorghum anthracnose is caused by the pathogen *Colletotrichum sublineolum* P. Henn., Kabát and Bubák and based on DNA analysis is considered a separate species from *C. graminicola* (Ces.) G.W. Wilson<sup>[1,2]</sup>. The pathogen is specific to *Sorghum* species and occurs worldwide, but is more prevalent in tropical and subtropical regions were hot and humid environmental conditions prevail<sup>[3,4]</sup>. The disease was first reported in Togo, West Africa in 1902<sup>[4]</sup> and occurs in many African nations<sup>[5-8]</sup> including Mozambique. In the United States, the disease was first observed in Texas in 1912<sup>[9]</sup>. Anthracnose is one of the most destructive diseases of sorghum resulting in yield losses of more than 50%<sup>[9,10]</sup>.

The pathogen is capable of infecting all above ground tissues of the sorghum plant<sup>[4]</sup>. The foliar disease is the most widely distributed, can occur at any stage of plant development and has been referred to as red leaf blight due to the accumulation of phytoalexins at the site of infection<sup>[4,11,12]</sup>. Symptoms of foliar anthracnose

infection are highly variable depending on the host plant, pathogen and environment<sup>[4,6]</sup>. Foliar disease symptoms general appear 40 days after seedling emergence. Characteristic disease symptoms on susceptible cultivars include circular to elliptical spots or elongated lesions and as the disease progresses, lesions coalesce covering most of the leaf tissue. As the fungus sporulates, fruiting bodies (acervuli) appear as black spots in the center of the lesions. Under favorable environmental conditions, coalescence of lesions will occur on susceptible cultivars resulting in leaf senescence and premature defoliation. Severe foliar infection can retard plant growth or result in plant death prior to maturity thus reducing grain development. Ali[13] reported grain yield losses due to foliar infection of 30% for a susceptible cultivar when inoculated with a virulent isolate of the pathogen. Grain yield losses from 41 to 67% for a susceptible cultivar from natural infection of foliar tissue were reported to occur in Mali, West Africa<sup>[14]</sup>. Grain yield losses from foliar infection are generally associated with a reduction in seed weight[13].

Corresponding Author: Dr. John E. Erpelding, USDA Agricultural Research Service, Tropical Agriculture Research Station, 2200 Pedro Albizu Campos Ave., Suite 201, Mayaguez, Puerto Rico 00680-5470 USA

The pathogen can also infect the stalk and has been referred to as red stalk rot<sup>[15]</sup>. Anthracnose stalk infection was first reported in 1943 and is considered an independent disease response from foliar infection, but foliar infection can contribute to the development of stalk rot<sup>[9,15,16]</sup>. Coleman and Stokes<sup>[15]</sup> identified a single dominant gene that conditioned resistance to anthracnose stalk rot, which was linked to a gene conditioning resistance to foliar infection. Yield losses in grain sorghum from anthracnose stalk rot are generally associated with lodging in susceptible cultivars. Yield losses as high as 100% have been reported when severe lodging prevents the mechanical harvest of the crop<sup>[9]</sup>. In addition, sclerotia that develop in the stalk can survive in plant debris and serve as a source of primary inoculum<sup>[17]</sup>.

Anthracnose infection in the peduncle, inflorescence and grain is referred to as panicle anthracnose and may be independent from foliar and stalk infection<sup>[4]</sup>. Panicle infection was first observed in Georgia<sup>[9]</sup>. Yield losses associated with panicle infection can be as high as 50%<sup>[4]</sup>. Infection of the peduncle will result in rotting of the stalk interior contributing to panicle breakage. Anthracnose infection of the inflorescence can result in death of some or all florets giving the appearance of sterility. Disease symptoms for inflorescence infection appear as lesions with acervuli on the glumes. Infection of the grain will appear as black streaking and anthracnose is one of many fungal species responsible for grain mold. Contaminated grain from panicle infection can contribute to the spread of the disease and possible introduction of novel pathotypes to new regions<sup>[3]</sup>.

The most economical method of controlling the disease is through the use of resistant cultivars. The development and utilization of resistant cultivars is complicated by variation within the pathogen and the occurrence of new virulent pathotypes. Extensive pathotype variation has been reported to occur within and between regions for the pathogen<sup>[3,18-20]</sup>. A loss of host plant resistance has also occurred in regions were disease pressure is high<sup>[21]</sup>. The pathogen is highly variable for many traits including cultural and spore morphology, symptom type, sporulation and growth rate, aggressiveness and virulence<sup>[6,10,19]</sup>. pathogenicity. Pathogen variability within and between regions has also been observed from molecular genetic analysis [21,22]. The perfect stage has not been confirmed the pathogen; therefore, asexual genetic recombination through parasexuality is considered the driving force in the development of new virulent pathotypes<sup>[4,21,23]</sup>.

Little information is known about the genetics of host plant resistance. Several inheritance studies have indicated that dominant and recessive genes condition resistance in the host plant [15,24,25], but no information is available on allelic variation for resistance or genetic diversity for resistance within the sorghum germplasm collection. Most sources of resistance are considered to harbor multiple resistance genes and tissue specific infection may be controlled by different resistance genes [4].

Additional sources of anthracnose resistance are needed for the development of resistant cultivars. Climatic conditions favorable to pathogen may enhance the development of anthracnose resistance in the host plant. Sorghum germplasm collections from these regions with a favorable environment for anthracnose should be evaluated to identify sources of host plant resistance. Therefore, the sorghum collection from Mozambique was evaluated for disease response to anthracnose to determine the variation in infection response, identify anthracnose resistant accessions, determine stability of resistance over environments, determine possible associations between resistance and phenotypic traits and identify resistant accessions for genetic evaluation.

#### MATERIALS AND METHODS

The Mozambique sorghum collection maintained by the US National Plant Germplasm System consists of 22 accessions. The collection was established in 1981 and donated to the US National Plant Germplasm System. Seed samples of the Mozambique germplasm collection were provided by the USDA-ARS Plant Genetic Resources Conservation Unit in Griffin, Georgia. The response to sorghum anthracnose infection was evaluated during the dry and wet growing seasons in 2004 at the USDA-ARS Tropical Agriculture Research Station in Isabela, Puerto Rico. The weather data for the two growing seasons is presented in Table 1. The anthracnose evaluation during the dry season was planted 22 January 2004 with the wet season evaluation planted 2 August 2004. The collection was planted in a randomized complete block design with three replications. The 22 accessions were planted in single rows 1.8 m in length with 0.9 m row spacing. The experiment was surrounded by border rows of anthracnose susceptible genotypes. Fertilizer was applied at a rate of 560 kg ha<sup>-1</sup> (15-5-10 NPK) at planting. A second application of fertilizer was performed approximately 30 days after planting for the anthracnose evaluation conducted in the dry season. Lorsban 15 G (O,O-diethyl O-(3, 5, 6-trichloro-2-pyridinyl) phosphorothioate) granular insecticide AgroSciences, Indianapolis, IN) was applied at a rate of 8 kg ha<sup>-1</sup> at planting for fire ant control. Supplemental irrigation was applied after planting for stand

Table 1: Total rainfall, number of days with rain, temperature and relative humidity during the dry and wet growing seasons

Weather variable <sup>a</sup>	Dry season	Wet season	
Total rainfall (mm)	166.1	376.7	
Number of days with rain	30	33	
Average temperature (°C)	24.2	25.4	
Minimum temperature (°C)	16.6	20.2	
Maximum temperature (°C)	29.6	32.1	
Average relative humidity (%)	80.1	82.6	
Minimum relative humidity (%)	40.1	45.9	
Maximum relative humidity (%)	97.7	96.0	

<sup>a</sup>Weather data was summarized after disease inoculation until the final anthracnose evaluation was conducted

establishment and on a weekly basis as necessary. No irrigation was applied after inoculation during the dry season and a single application was applied after inoculation during the wet season. Mechanical tillage and hand hoeing were used to control weeds. On 15 September 2004, a tropical storm passed over the island producing high winds and heavy rainfall. Damage was minor with some lodging, but the plants recovered prior to the evaluation conducted 33 days after inoculation. Two controls samples were included in the evaluation. The sorghum conversion line, SC748-6, was used as a resistant control. The line has been evaluated for several years at the study site in Puerto Rico and a stable resistance response has been observed. In addition, the line has been evaluated at several locations within the continental US and a resistant reaction to various anthracnose pathotypes has been observed. The germplasm line PI 609251 was used as a susceptible control. PI 609251 is a highly susceptible genotype selected from the sorghum germplasm collection from Mali, West Africa<sup>[26]</sup>.

Inoculations were conducted using anthracnose colonized sorghum seed. Anthracnose cultures were established from infected leaf tissue obtained from susceptible sorghum genotypes at the Isabela, Puerto Rico research location. The anthracnose pathotypes present at the research site are unknown; therefore, infected leaf tissue was randomly collected from several susceptible sorghum genotypes to represent the pathogen population at the research station. Infected leaf samples were collected prior to establishment of the experiments from naturally infected and inoculated sorghum plants identified from other evaluations. Anthracnose infected leaf tissue used in the first experiment was stored and used in the second evaluation, along with fresh infected leaf tissue. Infected leaf tissue was surface sterilized, transferred to half strength potato-dextrose agar (1/2 PDA) media and incubated at room temperature in the dark for 3-5 days. Small portions of the developing anthracnose cultures were excised and transferred to fresh 1/2 PDA media followed by incubation for 7 days at room temperature in the dark prior to seed

colonization. Sorghum seed was soaked overnight in tap water, placed in glass jars and autoclaved prior to inoculation with the anthracnose cultures. The outside edges of the 7 day old anthracnose cultures were excised and divided equally between the glass jars of sorghum seed to maintain the pathotypes present in the pathogen population. The anthracnose inoculated seed was incubated at room temperature in the dark for approximately 5 days for complete colonization. Inoculation with the anthracnose pathogen was conducted approximately 30 days after planting by placing colonized seeds in the leaf whorl. Approximately 10 anthracnose colonized seeds were placed in the leaf whorl of each plant and greater than 80% of the plants in the row were inoculated.

The response to anthracnose was determined 39, 53 and 73 days after inoculation for the evaluation conducted in the dry season. Disease evaluations are generally conducted approximately 30, 40 and 60 days after inoculation[26], but seven days of windy weather conditions after inoculation during the dry season resulted in no dew formation that slowed the development of the disease. During the wet season, disease response was assessed 12, 33, 45 and 60 days after inoculation. The response to anthracnose infection was evaluated using a 1-5 rating scale based on infection response observed on inoculated leaves and on the progression of the disease on non-inoculated leaves<sup>[26]</sup>. A rating of 1 = no symptoms or chlorotic flecks on leaves emerging from the inoculated whorl; 2 = hypersensitive reaction (reddening or red spots) on inoculated leaves with no acervuli formation; 3 = chlorotic lesions on inoculated leaves with acervuli in the center; 4 = necrotic lesions with acervuli on infected leaves with lesion elongation and infection spreading to non-inoculated leaf tissue and 5 = coalescence of lesions on inoculated leaves with abundant acervuli resulting in leaf death and most leaves infected including the flag leaf. Highly resistant accessions were rated as 1, resistant accessions as 2, moderately susceptible accessions as 3, susceptible accessions as 4 and highly susceptible accessions as 5. The rating scale was established to evaluate the qualitative response to anthracnose infection. Additionally, the percentage of infected leaf area was estimated during the final evaluation to assess potential quantitative responses to anthracnose infection.

Statistical analysis of the data was conducted using the disease response from the final rating. At this stage, plants are approaching maturity and further disease progression is associated with colonization of senescencing leaf tissue. Since photoperiod sensitivity delayed flowering and plants had not reached maturity for the final reading during the wet season, the last two ratings for this evaluation were compared to determine if further disease progression would result in a change in the disease rating. The Statistix software package (Analytical Software, Tallahassee, FL) was used to conduct the analysis of variance for the data. Statistical analysis of heterogeneous accessions that showed within-row variation for disease response was conducted using the most prevalent disease phenotype occurring within the row.

#### RESULTS

Table 2 shows the response to anthracnose infection and highly significant differences (p = 0.01) were detected between accessions (data not shown). The disease response was consistent within and between experiments for 16 accessions, with 12 accessions conditioning a resistant reaction and four accessions susceptible to the disease. Variation in anthracnose infection response within and between experiments was observed for six accessions. Within-accession variation was a factor contributing to the variable anthracnose disease response observed for three of the six accessions

during the dry growing season. Five accessions that showed a variable response to anthracnose infection during the dry season were rated as resistant during the wet season, which resulted in 17 accessions conditioning a resistant response during the wet season compared to 12 in the dry season. Only one accession showed a variable response to anthracnose infection between replications during the wet season. The anthracnose infection response for the control samples was consistent within and between experiments.

Anthracnose disease severity was higher during the dry growing season with an average of 32% infected leaf area for the four susceptible accessions compared to 6% observed during the wet growing season. Anthracnose infected leaf area ranged from 5 to 50% for the susceptible accessions during the dry growing season (data not shown). Disease severity during the wet season ranged from 1 to 20% of the leaf area infected for the susceptible accessions. The anthracnose infection response during the wet season for the four susceptible accessions was mainly confined to inoculated leaves with limited spread to non-inoculated leaves. Infection was generally associated with the leaf margins and midrib, while the

Table 2: Anthracnose disease response for 22 sorghum accessions from Mozambique, Africa and two control samples inoculated with Colletotrichum sublineolum and evaluated under field conditions in Puerto Rico during the dry and wet growing seasons

Accession <sup>a</sup>	Dry <sup>b</sup>	% Dry	Anthesis <sup>d</sup>	Wet	%Wet	Anthesis	Race	Color
PI 481828	2		59	2		75	Guinea	P
PI 481829	2\4*		59	2		75	Guinea	P\T
PI 481830	2		61	2		78	Guinea-Kafir	T
PI 481831	4\4\5	45	55	4	4	70	Guinea	T
PI 481832	2		55	2		90	Guinea	T\P
PI 481833	2\4\4	15	67	2\2\4	1	82	Guinea	T
PI 481834	2		87	2		107	Guinea	P
PI 481835	2		55	2		69	Guinea	T\P
PI 481836	2		59	2		70	Guinea	P
PI 481837	2\4*		59	2		70	Guinea	T\P
PI 481838	2		61	2		81	Guinea	P
PI 481840	4	25	60	4	4	91	Guinea-Kafir	P
PI 481841	4\4\5	38	61	4	5	91	Guinea-Kafir	P
PI 481842	4	20	70	4	12	99	Guinea	P
PI 481843	2		65	2		99	Guinea	P
PI 481844	2		63	2		82	Guinea	T\P
PI 481845	2\4*		63	2		99	Guinea	T\P
PI 481846	2\4\4	15	65	2		99	Guinea	P\T
PI 481847	2\2\4	5	65	2		99	Guinea	T\P
PI 481848	2		65	2		99	Guinea	P\T
PI 481849	2		55	2		99	Guinea	P
PI 481850	2		60	2		99	Guinea	P
SC748-6	2		62	2		68	Caudatum	P
PI 609251	5	95	56	5	60	63	Caudatum	R

Plant introduction numbers for the Mozambique sorghum accessions. SC748-6 = anthracnose resistant control. PI 609251 = anthracnose susceptible control. bRows with a single value indicate no variation for disease response within an experiment. An accession with more than one disease response value indicates variation within an experiment (data presented for the three replications) or within-accession variation (\*\*). Rating scale: 1 = no symptoms or chlorotic flecks on leaves emerging from the inoculated whorl; 2 = hypersensitive reaction (reddening or red spots) on inoculated leaves with no accervuli formation; 3 = chlorotic lesions on inoculated leaves with accervuli in the center; 4 = necrotic lesions with accervuli and infection spreading to non-inoculated leaf tissue and 5 = coalescence of lesions with abundant accrvuli resulting in leaf death and most leaves infected including the flag leaf.

Percentage of leaf area infection averaged over replications used to evaluate anthracnose disease severity for susceptible accessions.

<sup>&</sup>lt;sup>d</sup>Flowering date determined as days to 50% anthesis.

<sup>°</sup>Sorghum race classification[28].

Plant color determined based on pigment production associated with injured tissue. The predominant plant color is listed first for the accessions heterogeneous for plant color. P = purple. T = tan. R = red

senescence of lower leaves reduced the spread of the disease. Disease severity was also greater during the dry season for the susceptible control with nearly 100% infected leaf area compared to 60% during the wet growing season.

#### DISCUSSION

The Mozambique sorghum collection is a valuable source of anthracnose resistant germplasm with over 50% of the 22 accessions conditioning resistance to the disease. Twelve accessions conditioned a resistant response during the wet and dry growing seasons in Puerto Rico suggesting stable sources of resistance under differing environmental conditions. Four accessions from the collection were also evaluated in an anthracnose disease nursery in Georgia and found to be resistant to anthracnose infection, which would indicate that the germplasm may provide resistance to multiple pathotypes of the disease. The accessions included in the Georgia disease evaluation were selected prior to initial disease assessment during the dry season in Puerto Rico. Three of the four selected accessions conditioned a resistant response that was consistent during the dry and wet growing seasons in Puerto Rico. One accession produced a variable infection response during the dry growing season suggesting that the accession may be genetically heterogeneous. No within-accession variation was observed for this accession in the Georgia anthracnose evaluation. Previous studies have indicated pathotypic variation between locations[3,18], but at present no information is available on the pathotypes present within and between locations. Anthracnose infected leaf tissue has been collected from the locations to determine pathotype variation between locations. In addition, preliminary data from 300 sorghum accessions evaluated in Georgia and Puerto Rico would indicate pathotypic variation for the anthracnose pathogen between locations (data not shown). Ali and Warren<sup>[18]</sup> and Cardwell<sup>[3]</sup> identified two pathotypes within the anthracnose population in Puerto Rico. Guthrie<sup>[22]</sup> concluded that the Puerto Rican anthracnose population was the most homogeneous based on molecular marker analysis. Presently, no information is available on the pathotype diversity in Puerto Rico. It is possible that variation in the pathogen population may have contributed to the variation in disease response observed within and between experiments.

Climatic conditions are known to influence anthracnose infection response<sup>[4,7,27]</sup>. Genotype by environment interactions may have contributed to the variation in infection response observed within and

between experiments for six accessions. The difference in disease severity, as measured by the percentage of infected leaf area, observed between the wet and dry growing seasons for the susceptible accessions could also have resulted from variation in climatic conditions. Although environmental conditions may have enhanced disease expression in the dry season, the initial development of the disease may have been delayed by dry, windy conditions after inoculation. A susceptible response was only observed for one accession during the dry season at 39 days after inoculation. In comparison, four accessions showed susceptible responses at 33 days after inoculation during the wet season. All four of these accessions were rated as susceptible for the final evaluation in both experiments. The accumulation of phytoalexins associated with the hypersensitive reaction on inoculated leaves was clearly visible during the wet season at 12 days after inoculation, but limited anthracnose lesion development was observed. Neya and Le Normand<sup>[5]</sup> indicated that wetter climatic conditions enhanced disease severity for an evaluation conducted at three locations in Burkina Faso, West Africa. Hess<sup>[7]</sup> indicated that rainfall was a major factor influencing anthracnose severity for evaluations conducted at two locations in Mali, West Africa. Neya and Le Normand<sup>[5]</sup> and Hess<sup>[7]</sup> also observed differences in disease severity between growing seasons. Temperature, relative humidity and length of dew period can influence infection response and disease severity[8,27]. In addition to the interactions with climatic variables influencing disease development, other factors may have contributed to the reduction in disease severity observed during the wet growing season in Puerto Rico.

Sensitivity to day-length may have been a contributing factor for the reduction in anthracnose severity. During the wet growing season, anthesis was delayed by approximately 25 days compared to the dry season due to photoperiod sensitivity. Maintaining plants in a prolonged vegetative state may condition tolerance to anthracnose infection. In addition, the sorghum collection from Mozambique may also be a source of horizontal resistance, since infection severity and disease progression was lower compared to the susceptible control in both seasons. Infection was observed on the flag leaf for two susceptible accessions during the dry growing season, but disease severity averaged less than 50% and senescence of upper infected leaves was not observed. Microclimatic variables may have a greater influence on disease development in the presence of horizontal resistance as compared to vertical resistance. Thus, the interactions between host, pathogen and environment could significantly influence disease

response. Pande<sup>[19]</sup> concluded that pathogen-host interaction was a factor influencing aggression of a virulent pathotype. The combined interactions of the host and pathogen to environmental conditions would also influence the virulence of the pathogen under highly variable field conditions.

Plant color was also observed to be associated with anthracnose infection response for three accessions. Plant color is based on pigment production observed from tissue injury with purple, red and tan being the three major plant colors observed in sorghum. Withinaccession variation for plant color was observed for three accessions that were variable in infection response. The accessions were composed of tan and purple colored plants with the tan plants showing some susceptibility to anthracnose infection. Only three accessions within the Mozambique germplasm collection were uniformly composed of tan plants; therefore, no strong association between plant color and anthracnose susceptibility could be determined. Racial variation and the evolution of different sorghum races to environmental extremes could also contribute to variation in anthracnose infection response. The races of sorghum in Mozambique appeared to have some association with anthracnose susceptibility. Two sorghum races occur within the collection. Nineteen accessions were classified as race guinea with 11 showing resistance to anthracnose; whereas, one of the three accessions classified as race guinea-kafir conditioned anthracnose resistance. The number of accessions in the collection for each race is too small to conclude that race is a major factor influencing disease response. Race guinea has morphological characteristics that enhance survivability under high rainfall conditions and is commonly associated with wetter growing regions in Africa<sup>[28]</sup>. The pathogen is also more prevalent in hot, humid regions<sup>[4]</sup>, which may contribute to greater diversity within the pathogen population. Thus, disease pressure may contribute to greater genetic variation in host plant resistance for guinea sorghum.

Limited information is available on the genetics of host plant resistance to anthracnose in sorghum. LeBeau and Coleman<sup>[24]</sup> determined that anthracnose resistance was conditioned by a single dominant gene in two sorghum accessions from Sudan. A single dominant gene for resistance to anthracnose foliar infection and a linked dominant gene for anthracnose stalk rot resistance were determined for a sorghum genotype used for syrup production<sup>[15]</sup>. Recently, a recessive gene was identified in a sorghum conversion line, SC326-6, from Ethiopia<sup>[25]</sup>. Erpelding and Prom<sup>[26]</sup> indicated dominant and recessive genes conferred anthracnose resistance to Puerto Rican isolates for sorghum accessions from Mali, West Africa.

No information is available on genetic diversity or allelic variation for anthracnose resistance in sorghum. Because the pathogen is highly variable within and between regions, the Mozambique sorghum germplasm may provide unique sources of anthracnose resistance for use in sorghum improvement programs. Twelve sorghum accessions from Mozambique conditioned stable anthracnose resistance during the dry and wet growing seasons in Puerto Rico and may provide resistance to pathotypes in Georgia. The genetic inheritance of resistance will need to be determined to enhance utilization of the germplasm in breeding programs and to aid in the transfer of resistance genes to desirable breeding lines.

#### ACKNOWLEDGMENTS

The authors are grateful to Dr. Roger Monk and Pioneer Hi-Bred International, Inc. for conducting the anthracnose evaluation in Georgia.

#### REFERENCES

- Freeman, S., M. Pham and R.J. Rodriguez, 1993. Molecular genotyping of *Colletotrichum* sp. based on arbitrarily primed PCR, A+T-rich DNA and nuclear DNA analyses. Exp. Mycol., 17: 309-322.
- Sherriff, C., M.J. Whelan, G.M. Arnold and J.A. Bailey, 1995. rDNA sequence analysis confirms the distinction between *Colletotrichum graminicola* and *C. sublineolum*. Mycol. Res., 99: 475-478.
- Cardwell, K.F., P.R. Hepperly and R.A. Frederiksen, 1989. Pathotypes of *Colletotrichum graminicola* and seed transmission of sorghum anthracnose. Plant Dis., 73: 255-257.
- Thakur, R.P. and K. Mathur, 2000. Anthracnose. In: Compendium of Sorghum Diseases. (Frederiksen, R.A. and G.N. Odvody Eds.). The American Phytopathol. Soc., St. Paul, MN, USA, pp: 10-12.
- Neya, A. and M. Le Normand, 1998. Responses of sorghum genotypes to leaf anthracnose (*Colletotrichum graminicola*) under field conditions in Burkina Faso. Crop Prot., 17: 47-53.
- Marley, P.S., R.P. Thakur and O. Ajayi, 2001. Variation among foliar isolates of *Colletotrichum* sublineolum of sorghum in Nigeria. Field Crops Res., 69: 133-142.
- Hess, D.E., R. Bandyopadhyay and I. Sissoko, 2002. Pattern analysis of sorghum genotype x environment interaction for leaf, panicle and grain anthracnose in Mali. Plant Dis., 86: 1374-1382.

- Ngugi, H.K., S.B. King, G.O. Abayo and Y.V.R. Reddy, 2002. Prevalence, incidence and severity of sorghum diseases in western Kenya. Plant Dis., 86: 65-70.
- Harris, H.B., B.J. Johnson, J.W. Dobson Jr and E.S. Luttrell, 1964. Evaluation of anthracnose on grain sorghum. Crop Sci., 4: 460-462.
- Thomas, M.D. and R.A. Frederiksen, 1995. Dynamics of oval and falcate conidium production of Colletotrichum graminicola from sorghum. Mycologia, 87: 87-89.
- Snyder, B.A., B. Leite, J. Hipskind, L.G. Butler and R.L. Nicholson, 1991. Accumulation of sorghum phytoalexins induced by *Colletotrichum* graminicola at the infection site. Physiol. Mol. Plant P., 39: 463-470.
- Lo, S.C., K. de Verdier and R.L. Nicholson, 1999.
  Accumulation of 3-deoxyanthocyanidin phytoalexins and resistance to *Colletotrichum sublineolum* in sorghum. Physiol. Mol. Plant P., 55: 263-273.
- Ali, M.E.K., H.L. Warren and R.X. Latin, 1987.
  Relationship between anthracnose leaf blight and losses in grain yield of sorghum. Plant Dis., 71: 803-806.
- Thomas, M.D., I. Sissoko and M. Sacko, 1996.
  Development of leaf anthracnose and its effect on yield and grain weight of sorghum in West Africa. Plant Dis., 80: 151-153.
- Coleman, O.H. and I.E. Stokes, 1954. The inheritance of resistance to stalk red rot in sorghum. Agron. J., 46: 61-63.
- Frederiksen, R.A., 2000. Anthracnose Stalk Rot. In: Compendium of Sorghum Diseases. (Frederiksen, R.A. and G.N. Odvody Eds.). The American Phytopathological Society, St. Paul, MN, USA., pp: 27-28.
- Casela, C.R. and R.A. Frederiksen, 1993. Survival of Colletotrichum graminicola sclerotia in sorghum stalk residues. Plant Dis., 77: 825-827.
- 18. Ali, M.E.K. and H.L. Warren, 1987. Physiological races of *Colletotrichum graminicola* on sorghum. Plant Dis., 71: 402-404.
- Pande, S., L.K. Mughogho, R. Bandyopadhyay and R.I. Karunakar, 1991. Variation in pathogenicity and cultural characteristics of sorghum isolates of *Colletotrichum graminicola* in India. Plant Dis., 75: 778-783.

- 20. Casela, C.R., A.S. Ferreira and R.E. Schaffert, 1992. Physiological Races of *Colletotrichum graminicola* in Brazil. In: Sorghum and Millets Diseases: A Second World Review. (de Milliano, W.A.J., R.A. Frederiksen and G.D. Bengston Eds.). Patancheru, A. pp: 502–324, India: International Crops Research Institute for the Semi-Arid Tropics, pp: 209-212.
- Rosewich, U.L., R.E. Pettway, B.A. McDonald, R.R. Duncan and R.A. Frederiksen, 1998. Genetic structure and temporal dynamics of a *Colletotrichum* graminicola population in a sorghum disease nursery. Phytopathology, 88: 1087-1093.
- Guthrie, P.A.I., C.W. Magill, R.A. Frederiksen and G.N. Odvody, 1992. Random amplified polymorphic DNA markers: A system for identifying and differentiating isolates of *Colletotrichum* graminicola. Phytopathology, 82: 832-835.
- Souza-Paccola, E.A., L.C.L. Favaro, C.R. Casela and L.D. Paccola-Meirelles, 2003. Genetic recombination in *Colletotrichum sublineolum*. J. Phytopathol., 151: 329-334.
- LeBeau, F.J. and O.H. Coleman, 1950. The inheritance of resistance in sorghum to leaf anthracnose. Agron. J., 42: 33-34.
- Boora, K.S., R. Frederiksen and C. Magill, 1998.
  DNA-based markers for a recessive gene conferring anthracnose resistance in sorghum. Crop Sci., 38: 1708-1709.
- Erpelding, J.E. and L.K. Prom, 2004. Evaluation of Malian sorghum germplasm for resistance against anthracnose. Plant Pathol. J., 3: 65-71.
- 27. Pande, S., R.P. Thakur, R.I. Karunakar, R. Bandyopadhyay and B.V.S. Reddy, 1994. Development of screening methods and identification of stable resistance to anthracnose in sorghum. Field Crops Res., 38: 157-166.
- 28. Harlan, J.R. and J.M.J. de Wet, 1972. A simplified classification of cultivated sorghum. Crop Sci., 12: 172-176.