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Inheritance of Resistance to Fusarium Wilt in Cotton[1]

A. J. Kappelman, Jr.[2]

ABSTRACT

The inheritance of resistance to *Fusarium oxysporum* f. *vasinfectum* (Atk.) Snyd. & Hans. was investigated in seven families of cotton (*Gossypium hirsutum* L.). Each family contained P_1, P_2, F_1, F_2, F_3, B_1 and B_2 generations and each was evaluated in six different greenhouse tests. Plants were grown in fumigated soils and were artificially inoculated with known concentrations of inoculum.

Additive effects were significant in 34 of the 42 tests. Dominance effects were significant in two and epistatic effects were significant in eight tests; however, additive effects also were significant in each of those cases except one. Although estimates of additive effects were of much greater magnitude than were those of dominance or epistasis, it was not possible to draw direct inferences as to the relative importance of those effects, since they were made using the generation-mean analysis. In the families studied additive gene effects most satisfactorily explained the inheritance of resistance to fusarium wilt in cotton.

Additional index words: Fusarium oxysporum f. *vasinfectum, Gossypium hirsutum* L., Additive effects, Dominance effects, Epistatic effects.

FUSARIUM wilt [*Fusarium oxysporum* f. *vasinfectum* (Atk.) Snyd. & Hans.] of cotton (*Gossypium hirsutum* L.) can cause serious losses on sandy acid soils. Losses are increased where nematodes also are present, as is frequently the case on those soils. Although soil fumigation reduces the incidence of nematodes, no satisfactory method of field control of fusarium wilt other than the use of resistant varieties has been found. However, in view of the number of investigators and the length of time devoted to breeding for wilt resistance, it is difficult to account for the present scarcity of highly resistant, commercially acceptable varieties if one accepts the assumption of a simple scheme of inheritance for resistance. The objective of this study was to reevaluate the inheritance of resistance and obtain quantitative genetic estimates relating to the inheritance of this disease in cotton.

REVIEW OF LITERATURE

The inheritance of resistance to *F. oxysporum* f. *vasinfectum* has been investigated by several workers (5, 6, 7, 8, 9). Smith and Dick (9) in field experiments concluded that resistance in American Upland cotton, *Gossypium hirstum* L., was controlled by one major dominant gene with several modifying genes. Resistance in Sea Island cotton, *G. barbadense* L., was reported to be inherited as a dominant characteristic and controlled by two genes that were additive in effect. However, the evaluation of wilt resistance in their studies was complicated by the presence of nematodes, i.e., wilt resistance and nematode resistance were confounded variables.

Kelkar, Chowdhari, and Hiremath (8) determined that resistance in *G. herbaceum* L. to the "Indian" variety of the fungus was dominant and controlled by one gene. In Upland, Hopi, Sea Island, and Egyptian crosses investigated by Jenkins, Hall, and Ware (5) resistance also was determined to be inherited as a dominant characteristic. The F_1's of the crosses studied were reported to be as resistant as the more resistant parent.

Jones (6) found that one major gene with the absence of dominance determined wilt resistance in the F_2 of a cross between 'Delfos 425' × 'Half and Half' (*G. hirsutum* L.). In later studies he (7) reported results on a study of the inheritance of resistance in several plantings of the parental F_1, F_2, and F_3 populations of the above cross as well as in a cross between 'Coker 100 Ga.' × 'Half and Half.' In the latter study neither cross gave complete dominance for resistance or susceptibility in the F_1. In addition, the continuous variation among individual F_2 plants and F_3 line means in each cross led to the conclusion that resistance to wilt should be considered as a quantitative characteristic. The frequency of recovery of parental types in the F_3 of both crosses indicated that the parents differed by at least two, but probably no more than three, gene pairs. However, the behavior of the F_2 plants and the F_3 lines of the above crosses led to the suggestion that modifying genes also were involved in the inheritance of resistance to fusarium wilt.

MATERIALS AND METHODS

Seven families derived from eight inbred lines of cotton (Table 1) were evaluated for resistance to *F. oxysporum f. vasinfectum*. These lines had been bulk self-pollinated for six to eight generations prior to use in this test so that they were considered essentially homozygous. Each family was composed of the following generations: P_1, P_2, F_1, F_2, F_3, B_1, and B_2. The P_2 parent of all families was a homozygous susceptible selection from Hurley's 'Rowden.'

A randomized complete-block design with six replications was used for each family. Ten plants per plot were spaceplanted in rows on greenhouse benches (5 cm between plants in a row and 15 cm between rows) containing Cahaba sandy loam fumigated with methyl bromide. Six tests, each involving all families, were conducted during the period of July 1969

[1] Contribution from the Plant Science Research Division, ARS, USDA, and the Department of Agronomy and Soils, Auburn University Agricultural Experiment Station, Auburn, Ala. Received Feb. 19, 1971.

[2] Research Pathologist, Plant Science Research Division, ARS, USDA, in cooperation with the Dept. of Agronomy and Soils, Auburn Univ. Agr. Exp. Sta. Auburn, Ala. 36830.

to July 1970. Seed were hot-water treated at 80 C for 90 sec and then germinated at 30 C for approximately 20 to 24 hours prior to planting; thus, the seedlings were physically quite uniform. All tests were conducted under ambient greenhouse conditions. Air temperatures, therefore, varied greatly among tests with extremes of 24 to 40 C in the daytime and 16 to 24 C at night.

Plants were inoculated in a manner similar to that described by Bugbee and Presley (1). Two inoculations per plant were made at about the 1.5-cm aboveground level on the stem using a 23-gauge needle. Inoculum was a 1:1 mixture of two fusarium isolates. 3.5024 and R93-922, and contained approximately 3×10^6 microconidia/ml. Plants were inoculated 4 weeks after emergence and rated 2 weeks after inoculation. Plants exhibiting any foliar symptoms whatsoever were considered susceptible. The percentage of wilted plants per entry was then calculated. Plot means were converted using the arcsin transformation, and all populations were then analyzed using Hayman's (2) generation-mean analysis.

Expectations of the means of two inbred lines and their descendants from selfing and crossing have been described (4) as follows:

$$E(\overline{Y}_k) = m + \alpha_k d + \beta_k h + \alpha^2_k i + 2\alpha_k\beta_k j + \beta^2_k l$$

where $E(\overline{Y}_k)$ is the expectation of the mean of kth generation, m is a constant, α_k and β_k are coefficients dependent upon the generation, d = pooled additive effects, h = pooled dominance effects, i = pooled additive by additive effects, j = pooled additive by dominance effects, and l = pooled dominance by dominance effects.

Generation means of each family were analyzed using least-squares regression techniques. Increasingly complex models were fitted until all significant variation could be accounted for. Models fitted were:

Model 1 = $\overline{Y}_k = m + E_1$,

Model 2 = $\overline{Y}_k = m + \alpha_k d + E_2$,

Model 3 = $\overline{Y}_k = m + \alpha_k d + \beta_k h + E_3$,

Model 4 = $\overline{Y}_k = m + \alpha_k d + \beta_k h + \alpha^2_k i + E_4$,

Model 5 = $\overline{Y}_k = m + \alpha_k d + \beta_k h + \alpha^2_k i + 2\alpha_k\beta_k j + E_5$, and

Model 6 = $\overline{Y}_k = m + \alpha_k d + \beta_k h + \alpha^2_k i + 2\alpha_k\beta_k j + \beta^2_k l + E_6$

where E is the appropriate error for the given model.

RESULTS

Mean wilt percentages of the generations in all families (Table 2) varied from 13.2 to 79.6%. Although the F_1 means were approximately equal to the midparent value of its family, in all cases MP-F_1 values were positive, possibly indicating a tendency toward a lower F_1 susceptibility. Both F_2 and F_3 means were similar to that of the F_1 in their respective families. Backcrosses to the more resistant parent had means that averaged about 45% of the distance between the F_1 and the P_1 means, while backcrosses to the P_2 averaged only about 23% of the distance from the F_1 toward the more susceptible parent.

The pooled additive effects (mean squares for d) were significant ($P \leqslant .05$) in 34 of the 42 tests. Mean squares for the pooled dominance effects (h after adjustment for d) were significant ($P \leqslant .05$) in family 2 $\times$ 8, test 3 and in family 7 $\times$ 8, test 4. Epistatic effects were significant in eight tests. Pooled additive by additive effects were significant in family 1 $\times$ 8, test 4 ($P \leqslant .05$); in family 3 $\times$ 8, test 2 ($P \leqslant .05$); in family 6 $\times$ 8, test 2 ($P \leqslant .01$) and test 3 ($P \leqslant .05$); and in family 7 $\times$ 8, test 4 ($P \leqslant .05$). In addition, pooled additive by dominance epistatic effects in family 7 $\times$ 8, test 6 ($P \leqslant .05$); pooled dominance by dominance epistatic effects in family 1 $\times$ 8, test 5 ($P \leqslant .05$); and in family 2 $\times$ 8, test 4 ($P \leqslant .05$) also were significant. Dominance and epistatic effects although significant might not be real in that using a probability of .05, 1 time out of 20 a difference would be declared significant when, actually, it was not. In all cases except one where dominance or epistatic effects were significant, additive effects also were significant.

R^2 values (the proportion of the corrected sum of squares among generation means accounted for by a given component of variance) were calculated for all populations. In general, additive effects accounted for the greatest proportion of the genetic effects among generation means involved in resistance to fusarium wilt. However, since previous research has shown dominance gene action to best explain resistance to fusarium wilt, a comparison of R^2 values and levels of significance for additive and dominance effects as determined by studies herein are presented (Table 3).

DISCUSSION

Additive, dominance, and epistatic effects all were involved in the inheritance of resistance of cotton to fusarium wilt. However, in 34 of the 42 tests additive effects significantly affected the the inheritance of resistance. In two tests dominance effects and in eight

Table 1. Parental code and pedigrees of lines used to form the seven families studied.

Parental code	Pedigree (or designation)
1	Auburn 56 sel. DB
2	(DeKalb 108-5 × Auburn 56-49)-2 B_3F_7
3	Atlas 92 × Rex-4 B_3F_7
4	1059-27 Wiles
5	1059-31 Wiles
6	Auburn 56 sel. long staple (42-5)
7	Acala 442 × Empire S_5 (42-34)
8	Hurley's Rowden sel.

Table 2. Mean wilt percentage over six tests for each generation within seven families of cotton.

	Family						
Entry	1 × 8	2 × 8	3 × 8	4 × 8	5 × 8	6 × 8	7 × 8
P_1	16.0	44.7	37.1	26.4	16.8	13.2	31.1
P_2	65.8	77.7	66.3	76.4	71.8	79.6	67.4
F_1	39.3	56.1	47.9	43.6	43.8	35.4	46.3
F_2	39.5	56.3	39.0	43.9	54.3	32.8	41.0
F_3	38.9	60.1	39.1	49.0	40.6	32.3	47.0
B_1	23.5	50.2	37.5	40.0	29.7	26.8	48.0
B_2	49.0	58.1	46.1	52.0	47.3	49.9	58.2

Table 3. R^2 values indicating the proportion of the phenotypic variability among generation means attributable to additive and dominance effects.

	Family													
	1 × 8		2 × 8		3 × 8		4 × 8		5 × 8		6 × 8		7 × 8	
Test	A†	D‡	A	D	A	D	A	D	A	D	A	D	A	D
1	94.08**	0.34	65.27**	14.81	78.80**	1.46	73.03*	12.63	73.8*	1.00	72.95*	9.68	33.57	16.18
2	93.80**	2.54	42.25**	6.90	60.12**	4.14	86.70**	5.39	80.25**	1.12	88.43**	3.61	54.07	7.50
3	81.75**	0.69	67.01*	24.19*	78.98**	0.55	80.53**	9.54	76.69*	0.18	81.05**	10.47	39.39	0.45
4	60.37*	0.41	30.82	2.25	28.07	33.02	73.08**	16.27	68.78*	14.75	70.24*	13.86	83.88**	14.64**
5	97.17**	1.15	61.55*	0.00	74.60*	2.50	76.41*	1.01	80.74*	5.89	84.51**	0.02	85.45**	0.42
6	95.71**	0.70	77.79**	0.50	39.71	17.71	42.75	5.21	90.00**	0.09	84.66**	3.17	69.84*	0.71

*, ** Significant at the 0.05 and 0.01 levels of probability, respectively. † Additive effects. ‡ Dominance Effects

tests at least one type of epistatic effects also were found significant. However, in all of these tests except one, additive effects also were significant. Thus, in general, additive effects satisfactorily explained the majority of the inheritance of resistance to fusarium wilt in these crosses.

Results from previous studies [Smith and Dick (9), Kelkar et al. (8), and Jenkins et al. (5)] indicated that resistance to fusarium wilt of cotton was inherited as a dominant characteristic. However, all of these were field studies. Smith and Dick (9) conducted their studies in the presence of only reduced nematode populations, which might have complicated the true gene action of resistance to only the fusarium wilt organism. Jenkins et al. (5) felt that the varying infestation levels of different areas of the field where their studies were conducted might have affected the accuracy of their results. In contrast to these earlier studies, results presented herein were obtained from greenhouse plants grown in fumigated soil and inoculated with known amounts of fusarium microconidia. While pooled additive effects were significant in 34 of the 42 tests, this does not necessarily mean that the genes involved act in the additive genetic manner (in the classical sense).

Parameters obtained in the present study were estimated from means; thus, it was not possible to draw direct inferences about the relative magnitudes of additive and dominance effects. The distribution of genes having positive and negative effects results in varying degrees of concellation of effects in parameters estimated from means (10). Results obtained could also be biased due to the failure of cotton to meet one of the basic assumptions of generation mean analysis; that is, diploid inheritance. Even though American Upland cotton is a tetraploid, it behaves as an amphidiploid; and most qualitative traits studied thus far have been inherited as disomics. Parents were selected for either resistance or susceptibility to fusarium wilt. The P_2 parent of all families, Rowden, was the same. The variability in mean wilting of this parent in the different families could be due to several causes. First, these wilting percentages (Table 2) are means from six individual tests conducted over the period of a year under ambient greenhouse conditions. The variation in temperature could have greatly influenced disease development; thus, final wilting percentage. Secondly, each replication of each family was a randomized complete-block. Since plots were single rows, various degrees of competition may have existed and influenced wilting. Therefore, wilt percentages of the P_2 parent were not expected to be identical, as indeed they were not.

In addition, the P_1 parents of several families were related due to selection from or crossing with 'Auburn 56.' However, 13.2% of the most resistant parent in family 6 $\times$8 (averaged over all tests) were susceptible while 44.7% of the plants of the resistant parent in family 2 $\times$ 8 were lost because of susceptibility to fusarium wilt. Thus, families studied could not be considered as only the products of resistant $\times$ susceptible parents. Each of the above P_1 parents in families 2 $\times$ 8 and 6 $\times$ 8 contained Auburn 56 in their background. Earlier studies (9), in which resistance was found to be inherited as a dominant characteristic, were based only on two families. In both of those families Rowden was used as the susceptible parent, while in one of them 'Cook 307' (a parent of Auburn 56) was used as the resistant parent.

Where epistasis is a major factor, Hayman (3) has shown that no unbiased measurement of pooled additive or dominance effects can be obtained by using generation mean analyses, although this difficulty can be overcome when epistasis is relatively low. Epistatic effects were found significant but in only eight of the 42 tests; therefore, measurements for d and h are probably satisfactory.

Inheritance of fusarium wilt resistance was predominantly additive in the populations tested. Since cotton breeders have worked for many years to transfer resistance into commercial varieties apparently without a high degree of success, resistance either must be controlled by several genes or adequate evaluation procedures have limited progress. Studies to determine the number of genes involved in the inheritance of resistance to fusarium wilt in these familes currently are underway.

Since additive effects significantly contributed to inheritance of resistance to fusarium wilt, breeding for resistance is somewhat more difficult than if resistance were controlled by a single dominant gene. Differences between varieties indicate that some progress can be made through selection among varieties and utilization of the most resistant stocks. Increased resistance probably should occur among the F_1's from crosses between resistant varieties. Where additive effects are important, the backcross method of breeding is not too effective but is not precluded if selections are made in the F_3 or F_4 generations following crossing and if progeny testing is practiced. However, since additive effects were significant in most cases, additional resistance might be obtained from some type of recurrent selection program.

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