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ARTICLE

Inheritance of resistance to anthracnose stalk rot (*Colletotrichum graminicola*) in tropical maize inbred lines

Rodrigo Rodrigues Matiello[1*], Kátia Regiane Brunelli[2], Maria Teresa Gomes Lopes[3], Regina Mélo Sartori Coêlho Morello[4], Herberte Pereira da Silva[4] and Luis Eduardo Aranha Camargo[4]

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Abstract - *Generation means was used to study the mode of inheritance of resistance to anthracnose stalk rot in tropical maize. Each population was comprised of six generations in two trials under a randomized block design. Inoculations were performed using a suspension of 10^5 conidia mL^{-1} applied into the stalk. Internal lesion length was directly measured by opening the stalk thirty days after inoculation. Results indicated contrasting modes of inheritance. In one population, dominant gene effects predominated. Besides, additive x dominant and additive x additive interactions were also found. Intermediate values of heritability indicated a complex resistance inheritance probably conditioned by several genes of small effects. An additive-dominant genetic model sufficed to explain the variation in the second population, where additive gene effects predominated. Few genes of major effects control disease resistance in this cross. Heterosis widely differed between populations, which can be attributed to the genetic background of the parental resistant lines.*

Key words: *generation means analysis, disease resistance,* Zea mays*, anthracnose, heterosis.*

INTRODUCTION

Several fungi cause stalk rot in maize, a disease of global importance for its negative effects on grain yield, stalk breakage and premature death of plants (Jarvis et al. 1984). In the U.S., the causal agents of stalk rot species include *Gibberella zeae* (Schwein) Petch, *Colletotrichum graminicola* (Ces.) Wils, *Stenocarpella maydis* (Berk.) Sutton and species of the genus *Fusarium* (*F. verticillioides*, *F. proliferatum* and *F. subglutinans*) (Gath and Munkvold 2002). Of these pathogens, *Colletotrichum graminicola* causes significant annual yield losses estimated at greater than one billion U.S. dollars in the Americas alone (Oestreich 2005). In Brazil, the highest frequency of stalk rot concerns *C. graminicola*, *Stenocarpella maydis*, *S. macrospora*, *Fusarium graminearum* and *F. moniliforme* (Pereira and Pereira 1976, Balmer and Pereira 1987, Reis and Casa 1996, Denti and Reis 2003, Costa et al. 2010).

During the past several years, an expansion of area sown with maize (*Zea mays* L.) over western Brazil and the widespread adoption of the no-tillage cropping system and of a second late cropping have caused a progressive increase in the severity of diseases that once were considered of secondary importance (Lim and White 1978, Coêlho et al. 2001, Denti and Reis 2001, Denti and Reis 2003). Anthracnose stalk rot caused by *Colletotrichum graminicola* (Ces.) Wil. is an example of such disease.

Symptoms of the disease generally appear on the rind as narrow and longitudinal lesions, initially with a brownredish color that becomes dark brown and black with fruiting structures of the fungus (Dale 1963, Shurtleff 1980). Symptoms also include discoloration of pith and stalk breakage (Miles et al. 1980, Bergstrom and Nicholson 1999, Muimba-Kankolongo and Bergstrom 2011). Usually, these symptoms start immediately after flowering. The fungus also infects leaves, but there are indications that different genes control leaf and stalk anthracnose (Lim and White 1978, Zuber et al. 1981, Coêlho et al. 2001, Rezende et al. 2004). Studies concerning the mode of inheritance of genetic

1 Universidade Estadual de Ponta Grossa (UEPG), Departamento de Fitotecnia e Fitossanidade, 84.030-900, Ponta Grossa, PR, Brazil. *E-mail: rrmatiel@uepg.br

2 Sakata Seed Sudamerica, Estrada da Bocaina, s/n, 12.906-840, Bragança Paulista, SP, Brazil

3 Universidade Federal do Amazonas (UFAM), Departamento de Produção Animal e Vegetal, 69.077-000, Manaus, AM, Brazil

4 Universidade de São Paulo (USP/ESALQ), Departamento de Fitopatologia e Nematologia, 13.418-900, Piracicaba, SP, Brazil

resistance to stalk rot indicated that it is conditioned by few loci mainly of additive genetic effects (Lim and White 1978, Weldekidan and Hawk 1993). Estimates of the number of genes also indicate oligogenic control with additive and dominant genetic effects (Pereira et al. 1989, Toman and White 1993). Callaway et al. (1990) found significant heterosis and general and specific combining abilities, indicating that either additive or dominant effects contribute to resistance. While most studies involving this pathosystem indicated a quantitative mode of inheritance of resistance, Badu-Apraku et al. (1987) reported a single dominant gene in a cross between tropical and temperate lines.

Given that genetic resistance most efficiently controls the disease, knowledge about the mode of resistance inheritance allows the breeder to define the best selection method, thus increasing the efficiency of selection and maximizing the genetic gains during this procedure. This work reports the mode of resistance to anthracnose stalk rot inheritance in generations of crosses between tropical maize inbred lines.

MATERIAL AND METHODS

Three inbred lines (Das21, Das64, and Das86) were used to develop two populations from the crosses between Das86 x Das21 and Das86 x Das64. Das21 is resistant to anthracnose stalk rot. It is derived from a synthetic population of tropical lines with a prevalence of Suwan DMR germplasm. Suwan DMR was developed in Thailand through selection of tropical genotypes containing Caribbean flint and Tuxpeño dent germplasm (Lanza et al. 1999, Coêlho et al. 2001, Rezende et al. 2004). Das64 and Das86 were derived from a synthetic population (narrow genetic base) formed by lines of Amarillo dent and Caribbean flint populations. Both lines have semi-hard orange grains. Das64 is short and resistant to anthracnose stalk rot while Das86 is intermediate in height and highly susceptible (Coêlho et al. 2001).

Field trials for resistance to stalk rot evaluation were comprised of parental (P_1 and P_2), F_1, F_2 and the two backcrosses (BC_1 and BC_2) generations of plants.

The pathogen was isolated from pieces of maize stalk collected in Iraí de Minas (Minas Gerais, Brazil). The stalk fragments were surface-disinfected (30 seconds in 70 % alcohol, 2 min in 1 % sodium hypochlorite, and 1 min in distilled water) and transferred to Pirexâ Petri dishes (9 cm diameter) containing oatmeal agar medium (40 g oatmeal, 17 g agar, and 1 L distilled water). Plates were kept inside a growth chamber at 22 ± 2 ^{0}C under fluorescent light (12-hr light and 12-hr dark) until the fungus sporulated (approximately 14 days).

Inoculum was prepared by adding 20 mL of distilled water to the Petri dishes and scrapping the colonies in order to dislodge the conidia. The suspension was then filtered through double layers of cheesecloth. Before inoculation, the concentration of the spore suspension was adjusted to 1.8×10^5 conidia mL^{-1}.

Two trials were conducted in November (conventional planting season) and December (late planting season) 2001, in Indianópolis (Minas Gerais, Brazil) using a randomized complete block design. The trials were laid out in split plot design with three replications. Populations were assigned to main plots, and generations to subplots. The parental inbreds and the F_1 generations were sown in single row plots; F_2 generations, in four row plots, and the backcrosses (BC_1 and BC_2), in two row plots. Rows in each plot were 5m long, spaced 0.9cm apart, and contained approximately 20 plants.

Plants were inoculated at the flowering stage by injecting 1 mL of a 1.8×10^5 conidia mL^{-1} suspension into the first visible internode from above ground using a 50 mL veterinary syringe. Symptoms were evaluated 30 days after inoculation in all plants of the plots by removing leaves and ears and cutting the plants prior to longitudinally split them with an electric saw. Then, internal lesion length was measured (cm).

Analysis of variance (ANOVA) was performed for each trial as well as for the combined data using SAS (SAS Institute 2000) in order to detect differences between and within populations. Genetic effects were estimated through generation means analysis according to Mather and Jinks (1971). Initially, the genetic parameters were estimated by using a reduced genetic model comprised of additive and dominant effects as well as deviations from the model. In the presence of significant deviations, a more complete model was used, where additive x additive and additive x dominant epistatic interactions were included. Analysis of variance of the genetic parameters was performed according to Miranda Filho (1991).

Heterosis (H%) was calculated by the formula $H\% = (F_1 - MP) \times 100$, where MP represents the mean trait value between the parental lines. Broad sense heritability (h^2) was calculated by the formula: $h^2 = \left(\sigma^2_{F_2} - \sigma^2_e\right) \Big/ \sigma^2_{F_2}$, where $\sigma^2_{F_2}$ is the phenotypic variance among individuals from the F_2 generation and σ^2_e is the environmental variance estimated by grouping the sum of squares and degrees of freedom from the parental lines and from the F_1 generation (Hallauer and Miranda Filho 1988).

RESULTS AND DISCUSSION

The experimental conditions in both trials were appropriate for the development of the disease, as indicated by the high values of internal lesion length observed in the susceptible hybrid Das86 x Das21. For evaluating the severity of this disease, grading scales related either to the number of discolored internodes (Badu-Apraku et al. 1987, Weldekidan and Hawk 1993, Frey et al. 2011, Muimba-Kankolongo and Bergstrom 2011) or to the internal lesioned area of the stalk (Pereira et al. 1989) are normally used. In this work, however, internal lesion length was measured instead because it is a direct and non-subjective method of assessing disease severity. This method was effective in distinguishing resistant from susceptible individuals within generations and the populations.

The combined ANOVA of the two trials indicated significant interaction between trials and generations within populations. Therefore, it was opted to present and discuss the results based on the individual ANOVA of each trial. Significant differences between and within populations for mean of internal lesion length of *C. graminicola* were found in both trials (Table 1) likewise Pereira et al. (1989) and Rezende et al. (2004). The coefficients of variation of two trials were small magnitude and suitable for trials involving interaction pathogen-host in agreement with Zuber et al. (1981), Paterniani et al. (2000) and Rezende et al. (2004).

Table 1. Analysis of variance of mean lesion length of anthracnose stalk rot in the first and second trials

		Mean Square	
Sources of variation	**df**	**Trial 1**	**Trial 2**
Blocks	2	3.8	0.8
Population	1	2953.9 **	2466.3 **
Error a	2	11.5	20.7
Generation (Population)	10	781.5 **	582.8 **
Error b	20	16.9	7.8
Mean CV (%)		41.56 9.9	38.78 7.1

**, significant at $P < 0.01$

The susceptible line Das86 was the most susceptible while Das64 was the most resistant (Table 2). The F_1 cross Das86 x Das21 was more susceptible than the susceptible parental line Das86. The distribution of lesion length in the F_2 generation of this population was skewed towards susceptibility (Figure 1A, B). However, transgressive resistant individuals were found with approximately 29.6 and 16.2% of the individuals being more resistant than the resistant parent (Das21), in the two trials. On the other hand, the F_1 cross Das86 x Das64 was as susceptible as the susceptible parent (Das86). However, 15 and 21.7% of F_2 plants were more resistant (transgressive) than the resistant parental line (Das64) in trials 1 and 2, respectively (Figure 1C, D). Transgressive resistant individuals were observed as 15 and 21.7% of the F_2 plants were more resistant than the resistant parental line in trials 1 and 2, respectively (Table 2).

Table 2. Mean of internal lesion length (cm) of anthracnose stalk rot in six generations of two populations and estimates of heterosis and broad-sense heritability

	Das86 (P1) x Das21 (P2)		Das86 (P1) x Das64 (P2)	
Generations / Trials	**Trial 1**	**Trial 2**	**Trial 1**	**Trial 2**
P_1	35.4	46.0	39.9	38.2
P_2	23.9	30.1	16.4	18.5
F_1	77.4	79.3	37.8	38.3
F_2	57.2	40.9	33.5	32.7
BC_1	69.6	51.0	40.3	35.3
BC_2	40.2	30.6	27.1	24.5
Heterosis (%)	47.7	41.3	9.6	9.9
Broad-sense heritability (%)	85.7	44.6	29.2	51.5

In the population Das86 x Das64, the distribution of mean lesion length of the F_2 generation was less skewed towards susceptibility (Figure 1C, D). The level of susceptibility of the F_1 hybrids in both crosses suggests the presence of dominant alleles for susceptibility. The mean of backcross generations (BC_1 and BC_2) of both populations showed a typical behavior, that is, when the cross involved the susceptible genitor, the mean of internal lesion length of the generation was higher than when it involved the resistant one (Table 2). This corroborates earlier reports by Carson and Hooker (1981), Badu-Apraku et al. (1987), Callaway et al. (1990), as well as recent publication about this pathosystem by Frey et al. (2011) and Muimba-Kankolongo and Bergstrom (2011).

Genetic parameters were estimated by using the reduced model (additive-dominant) for Das86 x Das64 and the complete model for Das86 x Das21, due to the presence of significant deviations. Results indicated distinct modes of disease resistance inheritance between these populations. These results are consistent with those about anthracnose stalk rot, obtained by Lim and White (1978), Carson and Hooker (1981) and Pereira et al. (1989), as well as about anthracnose leaf blight, obtained by Badu-Apraku et al. (1987), Coêlho et al. (2001), Rezende et al. (2004). In Das86 x Das21, significant dominant and additive x dominant epistatic effects were detected in the first trial (Table 3). However, in the second trial additive, dominant, and both epistatic effects (additive x dominant and additive x additive) were significant. Notwithstanding, dominance effects were prevailed in both trials, accounting for 41.7 and 57.9% of

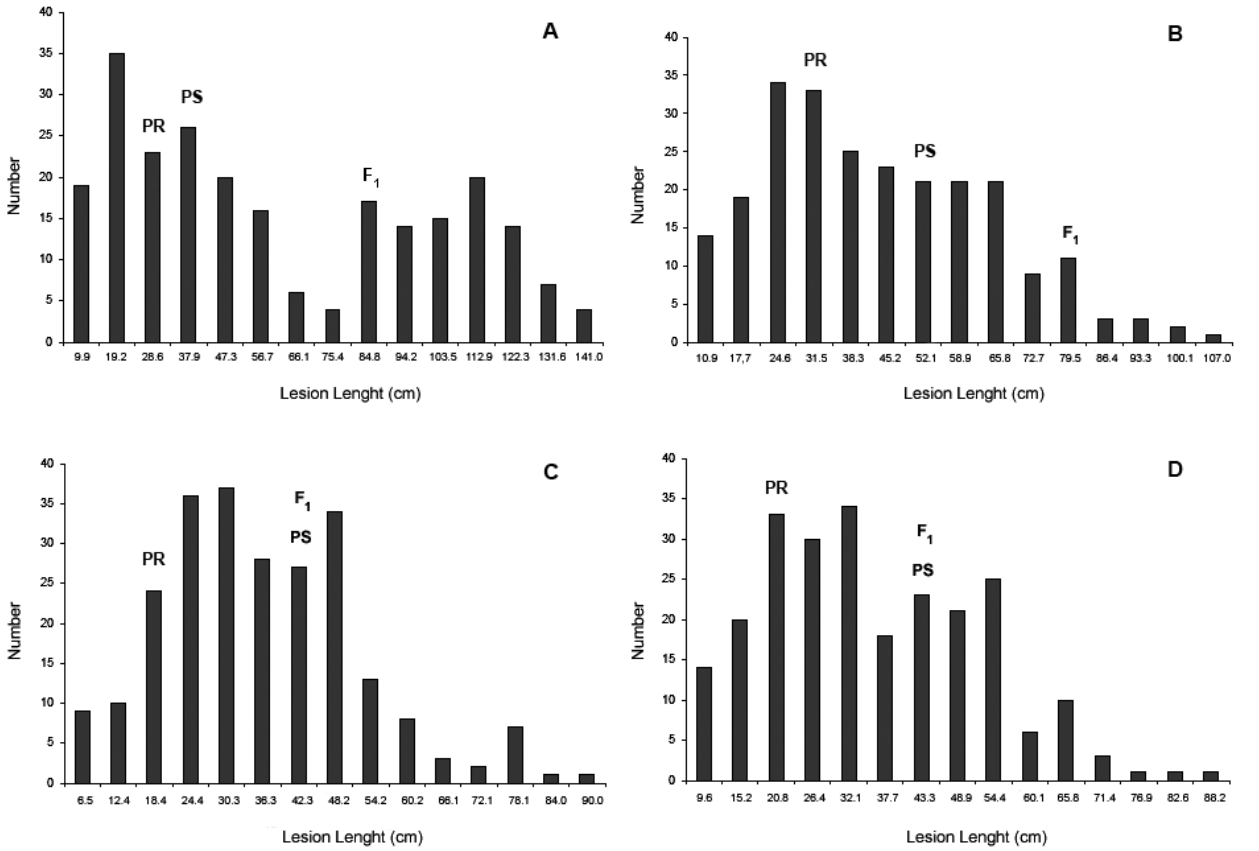

Figure 1. Number of plants in each class lesion length (cm) in two populations evaluated in two trials. **A** and **B**: population Das86 x Das21 trials 1 and 2, respectively; **C** and **D**: population Das86 x Das64 trials 1 and 2, respectively. **PR**, **PS** and $\mathbf{F_1}$ indicate resistant parent, susceptible parent and hybrids generation in each population.

the total genetic variance (Table 4). In the case of interactions, the additive x dominant explained 43.4% of genetic variance for the first trial, while on the second, the additive x additive interaction accounted for 23.4%. The other genetic effects estimated by the complete model were small.

On the other hand, in population Das86 x Das64 large additive effects, which accounted for more than 80% of the total genetic variation, were detected in both trials with only a small dominance effect being detected in the second trial. In this case, the additive-dominant reduced genetic model sufficed to explain the variations found between generations (Tables 3 and 4).

Analysis of generation means indicated a contrasting mode of inheritance between Das86 x Das21 and Das86 x Das64, which could be explained by differences in the genetic background of the resistant lines Das21 and Das64 (Badu-Apraku et al. 1987, Pereira et al. 1989, Lanza et al. 1999, Coêlho et al. 2001, Rezende et al. 2004). The prevalence of additive genetic action in the population Das86 x Das64 in both trials suggests that selection based on resistant individuals will result in genetic progress in this population. Carson and Hooker (1981) reported that the additive effects accounted for more than 90% of the total variation in crosses studied. The present results also showed that additive effects are an important source of variation (Das86 x Das64), especially when crossing lines contrasting for resistance expression. Weldekidan and Hawk (1993) found sum of squares for GCA 4-5 times larger than specific combining ability (SCA) indicating that additive effects were more important than nonadditive effects for ASR resistance.

In Das86 x Das21, however, most of the genetic variance was explained by dominance effects and digenic epistatic interactions. Even after adjusting the data to the complete model, deviations still remained significant, indicating that resistance is affected by higher order epistatic interactions involving more than two loci. These interactions were not estimated because it would require more generations. These results are in accordance with Badu-Apraku et al. (1987) and Rezende et al. (2004), who also reported dominance and epistatic effects

Table 3. Analysis of variance and partitioning of variation among generations of mean internal lesion length of anthracnose stalk rot of two populations evaluated in two trials

		Mean Square			
		Das86 x Das21		Das86 x Das64	
Sources of variation	**df**	**Trial 1**	**Trial 2**	**Trial 1**	**Trial 2**
Generations	6				
Parameters	5	3509.2**	2886.8 **	86.81**	64.60**
Additive (A)	1	66.7	127.5 **	364.45**	250.97**
Dominant (D)	1	214.5 **	876.1 **	68.17	57.70*
Deviation[a]	3	-	-	0.47	4.78
A x A	1	9.67	353.8 **		
A x D	1	222.9 **	61.7 *		
Deviation	1	0.21	94.6 **		
Error (b)[b]	20	5.62	2.61	5.62	2.61

*, ** significant at $P < 0.05$ and $P < 0.01$
[a] Deviation to the additive-dominant genetic model for Das86 X Das64
[b] Error term from the individual analysis of variance

Table 4. Percentage of variation in mean of internal lesion length of anthracnose stalk rot explained by additive, dominant, and epistatic effects and deviations from the adjusted models for two populations evaluated in two trials

	Das86 x Das21		Das86 x Das64	
Effects	**Trial 1**	**Trial 2**	**Trial 1**	**Trial 2**
Additive (A)	12.98	8.42	84.2	80.1
Dominant (D)	41.73	57.88	15.7	18.4
A x A	1.88	23.37	- [1]	- [1]
A x D	43.37	4.08	- [1]	- [1]
Deviations	0.04	6.25	0.1	1.5

[1] Epistatic interactions not estimated.

in some crosses analyzed. The higher susceptibility observed in this hybrid could result from the combination of dominant alleles conferring susceptibility present in both parents at different loci. This conclusion is supported by the fact that Das21 presented intermediate levels of resistance, indicating that it could carry some susceptibility alleles.

Broad sense heritability estimates were of intermediate magnitude and varied according to the population and to the trial (Table 2). Heterosis estimates were positive (i.e. more susceptibility) and highly variable both between trials and between populations (Table 2). Likewise, Pereira et al. (1989) reported that the direction of dominance was not consistent among crosses, but indicated a tendency of heterosis toward higher susceptibility to *C. graminicola*.

Recently, Frey et al. (2011) reported that the presence of major resistance gene to ASR (*Rcg1*) reduced the overall yield impact incurred by infection in inoculated plots by almost three fold in comparison to hybrids that lack the gene. Studies indicate small utilization of locus *Rcg*1 in modern maize in the world. Frey et al. (2011) observed that, in a screen of inbred lines that represent greater than 90% of the genetic diversity in public inbred lines of maize, *Rcg*1 was found in 5 out of 93 maize inbreds.

The present results indicated distinct modes of resistance to anthracnose stalk rot inheritance between two populations derived from crossing three tropical maize inbreds. Thus, different selection methods may be necessary depending on the genetic background of the parental lines. However, genetic improvement is possible by selecting and recombining transgressive resistant individuals in both populations of tropical maize.

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Herança da resistência a antracnose do colmo (*Colletotrichum graminicola*) em linhagens endogâmicas de milho tropical

Resumo - *Análise de médias foi usada para estudar o modo de herança da resistência a antracnose do colmo em milho tropical. Cada população foi composta de seis gerações que foram avaliadas para resistência em dois ensaios no delineamento de blocos ao acaso. As inoculações foram realizadas usando uma suspensão de 10^5 conídios mL^{-1} aplicados dentro do colmo. O comprimento interno de lesão foi medido diretamente após abertura do colmo 30 dias após a inoculação. Os resultados indicaram modos contrastantes de herança. Em uma população, os efeitos gênicos dominantes predominaram. Embora as interações, aditivo x dominante e aditivo x aditivo, também foram encontradas. Valores intermediários de herdabilidade indicaram herança da resistência complexa, provavelmente condicionada por vários genes de pequeno efeito. O modelo genético aditivo-dominante foi suficiente para explicar a variação na segunda população, onde os efeitos aditivos predominaram. Poucos genes de grande efeito controlam a resistência neste cruzamento. Heterose diferiu amplamente entre as populações o que pode ser explicado pelo background genético das linhagens genitoras resistentes.*

Palavras-chave: *análise de média de gerações, resistência a doenças,* Zea mays*, antracnose e heterose.*

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