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Mixed inheritance model for resistance to anthracnose leaf blight in maize

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ABSTRACT - *A separation of the effects of major genes from effects of polygenes is important to understand genetic inheritance of quantitative traits and predict the segregation of a crossing. The inheritance mode of resistance to anthracnose leaf blight (ALB) caused by* C. graminicola *was evaluated by a mixed model. P_1, P_2, F_1, F_2, BC_1, and BC_2 generations derived from four crossings between tropical maize inbred lines were used. Maximum likelihood was used to choose the best fitting inheritance model and to estimate genetic parameters. The mixed model indicated that the resistance to ALB was controlled by a major gene in all crosses and trials, and by polygenes in at least one trial. Additive and dominance effects were important for both major gene and polygenes. Both effects of the major genes were negative, indicating their contribution to disease resistance, while both effects of the polygenes were positive or negative, reflecting differences in the genetic background among inbred lines.*

Key words: *Colletotrichum graminicola*, disease resistance, inheritance of resistance, *Zea mays*.

INTRODUCTION

Anthracnose leaf blight caused by *Colletotrichum graminicola* (Ces.) Wils. is becoming increasingly predominant in tropical areas. The pathogen infects maize at various growth stages and causes both leaf blight and stalk rot (Badu-Apraku et al. 1987, Lin and White 1978, Zuber et al. 1981). Leaf blight is most evident in seedlings and mature plants after anthesis (Badu-Apraku et al. 1987). Yield losses up to 19 and 28% have been reported in maize hybrids and inbred lines, respectively (Smith 1976). Since genetic resistance is the most economic and efficient control method for this disease, knowledge about its mode of inheritance is essential for breeding programs. Previous reports indicated that resistance is controlled by few dominant genes. Among the reported additive and non-additive genetic effects, the former are more important (Carson and Hooker 1981a, b, Badu-Apaku et al. 1987, Lim and White 1978, Silva et al. 1986). Significant deviations from the additive-dominance model were also mentioned (Carson and Hooker 1981a, b), which are possibly result of the lack of an adequate scale to measure disease symptoms, epistasis, or of the failure to meet the assumptions of the generation mean analysis (Mather and Jinks 1971). A common feature of these reports are independently estimated parameters associated to polygenes (components of means and variances) and to major genes, i.e., not a mixed model that would include the effects of both major genes and polygenes.

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When a trait has continuous distribution, quantitative genetic models, which assume a large number of genes of equal effect, are commonly used. However, the validity of these models is severely compromised if major genes are present. Mixture models or mixed inheritance models provide a more sophisticated approach to discover major genes, assessing the agreement between phenotypic distribution and a mixture of normal distributions (Lynch and Walsh 1998). In this approach, each major gene genotype is expressed in an expected genotypic value, around which a variation occurs. This variation is due to environmental effects wich are or are not added by polygenic effects. A number of methods which analyse mixed inheritance models in human and animal populations have been described (Elston and Stewart 1973, Morton and MacLean 1974, Knott et al. 1991, Le Roy et al. 1990, Shoukri and McLachlan 1994, Janss et al. 1995). In addition, similar approaches through maximum likelihood were developed, to analyze major loci in segregating generations derived from crosses between inbred plant lines (Tourjee et al. 1995, Loisel et al. 1994, Jiang et al. 1994).

Often, EM algorithms are used to obtain maximum likelihood estimates (Dempster et al. 1977). When the sample is composed by categories with distinct variation, as is the case when different generations are analyzed, there is no apparent solution for the estimation of expressions which determine expectation (E) and maximization (M) steps, making adaptations necessary. In this case, the use of Newton-Raphson, Quase-Newton, and Powell numeric algorithms should be more appropriate (Silva 2003).

The objective of this study was to investigate the inheritance of resistance to anthracnose leaf blight in maize by a mixed inheritance model and inbred lines derived from tropical germplasm. Monogen version 0.1 (Silva 2003), which combines Quase-Newton and Powell methods to identify the best fitting inheritance model and to estimate genetic parameters by maximum likelihood, was the software used to analyse genetic data.

MATERIALS AND METHODS

Plant material

Four maize inbred lines, obtained by at least seven self-pollinations, were used in this study. Inbred DAS22 is susceptible to *C. graminicola* and has semiflint and orange kernels. It was derived from the Suwan DMR population developed in Thailand by selection from Caribbean flint and Tuxpeño dent populations. In Brazil, Suwan DMR is represented by CMS 05, a population of Embrapa Maize and Sorghum. The resistant inbred line DAS3 has flint and orange kernels and was derived from Suwan-3. This population was obtained by recurrent selection of Suwan-1. Finally, DAS6 is susceptible and DAS4 is resistant to *C. graminicola*. They both have semiflint and orange kernels, and were derived from a synthetic population of narrow genetic base composed by inbred lines from Amarillo dent and Caribbean flint populations, which are widely used in breeding programs throughout Asia.

The parental line, F_1, F_2, and BC_1, and BC_2 generations derived from the crosses DAS6 x DAS4, DAS6 x DAS3, DAS22 x DAS4, and DAS22 x DAS3 were obtained in 2000/01 to be used in this study.

Disease resistance evaluation

A pathogenic isolate of *C. graminicola* was grown on Petri dishes containing oatmeal agar (40 g oatmeal, 17 g agar, and 1 L distilled water) and incubated in a growth chamber at 22 ± 2 ºC, under fluorescent light (12 hours of light and 12 hours of darkness). Inoculum was prepared by flooding 2-3 week old colonized oatmeal agar plates with distilled water. The resulting spore suspension was filtered through a double gaze and the inoculum concentration adjusted to 5 x 10^5 conidia mL^{-1}. A drop of Tween 80® was added to each liter of inoculum.

Two trials were carried out in Jardinópolis (State of São Paulo, Brazil), in November/2001 (conventional planting season) and in December/2001 (late planting season). The split-plot design with three replications in use presented crosses in the plots and generations (P_1, P_2, F_1, F_2, BC_1, and BC_2) in the subplots. Subplots consisted of a single row for parental lines and F_1 generations, two rows for backcrosses, and four rows for F_2 generations. Each row was 5 m long with 25 plants, spaced 80 cm apart.

Seedlings were inoculated twice, at twenty and twenty-seven days after sowing, by spraying 5 mL of the spore suspension (5 x 10^5 conidia mL^{-1}) into the whorl of each plant. Disease severity of the youngest symptomatic leaf, generally the seventh from the bottom up, was assessed sixteen days after the second inoculation. A rating scale from 1 to 6 evaluated the disease: 1= absence of symptoms; 2 = up to 3 mm long chlorotic or necrotic points; 3 = 3 to 10 mm long necrotic lesions; 4 = 10 to 40 mm long necrotic lesions; 5 = 40 to 60 mm long necrotic lesions; 6 = coalescence of over 60 mm long necrotic lesions. Individual and joint variance analyses of disease severity means were carried out for both trials.

Genetic models and hypothesis testing

Estimates of genetic parameters were established and their tests run by the Monogen version 0.1 Software (Silva 2003). For the analyses, the model which presented a major gene, with additive and dominance effects, and polygenes, also with additive and dominance effects, was considered genetically most complete. Environmental variances (σ^2) were considered equal for all generations, and gene segregation was considered independent. According to this model, genotypic values for the major gene corresponding to homozygotes and the heterozygote are represented, respectively, by μ - A, μ + A and μ +D, where μ is a reference constant, *A* is the additive effect and *D* the dominance effect. Mean and variance components for the polygenes (Table 1) were calculated according to Mather and Jinks (1971).

Table 1. Polygenic components of means and variances of generations

Generation	Polygenic components of mean	Polygenic components of variance
P_1	- [a]	-
P_2	[a]	-
F_1	[d]	-
F_2	1/2 [d]	$V_A + V_D$
BC_1	-1/2 [a] + 1/2 [d]	$1/2\ V_A + V_D - S_{AD}$
BC_2	1/2 [a] + 1/2 [d]	$1/2\ V_A + V_D + S_{AD}$

[a]: additive component of polygene; [d]: dominance component of polygene; V_A: additive variance; V_D: dominance variance; S_{AD}: sum of products of additive-dominance effects.

From the complete genetic model (model 1, Table 2), simpler models were generated, i.e., models containing less parameters (models 2 through 9, Table 2). Genetic parameter estimates for the models were obtained by the maximum likelihood method. Hypotheses tests of the genetic parameters were performed based on the likelihood ratio (LR) between two models (Mood et al. 1974). LR statistics test whether a parameter added to a model leads to a significant increase in the variation quantity explained by the parameter. LR is given by:

$$LR = -2 \ln \frac{L(M_i)}{L(M_j)}$$

where $L(M_i)$ and $L(M_j)$ are likelihood functions for models i and j, and model i is hierarchic to model j. Roughly, this statistics follows a chi-square distribution where, for a test with probability α, H_0 is rejected if $LR > \chi^2_{(1-\alpha,v)}$, where v is the number of degrees of freedom given by the difference between the numbers of parameters in the models M_j and M_i.

Considering the hierarchization of the models, models 1 and 5 were initially confronted, with model 5 hierarchic to model 1. The LR between these models tests the hypothesis of monogenic inheritance. Then, models 1 and 7 were confronted, with model 7 hierarchic to model 1, to test the hypothesis of polygenic inheritance. The non-significance of one or both tests implies accepting the null hypothesis for the test in question, that is to say, there is no evidence of a major gene, in the first case, and no evidence of polygenes, in the second case. In cases where the null hypothesis for LR between models 1 and 5 was rejected, the dominance effects for the major gene were tested, confronting model 7 and model 8, with model 8 hierarchic to model 7. In cases where the null hypothesis for LR between models 1 and 7 was rejected, the dominance effects for the polygenes were tested, confronting models 5 and 6, with model 6 hierarchic to model 5. The model selected to explain the inheritance of resistance was the one that included all significant genetic effects.

The broad- ($\hat{H}^2$) and narrow- ($\hat{h}^2$) sense heritabilities were estimated by the following formulas:

$$\hat{H}^2 = \frac{V_A + V_D + A^2/2 + D^2/4}{V_A + V_D + A^2/2 + D^2/4 + \sigma^2}$$

$$\hat{h}^2 = \frac{V_A + A^2/2}{V_A + V_D + A^2/2 + D^2/4 + \sigma^2}$$

where V_A is the polygene additive variance, V_D the polygene dominance variance, A the additive effect of the major gene, D the dominance effect of the major gene, and σ^2 is the environmental variance.

RESULTS AND DISCUSSION

Disease resistance evaluation

The experimental precision in the first trial (VC_a = 14.30% and VC_b = 10.90%), in the second trial (VC_a = 12.57% and VC_b = 11.99%), and in the joint analysis (VC_a = 13.37% and VC_a = 11.93%) was superior to that previously reported for evaluation of leaf anthracnose (Zuber et al. 1981) and other maize diseases (Paterniani et al. 2000). Corroborating the experimental precision, no significant differences were observed among blocks and among blocks within a trial, which suggests that the disease incidence was uniform in the experiments. The crosses were statistically different in disease severity ($P < 0.05$) in the individual analyses, indicating that the genetic background of the lines, in relation to resistance genes, is different. Generations within crosses also differed ($P < 0.01$), manifesting a segregation of resistance genes to *C.*

Table 2. Genetic inheritance models and theirs parameters in the analysis of generations P_1, P_2, F1, F_2, BC_1 and BC_2

Model	Major gene	Polygenes	Genetic parameters
1. Mixed inheritance	Additive and dominant	Additive and dominant	μ, A, D, [a], [d], V_A, V_D, S_{AD}, σ^2
2. Mixed inheritance	Additive and dominant	Additive	μ, A, D, [a], V_A, σ^2
3. Mixed inheritance	Additive	Additive and dominant	μ, A, [a], [d], V_A, V_D, S_{AD}, σ^2
4. Mixed inheritance	Additive	Additive	μ, A, [a], V_A, σ^2
5. Polygenic inheritance	-	Additive and dominant	μ, [a], [d], V_A, V_D, S_{AD}, σ^2
6. Polygenic inheritance	-	Additive	μ, [a], V_A, σ^2
7. Monogenic inheritance	Additive and dominant	-	μ, A, D, σ^2
8. Monogenic inheritance	Additive	-	μ, A, σ^2
9. No genetic effects	-	-	μ, σ^2

μ: cross mean; A: additive effect of the major gene; D: dominance effect of the major gene; [a]: additive effect of the polygenes; [d]: dominance effect of the polygenes; V_A: polygene additive variance; V_D: polygene dominance variance; S_{AD}: sum of products of additive-dominance effects products; σ^2: environmental variance.

graminicola. The joint analysis of variance revealed that the effects of trial, trial x cross interaction, generation within a cross, and trial x generation interaction within a cross were significant ($P < 0.01$). However, the crosses did not significantly differ in the joint analysis.

In general, disease severity means (Table 3) of the trial installed in December were superior to those of November, 2001, indicating that the late season was more favorable to the development of the pathogen. This observation is in agreement with Bergstrom and Nicholson (1999), who mentioned the difficulty of controlling the disease in late plantings at endemic sites of the pathogen. There was an inversion in the behavior of susceptible lines between trials. Severity means for line DAS6 were superior in November, while the means for DAS22 were superior in December 2001, suggesting the presence of a genotype × environment interaction. Resistant lines (DAS4 and DAS3), on the other hand, did not present disease symptoms. The performance of lines DAS6 and DAS4 was similar to that observed by Coêlho et al. (2001), who for the first time reported a resistant line (DAS4) without infection symptoms. The absence of symptoms in this line suggests the presence of a major gene in the expression of the character.

F_1 and BC_2 means for all crosses were similar to those of the resistant parents. BC_1 and F_2 means, however, were intermediate to the parental means, while the F_2 mean tended towards the resistant mean. In the DAS6 x DAS4 cross, both the generation means and frequency distributions of the disease severity ratings indicate a dominance genetic effect (Figure 1). Even though two artificial inoculations per trial were performed, the high frequency of resistant individuals (rating 1) suggests that escapes might occur.

Table 3. Mean anthracnose leaf blight severities of six generations in four maize crosses evaluated in two trials

Generation	DAS6 x DAS4	DAS6 x DAS3	DAS22 x DAS4	DAS22 x DAS3
		November 2001		
P_1	3.78	3.38	2.75	2.58
P_2	1.00	1.00	1.00	1.00
F_1	1.00	1.08	1.00	1.03
F_2	1.28	1.32	1.19	1.23
BC_1	1.81	1.65	1.54	1.33
BC_2	1.00	1.05	1.03	1.00
		December 2001		
P_1	4.40	4.75	5.28	5.14
P_2	1.00	1.00	1.00	1.00
F_1	1.07	1.18	1.00	1.00
F_2	1.51	2.00	2.00	1.84
BC_1	2.39	2.74	3.13	3.08
BC_2	1.00	1.50	1.08	1.56

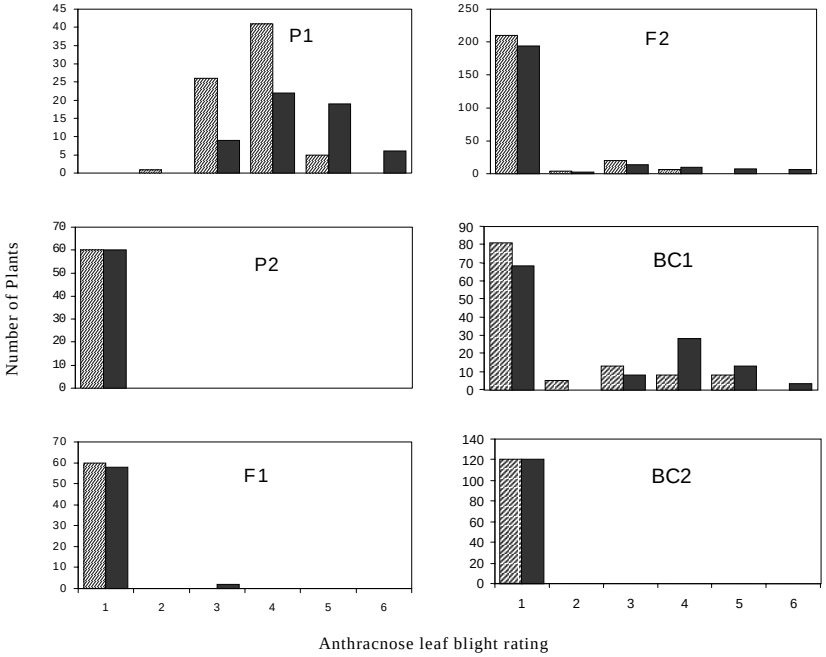

Figure 1. Frequency distributions of anthracnose leaf blight ratings of individual plants of maize parental inbred lines (P_1 and P_2) and the F_1, F_2, and backcross (BC_1 and BC_2) generations derived from the cross DAS6 x DAS4. Cross-hatched and solid bars represent data from November and December 2001, respectively. Plants were rated on a 1 to 6 scale.

Genetic inheritance models and hypothesis tests

Results of the hypothesis tests of the genetic models varied according to cross and trial (Table 4). In the DAS6 x DAS4 cross, a negative χ^2 value was obtained in the November 2001 trial for the likelihood ratio between models 1 and 5, which tests the evidence of segregation of a major gene. This negative value could be due to convergence problems with the likelihood functions, i.e., no parameter values were found that would reach the maximum likelihood point. Even though it was not possible to test the hypothesis of a major gene due to an intrinsic problem of this analysis type, there were evidences of the presence of this gene, since the likelihood ratio between models 7 and 8 was significant. This suggests a dominance genetic effect of a major gene. The tests for models 1 and 7 and models 5 and 6 were also significant, indicating the presence of polygenes with a dominance genetic effect. In December 2001, however, all tested hypotheses were significant, allowing us to draw inferences from the presence of a major gene with dominance genetic effect as well as polygenes with dominance. The model chosen for this cross was therefore the mixed inheritance model (model 1).

In the DAS6 x DAS3 cross, only the likelihood ratio tests between models 1 and 5 and between models 7 and 8 were significant in November 2001, indicating the presence of a major gene with dominance. However, in addition to the tests between models 1 and 5 and between models 7 and 8, the likelihood ratio tests between models 1 and 7 and between models 5 and 6, which provide evidence of dominant polygene segregation, were also significant in December 2001. Thus, the models that best fitted the data were models 7 and 1, for November and December, respectively.

All tested genetic hypotheses were significant in November 2001 for the DAS22 × DAS4 cross. However, only the likelihood ratio tests between models 1 and 5 and between models 7 and 8 were significant in the second trial, indicating monogenic segregation with a dominance effect. In this cross, the November data were better adjusted to model 1 and the December data better adjusted to model 7.

In the DAS22 x DAS3 cross, all tested genetic hypotheses were significant both for November and December 2001. Model 1, containing a major gene with dominance genetic effect, and polygenes with dominance, was therefore selected for this cross.

Model 1, which assumes the mixed inheritance of a greater effect gene and polygenes, was selected for all crosses in at least one of the trials. Model 7, on the other hand, which assumes monogenic inheritance, was selected in only one of the trials for crosses DAS6 x DAS3 and DAS22 x DAS4. The variation in the detection of polygenic effects in the two trials designed to evaluate these crosses can be attributed to environmental effects in their expression and to errors in the evaluation of severity, since the data for the six generations used in the analyses consisted of measurements of individual plants. In order to avoid this problem, data based on measurements of families derived from F_2, BC_1, and BC_2 could be used to reduce the experimental error, as demonstrated by Wang and Gai (2001), who utilized data from $F_{2:3}$ families. However, analyses of mixed models based on the evaluation of families derived from F_2 and backcrosses have not yet been developed. Another limitation of the analysis utilized in the present work is the fact that it does not consider the experimental design, and therefore does not remove the environmental variation component.

All models selected in the analyses of the four crosses evidenced the presence of a major gene with additive and dominance genetic effects. These results agree with those obtained by Coêlho et al. (2001), Badu-Apraku et al. (1987), and also with results from the phenotypic evaluation. The consistency of the observations allows us to draw conclusions on the authority of the analysis method utilized in the present work to study the inheritance of resistance to leaf anthracnose in maize.

Table 4. Chi-square values (χ^2) for hypothesis tests about genetic inheritance models of the four crosses evaluated in two trials.

Contrast	Hypothesis testing	df	Cross			
			DAS6 x DAS4	DAS6 x DAS3	DAS22 x DAS4	DAS22 x DAS3
			November 2001			
Model 1 x Model 5	Major gene	2	nv	397.1**	231.1**	1002.3**
Model 7 x Model 8	Dominant major gene	1	1064.7**	727.4**	535.0**	373.7**
Model 1 x Model 7	Polygenes	5	1458.2**	2.5	55.0**	1102.3**
Model 5 x Model 6	Dominant polygenes	3	2924.3**	-	322.6**	445.0**
			December 2001			
Model 1 x Model 5	Major gene	2	415.3**	200.7**	556.6**	225.6**
Model 7 x Model 8	Dominant major gene	1	890.6**	385.1**	1063.9**	437.5**
Model 1 x Model 7	Polygenes	5	292.7**	35.6**	9.3	111.8**
Model 5 x Model 6	Dominant polygenes	3	746.6**	224.0**	-	319.1**

nv: negative value, probably due to convergence problems; * and **: significant at 0.05 and 0.01 probability, respectively.

Genetic parameters for resistance to *C. graminicola*

Estimates of the genetic parameter for the selected models, degree of dominance, heritabilities, and percentages of variation explained by the additive and dominance effects are presented in Table 5. The genetic effects, both additive and dominant, of the major gene were always negative, indicating that they contribute to reduce disease severity (Figure 1). The additive and dominance effects of the polygenes were either positive or negative, depending on the cross and trial. From these results we infer that there are differences in the genetic makeup of the crosses in relation to the resistance polygenes. Some additive and dominance variance estimates (V_A and V_D) were equal to zero, despite the fact that the selected model indicated the presence of polygenic variation. Since these results do not make sense from a genetic point of view, it is assumed that these variances are of little magnitude, or that their estimates have a great associated error. In fact, it can be observed that these estimates presented a confidence interval (data not shown), with 95% probability, comprising both a positive and a negative value. Badu-Apraku et al. (1987) obtained a negative estimate of additive variance which was, for practical purposes, considered zero. The authors suggested some explanations for this estimate, which could be applied to the present work. One of the assumptions, both in the analysis of mixed models and the analysis of generation means, is that the environmental variation is the same within each generation. Different degrees of competition among plants within the plot, due to differences in vigor, could have provided conditions in which the plot environments were different for the generations in each replication. Another limitation of these analyses is that independent segregation of genes is assumed. If these assumptions are not met, the estimates may be biased.

The degree of dominance (D/A) ranged from 0.98 to 1.14. Dominance for resistance to *C. graminicola* has also been reported in other papers (Carson and Hooker 1981a, b, Lim and White 1978, Toman and White 1993, Badu-Apraku et al. 1987). The possibility of drawing inferences about allelic interaction is an advantage of the use of mixed models that separate genetic effects of the major gene from polygenes, since the D/A ratio is not suitable for this purpose, when two or more genes are considered (Mather and Jinks 1971). In this work, the degree of dominance was only estimated for the major gene. In polygenic models, some dominance effects can be negative and others positive, leading to reduced D values, even if these values are not small, individually. Similarly, value A could be small because due to the way genes are distributed between the parents, the algebraic sum of the contribution of the homozygous loci is small.

Broad-sense heritabilities ranged from 0.84 to 0.93, and 0.46 to 0.62 in narrow-sense. The additive genetic effects contributed to 54.6 to 67.4% of the total variation, and the dominance genetic effects to 32.6 to 45.4%. The heritability estimates of the four crosses suggest the possibility of genetic improvement by simple breeding methods, such as mass selection. However, selection based on progeny tests should be more effective, since the dominance components had an important participation in the total genetic variation. Due to the genotype x environment interactions, inbred lines or hybrid combinations must be tested in several environments to ensure a correct phenotypic evaluation.

Knowledge on the inheritance of a trait that discriminates major from minor genes is important to predict segregation of a cross in breeding programs (Jiang et al. 1994). The mixed models approach is different from the joint scale test, normally used in quantitative genetics (Mather and Jinks 1971). Mixed models regard the genetic system of a quantitative trait as an inheritance model containing major or minor genes. The joint scale test, in turn, considers a quantitative trait a polygenic system. Nevertheless, mixed models analysis can only test the presence of these genes, while QTL mapping models allow the identification, location, and quantification of their effects.

Table 5. The best fitting genetic model, estimates of genetic parameters, degree of dominance, heritabilities and contribution of genetic effects for the four crosses in the two trials

Crosses	Model	μ	A	D	[a]	[d]	V_A	V_D	S_{AD}	σ^2	D/A	H^2	h^2	a (%)	d (%)
November 2001															
DAS6 x DAS4	1	2.34	-1.27	-1.27	-0.07	-0.06	0.00	0.00	-0.10	0.10	1.00	0.92	0.62	66.67	33.33
DAS6 x DAS3	7	2.20	-1.19	-1.17	-	-	-	-	-	0.14	0.98	0.88	0.59	67.42	32.58
DAS22 x DAS4	1	1.91	-1.03	-1.03	0.10	0.19	0.00	0.00	-0.11	0.15	1.00	0.84	0.56	66.67	33.33
DAS22 x DAS3	1	1.81	-0.99	-0.99	0.11	0.24	0.00	0.00	-0.13	0.13	1.00	0.84	0.57	66.67	33.33
December 2001															
DAS6 x DAS4	1	2.67	-1.60	-1.61	-0.06	-0.05	0.00	0.00	-0.17	0.18	1.01	0.91	0.61	66.39	33.61
DAS6 x DAS3	1	2.83	-1.47	-1.67	-0.24	-0.3	0.00	0.20	0.20	0.36	1.14	0.85	0.46	54.63	45.37
DAS22 x DAS4	7	3.13	-2.09	-2.10	-	-	-	-	-	0.24	1.00	0.93	0.62	66.45	33.55
DAS22 x DAS3	1	3.09	-1.80	-1.78	-0.20	-0.27	0.00	0.53	0.29	0.23	1.00	0.93	0.51	55.06	44.94

μ: cross mean; A: additive effect of the major gene; D: dominance effect of the major gene; [a]: additive effect of the polygenes; [d]: dominance effect of the polygenes; V_A: polygene additive variance; V_D: polygene dominance variance; S_{AD}: Sum of products of additive-dominance effects; σ^2: environmental variance; D/A: dominance degree, H^2: broad-sense heritability; h^2: narrow-sense heritability; a (%): percentage of variation accounted by additive genetic effects; d (%): percentage of variation accounted by dominance genetic effects.

As demonstrated in this study, mixed models analysis detected the presence of maize resistance genes to *C. graminicola* of major and minor genetic effects. The gene action by both the major gene and by polygenes was additive and dominant, although additive gene action was more important.

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Modelo de herança mista para resistência à antracnose foliar em milho

RESUMO - *A separação dos efeitos de genes maiores dos efeitos de poligenes é importante para o entendimento da herança de caracteres quantitativos e para predizer a segregação de cruzamentos. Um modelo misto de herança foi usado para estudar a herança da resistência à antracnose foliar em milho causada por* C. graminicola*. Foram utilizadas as gerações P_1, P_2, F_1, F_2, RC_1 e RC_2 derivadas de quatro cruzamentos entre linhagens de milho tropical. O modelo genético mais adequado e as estimativas dos parâmetros genéticos foram obtidos pelo método da máxima verossimilhança. Os modelos mistos de herança indicaram que a resistência à antracnose foliar é controlada por um gene de efeito maior, em todos os cruzamentos e ensaios avaliados, e também por poligenes, em pelo menos um dos ensaios. Ambos os efeitos aditivos e dominantes foram importantes para os genes de efeito maior e poligenes. Os efeitos aditivos e dominantes dos genes de efeito maior foram negativos, indicando que eles contribuem para a resistência à doença, enquanto ambos os efeitos dos poligenes foram positivos ou negativos, refletindo diferenças na constituição genética entre linhagens.*

Palavras-chave: *Colletotrichum graminicola*, resistência a doenças, herança da resistência, *Zea mays*.

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