Genetic control of common bean reaction to angular leaf spot

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ABSTRACT

The Carioca MG (susceptible = P_1) and Pérola (resistant = P_2) cultivars were used as parents in a cross aiming at investigating the genetic control of the common bean plant reaction to *Phaeoisariopsis griseola.* Two hundred and fifty-one families (141 $F_{2:3}$, 53 F_2 RC_{11} and 57 RC_{12}) from this cross were evaluated for angular leaf spot severity in the leaves and pods, in two generations, under natural incidence of the pathogen. The genetic variance components, heritability in the broad (h_b^2) and narrow (h_n^2) senses and number of genes that control the reaction to angular leaf spot were estimated. The families were genetically different for level of resistance to the pathogen, and varied from 59.20% to 72.41% and varied from 19.32% to 73.79% for the leaf analysis. For reaction to the pathogen in the pods, the varied from 55.00% to 68.22% and was nil, indicating the presence of dominant alleles in the control of the trait, and of genes different from those responsible for the control of the disease in the leaves. Only one gene was estimated for the genetic control of the reaction to the pathogen in the leaves and two genes in the pods.

KEY WORDS: Angular leaf spot, genetic parameters, *phaseolus vulgaris*, number of genes.

INTRODUCTION

Among the various diseases that occur in the common bean crop, the angular leaf spot, caused by the *Phaeoisariopsis griseola* fungus, is the most frequent and generalized in almost all the cropping regions. For many years, this disease was considered of secondary importance because it was believed that it only occurred at the end of the crop cycle. However, after successive crops in the same area, it was verified that the pathogen affects the plant during all stages of development, mainly during the dry season and winter sowings, when they are spray irrigated (Sartorato and Rava, 1994). Grain yield losses can reach 80%, depending on the occurrence period and susceptibility of the cultivar (Brenes et al., 1983; Satorato and Rava, 1992; Vieira et al., 1998).

This disease can be controlled by planting pathogen-free seeds and by using chemical treatment, crop rotation and resistant cultivars. Resistance is certainly the most economic means for the farmer to control the disease because inoculum is available in the field at almost any time of the year. However, resistant genotypes in one location may be susceptible in another because the fungus presents high pathogenic variability (Sartorato and Rava, 1994; Pastor-Corrales et al., 1998; Nietsche et al., 2001).

Furthermore, obtaining resistant cultivars is difficult because of the inconsistency of data on the genetic control of the reaction of the common bean to the pathogen. Some studies have presented evidence that there are two or three independent genes, dominant or recessive, others studies have shown only one gene, and resistance may be due to a dominant allele or to a recessive allele (Barros et al., 1958; Singh and Saini, 1980; Carvalho et al., 1998; Ferreira et al., 2000; Nietsche et al., 2000). Such results evidently depend on the parents used and on the pathogenic variability, so it is important to know the genetic control of the most promising resistant sources to be used in breeding programs for the pathogen population of the region to obtain new resistant cultivars.

The objective of this study was to investigate the genetic control of the common bean reaction to *P. griseola* in the 'Carioca MG' x 'Pérola' cross in the south of Minas Gerais state, Brazil.

MATERIAL AND METHODS

One hundred and forty-one $F_{2:3}$, 53 F_2RC_{11} (F_1 x P_1) and 57 F_2RC_{12} (F_1 x P_2) families were obtained from the cross between the parents 'Carioca MG' (P_1) and 'Pérola' (P_2). 'Carioca MG' is highly susceptible and 'Pérola' has shown moderate field resistance (Marques Jr, et al., 1997; Carneiro 2002). These families were evaluated in the field, in the counties

of Lavras and Lambari, in the 1999 dry season, from February to May. The Carioca MG cultivar was sown as border about 15 days before setting up the experiments as a source of natural inoculum of the pathogen. A Federer augmented block design was used, in which the families were the regular treatments and the parents the common treatments. These treatments were repeated at every ten families. The experiment was spray irrigated, and the air humidity, although it had not been measured during the evaluation, it is normally low during those months in the South of Minas Gerais State, and the temperature is usually lower in Lambari than in Lavras.

The symptoms of the disease were evaluated in the leaves and pods, using a diagrammatic scale of scores 1 to 9, where 1 stands for complete resistance and nine for high susceptibility to the pathogen (Costa et al., 1990). In Lambari, the families were evaluated considering the severity of the disease in the leaves, in three assessment periods, the first at 55 days, the second at 65 days and the third at 75 days after emergence. The presence of the disease in the pods was evaluated at 80 days after emergence. Two evaluations of the disease severity in the leaves (65 and 75 days after emergence) and one in the pods at 80 days after emergence were made in Lavras.

Two criteria were adopted to perform the analysis of variance of the disease severity in the leaves. In the first case, analysis of variance was performed per location and each assessment in the leaves was considered a replication. The second criterion involved a joint analysis of locations (Lavras and Lambari). Evaluations mean per plot (mean disease severity) was used for leaf analysis in each location, and locations were considered as replications. In both cases a randomized complete block design was used. Only the second analysis of variance criterion could be adopted for disease severity in the pods, that is, locations (Lavras and Lambari) were considered as replications, because only one evaluation was done in each experiment.

The components of the genetic variance, the heritabilities in the broad (h_b^2) and narrow (h_n^2) senses and the number of genes, that control the reaction to *P. griseola* in the leaves and pods, were estimated from the analyses of variance (Ramalho et al., 1993). Furthermore, heterosis in the different assessments was estimated by the least squares method (Mather and Jinks, 1984) from the mean of the parents, the $F_{2:3}$ families and the F_2 backcross families.

In the subsequent generation, these same families (141 $F_{2:4}$ families, 53 F_3RC_{11} families and 57 F_3RC_{12} families) were evaluated in a simple 16 x 16 lattice design in the field, under natural pathogen incidence, in the 2000 dry season, in Lavras. Families were evaluated for disease severity in the leaves on two occasions, the first by one evaluator at 65 days after emergence and second by two evaluators at 75 days after emergence. The disease severity was also evaluated in the pods, by one evaluator at 80 days after emergence. The same diagrammatic score scale used in 1999 was applied.

Individual analyses of variance using the leaf disease severity data were performed for each period and evaluator, using the data of the area under the disease progress curve (AUDPC).

The AUDPC was estimated using the data for disease severity in each plot by the formula of Godoy (1995):

$$AUDPC = \sum_{i=1}^{n} [(x_i + x_{i+1}) / 2](t_{i+1} - t_i)$$

where x_i is the mean disease severity per plot (%) of the ith evaluation, n is the number of evaluations and $t_{i+1} - t_i$ is the interval between two consecutive evaluations.

The genetic variance components, the heritabilities in the broad and narrow senses and the number of genes that control the reaction to *P. griseola* in the leaves and pods were estimated from the analyses of variance (Ramalho et al., 1993). The heterosis in the different evaluations was also estimated, as already described, using the $F_{2:4}$ and F_3 of the backcrosses means adjusted for the parents. Furthermore, correlations between AUDPC and the different severity evaluations of angular leaf spot in the leaves and pods were estimated.

RESULTS AND DISCUSSION

In the 1999 experiment, the effects of the evaluation periods (replications) and families were significant for the angular leaf spot disease severity, according to the first criterion of analysis of variance in both locations, Lavras (CV = 11.77%) and Lambari (CV = 16.08%). The differences among replications indicated that there were differences among the assessment periods, due to the increased disease severity in the third evaluation period compared to the second and first periods in Lambari and increased

disease severity in the second period compared to the first period at Lavras. The mean values for leaf scores in Lambari were 1.90, 2.42 and 2.87 for the first, second and third evaluation periods, respectively. In Lavras, the mean leaf scores were 3.0 for the first period and 3.95 for the second evaluation period. The differences among the families indicated the presence of resistant and susceptible genotypes to the pathogen.

When the locations were treated as replications there were also significant effects ($P<0.01$) of locations and families on the leaf reaction scores. The evaluations made in the pods also showed (CV = 16.69%) differences between locations ($P<0.01$) and among families ($P<0.05$). In this case, the difference among replications indicated differences of mean disease severity scores in the two locations which were smaller in Lambari (mean leaf disease severity = 2.40; mean pod disease severity = 1.72) than in Lavras (leaf mean = 3.47; pod mean = 3.41) probably due to the lower temperatures found in Lambari. Furthermore, it is probable that different *P. griseola* races occurred in the two locations, and some races in Lavras may have been more aggressive.

Some surveys on pathogen races in various regions have shown that the pathogen races vary among the locations. Nietsche et al. (2001) assessed 30 *P. griseola* isolates collected in Minas Gerais state and identified 13 pathogen races, six identified in Lavras and three in Lambari. Two of the three races identified in Lambari were also reported in Lavras. Therefore it is probable that some of the pathogen races that occurred in Lavras and Lambari were different. When each location was considered a replication, marked family x race interaction was expected, which should inflate the experimental error and perhaps even prevent detection of genetic differences among families for angular leaf spot disease severity. However, the slight tendency to increase the experimental error and the experimental variation coefficient, as well as the 34% reduction among families variability suggest that different races in the two locations are probably contributing to the differences in disease severity and aggressiveness. Therefore due to the small family x race interaction, it is then inferred that the variability shown by the families was mainly due to horizontal resistance, although some variation due to vertical resistance common in the two locations may also exist. There are few studies of these two kinds of resistance in common bean, however they have been observed (Satorato and Rava, 1994; Abreu et al. 2002, Sartorato 2002). Consequently, the use of an individual experiment evaluation for the genetic parameter estimation was decided.

In the analyses carried out to evaluate leaf disease severity, in 2000, it was detected that the families showed different resistance levels ($P<0.01$) in both periods (CV = 17.97% for the first and CV = 14.24% for the second). In the latter case, the mean of scores from two evaluators was used. The AUDPC (CV = 13.80%) data analysis including all the evaluations was also carried out. There was also difference among the families ($P < 0.01$) when the incidence of the disease was evaluated in the pods, although the mean reaction was lower than that of the leaves. As the disease generally develops in the pods after the leaf occurrence, this low value was mainly due to low temperatures after the second leaf assessment. In addition, the genes that express resistance to *P. griseola* in the leaves and pods must be different.

The estimates of heterosis were nil for the incidence of the disease in the leaves in both the 1999 and 2000 experiments (Tables 1 and 2). This indicates that the genes that control the reaction to *P. griseola* have additive genetic action, which was confirmed by the estimates of the additive and dominant variances (Tables 1 and 2). The negative estimates of dominance variance can be considered zero (Silva, 2003). The positive estimates of additive variance indicated that it is the only cause of genetic variation that explains the reaction in the leaves. The heterosis estimate was also nil for pod reaction, and the estimates of additive and dominant variances were, respectively, negative and positive indicating presence of only dominance effect (Tables 1 and 2).

The number of genes controlling the reaction to the pathogen in the leaves was estimated as only one in all the disease severity evaluations. On the other hand, two genes were detected for the reaction to the pathogen in the pods (Tables 1 and 2). Most of the studies reported in the literature estimated the number of genes that control the reaction to *P. griseola* from leaf evaluations alone or from joint leaf and pod evaluations. The number of genes controlling the reaction to the pathogen in the leaves detected in this research was in line with that obtained by other authors working with other cultivars. For example, Nietsche et al. (2000) found that resistance to angular leaf spot in the Cornell 49-242 cultivar is controlled by a dominant allele. The same result was reported by Ferreira et al. (2000) for the MAR-2 cultivar and by Carvalho et al. (1998) in the AND-277 cultivar.

Considering the disease severity evaluations and the number of genes estimated for leaves and pods, it was inferred that the genetic control of the reaction to this pathogen must be different in each plant organ, mainly because only additive effects were detected for the reaction to the disease in the leaves and only dominant effects for the reaction in the pods. This was also confirmed by the low correlation of the mean reaction of the disease in the leaves and pods in the evaluated families (Table 3). Different kinds of common bean resistance to the pathogen have already been observed, like the resistance of leaves and pods in the 'Jalo' cultivar or resistance only in the pods in its descent line P 24 (Mendonça et al., 1999). Similar results have also been observed in the pathosystems *Phaseolus vulgaris x Xanthomonas axonopodis* pv. *phaseoli* (Valladares-Sanchez et al., 1983; Rodrigues et al., 1999).

In this study, both the AUDPC and the general mean of the evaluations were equivalent (Table 3). Therefore, both estimates were efficient for evaluating angular leaf spot disease severity. The correlation among the different evaluations of leaf severity, involving different evaluators and periods, indicate that only one evaluation is efficient (Table 3). However, it is important to point out that the use of the mean score of more than one evaluator reduces the inherent deviation of each evaluation, leading to more precise results as indicated by the smaller values of the experimental coefficient of variation (Marques Jr et al., 1997). Then, whenever possible, it is necessary to use more than one evaluation period and more than one evaluator.

Tables 1 and 2 show the heritability estimates in the broad and narrow sense for the severity of angular leaf spot in 1999 and 2000. These estimates indicate high possibility of gain from selection for resistance to *P. griseola* in the leaves. Once again, results for the AUDPC, which summarizes all the evaluations, were more efficient for superior genotype selection. The higher value for h_n^2 in the first evaluation was mostly due to the estimating error of the genetic variance components. Note that the corresponding h_b^2 is lower than the which is not common (Ramalho et al., 1993). Regarding pod disease incidence, the does not have any meaning because the dominance effects predominate (Tables 1 and 2). However, the also suggests possibilities of gain from selection. In the case of autogamous plants such as the common bean, the interest is for inbred lines that carry the alleles that express resistance to the pathogen, such as those of the 'Pérola' parent.

The small number of genes estimated, both for leaf and for pod resistance, suggest that 190 (P³95%) $F_{2:3}$ or $F_{2:4}$ segregating families are enough (Ramalho et al., 2001). However, as there is evidence of both vertical and horizontal resistance (Satorato and Rava, 1994) the size of the segregating population should have as many plants as possible, since modifier genes should also be present, although they were not detected in the present study.

CONCLUSIONS

The genes that express resistance to *P. griseola* in the leaves and pods have different genetic actions, and those that control the reaction in the leaves show additive effects while those that control the reaction in the pods show predominant dominant effects;

Only one gene was estimated in control of the leaf resistance reaction to *P. griseola* while two genes were expressing pod resistance reaction, and the alleles for resistance in both organs are present in the Pérola cultivar.

RESUMO

Controle genético da reação do feijoeiro à mancha angular nas vagens

Visando-se conhecer o controle genético da reação do feijoeiro ao *Phaeoisariopsis griseola*, foram cruzados os genitores 'Carioca MG' (suscetível) e 'Pérola' (resistente). Desse cruzamento foram obtidas 251 famílias (141 $F_{2:3}$, 53 F_2 RC_{11} e 57 RC_{12}), as quais foram avaliadas quanto a severidade da mancha angular nas folhas e vagens, sob incidência natural do patógeno. Foram estimados componentes de variância genética, herdabilidades nos sentidos amplo (h_a^2) e restrito (h_r^2) e número de genes que controlam a reação à mancha angular. Verificou-se que as famílias foram geneticamente diferentes em relação ao nível de resistência ao patógeno, sendo que a variou de 59,20% a 72,41% e a variou de 19,32% a 73,79%, para as folhas. Para a reação à mancha angular nas vagens, a variou de 55,00% a 68,22% e a foi nula, indicando a presença de alelos dominantes para controle do caráter, de genes diferentes daquele responsável pelo controle da reação à doença nas folhas. Foi estimado apenas um gene para o controle

Table 1. Mean reaction of the common bean 'Carioca MG' and 'Pérola' parents to *P.griseola* and estimates of genetic and phenotypic parameters for the severity of angular leaf spot in the 'Carioca MG' x 'Pérola' cross. Lavras and Lambari, 1999.

	Lavras		Lambari	
Genetic and phenotypic parameters	Leaves	Pods	Leaves	Pods
Mean of 'Carioca MG'	3.71± 0.62	3.94 ± 0.42	3.13 ± 0.49	1.87 ± 0.31
Mean of 'Pérola'	3.35 ± 0.62	3.15 ± 0.42	2.35 ± 0.49	1.66 ± 0.31
Heterosis	-0.24 ± 9.18	-0.60 ± 6.21	-0.45 ± 7.23	-0.22 ± 4.56
Additive variance	0.33	-0.01	0.10	-0.04
Dominance variance	-0.26	0.11	-0.01	0.05
Genetic variance of the $F_{2:3}$ families	0.27	0.02	0.10	-0.02
Genetic variance of the F_2 (RC) families	0.21	0.04	0.10	-0.01
Environmental variance	0.42	0.20	0.26	0.12
Heritability in the broad sense	62.98%	77.83%	72.41%	72.98%
Heritability in the narrow sense	68.22%	-2.49%	29.31%	-27.12%
Number of genes	1.0	2.0	1.0	2.0

Table 2. Mean reaction of common bean to *P. griseola* of the 'Carioca MG' and 'Pérola' parents and estimates of genetic and phenotypic parameters for incidence of angular leaf spot in the 'Carioca MG' x 'Pérola' cross. Lavras, 2000.

	Leaves			Pods
Genetic and phenotypic parameters	Period 1	Period 2	AUDPC[1]	
	Evaluator 1	Mean of 2 evaluators	Period 1 and 2	Evaluator 1
Mean of 'Carioca MG'	6.04 ± 0.82	6.18 ± 0.68	54.97 ± 5.8	4.10 ± 0.52
Mean of 'Pérola'	4.91 ± 0.82	5.13 ± 0.68	45.06 ± 5.80	2.88 ± 0.52
Heterosis	-2.94 ± 48.79	-2.88 ± 40.16	-25.49± 343.72	-0.39± 30.78
Additive variance	0.75	0.11	31.67	-0.10
Dominance variance	-0.57	-0.12	-17.02	0.04
Genetic variance of $F_{2:4}$ families	0.72	0.10	30.60	-0.10
Genetic variance of the F_3 (RC) families	0.68	0.09	29.54	-0.10
Heritability in the broad sense	61.03%	59.20%	67.86%	55.00%
Heritability in the narrow sense	73.79%	19.32%	56.36%	-38.10%
Number of genes	1.0	1.0	1.0	2.0
Error variances	0.837	0.567	41.536	0.333

[1/] Area under the disease progress curve

Table 3. Correlations (r) involving area under disease progress curve (AUDPC), period 1/evaluator 1, period 2/ mean of two evaluators, and mean of the three evaluations of angular leaf spot severity in the leaves and pods in the 'Carioca MG' x 'Pérola' cross. Lavras, 2000.

Correlation	r
Between AACPD and scores in the leaves in period 1/evaluator 1	0.97
Between AACPD and scores in the leaves in period 2/mean of 2 evaluators	0.99
Between AACPD and mean of the 3 evaluations in the leaves	0.94
Between AACPD and scores in the pods	0.61
Between scores in the pods and scores in the leaves in period 1/ evaluator 1	0.62
Between scores in the pods and scores in the leaves in period 2/mean 2 evaluators	0.56
Between scores in the pods and mean of the three evaluations in the leaves	0.62

genético da reação ao patógeno nas folhas e dois genes nas vagens.

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