ARTIGOS

Inheritance of soybean resistance to *Corynespora cassiicola*

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ABSTRACT

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The incidence of target spot, caused by *Corynespora cassiicola,* has gained increasing importance among the main soybean diseases in Brazil, and using susceptible cultivars can cause yield losses. Different susceptibility/resistance levels have been observed among cultivars in commercial crops but the genetics of the resistance to this pathogen is still unknown. To study the inheritance of soybean resistance to *C. cassiicola*, crosses were developed between cultivars including one cultivar resistant to target spot, BRS 316RR, one moderately resistant cultivar, BRS 184, and one susceptible cultivar BMX Potência RR. Parental generations, as well as F_2 and $F_{2:3}$ derived from their crosses, were evaluated as to severity and lesion size after inoculation with the pathogen. Quantitative analysis was applied to the data, and genetic models were adjusted for means and variances. Predominance of additive genetic effects controlling soybean resistance to *C. cassiicola* is suggested for the different crosses. The genetic models adjusted for the means detected an additive genetic effect more frequently. The additive variance D was detected only for the trait lesion size and had low heritability, indicating high environmental effect influencing the reaction. Based on mean and variance genetic models, further genetic gains are expected in the cross BRS 316RR x BMX Potência RR. The effect of genetic dominance was not important. The presence of significant epistasis in crosses between susceptible cultivars indicates the existence of at least two genes affecting resistance and that are interacting. The normal continuous distribution of the frequency of the number of individuals in different classes of resistance indicates that the resistance to *C. cassiicola* is quantitatively inherited and there is predominance of an additive genetic effect and low heritability.

Keywords: quantitative genetics, *Glycine max*, genetic parameters

RESUMO

Soares, R.M.; Arias, C.A.A. Herança de resistência da soja para *Corynespora cassiicola*. ***Summa Phytopathologica,*** v.46, n.2, p.85-91, 2020.

A incidência da mancha-alvo, causada pelo fungo *Corynespora cassiicola,* tem aumentado sua importância entre as principais doenças da soja no Brasil e o uso de cultivares suscetíveis pode ocasionar perdas de produtividade. Diferentes níveis de suscetibilidade/resistência têm sido observados entre as cultivares em cultivos comerciais, mas a genética da resistência para esse patógeno ainda é desconhecida. Para estudar a herança da resistência da soja a *C. cassiicola*, cruzamentos foram desenvolvidos entre cultivares, incluindo uma cultivar resistente a mancha-alvo, BRS 316RR, uma moderadamente resistente, BRS 184 e uma suscetível, BMX Potência RR. As gerações parentais, bem como as F_2 e $F_{2:3}$ derivadas dos seus cruzamentos foram avaliadas quanto a severidade e o tamanho de lesão após inoculação com o patógeno. A análise quantitativa foi aplicada aos dados e modelos genéticos foram ajustados para médias e variâncias. É sugerida uma predominância de efeitos genéticos aditivos controlando a resistência da soja a *C. cassiicola* entre os diferentes cruzamentos. O modelo genético ajustado para as médias, detectou efeito genético aditivo mais frequentemente. A variância aditiva D foi detectada somente para o tamanho de lesão e com baixa herdabilidade, indicando alto efeito ambiental influenciando a reação. Baseado nos modelos genéticos de médias e variâncias, ganhos genéticos adicionais são esperados no cruzamento BRS 316RR x BMX Potência RR. O efeito da dominância genética não foi importante. A presença de significante epistasia em cruzamento entre cultivares suscetíveis, indica existência de ao menos dois genes afetando a resistência e que eles estão interagindo. A distribuição normal contínua da frequência do número de indivíduos em diferentes classes de resistência, indica que a resistência a *C. cassiicola* é herdada quantitativamente, com predominância de efeito genético aditivo e baixa herdabilidade.

Palavras-chave: genética quantitativa, *Glycine max*, parâmetros genéticos.

Target spot caused by the fungus *Corynespora cassiicola* (Berk. & Curt.) Wei was first detected in Brazil in the state of São Paulo in 1976 (1). Its relative incidence has increased among the main soybean diseases in Brazil due to the lower sensitivity/resistance of the fungus to the fungicides most commonly used in soybean crops and to the use of susceptible cultivars; this disease is found in almost all regions of cultivation in Brazil. Losses from 18% to 32% were reported for susceptible cultivars (3).

The pathogen infects both the upper part and the root system of plants. Susceptible cultivars may undergo severe defoliation, showing brownish-red spots on the stem and pods. Typical target spot symptoms are roughly circular necrotic leaf lesions from little brown spots to typically large circular patches of dark brown color, which can reach up to 2 cm in diameter, showing a yellow margin and a dark point in the center surrounded by darker concentric rings (5).

The pathogen has a wide range of host plants, infecting more than

350 species in tropical and subtropical countries. The fungus survives and spreads mostly through infected seeds and crop debris on the soil surface. Favorable environmental conditions for target spot infection are temperatures above 25°C and high relative humidity (4).

For the control of target spot, resistant cultivars, seed treatment, rotation/succession of crops with grasses, and fungicide sprays are recommended (4). Teramoto et al. (12) described varied cultivar reaction to different isolates, reflecting the specificity of soybean resistance to the pathogen variability. In this study, soybean cultivars showing high resistance to *C. cassiicola* isolates were not observed, but there were cultivars with less target spot severity. Most soybean breeding programs do not select target spot resistant lines in their routine, and the least susceptible cultivars that are available have this characteristic by chance. This may be justifiable for the scarce knowledge of resistant sources and inheritance of soybean resistance to *C. cassiicola*.

Knowledge of the nature and magnitude of the gene effects that control a character is of paramount importance in the selection and prediction of the behavior of segregating generations. Thus, the genetic variability of the population and the genetic gains in the breeding program are indicated (7). Heritability is one of the most important properties of a character, as it measures the degree of correspondence between phenotypic value and genetic value (2).

Considering the relative importance of the disease and the need of further knowledge on the subject, this study was developed with the objective of studying the inheritance of soybean resistance to target spot caused by *C. cassiicola*.

MATERIAL AND METHODS

Genetic material

One cultivar resistant to target spot, BRS 316RR, one moderately resistant cultivar, BRS 184, and one susceptible cultivar, BMX Potência RR, were used in this study. These three cultivars received such classification based on preliminary assessments developed at Embrapa Soybean with the whole collection of Brazilian cultivars, Londrina, Paraná State, Brazil. These cultivars have good adaptation to the south and southeast regions of Brazil and belong to the maturity group VI. For this study, seeds from individual plants of each cultivar were used to discard the possibility of genetic variability within the parental generation, since these cultivars have never undergone intentional selection for this trait.

Experimental seed production

Three crosses were carried out with these cultivars during the 2017-2018 growing season in greenhouse environment: one of them was a resistant x moderate cross type (RM = BRS 316RR x BRS 184), another one was a resistant x susceptible cross type (RS = BRS 316RR x BMX Potência RR), and the third one was a moderate x susceptible cross type (MS = BRS 184 x BMX Potência RR). A portion of F_1 seeds was used to produce F_2 seeds in greenhouse in September 2016. The remaining F_1 seeds were stored in a cold chamber. Traits like flower and pubescence color were assessed to confirm true hybridizations. Parental and the remaining F_1 and F_2 generations were sown in a greenhouse in June 2017 to produce new seeds for parental, F_2 and $F_{2:3}$ generations, respectively. This procedure was carried out to obtain seeds of the same age for all generations. A sample of approximately 150 seeds was randomly collected from F_2 seeds to originate the $F_{2:3}$ families (F_3 family derived from a single F_2 plant).

Experimental design

The experiment was carried out in a completely randomized design under greenhouse conditions at Embrapa Soybean, Londrina, Paraná State, Brazil, to study the inheritance of resistance to *C. cassiicola*. The generations from RM, RS and MS crosses were grown and tested from October to December 2018. The assessments consisted of: 15 plants of each parental (R, M and S reaction type), 80 F2 plants derived from each of the three crosses, 150 F2:3 families from each cross, and each family was represented by three plants. Each plant was grown in individual pots corresponding to each plot – experimental unit. Pots of 4kg-soil capacity were filled with a mixture of soil, sand and manure at the proportion of 1:2:1 and treated with heated steam, at temperatures ranging from 100ºC to 150ºC.

Fungal source, maintenance and inoculation

A pathogenic isolate of *C. cassiicola*, stored in the microorganism collection of Embrapa Soybean (Collection of Microorganisms of Interest to Agriculture – CMES), named CMES 1883 and obtained from symptomatic soybean leaves collected from the municipality of Londrina, Paraná State, Brazil, was used to inoculate the plants.

The tests were carried out with potted sowing under greenhouse conditions. The plants were inoculated 20 days after sowing, in stage V2 (first trifolium), with a conidial suspension produced in the laboratory and sprayed at a concentration of 1.0×10^4 conidia/mL. Inoculum production was obtained in Petri dishes with modified V8 culture medium containing tomato extract (280 g) + agar (17 g) + calcium carbonate (4.5 g) + distilled water to complete one liter. The isolate was allowed to grow in the dishes for 12 days, until the whole surface of the medium was cover by the fungus. Inoculated plants were kept under humidity close to saturation for 48 hours, with automatic misting at 25°C -28°C.

Scoring method

The percentage of target spot severity was evaluated for the most infected trifoliate leaves from plants of each treatment at 28 days after inoculation, using a diagrammatic scale (8). Lesion size was also measured and expressed as millimeters for the largest lesion found on the most infected trifoliate leaf.

Inheritance analysis

Quantitative analysis (6) of the four generations (P_1, P_2, F_2 and $F_{2:3}$) for the means and variances allowed the estimation of up to four and five components of a genetic model, respectively. When fewer components were significant, a goodness-of-fit test of the model was performed. Estimation of mean components included the genetic component **m**, the additive effect [d], the dominance component [h], and the non-allelic interactions (additive by additive [i] or dominant by dominant [l]). Estimation of variance components included the additive (D) and dominance (H) genetic variance, and the environmental variance (E, E1 and E2). Narrow sense heritability was also estimated at plant level based on variance estimates.

The used model considered only the mean of the homozygous and the deviations of homozygous and heterozygous genotypes from the mean, and additive x additive epistasis. To verify if the model fit to the experimental data, the joint scale test was adopted (13). Based on these estimates, the expected values for the mean of the generations were obtained. Subsequently, the fitness of the proposed model was determined by comparing the observed and the expected values according to Chi-square ($\chi 2$) test (10), as the following expression:

$$\chi^2 = \Sigma\left[\left(obs-esp\right)^2 / esp\right],$$

with $n-1$ degrees of freedom, and n as the number of phenotypic classes (generations) of the model. If the result of the test is not significant, the proposed model explains the mean and variance phenotypic value of each generation.

RESULTS AND DISCUSSION

Phenotypic analysis – parental cultivars

Twenty days after inoculation, the severity level was enough to assess the materials, showing that the methodology used to develop the inoculum and the inoculation process were suitable for the objectives of this study. Means and variances derived from parental, F_2 and $F_{2:3}$ generations are summarized in Table 1. The cultivar BRS 316RR showed lower average for the traits severity and lesion size. Severity mean was 10.27% and individual scores varied from 0% to 30%, while lesion size averaged 1.47 mm, varying from 0 mm to 3 mm. BRS 184 showed higher mean for both traits (Table 1) with 21.73% severity (individual scores varying from 5% to 50%) and 2.53 mm lesion size (varying from 2.0 mm to 3.0 mm). BMX Potência RR also showed relatively higher mean for both traits: severity of 20.07% (individual scores varying from 4% to 40%) and lesion size of 2.20 mm (varying from 1.0 mm to 3.0mm).

Two parental cultivars, the resistant and the susceptible one, confirmed the classification observed in the preliminary experiments. The resistant cultivar BRS 316RR showed the expected reaction to *C. cassiicola* with mean for severity and lesion size significantly smaller ($p<0.05$) than that of the susceptible cultivar BMX Potência RR (Table 1). Therefore, the cultivar BRS 184, previously classified as moderately resistant, had mean for severity and lesion size significantly higher, compared to BRS 316RR, and did not differ ($p<0.05$) from the susceptible cultivar (Table 1). One hypothesis for the unexpected reaction of BRS 184 can be the high variability shown by different isolates of *C. cassiicola*, as evidenced in tests carried out to evaluate the reaction of soybean commercial cultivars against six isolates, where the reaction of the cultivars varied as the isolate changed (12). There is virulence variability among *C. cassiicola* isolates obtained from soybean plants, which may affect the effectiveness of control measures such as spraying with fungicides and the use of resistant varieties (9). Sexual reproduction fungi, as is the case for *C. cassiicola*, do not undergo regular recombination, and genetic variation results especially from the accumulation of spontaneous mutations (11). The diverse environments of soybean cultivation in Brazil, the predominance of cultivars susceptible to target spot, and the high exposure of the pathogen to fungicides may have favored the occurrence of mutations.

The variance values obtained for both traits considering the three parental cultivars did not differ ($p<0.05$) according to F-test (Table 1). As the three cultivars are genetically uniform to the trait, all these variances can be considered estimators of environmental variance. Both resistant and susceptible cultivars seem to suffer similar micro-environmental effects. But the variation coefficient (CV%) for lesion size was less than half the values for severity.

Phenotypic analysis – F_2 and $F_{2:3}$ generations

The mean severity for F_2 and $F_{2:3}$ generations derived from the cross between the cultivars BRS 316RR and BRS 184 and was 11.36 and 12.53, respectively (Table 1). These values are lower than the

Table 1. Degrees of freedom (DF), means ($\overline{X}$) and variances (S^2) of severity and lesion size of parental, F_2 and $F_{2:3}$ generations after inoculation with *Corynespora cassiicola*.

Generation	Severity				Lesion Size			
	DF	$\overline{X}$	S^2	CV (%)	DF	$\overline{X}$	S^2	CV (%)
BRS 316RR (R)[1]	14	10.27 a [4]	112.07 A [5]	103,1	14	1.47 a	0.55 A	50,5
BRS 184 (M)[2]	14	21.73 b	141.50 A	54,7	14	2.53 b	0.27 A	20,5
BMX Potência RR (S)[3]	14	20.07 b	128.07 A	56,4	14	2.20 b	0.31 A	25,3
BRS 316RR x BRS 184								
F_2	76	11.36	109.94	92,3	76	1.86	0.44	35,7
$F_{2:3}$	446	12.53	101.16	80,3	446	1.85	0.51	38,6
BRS 316RR x BMX Potência RR								
F_2	77	14.76	117.80	73,5	77	1.96	0,30	27,9
$F_{2:3}$	440	14.70	105.58	69,9	440	1.90	0,39	32,9
BRS 184 x BMX Potência RR								
F_2	79	18.24	131.53	62,9	79	2.04	0.59	37,7
$F_{2:3}$	443	16.45	118.79	66,3	443	1.99	0.43	32,9

[1, 2, 3] Resistant (R), moderate (M) and susceptible (S) soybean varieties.
[4]Parental means followed by the same letter for each cross do not differ according to t-test ($p<0.05$). [5]Parental variances followed by the same letter for each cross do not differ according to F-test ($p<0.05$).

Table 2.Genetic models adjusted to the means[1] and variances[2] for the traits severity and lesion size, evaluated for the crosses BRS 316RR x BRS 184 (RM), BRS 316RR x BMX Potência RR (RS) and BRS 184 x BMX Potência RR (MS) after inoculation with *Corynespora cassiicola*.

Genetic parameters	RM	RS	MS	RM	RS	MS
	Severity			Lesion Size		
m \1	12.53±0.43	14.73±0.44	16.90±0.47	1.86±0.03	1.91±0,03	1.99±0.03
[d]	5.332.04±	4.871.99±		0.58±0.11	0.34±0.11	
[h]						
[i]						0.38±0.10
[l]						
χ^2 / df / P \3	3.81 / 2 / 0.149	0.05 / 2 / 0.97	5.50 / 3 / 0.14	1.52 /2 / 0.47	1.15 /2 / 0.56	3.11 / 2 / 0.21
D \2					0.12±0.04	0.12±0,05
E	103.69±6.25	108.00±6.54	121.40±7.32	0.49±0.03	0.30±0.03	0.36±0.03
			-		-	-
χ^2 / df / P	1.49 / 4 / 0.83	1.32 / 4 / 0.86	1.01 / 4 / 0,90	2.16 / 4 / 0.71	6.20 / 3 / 0.10	7.65 / 3 / 0.05
h^2 \4	-	-	-	-	0.17	0.14

[1]Mean parameters include the mean of genetic and environmental effects for the cross (m), the additive genetic effect [d], dominance [h], and epistasis [i] and [l]; [2] ffect of additive genetic variance (D) and additive environmental variance (E); [3]Chi-square (χ^2) value for the model fit, degree of freedom (df), probability (P) associated with Chi-square; [4] Estimate of narrow sense heritability (h^2).

mean value obtained for the two parental cultivars in the same crossing (parental average = 16.0), evidencing possible presence of directional dominance to resistance or some type of non-allelic interaction, which can be confirmed in the genetic models. The same pattern was observed for the trait lesion size, which had means of 1.86 and 1.85 for F_2 and $F_{2:3}$ generations, respectively, lower than the average of parental cultivars of 2.0 (Table 1). Variance related to F_2 generation for the trait severity was lower than the mean variance of the two parental cultivars, indicating absence of significant genetic variation. For the trait lesion size, F_2 variance was slightly higher than the mean parental variance (mean of 0.38) and the presence of genetic effects need to be verified among the variance genetic models.

For the cross between the resistant cultivar BRS 316RR and the susceptible cultivar BMX Potência RR, the mean severity for F_2 and $F_{2:3}$ generations was 14.76 and 14.70, respectively (Table 1). These values are positioned next to the mean value for the two parental cultivars in the same assessment (parental average = 15.17), evidencing absence of directional dominance. For the trait lesion size, the means of 1.96 and 1.90 for F_2 and $F_{2:3}$ generations, respectively, are above the average for parental cultivars of 1.84 (Table 1).

The third cross between BRS 184 and BMX Potência RR had mean severity for F_2 and $F_{2:3}$ generations of 18.24 and 16.45, respectively (Table 1). These values are lower than the mean for the two parental cultivars that participated in the cross (parental average = 20.9), evidencing possible presence of directional dominance to resistance or some type of non-allelic interaction. For the trait lesion size, the means of 2.04 and 1.99 for F_2 and $F_{2:3}$ generations, respectively, are also below the average for parental cultivars of 2.37 (Table 1).

Mean and variance genetic models

The genetic models could be fit to the means and variances for both evaluated traits in the three crosses (Table 2). Besides fitting the models only to significant estimates, degrees of freedom were always used to verify the adequacy of the model according to Chi-square tests, which produced probabilities ranging from 5% to 97%; thus, no mean or variance genetic model was rejected ($p<0.05$). Priority was always given to simpler genetic models. For example, if an additive-dominant model was not rejected, other more complex models (e.g. including non-allelic effects) were avoided.

Cross BRS 316RR x BRS 184 (RM cross type)

Considering this cross including the resistant cultivar BRS 316RR and the moderately resistant cultivar BRS 184, the mean genetic model only with the mean parameter (**m**) and the additive genetic effect [d] was not rejected by the Chi-square test for the traits severity and lesion size (Table 2). The additive genetic effect was significant only in the mean models [d], while in the variance models the additive genetic variance (D) was not significant. The magnitudes of [d] estimates reached 42% and 31% for the mean **m**, considering the traits severity and lesion size, respectively. These magnitudes of genetic additive effect [d] can be considered higher than expected for a RM type cross but, as discussed above, the reaction of the moderately resistant BRS 184 to target spot was similar to that of the susceptible cultivar in this study, explaining the results. Still considering the mean models, non-additive genetic effects like dominance [h] or the non-allelic interactions [i] and [l] were not significant. The simple genetic model only with **m** and [d] can indicate some convenience for selective actions but the non-significance of the additive variance D in the variance model does not permit the estimation of heritability. Considering that [d] exists in the mean model, the absence of D in the variance model may have been caused by low experimental resolution. Increasing the number of assessed individuals per $F_{2:3}$ family could help obtain a significant estimate for D.

The conclusion is that there is genetic variability in this cross, but the heritability for the assessed characters is probably small, requiring strategies to reduce the environmental effect, such as using more repetitions or families with expressive number of plants, consequently avoiding the assessment of individual plants.

Observing the frequency distribution for the severity means of

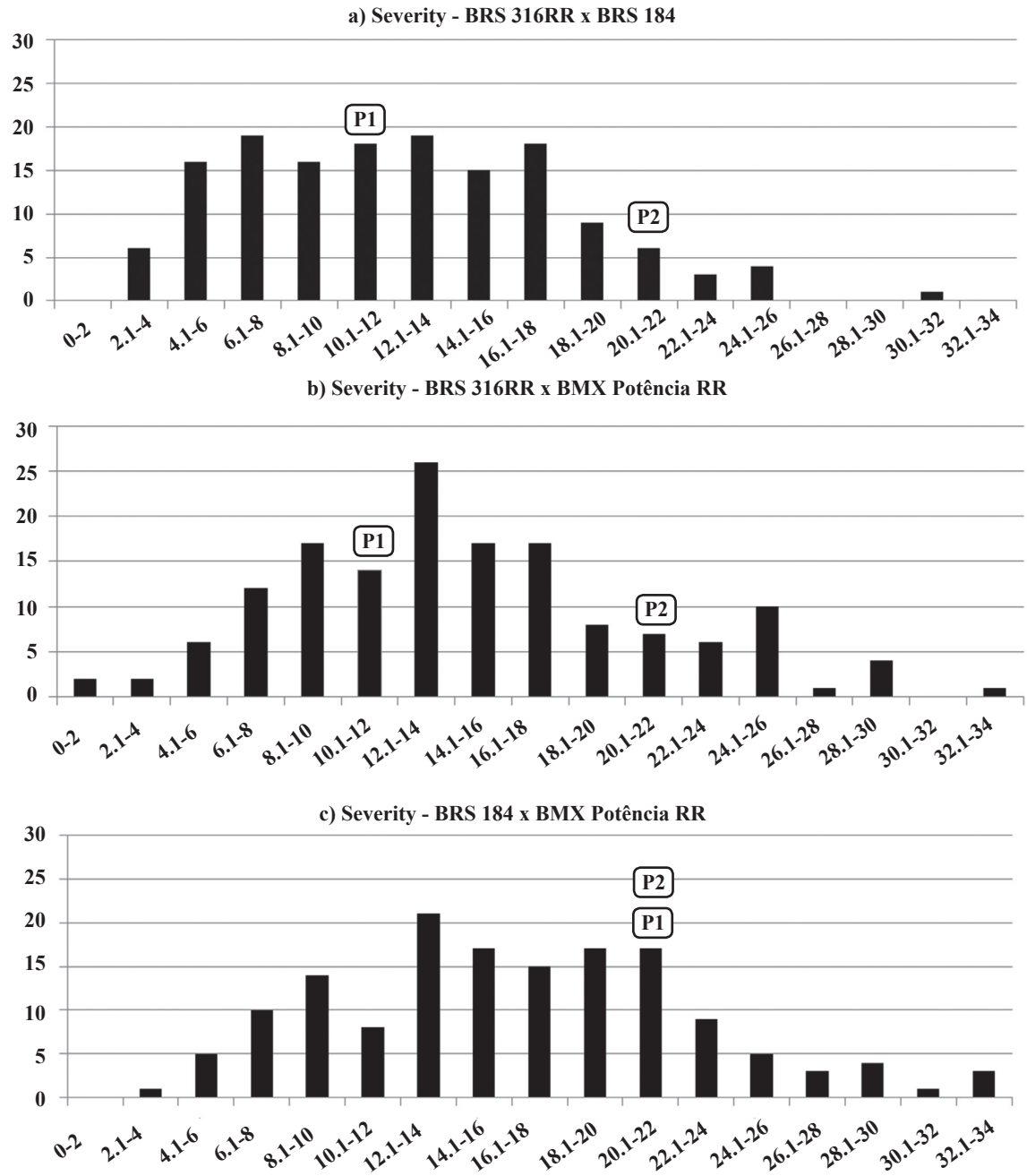

Figure 1. Distribution of frequency of $F_{2:3}$ families derived from the crosses BRS 316RR x BRS 184, BRS 316RR x BMX Potência RR and BRS 184 x BMX Potência RR for the trait severity of target spot (*Corynespora cassiicola*) and the relative average position of parents.

$F_{2:3}$ families (Figure 1a), there are 57 families (38% total families) positioned in the phenotypic classes below the resistant cultivar BRS 316RR. For the trait lesion type (Figure 2a), only eight families (5.8%) are classified into the first phenotypic group, below the resistant parent. The continuous pattern of the frequency distribution for both characters indicates that no major genes are involved in the genetic control, and the quantitative method is more suitable to study the resistance to *C. cassiicola*.

Cross BRS 316RR x BMX Potência RR (RS cross type)

This cross between a resistant and a susceptible cultivar (RS cross type) also presented predominance of additive genetic effect considering both mean and variance genetic models (Table 2). Additive genetic effect was significant in the mean models [d] for both assessed traits and in the variance models (D) only for lesion size. Higher values for the estimates of [d] were expected for this cross, because of higher genetic divergence between the parental cultivars. The magnitudes of [d] estimates reached 33% and 17.8% for the mean **m** for the traits severity and lesion size, respectively. These two proportions are smaller than that observed in the previous cross, which could mean a smaller comparative genetic divergence between parental cultivars. However, the significance of additive genetic variance D in the variance model for lesion size confirm the expected genetic divergence in this RS cross type. The smaller coefficient of variation and smaller estimate

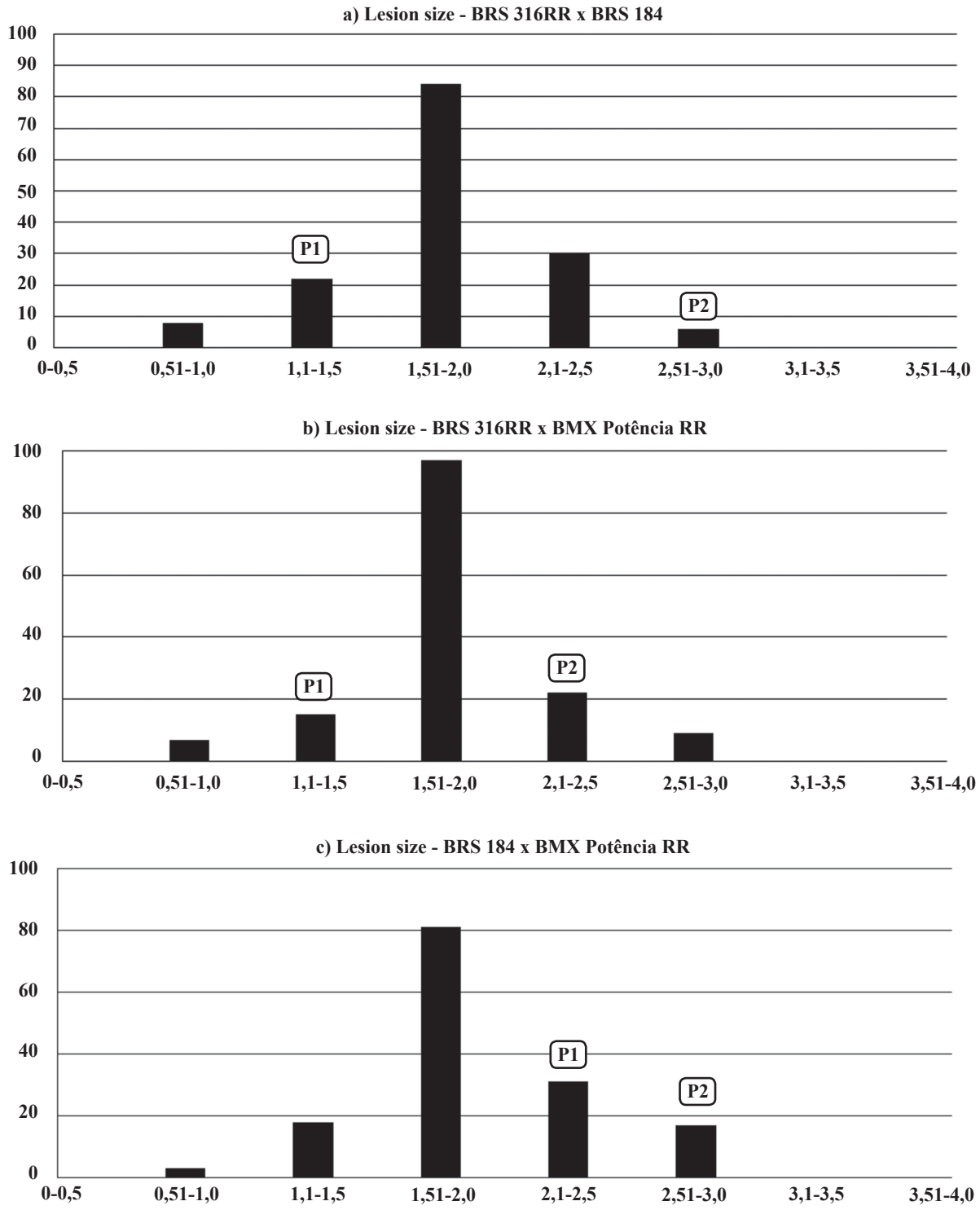

Figure 2. Distribution of frequency of $F_{2:3}$ families derived from the crosses BRS 316RR x BRS 184, BRS 316RR x BMX Potência RR and BRS 184 x BMX Potência RR for the trait lesion size of target spot (*Corynespora cassiicola*) and the relative average position of the parents.

of environmental variance (E) observed for lesion size probably facilitates the detection of D in comparison to severity, which had a greater associated experimental error. Non-additive genetic effects like dominance [h] or non-allelic interactions [i] and [l] were not significant for this cross, which can facilitate selective processes in breeding programs. The significant estimates of D and E in the variance model for lesion size allow estimating the narrow sense heritability at individual plant level, obtaining the value of 0.173% or 17.3%. This low value for heritability reinforces the difficulty that breeding programs will have to develop cultivars that are more resistant to the disease. The significant participation of environmental factors in the determination of the genotype reaction is a problem that can be mitigated with good experimental designs.

There are 39 families (26% total families) positioned into

the phenotypic classes below the resistant cultivar BRS 316RR in the frequency distribution for severity (Figure 1b). For the trait lesion size (Figure 2b), only seven families (4.7%) are classified into the first phenotypic group, below the resistant parental cultivar. For this cross, a continuous pattern for the frequency distribution was also observed for both traits, showing that only minor genes are involved in the genetic control of the resistance to *C. cassiicola*.

Cross BRS 184 x BMX Potência RR (MS cross type)

For the trait severity, only the mean **m** was observed in the mean model and the environmental variance (E) among the significant parameters; thus, no genetic effect was detected for this trait (Table 2). This result could be expected since the two cultivars used as parental cultivars for this cross performed like susceptible genotypes. However, considering the character lesion size, epistasis of the type additive by additive [i] was significant, showing that non-allelic genes are interacting to define the final reaction to *C. cassiicola* in this cross. These genes would be dispersed between the two parental cultivars, since the additive genetic effect [d] was non-significant. To reinforce the existence of the additive genetic effect dispersed between these two parental cultivars, the additive genetic variance (D) was significant in the variance model for this character. The estimated heritability was also of small magnitude (14%) for the trait lesion size, indicating that there is genetic variability to be explored in this cross but the environmental effects predominate for the character definition.

There are 16 families (11% total families) positioned into the phenotypic classes below the resistant cultivar BRS 316RR in the frequency distribution for severity (Figure 1c). For the trait lesion size (Figure 2c), only three families (2.0%) are classified into the first phenotypic group, below the resistant cultivar BRS 316RR. For this cross, a continuous pattern for the frequency distribution was also observed for both traits, showing that only minor genes are involved in the genetic control of resistance to *C. cassiicola*.

General analysis considering the three crosses

Considering both mean and variance models (Table 2), there was predominance of additive genetic effects [d] or D in all crosses, except the MS cross for the trait severity, which did not present any genetic parameter in both models. That was observed even in the cross between moderate and susceptible parents (MS cross type), indicating the presence of minor genes dispersed between these cultivars. Although the additive genetic effects were significant, the magnitude of these effects did not appear to be very large, explaining the presence of [d] combined with the absence of D in two crosses for severity and in one cross for lesion size, which requires a great deal of research effort to evaluate and select the most resistant individuals.

On the other hand, other complicating effects for selection of resistant genotypes such as effects of dominance or genotype by microenvironment interaction are absent. Other non-additive effects like additive-by-additive epistasis [i] can less frequently participate in genetic control. The occurrence of interaction between genes or non-allelic epistasis is indicative of at least two genes controlling the character, which interact somehow.

Estimation of narrow sense heritability was possible only in two crosses for the trait lesion size, both showing small values, which confirms that lower genetic gains would be expected along the selection processes. To explore molecular markers linked to resistance QTLs, the cross BRS 316RR x BMX Potência RR would be most advantageous since it has both [d] and D among the significant parameters and minor E values among variance models for the trait lesion size, which had the smallest experimental error associated.

The number of individuals into the several phenotypic classes and the proportions of individuals showing mean values inferior, equal or superior to that of the resistant parents to both severity and lesion size evidences that the inheritance of resistance to *C. cassiicola* is of quantitative type (Figures 1 and 2).

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