

Inheritance of Resistance in Sorghum to Three Pathotypes of *Peronosclerospora sorghi*

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ABSTRACT

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The F₁, F₂, and F₃ generations of the cross of sorghum (*Sorghum bicolor*) inbred line SC 414-12 (which is resistant to pathotypes 1, 2, and 3 of *Peronosclerospora sorghi*) and the universally susceptible sorghum line Tx 412 were used to determine inheritance of resistance to *P. sorghi*. In SC 414-12, resistance was expressed as an incompatible host/pathogen interaction which inhibited pathogen development and sporulation in

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leaves inoculated with conidia of *P. sorghi*. The reactions of the parental lines and progenies to conidial inoculum of the three pathotypes supported the hypothesis that resistance of *P. sorghi* to these pathotypes was conferred by a single dominant gene. The F₂ phenotypic ratios were 3 resistant : 1 susceptible; F₂ genotypic ratios were 1 homozygous resistant : 2 heterozygous : 1 homozygous susceptible.

Sorghum downy mildew, caused by *Peronosclerospora sorghi* (Weston and Uppal) C. G. Shaw, is an internationally important disease of sorghum (*Sorghum bicolor* (L.) Moench.) and corn (*Zea mays* L.) (6). In the United States, the disease has caused severe damage to sorghum production in Texas (5). Host resistance is an effective means of controlling sorghum downy mildew and several resistant sorghum hybrids adapted to Texas have been developed (5). Although sorghum genotypes resistant to downy mildew have been identified and used successfully to produce resistant sorghum hybrids, few studies on the mode of inheritance of resistance have been reported. Puttarudrappa and co-workers (7) reported that resistance to *P. sorghi* in sorghum cultivars IS 84 and IS 2941 was conferred by two recessive genes. Frederiksen et al (6) reported that resistance to *P. sorghi* in three sorghum lines ranged from complete to intermediate dominance and that it was conditioned either by multiple genes or by a major gene with modifiers.

Recently, pathotypes of *P. sorghi* capable of inducing sorghum downy mildew in previously resistant sorghum inbred lines and hybrids were discovered in Texas (3,4). The pathotypes 1, 2, and 3 identified in Texas are the only reported pathotypes of *P. sorghi*. These pathotypes were differentiated by the sorghum lines Tx 412, CS 3541, and Tx 430. Pathotype 1 was virulent to Tx 412 and avirulent to CS 3541 and Tx 430. Pathotype 2 was virulent to Tx 412 and CS 3541 and avirulent to Tx 430. Pathotype 3 was virulent to each of the differentials. Compatible host/pathotype interactions were characterized by the ability of the pathogen to sporulate on leaves inoculated with conidia. Incompatible interactions were characterized by the inhibition of pathogen sporulation on inoculated leaves.

Several inbred lines of sorghum developed by the Texas Agricultural Experiment Station were tested for reaction to each of the three pathotypes of *P. sorghi*. Only inbred line SC 414-12 was resistant to all pathotypes.

The purpose of the research reported in this paper was to determine the mode of inheritance of resistance to *P. sorghi* in SC 414-12.

MATERIALS AND METHODS

Two sorghum inbred lines, SC 414-12 and Tx 412, were used as parental lines. The reactions of these lines to *P. sorghi* had been determined by tests in the field and greenhouse. The inbred SC 414-12 was resistant to pathotypes 1, 2, and 3 of *P. sorghi*; Tx 412 was universally susceptible. The parental lines were crossed and the F₁, F₂, and F₃ progenies were produced in fields where sorghum downy mildew was not observed.

The populations of pathotypes 1, 2, and 3 of *P. sorghi* used in this study originated from diseased plants collected in field nurseries. The pathotypes were maintained in the greenhouse on susceptible sorghum hybrids infected by conidial inoculation of freshly germinated seeds (6). The greenhouse populations of the pathotypes were tested at monthly intervals with the sorghum differentials to ensure that their original virulence characteristics were retained.

Reactions of the parental lines and the F₁, F₂, and F₃ progenies to *P. sorghi* were determined by inoculating seedling plants at the 1.5 leaf stage of growth. The conidial inoculum was secured from the diseased plants used to maintain the pathotypes. Infected leaves from these plants were placed above the sorghum seedlings under environmental conditions conducive to sporulation of *P. sorghi*. The conidia produced on these leaves were distributed over the seedlings by using controlled air currents as previously described (2). The inoculated plants were grown in the greenhouse for 6 days after inoculation and then incubated for 17-24 hr in a moist chamber at a temperature of 20 C and RH maintained above 95% with a humidifier. The plants were removed from the moist chamber and observed macroscopically for sporulation of *P. sorghi* on the inoculated leaves. The plants on which sporulation occurred were classed as susceptible; plants without sporulation were classed as resistant (4).

Populations of the parental lines and the F₁ and F₂ generations were tested for reaction to each of the three pathotypes of *P. sorghi*. The genotypes present in the F₂ generation were determined by the reactions of F₃ families produced by selfing a random sample of F₂ plants. Twenty plants of each of 136 F₃ full-sib families were tested for reaction to pathotype 1. The F₃ families were classified as homogeneous resistant, homogeneous susceptible, or heterogeneous. To determine the relationships among genetic factors conditioning resistance to the different pathotypes, 22 F₃ families homogeneous for resistance to pathotype 1 and 41 F₃ families heterogeneous for reaction to pathotype 1 were tested for reaction to pathotypes 2 and 3, respectively. Chi-square tests were used to

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TABLE 1. Reactions of the sorghum inbreds SC 414-12 and Tx 412 and their F₁ and F₂ progenies to pathotypes 1, 2, and 3 of *Peronosclerospora sorghi*

Pedigree	Pathotype ^b	Number of plants ^a		Expected ratio	P-value
		R	S		
SC 414-12	1,2,3	20	0		
Tx 412	1,2,3	0	20		
Tx 412 × SC 414-12	1,2,3	20	0		
(Tx 412 × SC 414-12)F ₂	1	357	113	3:1	0.25-0.50
(Tx 412 × SC 414-12)F ₂	2	320	89	3:1	0.10-0.20
(Tx 412 × SC 414-12)F ₂	3	307	99	3:1	0.50-0.75

^aR = resistant plants, and S = susceptible plants.

^bReactions to each pathotype were tested separately.

test the goodness of fit of the segregation ratios observed in the F₂ and F₃ populations.

RESULTS AND DISCUSSION

The F₁ plants produced by crossing susceptible inbred Tx 412 and resistant inbred SC 414-12 were resistant to the three pathotypes of *P. sorghi* (Table 1). The reactions of the F₂ population indicated that resistance to each pathotype of *P. sorghi* was conditioned by a single dominant genetic factor.

The use of backcross progenies to test hypothesis of F₂ genotypic ratios in sorghum is impractical because of the large number of manual floral emasculations required to produce an adequate backcross population. However, F₃ families produced by self-pollinated F₂ plants provide the means of identifying F₂ genotypes. In tests of F₃ families, homogeneity of reaction in a 20-plant sample was accepted as proof that the F₂ parental plant was homozygous for the genetic factor controlling reaction to *P. sorghi*. Assuming that resistance was monogenic and dominant, the probability of failing to detect heterogeneity with a 20-plant sample of an F₃ progeny was less than 1%. The observed ratio of resistant:heterogeneous:susceptible F₃ progenies was an acceptable approximation of the 1:2:1 ratio expected if resistance was conditioned by a single, dominant, genetic factor (Table 2).

None of the 63 F₃ families tested for reaction to each of the three pathotypes differentiated the pathotypes. If resistance to the three pathotypes was not conferred by the same gene or very closely linked genes, recombinations among loci in the F₂ generation should produce F₃ families with differential reactions to the different pathotypes. The absence of such differential genotypes in the sample taken indicated that if linked, differential loci were involved, the linkage was too close to be detected by the sample size used in this study. Tests of a sample of 63 F₃ families had a 98% probability of detecting linkages with crossover frequencies as low as 3%. The simplest hypothesis that fits the data is that universal resistance in SC 414-12 to the three pathotypes of *P. sorghi* is conferred by a single dominant gene.

Our study of the inheritance of resistance to *P. sorghi* differed from previous studies in the method used to identify resistant genotypes and in the concept of what constituted resistance to *P. sorghi*. Earlier studies of the inheritance of resistance to *P. sorghi* in sorghum (6,7) used disease nurseries in the field to identify resistant and susceptible genotypes. In these field tests, the plants were

TABLE 2. Reactions of (Tx 412 × SC 414-12)F₃ sorghum progenies to pathotypes of *Peronosclerospora sorghi*

F ₃ progenies	Pathotype	Reaction ^a			Expected ratio	P-values
		R	H	S		
Random sample	1	25	77	34	1:2:1	0.10-0.20
Resistant to pathotype 1	2,3	22	0	0		
Heterogeneous to pathotype 1	2,3	0	41	0		

^aR = number of F₃ families homogeneous for resistance, H = number of F₃ families heterogeneous for reaction to *P. sorghi*, and S = number of F₃ families homogeneous for susceptibility.

exposed to naturally occurring inoculum, and plants that did not develop the systemic phase of sorghum downy mildew (5) were assumed to be resistant. In our experience with field nurseries, we found that significant numbers of susceptible plants escape detection because of the erratic disposition of inoculum and the occurrence of environmental factors unfavorable to the development of systemic sorghum downy mildew (1). In the inheritance study reported by Puttarudrappa and co-workers (7), 25% of the plants of the susceptible parental sorghum line escaped infection in the test nursery used to determine the reactions of parental lines and progenies. Controlled conidial inoculation of plants in the greenhouse was a more efficient method of detection of resistant genotypes.

Resistance to *P. sorghi*, by our standards, was expressed as an incompatible, host-pathogen relationship in which colonization of the host tissue by the pathogen was insufficient to produce sporulation by the pathogen (4). To induce the systemic phase of sorghum downy mildew from conidial inoculation, the pathogen must progress from its initial invasion point in the outer leaves to the immature foliage tissues enclosed in the leaf whorl. Histological studies of the infection of susceptible and resistant sorghum genotypes by *P. sorghi* revealed that the inability of *P. sorghi* to grow in leaf tissues of resistant genotypes was the major component of resistance to sorghum downy mildew in the field (8).

LITERATURE CITED

- Balasubramanian, K. A. 1974. Role of date of seeding, soil moisture, temperature and pH in the incidence of downy mildew of sorghum. Plant Soil 41:233-241.
- Craig, J. 1976. An inoculation technique for identifying resistance to sorghum downy mildew. Plant Dis. Rep. 60:350-352.
- Craig, J., and Frederiksen, R. A. 1980. Pathotypes of *Peronosclerospora sorghi*. Plant Dis. 64:778-779.
- Craig, J., and Frederiksen, R. A. 1983. Differential sporulation of pathotypes of *Peronosclerospora sorghi* on inoculated sorghum. Plant Dis. 67:278-279.
- Frederiksen, R. A. 1980. Sorghum downy mildew in the United States: Overview and outlook. Plant Dis. 64:903-908.
- Frederiksen, R. A., Bockholt, A. J., Clark, L. E., Casper, J. W., Craig, J., Johnson, J. W., Jones, B. L., Matocha, P., Miller F. R., Reyes, L., Rosenow, D. T., Tuleen, D., and Walker, H. J. 1973. Sorghum downy mildew, a disease of maize and sorghum. Tex. Agric. Exp. Stn. Res. Monogr. 2. 32 pp.
- Puttarudrappa, A., Kulkarni, B. G., and Kajjari, N. B. 1972. Inheritance of resistance to downy mildew (*Sclerospora sorghi*) in sorghum. Indian Phytopathol. 25:471-473.
- Yey, Y., and Frederiksen, R. A. 1980. Sorghum downy mildew: Biology of systemic infection by conidia and of a systemic response in sorghum. Phytopathology 70:372-376.

TABLE 5. Relative numbers^a of uredinia of *Puccinia recondita* produced on seedling leaves of wheat line LR2C(W1) and its recurrent parent cultivar, Wichita

Wheat line or cultivar	Cultures of <i>P. recondita</i>	
	PRTUS6 ^a	PRTUS4 ^a
LR2C(W1)	68	90
Wichita	100	100