Research Article

Genetic control of anthracnose stalk rot resistance in tropical maize[1]

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ABSTRACT

The genetic resistance to diseases in plants represents an important support pillar in modern agriculture. This study aimed to determine the genetic inheritance to anthracnose stalk rot resistance in tropical maize. Nine segregating families were obtained from contrasting inbred lines crosses. The parental lines and the filial generations (F_1, F_2, BC_1 and BC_2) were evaluated for antrachnose stalk rot resistance in an experiment in randomized blocks, with three replications. The treatments were arranged in a split-plot design, with family effect in the plots and generation effect in the split-plots. The results showed a similar inheritance among the families, with predominance of additive genetic effects. The inbreed lines (L_R 04-2, L_R 03-2 and L_R 23-1) were very effective in transmitting resistance genes to their descendants because they allowed sharp decreases in the lesions length in the stalks. It was also possible to notice an oligogenic inheritance involved in the anthracnose stalk rot resistance for the evaluated families. It may be inferred that genetic gains from artificial selection could be successful for developing maize inbred lines more resistant to anthracnose stalk rot.

KEYWORDS: *Zea mays*, *Colletotrichum graminicola*, gene action, heritability, heterosis.

RESUMO

Controle genético da resistência à antracnose do colmo em milho tropical

A resistência genética a doenças em plantas representa um importante pilar de sustentação na agricultura moderna. Objetivou-se determinar a herança genética da resistência à antracnose do colmo em milho tropical. Nove famílias segregantes foram obtidas a partir de cruzamentos de linhagens endogâmicas contrastantes. As linhagens parentais e as gerações filiais (F_1, F_2, BC_1 e BC_2) foram avaliadas quanto à resistência à antracnose do colmo em um experimento em blocos casualizados, com três repetições. Os tratamentos foram arranjados em delineamento de parcelas subdivididas, com efeito de famílias nas parcelas e de gerações nas subparcelas. Os resultados mostraram herança semelhante entre as famílias, com predominância de efeitos genéticos aditivos. As linhagens endogâmicas (L_R 04-2, L_R 03-2 e L_R 23-1) foram muito eficazes na transmissão de genes de resistência a seus descendentes, pois permitiram diminuições acentuadas no comprimento das lesões nos colmos. Também observou-se uma herança oligogênica envolvida na resistência à antracnose do colmo nas famílias avaliadas. Pode-se inferir que os ganhos genéticos com a seleção artificial poderiam ter sucesso no desenvolvimento de linhagens endogâmicas de milho mais resistentes à antracnose do colmo.

PALAVRAS-CHAVE: *Zea mays*, *Colletotrichum graminicola*, ação gênica, herdabilidade, heterose.

INTRODUCTION

In Brazil, the maize-on-maize succession and increase in no-till crop areas over crop residues, without crop rotation, have contributed to a favorable environment for development of stalk rot diseases (*Fusarium verticillioides*, *Giberella zeae*, *Stenocapella* sp., *Colletotrichum graminicola*) that previously had been considered of secondary importance for the crop (Costa et al. 2008, Matiello et al. 2012).

Anthracnose stalk rot, caused by *Colletotrichum graminicola* (Ces.) Wils, can bring significant crop damage and considerable economic losses. In Brazil, a predominance of *C. graminicola* (62.7 %) was observed among the main pathogens associated with stalk rot in maize (Costa et al. 2008).

Damage caused by rotting at the stalk base is still not common in Brazil. In the Paraná state, Nazareno (1989) reported damage to yield of 12-40 %, depending on the year, location and hybrid used. Matiello et al. (2013) estimated a significant

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damage for grain yield only for the susceptible hybrid H8420 (16.1-20.2 %). In addition, Cota et al. (2009) found a reduction of 34.4 % for ear weight in plants inoculated with *C. graminicola*.

The use of genetic resistance is one of the most efficient methods to control plant diseases. Studies indicate that a few genes with additive effects condition the genetic control of maize resistance to *C. graminicola*, although some resistance genes of predominantly dominant gene action have also been identified (Carson & Hooker 1981, Badu-Apraku et al. 1987a, Toman & White 1993, Weldekidan & Hawk 1993, Jung et al. 1994).

Carson & Hooker (1981) concluded that additive genetic effects are responsible for more than 90 % of variation among the mean values of generations, with significant dominance effects in some populations. Matiello et al. (2012) studied resistance in tropical maize from six generations of two populations and reported a different mode of inheritance among them. For DAS21 x DAS86, there was a predominance of dominant gene action, whereas, in DAS64 x DAS86, there was a greater contribution of additive gene action. Badu-Apraku et al. (1987b) first confirmed a monogenic type resistance with dominant gene action in relation to stalk rot. Jung et al. (1994) used the RFLP molecular marker and observed only one genomic region located in chromosome 4 with strong association to a QTL responsible for more than 50 % of the variation for resistance. Frey et al. (2011) tested four combinations of maize hybrids that were near isogenic (presence or absence) for the resistance gene *Rcg1*. Results showed that the hybrids that contained *Rcg1* that were inoculated with *C. graminicola* produced 1,106 kg ha^{-1} more than the hybrids without the resistance gene. *Rcg1* seems to delay the disease development in the plant, raising the hypothesis that less inoculum would be produced for the next crop season. This would thus reduce the chances of the pathogen adapting to the presence of the gene, which may provide longer-lasting resistance in the field (Frey et al. 2011).

Since the genetic inheritance of a trait is the most important understanding for selection of resistant genotypes in a maize breeding program, this study aimed to determine the inheritance to anthracnose stalk rot (*C. graminicola*) resistance in descendent generations of nine crosses among contrasting inbred lines of tropical maize.

MATERIAL AND METHODS

The experiment was conducted in the 2015/2016 crop season, at the Universidade Estadual de Ponta Grossa, Paraná state, Brazil (25º5'49''S to 26º18'S, 48º34'W to 52º32'W and 950 m of altitude). The climate in the region is Cfb, with mean temperature in the coldest month below 18 ºC, and in the hottest month above 22 ºC (Iapar 1994).

Inbred lines of tropical maize, belonging to the *C. graminicola* resistance breeding program of the Universidade Estadual de Ponta Grossa, originated from Brazilian south maize landraces, were initially screened for stalk rot. The lines L_R 04-2, L_R 03-2 and L_R 23-1 were resistant, and the lines L_S 71-1, L_S 95-1 and L_S 99-4 were susceptible. These six contrasting inbred lines were crossed to obtain nine families. Each family was constituted by the following generations: P_1 resistant line; P_2 susceptible line; F_1 filial generation; F_2 segregating generation obtained by self-pollination of F_1; RC_1 backcross of the F_1 generation with the resistant line (P_1); and RC_2 backcross of the F_1 generation with the susceptible line (P_2).

The experiment was conducted in a randomized blocks design, with three replications. The treatments were arrenged in a split-plot, the families effect was evaluated in the plot and the generations effect in the split-plot. The genetically uniform generations (P_1, P_2 and F_1) were represented by one row per replication, the segregating populations of the F_2 generation with 4 rows per replication, and the backcrosses generations (RC_1 and RC_2) with 2 rows per replication. Each row was composed of 5 m in length, with approximately 25 plants per row, spaced 0.8 m apart.

The isolate "Ori" was provided by Dow AgroSciences Ltda. (Jardinópolis, São Paulo state, Brazil), as culture medium discs with fungus colonies. The pathogen multiplication was performed in oat-agar culture medium (10 g of oat flour, 2.5 g of agar and 250 mL of distilled water), being maintained at room temperature on the lab bench, until complete sporulation of colonies. For the inoculum preparation, 20 mL of sterile distilled water were added to Petri dishes containing the sporulated colonies, followed by surface scraping, in order to release the conidia. The suspension was adjusted to 5 x 10^5 conidia mL^{-1}, with a Neubauer chamber (Matiello et al. 2012). One drop of spreader Tween 80™ was added to each liter of the conidial suspension. The spore suspension was

adjusted to 5 x 10^5 conidia mL^{-1}. In the full flowering stage (R1), the plants were inoculated through injection of 1 mL of conidia suspension in the middle of the first visible internode above the soil surface. In the milk stage (R3), the stalks were cut longitudinally and evaluated regarding the internal length of the lesion (ILL). For the number of discolored internodes (NDI), the first six internodes of the stalk from the point of inoculation were evaluated, with 1 on the scale representing the most resistant to anthracnose stalk rot and 6 representing the most susceptible, according to a scale adapted from Keller & Bergstrom (1988).

The ILL and NDI data were submitted to analysis of variance. In the presence of a significant effect for generations (families), the generations within each family were decomposed. The mean values of the analyzed variables were compared by the Tukey test at 5 % of significance, using the Sisvar 5.3 software (Ferreira 1999).

The genetic effects were estimated by analysis of mean values of generations according to the model by Mather & Jinks (1971). Based on the ILL and NDI phenotypic variances, were estimated the phenotypic variance $\hat{\sigma}^2_{f\,(F2)} = \hat{\sigma}^2_{F2}$, in which $\hat{\sigma}^2_{F2}$ is the total phenotypic variance of the F_2 generation; genotypic variance $\hat{\sigma}^2_{g(F2)} = \hat{\sigma}^2_{f\,(F2)} - \hat{\sigma}^2_{m(F2)}$, in which $\hat{\sigma}^2_{m(F2)}$ is the environmental variance; additive genetic variance $\hat{\sigma}^2_a = 1/2a^2_{f\,(F2)} = 2\hat{\sigma}^2_{g(F2)} - [\hat{\sigma}^2_{g(RC1)} + \hat{\sigma}^2_{g(RC2)}]$, in which â represents the variance due to the additive effects and $\hat{d}$ the variance due to the dominance deviations $\hat{\sigma}^2_d = 1/4d^2 = \hat{\sigma}^2_{g(F2)} - \hat{\sigma}^2_a$. The narrow sense heritability was estimated according to Cruz (2009) by

$$\hat{h}^2_{ns} = \frac{\hat{\sigma}^2_{a(F2)}}{\hat{\sigma}^2_{f(F2)}} = \frac{\hat{\sigma}^2_{a(F2)}}{\hat{\sigma}^2_{a(F2)} + \hat{\sigma}^2_{d(F2)} + \hat{\sigma}^2_{m(F2)}},$$

while the heterosis percentage was estimated by $\hat{H}(\%) = (\hat{H} * 100)/PA$, where $\hat{H}$ was estimated by the fenotipical F_1 average minus the parental average (PA). Estimates of the minimum number of effective genes (*n*) were calculated according to the formula $n = [(D^2(1 + 0.5K^2)]/8\,\sigma^2_g$, in which D is the amplitude between the mean values of the parental lines, and K represents the mean degree of dominance based on variances, which was estimated by $K = \sqrt{2\sigma^2_d/\sigma^2_a}$ by the Genes 7.0 software (Cruz 2009).

RESULTS AND DISCUSSION

For evaluation of the anthracnose stalk rot severity in maize, most studies use a scoring scale relative to the number of discolored internodes (Badu-Apraku et al. 1987b, Weldekidan & Hawk 1993, Nicoli et al. 2016) or the area of the internal lesion of the stalk (Pereira et al. 1989). In this study, in addition to the evaluation of the number of discolored internodes (NDI), the internal length of the lesion (ILL) was used, since it is a direct and non-subjective method of determination of the anthracnose stalk rot severity. The analysis of variance results showed a highly significant effect ($p < 0.01$) for families, generations and generations inside the families for the variables ILL and NDI (data not shown).

The inoculation of the maize stalks with *C. graminicola* in the different generations of the respective families confirmed, according to the ILL and NDI variables, that the pathogen isolate was pathogenic, because it always conferred a greater length of lesions in the stalks in susceptible lines (L_S 71-1, L_S 95-1 and L_S 99-4). The analysis of the mean values for ILL and NDI confirmed highly contrasting differences among the maize inbred lines used in the families development, in relation to the reaction to *C. graminicola* (Table 1).

For ILL, the resistant lines (L_R 04-2, L_R 03-2 and L_R 23-1) exhibited low values (6.3-15.4 cm), confirming the high potential of these inbred lines as sources of genes for resistance to *C. graminicola*. The line L_R 04-2 stood out positively, with a maximum of 8.3 cm for internal lesion (Table 1). Of the three lines susceptible to anthracnose stalk rot, L_S 71-1 exhibited the highest ILL (146.9-151.6 cm), with a mean of 150 cm, whereas the others had averages of 124.9 (L_S 95-1) and 118.9 cm (L_S 99-4) (Table 1). Matiello et al. (2012) also used the ILL to evaluate maize inbred lines for anthracnose stalk rot resistance. The authors observed a pattern of susceptibility much lower than the susceptible line DAS86, which exhibited an average of 46 cm for internal lesion. In the F_1 generation of the nine families, the ILL ranged from 7.9 to 24.4 cm, confirming the pattern of resistance that the lines L_R 04-2, L_R 03-2 and L_R 23-1 transmit to the hybrid generation (F_1) (Table 1). The maximum segregation generation (F_2) exhibited ILL ranging from 30.2 to 73.8 cm, confirming an intermediate performance, when compared the parental lines. However, when L_S 99-4 was used as a susceptible parental line, the average value was a little higher (46.1 cm), if compared to the other susceptible lines used in the crosses (Table 1).

The mean ILL values for the backcross generations (RC_1 and RC_2) showed a classic

Table 1. Decomposition effect of the generations in the respective families for the variables internal length of the lesion and number of discolored internodes.

Generations	L_R04-2 x L_S71-1	L_R04-2 x L_S95-1	L_R04-2 x L_S99-4	L_R03-2 x L_S71-1	L_R03-2 x L_S95-1	L_R03-2 x L_S99-4	L_R23-1 x L_S71-1	L_R23-1 x L_S95-1	L_R23-1 x L_S99-4
				Internal lenght of the lesion (cm)					
L_R	6.3 B	8.3 C	8.0 C	10.9 C	14.9 C	15.4 C	12.7 D	13.7 C	12.7 C
L_S	151.6 A	127.7 A	103.2 A	151.6 A	120.5 A	116.1 A	146.9 A	126.5 A	137.3 A
F_1	10.4 B	10.6 C	7.9 C	11.4 C	24.4 C	11.3 C	20.2 CD	13.2 C	19.0 C
F_2	31.5 B	30.2 BC	31.5 C	31.7 C	35.8 C	33.1 C	40.0 C	59.9 B	73.8 B
RC_1	11.9 B	10.6 C	9.0 C	7.4 C	20.1 C	14.1 C	16.4 CD	29.0 C	13.9 C
RC_2	137.7 A	40.6 B	73.9 B	71.7 B	68.8 B	80.3 B	78.7 B	128.5 A	36.9 C
				Number of discolored internodes					
L_R	1.1 C	1.3 C	1.3 B	1.5 BC	1.4 C	1.4 C	2.5 C	1.6 C	1.6 C
L_S	5.9 A	5.9 A	5.7 A	5.7 A	5.7 A	5.5 A	5.8 A	5.8 A	5.8 A
F_1	1.3 C	1.2 C	1.3 B	1.3 C	1.4 C	1.2 C	1.5 D	1.4 C	1.6 C
F_2	2.5 B	2.2 B	2.1 B	2.4 B	3.3 B	2.4 B	4.1 B	3.3 B	3.8 B
RC_1	1.5 C	1.3 C	1.3 B	2.0 BC	1.8 C	1.5 C	1.6 D	1.8 C	1.5 C
RC_2	5.4 A	5.4 A	5.4 A	5.5 A	5.5 A	5.4 A	5.7 A	5.6 A	5.7 A

* Means followed by the same letter in the column do not differ statistically from each other by the Tukey test at 5 % of probability.

segregation, i.e., when the backcross involved the resistant lines (L_R 04-2, L_R 03-2 and L_R 23-1), the mean value for the RC_1 generation tended toward a greater resistance, with ILL ranging from 7.4 to 29.0 cm; and when the susceptible lines (L_S 71-1, L_S 95-1 and L_S 99-4) were present in the backcross (RC_2), the ILL was much higher, with amplitude of 36.9-137.7 cm (Table 1). These results corroborate other studies related to genetic control of anthracnose stalk rot resistance (Carson & Hooker 1981, Toman & White 1993, Matiello et al. 2012).

Few studies on genetic control of maize resistance to stalk rot have evaluated the disease based on area of the stalks with lesions (Pereira et al. 1989). The big limitation of this way of evaluation concerns possible errors associated with determination of the area with lesions, the fact that the colonization of the pathogen is not uniform within the stalk, and the variation in the diameter of the internodes due to the position on the stalk (Pereira er al. 1989). The ILL showed to be a better way to evaluate the resistance, since the error input is reduced (Matiello 2004). In general, most studies use a scoring scale relatively to the number of discolored internodes (Hooker 1976, Carson & Hooker 1981, Holley 1988, Keller & Bergstrom 1988, Toman & White 1993, Jung et al. 1994, Denti & Reis 2001, Blum et al. 2003, Yang et al. 2005, Frey et al. 2011). Thus, the choice was made to also evaluate resistance to anthracnose stalk rot using the NDI.

Evaluations of the NDI variable showed that the resistant lines L_R 04-2, L_R 03-2 and L_R 23-1 had a high resistance pattern to *C. graminicola*, since the discoloring of the internodes of these lines was restricted to 1.49 internodes, on average (Table 1). These results corroborate Carson & Hooker (1981), who observed that the resistant line A556 exhibited a NDI of 1.04-1.34. Likewise, Badu-Apraku et al. (1987b) reported that the LB31 line resistant to anthracnose stalk rot did not exceed 1.0 discolored internode. Frey et al. (2011) found 1.6 discolored internodes, on average, for the lines that contained the resistance gene *Rcg1*. In this study, the susceptible lines L_S 71-1, L_S 95-1 and L_S 99-4 exhibited NDI with an amplitude of 5.5-5.9 (Table 1). As the NDI evaluation was limited to the sixth internode of the stalks, many plants of the susceptible lines went beyond the maximum score of the scale (score 6), emphasizing the high susceptibility of these lines to anthracnose stalk rot.

The F_1 generation of all the crosses had a performance very similar to the resistant lines (L_R 04-2, L_R 03-2 and L_R 23-1), with NDI ranging from 1.2 to 1.6 (Table 1). For the F_2 generation, it was found that all the crosses had an intermediate NDI, if compared to the parental generations (L_R and L_S), with amplitude from 2.1 in the cross L_R 04-2 x L_S 99-4 to 4.1 in L_R 23-1 x L_S 71-1 and L_R 23-1 x L_S 99-4 (Table 1). When L_R 23-1 was used in the crosses, there was a tendency toward a greater

susceptibility, with the mean NDI ranging from 3.3 to 4.1 (Table 1). Badu-Apraku et al. (1987b) found similar results for the cross LB31 (resistant) x B37 (susceptible), for anthracnose stalk rot. The authors reported that the F_1 generation was totally resistant, and, in the F_2 generation, 71.4 % of the individuals were ranked as resistant.

The genetic parameters estimates allow a better guidance to choose the method to obtain more resistant genotypes in breeding programs. The decomposition of genetic variance (Table 2), in relation to the nine families, for the ILL, showed that the variance due to additive effects explained 85.1 (L_R 04-2 x L_S 95-1) to 98.07 % (L_R 23-1 x L_S 95-1) of the variation found among the families. The other effects estimated from the model were irrelevant and of low magnitude. Seven families exhibited additive genetic effects higher than 90 % (Table 2). The prevalence of additive effects in genetic resistance control indicate a condition quite favorable to breeding, without difficulties in the identification and selection of genotypes, with a greater concentration of favorable alleles. These results corroborate Carson & Hooker (1981) and Toman & White (1993), who reported additive effects explaining more than 90 % of the variation for the mean values of the generations. In the same way, Jung et al. (1994) found that additive effects were mainly responsible for genetic variation in the studied populations, with indices ranging from 80 to 99 %.

The evaluation of resistance to anthracnose stalk rot by the number of discolored internodes (NDI) is considered an indirect quantification method of the disease, because it does not take into consideration the true length of the lesion caused

Table 2. Variation percentage for internal length of the lesion and number of discolored internodes of anthracnose stalk rot explained by additive effects (â), dominance effects ($\hat{d}$) and epistatic interactions ($\widehat{aa}$, $\widehat{ad}$ and $\widehat{dd}$) for each family and estimates of the coefficient of broad sense heritability ($\hat{h}^2_a$), narrow sense heritability ($\hat{h}^2_r$), heterosis ($\hat{H}$), heterosis percentage ($\hat{H}_{\%}$) and number of genes resistant to anthracnose stalk rot for the variables internal length of the lesion and number of discolored internodes for each family.

Effect	L_R04-2 x L_S71-1	L_R04-2 x L_S95-1	L_R04-2 x L_S99-4	L_R03-2 x L_S71-1	L_R03-2 x L_S95-1	L_R03-2 x L_S99-4	L_R23-1 x L_S71-1	L_R23-1 x L_S95-1	L_R23-1 x L_S99-4
				Internal lenght of the lesion					
M	0.79	6.15	4.48	2.55	0.57	3.75	4.74	0.68	6.32
Â	97.10	85.10	93.12	92.63	94.36	89.31	91.54	98.07	87.09
$\hat{d}$	0.014	2.81	0.06	0.04	0.65	0.19	0.03	0.11	1.74
$\widehat{aa}$	1.22	0.34	0.08	0.98	2.36	0.15	0.01	0.07	1.99
$\widehat{ad}$	0.52	3.90	1.70	3.72	0.55	6.53	3.14	0.15	2.42
$\widehat{dd}$	0.35	1.69	0.55	0.08	1.51	0.07	0.54	0.90	0.43
				Number of discolored internodes					
M	0.05	1.57	1.64	2.90	2.07	0.54	1.75	5.21	4.97
Â	99.66	96.99	97.57	91.42	95.95	95.37	97.49	92.97	89.89
$\hat{d}$	0.05	0.36	0.026	0.74	0.008	0.70	0.002	0.45	0.97
$\widehat{aa}$	0.10	0.03	0.072	0.16	0.032	1.61	0.06	0.59	1.10
$\widehat{ad}$	0.01	0.88	0.64	4.40	1.82	0.02	0.33	0.77	2.84
$\widehat{dd}$	0.13	0.17	0.044	0.37	0.11	1.75	0.36	0.0002	0.22
Estimates	L_R04-2 x L_S71-1	L_R04-2 x L_S95-1	L_R04-2 x L_S99-4	L_R03-2 x L_S71-1	L_R03-2 x L_S95-1	L_R03-2 x L_S99-4	L_R23-1 x L_S71-1	L_R23-1 x L_S95-1	L_R23-1 x L_S99-4
				Internal lenght of the lesion					
$\hat{h}^2_{ns}$ (%)	71.51	88.98	95.61	59.09	50.49	55.01	41.64	34.33	33.63
$\hat{H}$ (cm)	- 68.55	- 57.40	- 47.70	- 69.85	- 43.30	- 54.45	- 59.60	- 56.90	- 56.00
$\hat{H}$ (%)	- 86.83	- 84.41	- 85.79	- 85.97	- 63.96	- 82.81	- 74.69	- 81.17	- 74.66
Nº of genes	3.18	2.34	2.39	4.49	3.64	4.02	3.74	4.36	3.47
				Number of discolored internodes					
$\hat{h}^2_{ns}$ (%)	39.63	69.80	54.24	70.20	50.90	57.71	37.48	53.24	30.64
$\hat{H}$ (cm)	- 2.20	- 2.40	- 2.20	- 2.30	- 2.15	- 2.25	- 2.65	- 2.30	- 2.10
$\hat{H}$ (%)	- 62.86	- 66.67	- 62.86	- 63.89	- 60.56	- 65.22	- 63.85	- 62.16	- 56.76
Nº of genes	2.41	1.47	1.64	1.32	1.58	1.49	2.32	1.22	2.10

by *C. graminicola* (Matiello et al. 2012). Although the evaluation was based on a scoring scale, the results led to a greater uniformity in the phenotypic characterization of the families/generations in the experiment. This uniformity directly affected the magnitude of the estimates of the genetic parameters involved in genetic control resistance in these families. The families derived from L_R 04-2 exhibited a mean additive effect of 98.1 %, whereas L_R 03-2 explained 93.45 % and L_R 23-1 94.25 % (Table 2).

The predominance of additive gene action in the nine families is a good indicator that selection based on resistant individuals will result in a significant genetic progress from artificial selection (Table 2). If the aim is to increase the resistance of segregating populations of the program, the mass selection method would allow a rapid genetic gain. In contrast, if the purpose is to obtain inbred lines resistant to anthracnose stalk rot, the genealogical and backcross methods could be recommended.

Narrow sense heritability ($\hat{h}^2_{ns}$) quantifies the relative importance of phenotypic variance, in relation to the additive proportion of total genetic variance, and its estimation is of great interest to the breeder, because it allows an effective gain in the selection process to be estimated (Hanson 1963). Estimates of narrow sense heritability for ILL revealed an amplitude of 33.63-95.61 %, and the families derived from L_R 04-2 and L_R 03-2 exhibited a coefficient of at least 50.49 %, on average. The lowest heritability coefficients were estimated for the families derived from the source of resistance L_R 23-1 (Table 2).

For NDI, the heritability coefficients were of lower magnitude, if compared to the ILL. Likewise, the lowest estimate was observed for the families derived from the L_R 23-1 source, with a mean heritability of 40.45 % (Table 2). The prediction of gain from selection before carrying out selection assists in defining the best selection strategy, since the estimated value of narrow sense heritability will guide the magnitude of genetic progress from artificial selection (Fehr 1987, Ramalho et al. 1993). The high coefficients of narrow sense heritability, associated with the larger contribution of genetic variance due to the additive effects for resistance inheritance, indicate a greater ease in the artificial selection process of breeding programs that aim at genetic resistance for this pathosystem.

Heterosis is used to characterize the mean superiority of the F_1 generation, in relation to the mean of the parental lines, and it is observed when the trait evaluated in the hybrid is greater (positive) or less (negative) than the mean of the parents (Toman & White 1993). The estimates for heterosis percentage had negative values of large magnitude (Table 2). Regardless of the variable used (ILL and NDI) to quantify the reaction of the families/generations to anthracnose stalk rot, the F_1 generation always showed a significant decrease in lesions from the pathogen, if compared to the susceptible parents.

The estimates for heterosis percentage ($\hat{H}_{\%}$) of the ILL variable indicated coefficients ranging from -63.96 to -85.97 %. In general, all the lines used as a source of resistance (L_R 04-2, L_R 03-2 and L_R 23-1) led to quite significant reductions in filial generations, with decreases of 43.3-69.85 cm for ILL (Table 2). For NDI, the heterosis percentage indicated lower indices, with amplitude from -56.76 to -66.67 % (Table 2). This performance was predictable, because the evaluation of the families/generations by the NDI was based on a scoring scale, which, in a certain way, standardized the disease quantification of the parental and filial generations, among the families studied. In general, the sources of resistance showed the same reduction in the number of discolored internodes of the respective F_1 generations, in relation to the susceptible lines, with amplitude of -2.1 to -2.65 for NDI (Table 2).

The aim of the number of genes estimation or gene blocks is to assist the breeder regarding the best selection method to be used (Matiello 2004), as well as provide an indicator of the degree of difficulty to reach a desired goal. However, it is considered only an indicator, because it is based on a series of genetic presuppositions that normally are not met (Cruz & Regazzi 1997, Lobo et al. 2005). The results found in this study indicated that the estimated number of genes involved in genetic control of maize resistance to anthracnose stalk rot, regardless of the manner of evaluation and the source of resistance, corroborates the hypothesis of oligogenic control of maize resistance to *C. graminicola* in the nine families studied.

For ILL, the estimates related to number of genes revealed an amplitude of 2.3-4.5 (Table 2). It is important to highlight that all the families had quite similar estimates, on average. When L_R 04-2 was used as a source of resistance in the crosses, the estimated mean number of genes was 2.6; 4.1 for the line

L_R 03-2; and 3.86 for L_R 23-1 (Table 2). In relation to NDI, the estimates may have underestimated the number of genes that control the resistance to anthracnose stalk rot, if compared to the ILL (Table 2). The estimated minimum number was 1.2 for the family L_R 23-1 x L_S 95-1 and ranged to, at most, 2.4 genes for the cross L_R 04-2 x L_S 71-1. The lower number of genes for the NDI variable may have been a result of the low amplitude revealed among the parental lines (resistant and susceptible), since the use of the scale limited the quantification of the disease in the susceptible lines to at most six discolored internodes. In fact, for the nine families, the mean number of genes estimated for the NDI was only 1.6, and 3.5 for the ILL (Table 2).

Both methodologies (ILL and NDI) for anthracnose stalk rot quantification were effective in discriminating resistant/susceptible individuals in the nine families of the respective segregating generations. Despite the easiness in evaluating the NDI, there is a greater precision in the use of the ILL as an estimate of resistance to anthracnose stalk rot in maize. Regardless of the form of evaluation (ILL or NDI), the experimental results showed a large phenotypic contrast for resistance among the inbred parental lines. The magnitude of this contrast is a priority for any study that aims to analyze the genetic control of this pathogen x host interaction.

From the genetic parameters estimated for the set of inbred lines of tropical maize, it can be affirmed that genetic control of maize resistance to *C. graminicola* is oligogenic, with an overall mean of 2.6 genes, regardless of the way of evaluation of the disease and of the source of resistance used. The predominance of additive gene action, explaining an average of 93.7 % of the genetic variation among the means of the generations (Table 2), reinforces the importance of this gene action on the determination of the trait in this set of families. Additive variance is one of the determinant factors of covariance among parental lines, and, for that reason, its existence/magnitude reinforces the relationship degree of the unit of selection and the unit to be improved (Cruz & Regazzi 1997). The prevalence of additive gene action and the small number of genes controlling resistance, associated with high coefficients of narrow sense heritability, indicate a condition very favorable to breeding programs, easily increasing the number of individuals with a greater frequency of resistance alleles.

CONCLUSIONS

1. The resistance sources L_R 04-2, L_R 03-2 and L_R 23-1 are very effective in transmitting resistance genes to their descendants, because they allow sharp decreases in the length of lesions in the stalks;
2. The genetic control of tropical maize resistance to anthracnose stalk rot is oligogenic, with additive gene action allowing genetic progress from artificial selection for resistance to anthracnose stalk rot.

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