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# Inheritance of resistance to *Colletotrichum gossypii* var. *cephalosporioides* in cotton

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**ABSTRACT** - *The objective of this study was to analyze the inheritance of the resistance to cotton ramulosis. For this purpose, two groups of lines with contrasting performance for the evaluated trait were crossed. The disease-susceptible parents were Delta Opal, CNPA 999 and CNPA 2161, and those with resistance BRS Facual, CNPA 2043 and CNPA 2984, resulting in nine crosses, always of one resistant and one susceptible parent, totalizing 42 treatments. The experiment was set up in a randomized complete block design with three replications. It was verified that the genetic control of ramulosis resistance is predominantly oligogenic, and the number of genes involved depends on the parents that participate in each cross, due to the possibility of differential loci fixation. Evidence of partial dominance in the sense of increasing disease resistance was found, but there were also indications that dominance is not unidirectional.*

**Key words**: cotton, ramulosis, genetics control, *Gossypium hirsutum*.

## INTRODUCTION

One of the major cotton diseases in Brazil is ramulosis, caused by *Colletotrichum gossypii* var. *cephalosporioides* (Costa and Fraga Junior 1939). It occurs in all cotton producing areas in the Midwest region of the country, since the environmental conditions of this region are favorable for the development of the disease, which is restricted to South America (Malaguti 1955, Mathieson and Mangano 1985, Lima et al. 1999). The etiologic agent attacks the entire above-ground part of the plant, in all development stages. If it occurs in the early growth period, losses in fiber production can reach up to 80% (Lima et al. 1999). Symptoms of this disease are necrotic lesions in young tissues, mainly of leaves, and at more advanced stages, the uncontrolled growth of lateral shoots, caused by the loss of apical dominance when the apical meristem of the plant is affected (Suassuna and Coutinho 2007).

The management of ramulosis involves cultural practices, particularly crop rotation, as well as the use of pathogen-free seed, chemical seed treatment and fungicide application to the above-ground plant parts. However, crop rotation is not always implemented, resulting in an increase in initial inoculum for the following growing seasons. In these cases chemical control is the only practice used to control the disease. The use of genetic resistance is a quick and cost-effective disease control for cotton (Lima et al. 1984). In Brazil, studies to obtain varieties resistant to ramulosis were initiated soon after the first report of the disease

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in the State of Sao Paulo in 1936 (Costa and Fraga Junior 1939).

To understand the inheritance of resistance to cotton ramulosis, Carvalho et al. (1988) crossed a line selected from the resistant cultivar HR21 T 16 and the highly susceptible genotype SU 0450-8909. They concluded that susceptibility is conferred by an allele with partial dominance, and the mean dominance degree was found to be 0.95 and heritability 0.51. Another important result of this study was that the leaf hairiness is positively correlated with susceptibility to the disease. Genotypes without or with little intensity of hair growth are more resistant. Carvalho et al. (1994) corroborated the previous results, using four cultivars of upland cotton (*Gossypium hirstum* L.). Crosses of a resistant variety (HR 102) with three susceptible lines (SU0450-8909, CNPA 3H, IAC 20) were performed, and diallel analysis by the model of Hayman (1954) showed that susceptibility is controlled by dominant alleles. Apart from that, no effect of extra-nuclear genetic factors was observed and the environmental influence on phenotypic variation was relatively low. Zandoná et al. (2006) also studied the inheritance mechanism of resistance to ramulosis, in two resistant cultivars (BRS Antares and IAC 23), crossed with the susceptible cultivar Stoneville 474. They found that resistance in the cross involving the BRS Antares was conditioned by a dominant allele, and in the cross with IAC 23 by dominant alleles in two independent genes with duplicated effect.

Therefore there is no consensus about the number of genes involved in controlling the trait, nor if resistance is controlled by dominant(s) and/or recessive(s) allele(s). In this context, the objective of this study was to analyze the inheritance pattern of genetic resistance to ramulosis of upland cotton, by biometric approaches based on components of means and variances of generations.

## MATERIAL AND METHODS

The crosses were performed at the experimental station of the Secretaria da Agricultura Pecuária e Abastecimento do Estado de Goiás (Seagro, GO), in Senador Canedo, GO. Two groups of lines/cultivars with contrasting performance for the trait were intercrossed. The parents susceptible to ramulosis were cultivar Delta Opal and the experimental lines CNPA 999 and CNPA 2161. The parents with high resistance level to the disease were the cultivar Facual and the test lines CNPA 2984 and CNPA 2043. It was therefore possible to obtain nine hybrid combinations, crossing only resistant with susceptible parents. Upon hybridization, an open bulk of each $F_1$ plant was collected, and the seeds were sown for selfing and to obtain $F_2$ plants. The backcrosses $BC_1$ and $BC_2$ were performed at the same time.

After establishing the generations, a field experiment was installed at the Experimental Station of the Fundação Goiás in Santa Helena de Goiás (lat 17.8 º S, long 50.6 ° W, 485 m asl). The test was arranged in a randomized complete block design with 42 treatments (the generations $F_1$, $F_2$, $BC_1$ and $BC_2$ of each cross, plus six parents) and three replications. Seeds were sown on December 28, 2007. The plot sizes differed according to the test generation and the genetic variability expected in each. Plots of two 5-m rows were therefore used for the parents and for each $F_1$ and backcross generation, and plots of four 5-m rows for the $F_2$ generations. The spacing between rows was 0.9 and the plant density nine plants per meter.

The plants were artificially inoculated with inoculum suspension ($5 \times 10^4$ conidia $mL^{-1}$) five times, the first on February 12, 2008, and the others at weekly intervals. The inoculum suspensions were prepared with three isolates of the pathogen from the municipalities of Acreúna - GO (CNPA 0095), Santa Helena de Goiás - GO (CNPA 0104) and Campo Verde - MT (CNPA 0116). The isolates were cultured on potato dextrose agar (PDA) in Petri dishes. The dishes were maintained in an incubator at 25 °C, photoperiod of 12 h. After ten days, 10 mL of sterile distilled water were added to each plate, and the conidia removed carefully with a brush.

Severity was evaluated on April in 13, 2008, assessing all plants of each plot (a total of 10,720 observations) based on the score/grade scale proposed by Suassuna et al. (2008), where 1 = no symptoms, 2 = necrotic lesions on young leaves, 3 = leaf lesions, shortened internodes and early growth of lateral shoots (up to three new shoots), 4 = leaf lesions, shortened internodes, lateral shoot growth (between three and ten shoots), little vegetative growth, and 5 = leaf lesions, shortened internodes, lateral shoot growth (more than 10 shoots) and dwarfism. Data from the test were subjected to analysis of variance, using the following statistical model:

$Y_{ijk} = m + b_j + t_i + e_{ij} + p_{(k)\,ij}$

where:

$Y_{ijk}$: measured value of plant k, of treatment i in block j;

m: overall mean;

$t_i$: fixed effect of treatment i;

$b_j$: random effect of block j;

$e_{ij}$: experimental error associated with the ij$^{th}$ plot, assumedly i.i.d.~ N(0, $\sigma^2$); and

$p_{(k)\,ij}$: effect of plant k in the ij$^{th}$ plot.

To detail the treatment effects, the degrees of freedom (DF) and sums of squares (SS) associated with these effects were orthogonally decomposed in effects of cross and crosses within-generation. The former were also decomposed in effects of parents, parents vs. progenies and progenies. The effects of crosses within-generation were however decomposed for each cross, which, individually, were also investigated in the following contrasts: $F_1$ vs $F_2$, $F_1$ vs $BC_1$, $F_1$ vs $BC_2$. These contrasts provide information related to the trait inheritance and can deepen the understanding of its genetic control. The analysis was performed using SAS software (SAS Institute 2002).

The full statistical model used to estimate the additive genetic effects (*a*) and dominance (*d*), including the effects of epistasis (*aa*, *ad*, *dd*), was the one proposed by Mather and Jinks (1984). It was adjusted for each cross, based on the method of weighted least squares (Rowe and Alexander 1980). The components of genetic variance ($\sigma_g^2 = \sigma_A^2 + \sigma_D^2$, where $\sigma_A^2$ is the additive variance associated with mean gene effects and $\sigma_D^2$ the variance of dominance associated with effects of allelic interaction) and the environmental variance ($\sigma_e^2$) were estimated by the method of weighted least squares. The iterative procedure was used, due to the use of a weight matrix in the system of normal equations, which are functions of variances and covariances between and within progenies.

The mean dominance degree (MDD) was estimated by means of two expressions, as described by Cruz and Regazzi (1997):

$$MDD_v = \sqrt{\frac{2\hat{\sigma}_D^{\,2}}{\hat{\sigma}_A^{\,2}}} \quad \text{and} \quad MDD_m = \frac{2\overline{F}_1 - \left(\overline{P}_1 + \overline{P}_2\right)}{\overline{P}_1 - \overline{P}_2}$$

The use of the two expressions, based on the variances ($MDD_v$) and means ($MDD_m$), is justified since the concept of the first, despite more widespread, provides no information about the direction of dominance, and also make it impossible to estimate the degree of dominance in cases of negative estimates of the variances involved.

The estimates of the coefficient of determination in the broad and narrow senses were also obtained, with their standard errors, by the expressions proposed by Cruz and Regazzi (1997):

$$\hat{h}_a^2 = \frac{100\hat{\sigma}^2_{g(F_2)}}{\hat{\sigma}^2_{F_2}} \quad \text{and} \quad \hat{h}_r^2 = \frac{100\hat{\sigma}^2_{A}}{\hat{\sigma}^2_{F_2}}\ ; \text{ with:}$$

$$s\left(\hat{h}_a^2\right) = \left\{\left(\frac{2}{9}\right)\left[\left(\frac{1}{\left(\hat{\sigma}^2_{F_2}\right)^2}\right)\left(\frac{\left(\hat{\sigma}^2_{Pi}\right)^2}{n_1+2} + \frac{\left(\hat{\sigma}^2_{Pj}\right)^2}{n_2+2} + \frac{\left(\hat{\sigma}^2_{F_1}\right)^2}{n_3+2}\right) + \left(\frac{1}{n_4+2}\right)\left(3-\hat{h}_a^2\right)^2\right]\right\}^{\frac{1}{2}}$$

$$s\left(\hat{h}_r^2\right) = \left\{2\left[\left(\frac{1}{\left(\hat{\sigma}^2_{F_2}\right)^2}\right)\left(\frac{\left(\hat{\sigma}^2_{Rc1}\right)^2}{n_5+2} + \frac{\left(\hat{\sigma}^2_{Rc2}\right)^2}{n_6+2}\right) + \left(\frac{1}{n_4+2}\right)\left(2-\hat{h}_r^2\right)^2\right]\right\}^{\frac{1}{2}}$$

Finally, the number of genes (k) involved in the genetic control of the trait was estimated by the expression proposed by Burton (1951), considering the probable existence of dominance effects:

$$k = \frac{\frac{1}{4}\left(\frac{3}{4} - \mu + \mu^2\right)\left(\overline{P}_2 - \overline{P}_1\right)^2}{\sigma^2 F_2 - \sigma_e^{\,2}}\ ;$$

where: $\mu = \dfrac{\left(4\overline{F}_2 - 3\overline{P}_1 - \overline{P}_2\right)/2}{\overline{P}_2 - \overline{P}_1}$.

The use of this estimator requires some assumptions such as: completely contrasting parents, absence of epistasis, absence of linkage; assessment in similar environmental conditions (Ramalho et al. 1993). Therefore, in view of the possible deviations from these assumptions, the results should not be considered definitive.

## RESULTS AND DISCUSSION

The genotypes differed in ramulosis severity (Table 1), indicating the existence of wide genetic variation between treatments ($p \leq 0.01$) for resistance to *C. gossypii* var. *cephalosporioides*. The mean score was 2.09, which shows predominance of genotypes with some level of disease resistance (1 to 5 scale, where score 1 = no symptoms and 5 = full expression). This was most likely due to the chosen parents, which were

**Table 1.** Summary of the analysis of variance for the variable score of ramulosis severity, in an inheritance study of genetic resistance in cotton, including estimates of mean contrasts ($\hat{Y}$) and probabilities of significance of statistical tests (F-Snedecor for the sources of variation with more than one degree of freedom, and t-Student, for those with only one degree of freedom)

| Source of variation | DF | MS | $\hat{Y}$ | p-value |
|---|---|---|---|---|
| Blocks | 2 | 2.899 | - | 0.0118 |
| Treatments | 41 | 17.284 | - | <0.0001 |
| Crosses (including parents) | 14 | 20.685 | - | <0.0001 |
| Parents vs Progenies | 1 | 3.858 | -0.0682 | 0.0037 |
| Parents (G) | 5 | 29.806 | | <0.0001 |
| Susceptible (S) | 2 | 19.399 | | <0.0001 |
| Resistant (R) | 2 | 18.084 | | <0.0001 |
| S vs R | 1 | 105.022 | 0.675 | <0.0001 |
| Progenies (D) | 8 | 29.806 | | <0.0001 |
| Generation (Cross) | 27 | 13.172 | | <0.0001 |
| Delta Opal x BRS Facual | 3 | 9.430 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 4.622 | -0.182 | 0.0015 |
| $F_1$ vs $Bc_1$ | 1 | 9.497 | -0.302 | <0.0001 |
| $F_1$ vs $Bc_2$ | 1 | 2.498 | 0.153 | 0.0196 |
| Delta Opal x CNPA 2043 | 3 | 31.256 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 73.791 | -0.704 | <0.0001 |
| $F_1$ vs $Bc_1$ | 1 | 1.956 | -0.137 | 0.0389 |
| $F_1$ vs $Bc_2$ | 1 | 11.708 | -0.341 | <0.0001 |
| Delta Opal x CNPA 2984 | 3 | 8.374 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 3.723 | -0.161 | 0.0044 |
| $F_1$ vs $Bc_1$ | 1 | 20.288 | -0.457 | <0.0001 |
| $F_1$ vs $Bc_2$ | 1 | 0.008 | -0.010 | 0.8948 |
| CNPA 999 x BRS Facual | 3 | 15.613 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 21.302 | 0.362 | <0.0001 |
| $F_1$ vs $Bc_1$ | 1 | 2.669 | -0.165 | 0.0158 |
| $F_1$ vs $Bc_2$ | 1 | 9.683 | 0.296 | <0.0001 |
| CNPA 999 x CNPA 2043 | 3 | 14.543 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 39.980 | -0.574 | <0.0001 |
| $F_1$ vs $Bc_1$ | 1 | 26.635 | -0.544 | <0.0001 |
| $F_1$ vs $Bc_2$ | 1 | 14.017 | -0.369 | <0.0001 |
| CNPA 999 x CNPA 2984 | 3 | 18.558 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 44.501 | -0.557 | <0.0001 |
| $F_1$ vs $Bc_1$ | 1 | 9.029 | -0.284 | <0.0001 |
| $F_1$ vs $Bc_2$ | 1 | 0.893 | -0.093 | 0.1629 |
| CNPA 2161 x BRS Facual | 3 | 13.475 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 17.524 | -0.363 | <0.0001 |
| $F_1$ vs $Bc_1$ | 1 | 0.002 | -0.005 | 0.9401 |
| $F_1$ vs $Bc_2$ | 1 | 0.778 | 0.091 | 0.1928 |
| CNPA 2161 x CNPA 2043 | 3 | 3.452 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 1.727 | -0.105 | 0.0523 |
| $F_1$ vs $Bc_1$ | 1 | 0.574 | 0.072 | 0.2634 |
| $F_1$ vs $Bc_2$ | 1 | 5.137 | -0.220 | 0.0008 |
| CNPA 2161 x CNPA 2984 | 3 | 3.846 | | <0.0001 |
| $F_1$ vs $F_2$ | 1 | 2.653 | -0.139 | 0.0162 |
| $F_1$ vs $Bc_1$ | 1 | 7.348 | -0.276 | <0.0001 |
| $F_1$ vs $Bc_2$ | 1 | 0.075 | 0.028 | 0.6851 |
| Error (among) | 82 | 8.592 | - | - |
| Plant/plot/block | 10719 | 0.458 | - | - |

commercial cultivars and promising experimental lines; that is, some genotypes that were already improved (adapted) and, naturally, with a certain degree of disease resistance to regional cotton diseases. Nevertheless, the contrast between means of susceptible (S) and resistant (R) genotypes, although of small magnitude (0.675), was positive and highly significant ($p < 0.01$), indicating that this choice was adequate for the discrimination of most parents.

Statistically significant differences were found in almost all other contrasts (Table 1). Differences between some of these generations had in fact been expected due to their different allelic segregation. The parents of some hybrids were however not highly contrasting (e.g., CNPA 2161 x CNPA 2043), or influenced by environmental effects (experimental error) of major magnitude, so some pairs of means were not statistically discriminated. The cross CNPA 2161 x CNPA 2043 was excluded from the analysis of the genetic components of means and variances, because the estimated difference between the means of both parents was not significant.

Considering only one locus with two alleles (A and a), and assuming the condition of complete dominance in the $F_1$ generation of crosses between contrasting lines in diploid species, heterozygous plants are expected, which will have one of the alleles of the dominant parent $P_i$ (allele A) and the other of the recessive parent $P_j$ (allele a); that is, a genotypic constitution Aa. Assuming also that the allele for resistance (A) dominates the susceptibility allele (a), the occurrence of only one phenotype in the $F_1$ and two phenotypes in the $F_2$ generation can be explained, with the genotypic proportion of 3A_: 1aa. With the result of the cross test (first backcross for the parent with recessive alleles), the proportion is expected to be 1Aa: 1aa. In this case, the phenotype of the progenies depends only on the expression of alleles present in the gametes of heterozygous plants of $F_1$ generation (Ramalho et al. 2004).

Therefore, and considering the first parent ($P_1$) of each cross as susceptible when testing the mean contrasts $F_1$ vs $Bc_1$, under the proposed hypothesis, estimates with negative differences ($\overline{F}_1 - \overline{Rc}_1 < 0$) were expected. This result was statistically confirmed in seven of the crosses, and in the other two the expectation was not inverted either, but rather the non-detection of significance in the contrast. Taking another contrast, $F_1$ vs $Bc_2$, where the expectation under the previous hypothesis is non-significance (Aa x AA ⇛ 1AA:1Aa), it was observed that four of the nine crosses produced the expected outcome. Three others resulted in positive contrasts ($p < 0.05$) and the last two, in negative contrasts ($p < 0.05$). This may prove that the inheritance under study is not influenced by the action of complete dominance or that more genes are involved in the control of the trait.

Moreover, when analyzing the estimates of mean contrasts between the $F_1$ and $F_2$ generations, it was observed that seven of them were statistically negative ($p < 0.05$), that is, in these crosses, the $F_1$ plants are on average more resistant than the $F_2$ plants. This fact is also expected under the hypothesis of monogenic inheritance (the dominant allele determining the resistance), although it may be due to a certain degree of inbreeding depression in the expression of resistance. The occurrence of inbreeding depression in cotton is ascribed to its polyploid nature and to the development of genomes typical to autogamous plants during domestication (Young and Murray 1966).

To assess the contribution of different genetic effects derived from the generation means (*m, a, d, aa, ad, dd*), a non-orthogonal decomposition was performed, according to the method of Gauss, described by Cruz and Regazzi (1997). According to these authors, although this decomposition is not orthogonal, the estimated relative contribution (RC) indicates the importance of a particular genetic effect on the available variability in the traits studied. Thus, aside from the mean effect, the genetic effect was the most important in determining inheritance of ramulosis severity (Table 2). This was observed in all crosses, except for CNPA 2161 x BRS Facual, where the additive-additive epistatic effect was the most important (15.37%). This indicates the possibility of obtaining superior homozygous genotypes by selection from the $F_2$ generation, and satisfactory gains in the selection cycles following these crosses, given the high additive effect.

In crosses in which the addictive action prevailed, it was verified that at least one of the epistatic interactions was significantly different from zero, except in the cross Delta Opal x CNPA 2984. Thus, it can be inferred that the trait inheritance is determined by genes with additive effect, with the presence of genetic effects of dominance tending to increase ramulosis resistance, as well as epistatic effects, although small in magnitude, in seven of eight crosses (Table 2).

The estimates of genetic variance components of generations are presented in Table 3. In all crosses, the point estimates of the variance of dominance tended to be lower than the additive variance, which confirms most of the results discussed above. On the other hand, negative estimates of the dominance variance suggest that this component can be parametrically zero ($\sigma_D^2 = 0$). Therefore, these results should be interpreted with caution, since the variances are not significantly different from zero (Table 3). This may however be due to the errors associated with estimates of variance, usually very high, which reduce the power of statistical tests, contrary to the approach of mean contrasts included here (Table 1). Problems with the genetic analysis of variance were also pointed out by Fuzatto et al. (2006).

The number of genes involved in the trait control, although the estimates were affected by considerable environmental influence on the expression (on average 54%), ranged from 1 to 21 (Table 3). For these estimates one should also consider the fact that the estimator of

**Table 2.** Non-orthogonal decomposition of the sums of squares of parameters for the additive-dominant model with epistatic effects (*m*, *a*, *d*, *aa*, *ad*, *dd*), by the method of Gauss, for the trait severity to ramulosis, in crosses of upland cotton, with the respective sums of squares of the deviations and Relative Contribution (RC%) of each parameter

| **Parameters**[1] | **Delta Opal x BRS Facual** | | **Delta Opal x CNPA 2043** | |
|---|---|---|---|---|
| | **SSdeviation** | **RC (%)** | **SSdeviation** | **RC (%)** |
| *m/a.d.aa.ad.dd* | 142.96** | 41.96 | 298.23** | 54.51 |
| *a/m.d.aa.ad.dd* | 187.478** | 55.02 | 101.298** | 18.51 |
| *d/m.a.aa.ad.dd* | 2.735$^{ns}$ | 0.8 | 34.94** | 6.39 |
| *aa/m.a.d.ad.dd* | 4.568* | 1.34 | 62.98** | 11.51 |
| *ad/m.a.d.aa.dd* | 2.018$^{ns}$ | 0.59 | 35.42** | 6.47 |
| *dd/m.a.d.aa.ad* | 0.973$^{ns}$ | 0.29 | 14.29** | 2.61 |
| Total | 340.73 | 100 | 547.158 | 100 |
| **Parameters** | **CNPA 999 x BRS Facual** | | **CNPA 999 x CNPA 2043** | |
| | **SSdeviation** | **RC (%)** | **SSdeviation** | **RC (%)** |
| *m/a.d.aa.ad.dd* | 11.96** | 5.92 | 191.36** | 77.07 |
| *a/m.d.aa.ad.dd* | 101.22** | 50.13 | 42.08** | 16.95 |
| *d/m.a.aa.ad.dd* | 33.85** | 16.76 | 0.005$^{ns}$ | 0 |
| *aa/m.a.d.ad.dd* | 32.55** | 16.12 | 6.65** | 2.68 |
| *ad/m.a.d.aa.dd* | 0.57$^{ns}$ | 0.28 | 2.63$^{ns}$ | 1.06 |
| *dd/m.a.d.aa.ad* | 21.76** | 10.78 | 5.58* | 2.25 |
| Total | 201.91 | 100 | 248.305 | 100 |
| **Parameters** | **Delta Opal x CNPA 2984** | | **CNPA 2161 x BRS Facual** | |
| | **SSdeviation** | **RC (%)** | **SSdeviation** | **RC (%)** |
| *m/a.d.aa.ad.dd* | 71.04** | 32.65 | 299.58** | 61.94 |
| *a/m.d.aa.ad.dd* | 138.63** | 63.71 | 54.88** | 11.35 |
| *d/m.a.aa.ad.dd* | 1.5293$^{ns}$ | 0.7 | 31.34** | 6.48 |
| *aa/m.a.d.ad.dd* | 2.72$^{ns}$ | 1.25 | 74.34** | 15.37 |
| *ad/m.a.d.aa.dd* | 0.163$^{ns}$ | 0.07 | 5.87* | 1.21 |
| *dd/m.a.d.aa.ad* | 3.502$^{ns}$ | 1.61 | 17.65* | 3.65 |
| Total | 217.5843 | 100 | 483.66 | 100 |
| **Parameters** | **CNPA 999 x CNPA 2984** | | **CNPA 2161 x CNPA 2984** | |
| | **SSdeviation** | **RC (%)** | **SSdeviation** | **RC (%)** |
| *m/a.d.aa.ad.dd* | 280.134** | 63.47 | 115.021** | 80.56 |
| *a/m.d.aa.ad.dd* | 66.47** | 15.06 | 21.13** | 14.8 |
| *d/m.a.aa.ad.dd* | 27.05** | 6.13 | 0.25$^{ns}$ | 0.18 |
| *aa/m.a.d.ad.dd* | 50.58** | 11.46 | 0.12$^{ns}$ | 0.08 |
| *ad/m.a.d.aa.dd* | 6.37** | 1.44 | 4.97* | 3.48 |
| *dd/m.a.d.aa.ad* | 10.73** | 2.43 | 1.29$^{ns}$ | 0.9 |
| Total | 441.33 | 100 | 142.78 | 100 |

[1] the bar indicates that the preceding parameter was adjusted to the other following parameters.
** significant at 1% probability, by the t test; * significant at 5% probability, by the t test; ns: non-significant

**Table 3.** Estimates of the components of variance and their respective standard deviations, of the mean dominance degree (*MDD*)[1], number of genes and coefficient of genotypic determination, in the broad and narrow sense ($h^2_a$ and $h^2_r$), for the trait reaction to *C. gossypii* var. *cephalosporioides*

| Parameter | Estimate | Error | Prob > t | Parameter | Estimate | Error | Prob > t |
|---|---|---|---|---|---|---|---|
| | **Delta Opal x BRS Facual** | | | | **CNPA 999 x CNPA 2043** | | |
| $\sigma^2_A$ | 0.259 | 0.115 | 0.265 | $\sigma^2_A$ | 0.156 | 0.347 | 0.731 |
| $\sigma^2_D$ | -0.103 | 0.089 | 0.429 | $\sigma^2_D$ | -0.122 | 0.281 | 0.786 |
| $\sigma^2_e$ | 0.414 | 0.031 | 0.046 | $\sigma^2_e$ | 0.480 | 0.112 | 0.153 |
| $h^2_a$ (%) | 27 | 0.515 | - | $h^2_a$ (%) | 7 | 0.093 | - |
| $h^2_r$ (%) | 45 | 2.084 | - | $h^2_r$ (%) | 30 | 1.331 | - |
| *MDD* | -0.181 | - | - | *MDD* | -1.001 | - | - |
| *no. of genes (k)* | 2.48 | - | - | *no. of genes (k)* | 1.37 | - | - |
| | **Delta Opal x CNPA 2043** | | | | **CNPA 999 x CNPA 2984** | | |
| $\sigma^2_A$ | 0.14 | 0.364 | 0.766 | $\sigma^2_A$ | 0.182 | 0.376 | 0.713 |
| $\sigma^2_D$ | 0.113 | 0.294 | 0.811 | $\sigma^2_D$ | -0.1 | 0.302 | 0.780 |
| $\sigma^2_e$ | 0.339 | 0.068 | 0.117 | $\sigma^2_e$ | 0.487 | 0.111 | 0.141 |
| $h^2_a$ (%) | 43 | 0.771 | - | $h^2_a$ (%) | 15 | 0.239 | - |
| $h^2_r$ | 26 | 1.007 | - | $h^2_r$ (%) | 32 | 1.470 | - |
| *MDD* | -0.85; 1.27 | - | - | *MDD* | -0.511 | - | - |
| *no. of genes (k)* | 0.25 | - | - | *no. of genes (k)* | 1.05 | - | - |
| | **Delta Opal x CNPA 2984** | | | | **CNPA 2161 x BRS Facual** | | |
| $\sigma^2_A$ | 0.043 | 0.252 | 0.892 | $\sigma^2_A$ | 0.187 | 0.396 | 0.718 |
| $\sigma^2_D$ | -0.022 | 0.219 | 0.876 | $\sigma^2_D$ | -0.069 | 0.317 | 0.923 |
| $\sigma^2_e$ | 0.424 | 0.073 | 0.104 | $\sigma^2_e$ | 0.383 | 0.102 | 0.180 |
| $h^2_a$ (%) | 5 | 0.071 | - | $h^2_a$ (%) | 23 | 0.594 | - |
| $h^2_r$ (%) | 10 | 0.372 | - | $h^2_r$ (%) | 37 | 1.679 | - |
| *MDD* | -0.751 | - | - | *MDD* | 0.781 | - | - |
| *no. of genes (k)* | 21.39 | - | - | *no. of genes (k)* | 1.08 | - | - |
| | **CNPA 999 x BRS Facual** | | | | **CNPA 2161 x CNPA 2984** | | |
| $\sigma^2_A$ | 0.272 | 0.291 | 0.521 | $\sigma^2_A$ | 0.059 | 0.413 | 0.911 |
| $\sigma^2_D$ | -0.245 | 0.25 | 0.492 | $\sigma^2_D$ | -0.039 | 0.352 | 0.909 |
| $\sigma^2_e$ | 0.563 | 0.113 | 0.124 | $\sigma^2_e$ | 0.364 | 0.125 | 0.203 |
| $h^2_a$ | 5 | 0.074 | - | $h^2_a$ (%) | 5 | 0.063 | - |
| $h^2_r$ | 46 | 1.952 | - | $h^2_r$ (%) | 15 | 0.620 | - |
| *MDD* | 0.721 | - | - | *MDD* | -0.331 | - | - |
| *no. of genes (k)* | 14.83 | - | - | *no. of genes (k)* | 0.75 | - | - |

[1] : Mean dominance degree calculated as $GMDm = \left|2\overline{F}_1 - \left(\overline{P}_1 + \overline{P}_2\right)\right| / \left(\overline{P}_1 + \overline{P}_2\right)$, because negative estimates of $\sigma^2_D$, in each cross; in the cross Delta Opal x CNPA 2043, the traditional expression $GMDv = \sqrt{2\hat{\sigma}_D^{\,2} / \hat{\sigma}_A^{\,2}}$ (second estimate) was used as well

Burton (1951), although recommended in the presence of dominance, is not indicated in the case of epistasis (Ramalho et al. 1993). The most reliable estimates are therefore those related to the crosses Delta Opal x BRS Facual (2 genes), Delta Opal x CNPA 2984 (21 genes), CNPA 999 x CNPA 2043 (1 gene) and CNPA 2161 x CNPA 2984 (1 gene), which indicate predominantly oligogenic genetic inheritance.

The difference in the number of genes controlling the trait, from one cross to another, although the cause may be a methodological, and/or environmental effect, can also be due to differentiated fixation of alleles in the parents involved in certain crosses. Thus, a particular pair of parents (e.g. CNPA 2161 and CNPA 2984) can share a single non-fixed locus with different alleles, while other pairs of parents (e.g. Delta Opal and BRS Facual or Delta Opal and CNPA 2984) can share a greater number of these loci.

Mean dominance degree (*MDDm*) estimates with absolute values between 0,0 and 1,0 were found in

almost all crosses (Table 3). This confirms previous studies that reported the presence of partial dominance in the genes that control the trait. Negative $MDD_m$ estimates in most crosses also indicate dominance for a reduced phenotypic expression of the trait (Cruz and Regazzi 1997), that is, tending to the lowest grades of disease severity, ie, to more resistant genotypes.

On the other hand, in the case of the cross Delta Opal x CNPA 2043, the discrepancy in the *MDD* based on the estimators $MDD_v$ and $MDD_m$ indicates that the dominance deviations are possibly not unidirectional. This hypothesis was reinforced by the positive *MDD* estimates in the crosses CNPA 999 x BRS Facual and CNPA 2161 x BRS Facual, in which the parents can differ from each other, especially in loci with dominance expression of the highest grades, that is, tending to more susceptible genotypes. This explanation could also justify the inconsistencies in results reported in the literature. For example, Carvalho et al. (1994) observed dominance tending to susceptibility to ramulosis, while Zandoná et al. (2006) concluded that dominance tends towards disease resistance, as observed predominantly in this study.

# Herança da resistência à ramulose do algodoeiro

**RESUMO** - *O objetivo deste estudo foi analisar a herança genética da resistência à ramulose do algodoeiro. Para isso, foram realizados cruzamentos entre dois grupos de linhagens contrastantes para o caráter. Os genitores suscetíveis à doença foram Delta Opal, CNPA 999 e CNPA 2161, e aqueles com elevado nível de resistência, BRS Facual, CNPA 2043 e CNPA 2984. Assim, nove cruzamentos foram obtidos, sempre entre um genitor resistente e um suscetível, totalizando-se 42 tratamentos. O delineamento experimental foi em blocos completos casualizados com três repetições. Verificou-se que o controle genético da resistência à ramulose do algodoeiro é predominantemente oligogênico, sendo que o número de genes envolvidos é dependente dos genitores que participam em cada cruzamento, haja vista a possibilidade de fixação diferenciada de locos entre eles. Há evidências de dominância parcial, especialmente no sentido de aumentar a resistência à doença, embora haja também indícios de que esta não seja unidirecional.*

**Palavras chave:** ramulose do algodoeiro, controle genético, *Gossypium hirsutum*.

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