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Inheritance of resistance to reddish wilting in cotton

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ABSTRACT - *The control of genetic resistance to the anomaly reddish wilting in cotton was studied using diallel crosses, by the method of Hayman, and also by specific designs for analyses of means and variances, by the method of Mather and Jinks. Results indicated that the resistance to this anomaly is predominantly due to additive effects, based on the action of three to four genes. The mode of gene action and good heritability level of the factors concerned indicated a high probability of success in the selection for resistance to this anomaly.*

Key words: cotton, reddish wilting, genetic resistance mechanism.

INTRODUCTION

The anomaly reddish wilting was first detected in the region of Londrina, state of Paraná, in the growing season 1992/93. One year later it already affected plantations across practically all cotton-producing regions of the states of Paraná, São Paulo, Minas Gerais and Goiás, where the greatest part of the production in the southern cultivation region of this fiber in Brazil was then concentrated. This meant not only damages to the plantations, but also a barrier in the studies of genetic improvement of cotton, as in the program developed by the Instituto Agronômico de Campinas, where around 50% of the study germplasm was affected by the anomaly at the time.

While the dynamics of occurrence of this anomaly became quite clear as the anatomic alterations (Queiroz–Voltan 1995), the symptomology and effects on the fiber and seed yield and quality of the affected plants (Chiavegato et al. 1994, Fuzatto et al. 1997a), all attempts to determine its nature and causes were to date unsucessful. In spite of some similarity with a disease observed in Argentina (Follin and Campagnac 1981), supposedly caused by a virus and transmitted by aphids, the hypothesis that the Argentinean and the Brazilian anomaly are the same was not confirmed, although the manifestation of the anomaly in Brazil is always associated to the incidence of aphids as well, mainly at high degrees, in the affected plantations. Evidence that it could be the disease bronze wilt, caused by species of *Agrobacterium* biovar I, as reported by Bell (1999, 2000) was not found either.

Still, whatever the cause– biotic or abiotic –the evidences of genetic diversity for resistance to this anomaly among cultivated or study genotypes in Brazil were unquestionable (Cia et al. 1999, Fuzatto et al. 1997b). This framed the solution of the problem through the use of resistant cultivars, which required either an identification of such cultivars for immediate use, or

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their development by adequate methods of genetic improvement. For this latter purpose, studies were performed to gain insights on the mechanisms involved in the inheritance of resistance to this anomaly, whose results are reported here.

MATERIAL AND METHODS

In the growing season 1993/94, a high but differentiated incidence of "reddish wilting"occurred in a trial of lines set up in Ribeirão Preto, state of São Paulo, as part of the genetic improvement program of cotton conducted by the Instituto Agronômico de Campinas. Three of these lines, with on average zero, 42 and 80% affected plants, respectively, were then collected for the establishment of a complete diallel mating system. The nine resulting treatments (parentals, F_1 and reciprocal) were studied in the growing season 1995/96, in Londrina-PR, in a field experiment arranged in random blocks with four replications. The arcsin $\sqrt{\%}$ - transformed data of percentage of affected plants underwent analysis of variance and were analyzed by Hayman (1954)'s methodology for the determination of genetic parameters.

In a next stage, the two most contrasting lines of the previous experiment were used in a scheme aiming at the genetic analysis of means and variances. The six populations obtained (parental, F_1, F_2, RC_1 and RC_2) were studied in an experimental design of complete random blocks, with three replications, conducted in Campinas, in the growing season 1996/97. In individual evaluations, the plants were rated by grades from 1 to 5, increasing with the symptom intensity, as proposed by Chiavegato et al. (1994). For the statistical analyses the data were $\sqrt{x}$ -transformed, using the method of Mather and Jinks (1977), to evaluate nature and magnitude of the gene effects concerned. To estimate the number of genes participating in the trait expression, the additive variance and data amplitude in the F_2 generation were used, as indicated by Cruz and Regazi (1997).

The experimental plots in both experiments consisted of one 5 m long row each, with 25 to 35 plants, according to the population. Aside from the deliberate absence of aphid control, to warrant the occurrence of the anomaly, the cultural practices were the ones normally used in experiments with cotton genotypes.

RESULTS AND DISCUSSION

Diallel experiment

The results obtained with the parental and F_1 populations, expressed in percentages of plants without anomaly symptoms, are shown in Table 1. Since the differences between the reciprocal were not significant –0.31, 6.41 and 6.93, respectively, for the three crosses, in view of the minimum significant difference of 19.31, by the Tukey test at 5% – their means are presented in each cross. For each parental the values Wr + Vr (respectively covariance between the values of the genotypes and those of their crosses, and variance of the rows in the diallel table) are also presented.

With exception of parent $\mathbf{P_2}$, which was just as susceptible as $\mathbf{P_3}$, the data of this experiment confirmed the original trial of lines. The analysis of variance presented highly significant differences between the genotypes (F = 9.77), discriminating, by the test Tukey at 5%, parent P_1, from P_2 as well as from P_3. The variation coefficient was 13.3% and the relation CVg/CVe was 1.71. The intraclass correlation of $r_I = 0.74$, presented a reasonable data consistency.

The equation Wr = 72.68 + 0.96 Vr (F = 70.1) was established in the diallel analysis and the regression coefficient in the sufficiency test of the model did not differ from the unit (t = 0.43). This evidenced an absence of epistatic effects and an agreement with the additive/ dominant model. The data of the genetic parameters obtained in this analysis are shown in Table 2. The results demonstrate the predominance of the additive effects in the genetic control of the trait. The coefficients of genotypic determination (narrow and broad-sense heritability) were, respectively, 0.90 and 0.92, which shows the high heritability of the trait. The dominance effect, besides its small magnitude (mean degree = 0.28) and statistical insignificance, was not coherent. Actually, the comparison of the values Wr + Vr and the means (Table 1) showed that in the P_1 x P_3 cross, the dominance effect is associated to resistance to reddish wilting, while in cross P_1 x P_2 the dominance occurred for susceptibility. This fact also impaired the analyses

Table 1. Mean percentage of cotton plants without symptoms of reddish wilting in populations of a diallel cross and Wr+Vr values for the parental

Genotypes	Means	Wr + Vr
P_1	92.3	305.1
P_2	47.7	171.6
P_3	45.1	359.3
$P_1 \times P_2$	63.5	——
$P_1 \times P_3$	76.5	——
$P_2 \times P_3$	38.9	——

Table 2. Genetic and statistical parameters[1] obtained in a diallel system for the study of reddish wilting in cotton

	Estimate	Variance	t value
E	15.71	6.2553	
D	315.79	25.0211	51.55*
H_1	25.16	252.9906	1.29
H_2	20.10	225.1895	1.09
h^2	-1.74	105.0266	-0.14
F	-39.66	177.9275	-2.42*
$D-H_1$	290.63	211.2889	19.98 (<0.05)

[1] E= Component of the environmental variation; D = Additive variance; H_1= Dominance variance; H_2= Dominant component with corrected allele distribution in the parental; h^2= Sum of the heterozygous effects; F= Covariance between the additive and dominant genetic effects

Table 3.Mean grades[1] and variances obtained in the study of the genetic resistance mechanism to reddish wilting in cotton

Generation	N	Mean	Variance
P_1	124	1.0711	0.0303
P_2	142	1.3963	0.1923
F_1	82	1.1817	0.0801
F_2	132	1.1999	0.1062
RC_1	103	1.0968	0.0413
RC_2	96	1.2065	0.1184

[1] grades from 1 to 5, increasing with rising symptom intensity, transformed by $\sqrt{x}$

Table 4. Genetic parameters[1] obtained in the mean analysis performed in the study of resistance to reddish wilting in cotton

Effect	Estimate	t value	R^2%
m	1.4271	10.15 **	57.32
a	0.162	68.13 **	36.46
d	-0.6631	-1.96 *	2.14
aa	-0.1934	-1.38	1.07
ad	0.1057	1.17	0.77
dd	0.4177	1.98 *	2.23

[1] m= mean of the homozygous of all genes; a= additive effects; d= dominance effects; aa= additive x additive interactions; ad= additive x dominant interactions; dd= dominant x dominant interactions

of other genetic information that depends on the parameters associated to the dominance effects. Lastly, it must be emphasized that the efficacy of Hayman´s methodology can be affected when a small number of parentals is used, as in the present case.

Experiment with parents and segregating generations

This experiment was somewhat impaired by an inconsiderate pesticide application, which controlled the aphids before the plague had infested the entire trial – as desired – intensely and evenly. The possible consequences of this fact are discussed below. The analysis of variance of the mean grades of the plots presented CV = 8.73, r_I = 0.55 and a ratio CVg/CVe = 1.10, which indicates reasonable precision and consistency of the trial. The F value for genotypes was 5.89** and Tukey's test at 5% confirmed the difference of resistance among the parents and also among the susceptible parental and the backcross to the resistant parent. The contrasts $Ya = 2RC_1 - P_1 - F_1$, $Yb = 2RC_2 - P_2 - F_1$ and $Yc = 4F_2 - 2F_1 - P_1 - P_2$, with t values of 0.88, 0.22 and 0.16, respectively, were not significant, which showed the absence of epistatic effects, as presumed by the method used for genetic analysis of the means and variances (Mather and Jinks 1977).

In the genetic analysis of the means (Tables 3 and 4), the results were similar to those obtained in the diallel experiment. The additive effects were predominant in the genetic control of the trait (Table 4). The results of dominance, despite within the limit of significance, were of small magnitude (mean degree = 0.32). Furthermore, they occurred in the direction of susceptibility, an opposite result to the one obtained in the cross of these same parents in the diallel experiment. This confirms the above restrictions on any inferences based on the dominance effects in the data analysis of these experiments.

The genetic analysis of the variances (Table 5) presented the same tendencies as before, especially the

Table 5. Genetic parameters of F_2 population, obtained in the analysis of variances in a study of the genetic resistance mechanism of cotton to reddish wilting

Parameter	Estimate
Phenotypic variance	0.1062
Environmental variance	0.0303
Genotypic Variance	0.0759
Additive variance	0.0539
Variance of Dominance	0.0220
Broad-sense heritability	0.72
Narrow-sense heritability	0.51
G.M.D. (based on variances)	0.90
G.M.D. (based on means)	0.32
Nr. of Genes (based on variances)	3.54

predominance of additive effects, though in lower magnitude. Some data are, however, questionable, such as the discrepancy in the values of the variances of the parental and F_1 populations, which should basically express only the environmental effect. Likewise, the mean degree of dominance of 0.90 is not concordant with the means presented by the parents and F_1 generation. A possible explanation for the lower precision of these data would be the above-mentioned inhomogeneity of the aphid incidence in the experiment, and consequently of the occurrence, also irregular, of the anomaly under study. This would introduce an error into the evaluations that could affect the different populations irregularly, especially the most susceptible ones. In fact, the most discrepant variance is the one of the susceptible parent (P_2) (Table 3). The variance of the resistant parental was therefore adopted - which did not differ much from the residual mean squares in the analysis of variance of the plot means– was therefore adpted to represent the enviromental effect in in the genetic analyses. Incidentally, problems in the use of the environmental variance in studies of this type have already been described by Warner (1952), who proposed a method of estimating heritability without the cited parameter. It is noteworthy that this method, applied to the data of the present experiment, resulted in a similar estimate of the narrow-sense heritability (0.50) as obtained with the adopted procedure (0.51). Finally, one must not forget the difficulty of one-time evaluations, bearing in mind that the appearance of plants with the anomaly in the same population can last up to three weeks, so during this period, plants with initial symptoms and others that have already recovered from the disease grow side by side. Obviously, the errors caused by the described events, above all in the plant-by-plant evaluation, affect the variances more than the means. This is why special care must be taken in the interpretation of the genetic information derived, in the present case, from the study of variances.

Summing up, bearing the reservations discussed above in mind, the experiments reported here allow the conclusion that: a) the resistance of cotton to the anomaly reddish wilting is an inheritable trait, predominantly controlled by additive genetic effects; b) three to four genes participate in the trait control; c) the mode of gene action and the good heritability level of the factors concerned indicate a good chance of success in the selection for resistance to this anomaly.

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