Inheritance of Resistance to Pathotypes 1, 2, and 3 of *Peronosclerospora sorghi* in Sorghum

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ABSTRACT

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The relationship between the genes for resistance to *Peronosclerospora sorghi* pathotypes 1, 2, and 3 in the sorghum lines QL3-India and SC414-12 were investigated by making reciprocal crosses between them and susceptible lines. The results supported the hypothesis that QL3-India has two genes conditioning resistance to each of the three pathotypes, whereas SC414-12 has one gene for resistance to the same three pathotypes. There appears to be no linkage between those genes, since the observed segregation in the F_2 of the cross between the resistant parents was in agreement with a 63:1 ratio. Management of these three genes to prolong the life of sorghum cultivars is advocated.

Peronosclerospora sorghi (Weston & Uppal) C. G. Shaw is the causal agent of sorghum downy mildew, a disease of sorghum and maize. Sorghum downy mildew frequently devastates these crops in the tropics and subtropics where cool, humid conditions occur during early stages of plant growth (6,7). Control of sorghum downy mildew is obtained by the use of chemicals, cultural practices, and resistant varieties (8). Host resistance is the method most frequently used.

The ability of the fungus to overcome host resistance has been demonstrated. In 1979 and 1980, pathogenic variation of the fungus was detected on sorghum in Texas (3,4). Three pathotypes of *P. sorghi* (P_1, P_2, P_3) are known in Texas. P_1, the original pathotype, is widely distributed in Texas, whereas P_2 is rare and reported occasionally from only two locations, near Beeville and Mathis. P_2 differs from P_1 isolates of *P. sorghi* in the ability to infect sorghum cultivars such as CS3541 and SC170-6-17. P_3, however, has a much broader host range, attacking a number of sorghum cultivars, particularly the popular commercially used RT × 430. P_3 of *P. sorghi* is now widely distributed in an area along the upper coastal plains of southern Texas. Fortunately, additional sources of resistance to the new pathotypes were identified (10). The sorghum lines SC414-12 and QL3-India are resistant to three known Texas pathotypes of *P. sorghi*. Because germ plasm from only a few sources of resistance is used over a wide area of Texas, the appearance of new pathogenic variants of the fungus seems likely. The identification of different genes for resistance is needed for development of sorghum cultivars with durable resistance to sorghum downy mildew.

Studies on the inheritance of resistance (5) provided information on the number of genetic factors in SC414-12 involved in resistance to the three pathotypes. QL3-India was identified as resistant to 16 isolates of *P. sorghi* from different parts of the world (9) and has great potential as a source of resistance (11).

The present work reports the identification of the nature and number of genes in QL3-India involved in resistance to three pathotypes of *P. sorghi* and investigation of their relationship to the genes that condition resistance in SC414-12.

MATERIALS AND METHODS

Eight sorghum inbred lines were used as parents. Of these, two (QL3-India and SC414-12) are resistant to all three pathotypes; three are differentials (Tx430 is resistant to P_1 and P_2 and susceptible to P_3, and SC170-6-17 and CS3541 are susceptible to P_2 and P_3 and resistant to P_1); and three (Tx412, Tx7078, and Tx2536) are "universally" susceptible.

Parental lines were screened for homogeneity of reaction by separately inoculating the progeny of selfed plants with each pathotype. Only plants whose progeny gave an homogeneous reaction were used as parents. Each of the resistant lines, SC414-12 and QL3-India, were crossed with the remaining lines and between each other. Reciprocal crosses were also obtained. F_1 seed was produced by hand emasculation, F_1 progenies were identified by using genetic markers from the paternal plant, and F_1 plants were advanced to the F_2.

Populations of the three pathotypes used in this study were obtained from J. Craig (USDA, College Station, TX). Inoculum was maintained on the susceptible sorghum hybrid Tophand by exposing 1-day-old germinated seeds to conidial showers under conditions of 90% relative humidity and 20 C. Seedlings infested with different pathotypes were separated in the greenhouse to avoid pathotype mixtures. To verify pathotype identification, sorghum differential lines were inoculated with *P. sorghi* from these seedlings.

Parental lines, F_1, and F_2 populations were tested for reaction to each pathotype at the second leaf stage (2). Inoculated plants remained in the greenhouse for 6 days, followed by incubation at 20 C and greater than 90% relative humidity for 18 hr. The abaxial side of the second leaf then was observed for sporulation of the pathogen, which indicates a compatible host-pathogen interaction (4).

A chi-square test was used to analyze the goodness of fit observed to the expected segregation ratios.

RESULTS AND DISCUSSION

QL3-India appears to have two dominant genes conditioning resistance to each of the three pathotypes tested

Table 1. Reactions of parental lines and of F_1 and F_2 progenies of crosses between QL3-India and susceptible lines to pathotypes 1, 2, and 3 of *Peronosclerospora sorghi*

Sorghum pedigree	Pathotype[a]	Number of plants		Expected ratio	*P* value
		Resistant	Susceptible		
Tx412	1,3	0	20,20		
SC170-6-17	2	0	20		
QL3-India	1,2,3	20,20,20	0		
(QL3-India × Tx412)F_1	1	15	0		
(SC170-6-17 × QL3-India)F_1	2	15	0		
(Tx412 × QL3-India)F_1	3	19	0		
(QL3-India × Tx412)F_2	1	201	11	15:1	0.50–0.80
(SC170-6-17 × QL3-India)F_2	2	202	17	15:1	0.20–0.50
(Tx412 × QL3-India)F_2	3	455	40	15:1	0.05–0.10

[a] Reactions to each pathotype were tested separately.

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Table 2. Reactions of QL3-India reciprocal crosses to *Peronosclerospora sorghi* pathotypes

Sorghum pedigree	Pathotype	Number of plants	
		Resistant	Susceptible
(Tx412 × QL3-India)F_1	1	13	0
(QL3-India × TX412)F_1	1	10	0
(Tx412 × QL3-India)F_1	2	9	0
(QL3-India × Tx412)F_1	2	9	0
(Tx412 × QL3-India)F_1	3	11	0
(QL3-India × Tx412)F_1	3	10	0

Table 3. Reactions of QL3-India, SC414-12, and their F_1 and F_2 progenies to pathotypes 1, 2, and 3 of *Peronosclerospora sorghi*

Sorghum pedigree	Pathotype[a]	Number of plants		Expected ratio	*P* value
		Resistant	Susceptible		
SC414-12	1,2,3	30,30,30	0		
QL3-India	1,2,3	30,30,30	0		
(QL3-India × SC414-12)F_1	1,2,3	15,15,15	0		
(QL3-India × SC414-12)F_2	1	280	8	63:1	0.05–0.20
(QL3-India × SC414-12)F_2	2	231	1	63:1	0.05–0.20
(QL3-India × SC414-12)F_2	3	449	5	63:1	0.20–0.50
(QL3-India × SC414-12)F_2[b]	1,2,3	960	14	63:1	0.75–0.90

[a] Reactions to each pathotype were tested separately.
[b] Data from the three tests combined.

(Table 1). Cytoplasmic inheritance was not detected on the basis of analysis of ratios from reciprocal crosses (Table 2). Crosses of SC414-12 and susceptible lines (data not shown) indicated that this resistant sorghum line has a single dominant factor conditioning resistance to each pathotype, as reported before (5). The F_2 population from the cross between the two resistant lines was inoculated separately with each of three pathotypes. The number of F_2 plants tested with P_2 is too few to satisfy a chi-square test, which requires a minimum of five in the expected classes. However, since both parents are resistant to all three pathotypes, it is possible to combine data from the three tests for chi-square analysis. The results agree with the hypothesis that both lines differ in at least three genetic factors involved in resistance (63:1) (Table 3).

Other studies involving QL3-India (1) have identified a set of six genes coding for resistance to an Indian isolate of *P. sorghi*. In this study, only two factors conditioning resistance were identified. The results of Bhat's study (1) and our investigation are not directly comparable because of differences in the isolate of *P. sorghi* used, inoculation techniques, and criteria for reaction type. Bhat's study (1) was dependent on inoculum produced in infected spreader rows, and disease incidence was recorded as the number of plants with systemic infection. These procedures produced only 89.9–90.9% frequency of infected plants in the susceptible parent. This indicated either failure to achieve uniform inoculation, heterogeneity in the population of the susceptible parent, or failure of inoculation of susceptible plants to induce the systemic phase of downy mildew due to causes other than genetic factors. In this study, sporulation on the inoculated leaf was accepted as a sign of a compatible host-pathogen interaction (4). This criterion is more reliable for identification of susceptible genotypes than scoring only systemically infected plants (5) as susceptibles. Genetically susceptible plants often escape systemic infection (2).

The information obtained can be applied to control the sorghum downy mildew pathogen. The three genes identified in the present study can be utilized in planning different strategies of deployment such as gene pyramiding and gene rotation. These measures can complement the use of chemical control and other cultural practices to further decrease the pathogen population, which should reduce the probability of recurrence of new races.

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LITERATURE CITED

1. Bhat, M. G. 1981. Studies on inheritance of resistance to downy mildew *Peronosclerspora sorghi* (Weston and Uppal) Shaw in sorghum. M.S. thesis. Department of Agricultural Science, Bangalore, India.
2. Craig, J. 1976. An inoculation technique for identifying resistance to sorghum downy mildew. Plant Dis. Rep. 60:350-352.
3. Craig, J., and Frederiksen, R. A. 1980. Pathotypes of *Peronosclerospora sorghi*. Plant Dis. 64:778-779.
4. Craig, J., and Frederiksen, R. A. 1983. Differential sporulation of pathotypes of *Peronosclerospora sorghi* on inoculated sorghum. Plant Dis. 67:278-279.
5. Craig, J., and Schertz, K. F. 1985. Inheritance of resistance in sorghum to three pathotypes of *Peronosclerospora sorghi*. Phytopathology 75:1077-1078.
6. Fernandez, B. A., Malaguti, G., and Hass, H. 1975. *Sclerospora sorghi* Weston and Uppal, grave patogeno del sorgo en Venezuela. Agron. Trop. 25:367-380.
7. Frederiksen, R. A., Amador, J., Jones, B. L., and Reyes, L. 1969. Distribution, symptoms and economic loss of downy mildew caused by *Sclerospora sorghi* (Kulk.) Weston and Uppal in grain sorghum in Texas. Plant Dis. Rep. 53:995-998.
8. Odvody, G. N., Frederiksen, R. A., and Craig, J. 1983. The integrated control of downy mildew. Proc. Annu. Corn Sorghum Res. Conf. 38:28-36.
9. Pawar, M. N., Frederiksen, R. A., Mughogho, L. K., Craig, J., Rosenow, D. T., and Bonde, M. R. 1985. Survey of virulence in *Peronosclerospora sorghi* isolates from India, Ethiopia, Nigeria, Texas (USA), Honduras, Brazil, and Argentina. Pages 130-133 in: Proc. Bienn. Grain Sorghum Res. Util. Conf. 12th.
10. Rosenow, D. T. 1983. Sources of resistance to pathotype 3 of *Peronosclerospora sorghi*. Pages 8-9 in: Integrated control of sorghum downy mildew. Texas Agric. Exp. Stn. MP-1535.
11. Williams, R. J., Dange, S. R. S., Mughogho, L. K., and Rao, K. N. 1982. Identification of QL-3 sorghum, a source of resistance to *Peronosclerospora sorghi*. Plant Dis. 66:807-809.