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Inheritance of Anthracnose Resistance for the Sorghum Cultivar Redlan

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Abstract: To determine the inheritance of resistance in Redlan, a F_2 population was developed and evaluated at the USDA-ARS Tropical Agriculture Research Station in Isabela, Puerto Rico in 2005. Results of the disease evaluation indicated that foliar anthracnose resistance observed in Redlan is controlled by a single dominant gene. Anthracnose infection of the leaf midrib was also observed in the F_2 population and results indicate resistance for midrib infection is controlled by a single recessive gene. Segregation analysis, based on the frequency of recombinants for foliar and midrib resistance, indicated that the two genes are unlinked.

Key words: *Sorghum bicolor*, *Colletotrichum sublineolum*

INTRODUCTION

Anthracnose caused by *Colletotrichum sublineolum* P. Henn., Kabát and Bubák (Crouch *et al*., 2006) is considered one of the most important diseases of sorghum and was first reported in the United States in 1912 (Harris *et al*., 1964). The disease is commonly found in tropical and subtropical environments where warm, humid climatic conditions enhance the development and spread of the disease (Thakur and Mathur, 2000). Anthracnose infection can be observed on all above ground tissues of susceptible sorghum plants including the leaf, stalk, panicle and seed. Foliar infection is the most commonly observed symptom, generally appearing 40 days after seedling emergence. Symptoms on susceptible cultivars include circular to elongated lesions with the formation of black spots (acervuli) in the center of the lesions during sporulation. Under epidemic conditions, grain yield losses of more than 50% have been reported for susceptible cultivars (Harris *et al*., 1964; Thomas *et al*., 1996; Thakur and Mathur, 2000).

The successful management of sorghum anthracnose using resistant varieties has been hindered by variation within the pathogen population and the occurrence of new pathotypes of the disease. Numerous studies have indicated that the pathogen is highly variable with multiple pathotypes present within and between sorghum production regions (Ali and Warren, 1987; Cardwell *et al*., 1989; Pande *et al*., 1991; Guthrie *et al*., 1992; Rosewich *et al*., 1998; Marley *et al*., 2001; Valerio *et al*., 2005). The ability to effectively evaluate pathotype variation requires the selection of a differential set of sorghum lines to characterize pathogen virulence. Valerio *et al*. (2005) used 10 sorghum breeding lines to differentiate pathogen virulence for anthracnose pathotypes occurring in Brazil. Redlan (BTx378) was included in this set of 10 sorghum anthracnose differential lines. Redlan (NSL 4025, USDA-ARS National Genetic Resources Program, 2006) is a grain sorghum cultivar released in the United States in 1954 (Karper, 1954). Variation in pathogen virulence using Redlan as a host differential line has been observed in Texas, Puerto Rico and Brazil (Cardwell *et al*., 1989; Valerio *et al*., 2005). The anthracnose disease response of Redlan has been evaluated over multiple growing seasons in Isabela, Puerto Rico and a resistant disease reaction has been observed under field conditions for all evaluations (Erpelding and Prom, 2004).

The genetics of host plant resistance for the set of sorghum anthracnose differential lines will be essential to effectively evaluate pathogen virulence within and between regions and to evaluate the occurrence of new virulent pathotypes within the pathogen population. Presently, no information is available on the genetic inheritance of host plant resistance for the sorghum cultivar Redlan. Thus, an evaluation was conducted to determine the genetics of resistance for Redlan to anthracnose pathotypes occurring in Isabela, Puerto Rico.

MATERIALS AND METHODS

Redlan was used as the male parent in a cross with PI 609251 to generate a segregating F_2 population to evaluate inheritance of anthracnose resistance. PI 609251 is a sorghum accessions from Mali, West Africa and is highly susceptible to anthracnose infection (Erpelding and Prom, 2004; Erpelding and Prom, 2006). Anthracnose infection is typically observed on PI 609251 approximately 40 days after emergence and by 90 days the disease has resulted in complete defoliation with severe stalk breakage

and lodging. Rapid disease progression on inoculated leaves has been observed for PI 609251 with the disease spreading to all leaves resulting in an average infected leaf area ranging from 70 to 100% at maturity. Anthracnose lesion development is typically observed in the leaf midrib for PI 609251. Although stalk breakage is typically observed from foliar anthracnose infection of PI 609251, the development of microsclerotia within the stalk has not been frequently observed. For Redlan, a hypersensitive response is typically observed after inoculation with no disease development observed at maturity. The hypersensitive response is characterized as red spots on inoculated leaves. Senescence of leaf margins and tips has been observed under severe epidemic conditions, but no lesion development has been observed in healthy leaf tissue. Anthracnose infection of the midrib and stalk has not been observed for Redlan under field conditions from inoculation of foliar tissue.

Seed from the cross was planted in the field and panicles from the F_1 plants were bagged prior to anthesis to produce F_2 seed for population evaluation. Seed obtained from the F_1 generation was planted in growing trays in the greenhouse to generate single F_2 plants. A single seed was planted in a 30 mm peat pellet with a total of 564 seeds planted. Redlan and PI 609251 were also planted to compare infection response of the parents with the population. The seedlings were grown in the greenhouse for approximately 14 days before being transplanted to the field for the anthracnose disease evaluation. A total of 479 vigorous F_2 plants were transplanted in the field on 7 July 2005 at the USDA-ARS Tropical Agriculture Research Station in Isabela, Puerto Rico. Fertilizer was applied at a rate of 560 kg ha^{-1} (15-5-10 NPK) 11 days after planting. Irrigation was conducted as necessary until plants were inoculated with the anthracnose pathogen. Weeds were controlled by hand hoeing. Plants were inoculated with anthracnose colonized seed 29 days after transplanting. Preparation of anthracnose cultures, inoculation and disease evaluation were as described by Erpelding and Prom (2006). Anthracnose infection response was evaluated approximately 30 and 60 days after inoculation using a 1-5 rating scale with highly resistant plants rated as 1, resistant plants rated as 2, moderately susceptible plants rated as 3, susceptible plants rated as 4 and highly susceptible plants rated as 5. Plants were also evaluated for anthracnose infection of the leaf midrib, percent infected leaf area, lodging and leaf senescence. Chi-square analysis was conducted on the disease response data for the population to determine the genetic inheritance of anthracnose resistance.

RESULTS

Reddening of leaf tissue, characteristic of the hypersensitive response to anthracnose infection, was observed on all F_2 plants within seven days after inoculation followed by the development of anthracnose lesions on susceptible plants approximately 10 day after inoculation. For 365 F_2 plants, no change in the hypersensitive response was observed 60 days after inoculation and the plants were rated as resistant (Table 1). Senescence of leaf margins and tips was observed for 179 plants rated as resistant and 54 anthracnose resistant plants showed some lesion development in the senesced tissue or at the margins of the senescencing leaf tissue. Lesion development was limited to inoculated leaves for these plants and no progression of the disease was observed 60 days after inoculation to rate the plants as susceptible. Complete senescence of leaf tissue was observed for 14 plants 60 days after inoculation, but no anthracnose lesion development was observed in healthy leaf tissue prior to leaf senescence to indicated susceptibility to anthracnose infection. Lodging was observed on seven plants with completed leaf senescence, suggesting possible anthracnose stalk rot symptoms. Plant death was observed for 10 of the 14 plants with complete leaf senescence. No anthracnose lesion development was observed on these plants 30 days after inoculation and little infection was observed in the senesced leaf tissue 60 days after inoculation. Due to the lack of lesion development in healthy leaf tissue and no disease progression, the plants were rated as resistant. In contrast, four plants rated as resistant 30 days after inoculation showed lesion development 60 days after inoculation. Disease progression was observed in healthy leaf tissue for these plants with infected leaf area ranging from 1 to 10%. As a result, these plants were rated as susceptible 60 days after inoculation.

Susceptibility to anthracnose infection was observed for 114 F_2 plants 60 days after inoculation (Table 1). No plants were given a rating of 3 (moderately susceptible), since all plants showed necrotic lesion development and progress of the disease to non-inoculated leaf tissue. Eighty-six susceptible plants were given a rating of 4 with an average infected leaf area of 20%. Twenty-eight plants were rated as 5, indicating a highly susceptible response, with an average infected leaf area of 75%. Infected leaf area ranged from 1 to 100% for the 114 anthracnose susceptible F_2 plants with an average infected leaf area of 34%. Sixteen plants were rated as highly susceptible 30 days after inoculation and 12 plants rated as susceptible 30 days after inoculation showed rapid disease progression on all leaves resulting in a highly

Table 1: Anthracnose disease response for the PI 609251 x Redlan F_2 population evaluated in Isabela, Puerto Rico in 2005

Disease response[1]	Foliar rating 1[2]	Foliar rating 2[3]	Midrib[4]	Stalk lodging[5]	Leaf senescence[6]
Resistant	369	365	115	7	14
Susceptible	110	114	364	17	20

[1]The 479 plants for the F_2 population were inoculated with the anthracnose pathogen and infection response was used to rate the plants as resistance or susceptible to anthracnose foliar and midrib infection, [2]The first rating of foliar anthracnose infection was conducted 30 days after inoculation, [3]The second foliar anthracnose evaluation was conducted 60 days after inoculation, [4]Number of F_2 plants showing a resistant or susceptible response to anthracnose infection of the leaf midrib, [5]Number of plants showing symptoms of stalk rot resulting in lodging for the 479 F_2 plants rated as resistant and susceptible to anthracnose foliar infection, [6]Number of plants showing complete leaf senescence 60 days after inoculation for the F_2 plants rated as resistant and susceptible to anthracnose foliar infection

susceptible disease response being observed 60 days after inoculation. Complete senescence of leaf tissue was observed 60 days after inoculation for two plants rated as susceptible. Eight highly susceptible plants showed nearly complete leaf senescence 30 days after inoculation with complete leaf senescence observed for two plants. Eighteen plants rated as highly susceptible 60 days after inoculation showed complete senescence of leaf tissue resulting in plant death. Infected leaf area was greater than 90% for 15 highly susceptible plants. Lodging resulting from stalk rot was observed for 17 plants rated as highly susceptible. No lodging was observed for the 86 plants with a susceptibility rating of 4.

Six plants of PI 609251 and Redlan were included as controls in the evaluation of the F_2 population. Reddening of inoculated leaves associated with the hypersensitive response was observed for PI 609251 and Redlan within seven days after inoculation. No change in the hypersensitive response was observed for Redlan 60 days after inoculation. The hypersensitive response was also observed at maturity for Redlan. For PI 609251, anthracnose lesion development was clearly visible 10 days after inoculation. PI 609251 was rated as susceptible 30 days after inoculation with complete senescence of lower leaves and the tips of the upper leaves. Anthracnose infection was also observed in the leaf midrib 30 days after inoculation. Sixty days after inoculation, PI 609251 was rated as highly susceptible with most leaves completely senesced and approximately 50% of the leaf area infected with anthracnose.

Infection of the leaf midrib was also evaluated for the F_2 population (Table 1). Midrib infection was observed for 364 F_2 plants with infection of the midrib observed for 260 plants rated as resistant to anthracnose foliar infection. Midrib infection was only observed on inoculated leaves for the plants rated as resistant to anthracnose foliar infection. Only a few anthracnose lesions were observed in the midrib for these plants and generally one lesion was present in the leaf midrib. The majority of the plants rated as susceptible or highly susceptible to foliar infection showed anthracnose infection of the leaf midrib. Nearly all the plants rated as susceptible showed midrib infection (83 of 86 plants); whereas, 21 of the 28 highly susceptible plants showed midrib infection.

Chi-square analysis using a Mendelian 3 (resistant):1 (susceptible) genotypic ratio for the F_2 segregation data from the PI 609251 x Redlan population indicated that foliar anthracnose resistance observed for Redlan is conferred by a single dominant gene ($\chi^2 = 0.40$, $p = 0.5440$). Midrib infection has been observed in the germplasm line PI 609251, but has not been observed in Redlan under field conditions at Isabela, Puerto Rico. Chi-square analysis using a Mendelian 3 (susceptible):1 (resistant) genotypic ratio for the F_2 segregation data would suggest that resistance to midrib infection associated with Redlan is conditioned by a single recessive gene ($\chi^2 = 0.28$, $p = 0.6162$). Resistance to anthracnose infection of the leaf lamina and midrib, similar to the Redlan phenotype, was observed for 105 F_2 plants. Anthracnose susceptibility of the leaf lamina and midrib similar to the phenotype of PI 609251 was observed for 104 F_2 plants. Non-parental phenotypes for infection of the leaf lamina and midrib were observed for 270 plants suggesting recombination between the two resistant genes. Segregation analysis, based on recombinant phenotypes, would suggest that the dominant gene for leaf resistance and the recessive gene for midrib resistance are unlinked and inherited independently.

DISCUSSION

Redlan was derived from a cross between C.I. 1090 (kafir x milo) and C.I. 71 (Standard Blackhull kafir) (Karper, 1954). Anthracnose infection has been observed on Standard Blackhull kafir in Isabela, Puerto Rico; thus, it is assumed that the source of anthracnose resistance for Redlan is from C.I. 1090. A considerable range of phenotypic variation in anthracnose infection response was observed for the PI 609251 x Redlan F_2 population. Segregation analysis for the population would indicate foliar anthracnose resistance derived from Redlan is controlled by a single dominant gene. A resistant response to foliar anthracnose infection was also observed for the F_1 plants from the cross suggesting resistance was inherited as a dominant trait. Single dominant and recessive genes conferring foliar anthracnose resistance have been identified in other sorghum breeding lines (LeBeau and Coleman, 1950; Boora *et al*., 1998; Singh *et al*., 2006). Dominant sources

of anthracnose resistance have greater potential in sorghum hybrid breeding programs, since resistance can be introgressed into one parent for expression in the hybrid. Redlan also confers resistance to anthracnose isolates occurring in Texas and presently a recombinant inbred population is being developed from the PI 609251 × Redlan F_2 population to evaluate whether the single dominant gene conferring resistance to anthracnose isolates in Puerto Rico confers resistance to anthracnose isolates from other regions. The development of resistant and susceptible inbred lines from the population would also be useful as differential lines for evaluating pathogen virulence within and between anthracnose populations.

Anthracnose evaluations have indicated that the site of infection in the host plant can be controlled by different host resistance genes (Thakur and Mathur, 2000). Coleman and Stokes (1954) indicated that resistance to anthracnose foliar and stalk infection were controlled by two linked, single dominant genes. Results of this study indicate resistance to anthracnose foliar infection of the leaf lamina is controlled by a single dominant gene unlinked to a single recessive gene conferring resistance to anthracnose leaf midrib infection. Stalk rot and lodging were also observed in the population, but the low number of susceptible plants would indicate resistance may be controlled by multiple genes. To determine the genetic inheritance of anthracnose stalk infection, stalk inoculation will be essential to confirm infection response. The recombinant inbred population will be beneficial in evaluating resistance to tissue specific infection through the use of replicated evaluations and controlled inoculation of specific tissues.

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