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REVIEW

Genetic control of Asian rust in soybean

Aliny Simony Ribeiro · José Ubirajara Vieira Moreira · Pedro Henrique Braga Pierozzi · Breno Francovig Rachid · José Francisco Ferraz de Toledo · Carlos Alberto Arrabal Arias · Rafael Moreira Soares · Claudia Vieira Godoy

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Abstract The objective of this study was to investigate the genetic mechanisms of soybean resistance to Asian rust (*Phakopsora pachyrhizi* Syd. & P. Syd). F2 and F3 generations from 15 diallel crosses involving six soybean cultivars, FT-2, EMBRAPA 48, BRS 154, BRS 184, BRS 214, BRS 231, were used to analyze the genetic control of Asian rust resistance in the soybean parents tested. Genetic models were fitted to means and variances of the generations tested in a completely randomized field experiment with 5,700 hill plots. The experiment was spray-inoculated twice with an isolate that was first detected during the 2002/03 season in Mato Grosso (MT) State and presently prevail in Central Brazil, at a six-day interval, on borders rows and on the useful area, respectively, with a 10^4 spores/ml distilled H_2O suspension. Assessments were made using a diagrammatic scale for disease severity at seven and 39 days after the first detection of Asian rust in the experiments. Evaluations made in the second assessment (39 days) discriminated better between genotypes. Selection at early plant developmental stages may not result in adult resistant plants. Cultivar FT-2, which had presented monogenic resistance to a rust isolated that prevailed in the first two years of rust occurrence in Brazil, showed no resistance to the MT State rust strain used in this experiment, but eleven crosses showed genetic variability for resistance in the second assessment. Soybean rust resistant genes showing predominantly additive effects are dispersed among parents. Narrow sense heritability values ranging from 0.42 to 0.74 at the F3 family level in the second assessment suggested that selection of resistant genotypes is feasible.

Keywords Asian rust · Disease resistance · Genetic analysis · Genetic inheritance · *Phakopsora pachyrhizi*

A. S. Ribeiro
Maringa State University, Maringá, PR, Brazil

J. U. V. Moreira · B. F. Rachid
J. Francisco F. de Toledo (✉) ·
C. A. A. Arias · R. M. Soares · C. V. Godoy
Genetics and Breeding, Embrapa Soybean, Carlos Joao Strass Road, Orlando Amaral Access,
P.O. Box 231, 86001-970 Londrina, PR, Brazil
e-mail: toledo@cnpso.embrapa.br

P. H. B. Pierozzi
Londrina State University, Londrina, PR, Brazil

Introduction

Asian rust (*Phakopsora pachyrhizi* Syd & P. Syd) is a soybean disease initially described in China that has spread rapidly in several Brazilian cropping States (Yorinori et al. 2004).

The ideal conditions that trigger the process of infection are 10 h of moisture on the leaf surface (rain, dew or irrigation) and day temperatures ranging from 15 to 28°C. Symptoms start with smaller than 1 mm diameter greenish to gray colored spots where the reproductive structures are formed (uredia and uredospores). As the disease progresses, the leaf tissues around the spots become pale brown (''TAN'' type lesion) or reddish brown (''RB'' type lesion) in susceptible and resistant genotypes, respectively. The damage caused by leaf necrosis and early leaf fall can significantly reduce yield levels. It is estimated that in the 2001/2002 to 2003/2004 growing seasons, the total grain loss in Brazil caused by Asian rust was greater than 8.5 million tons, equivalent to US$ 2.06 billion (Yorinori et al. 2004; Henning et al. 2005).

Brazilian breeding programs have not yet developed resistant cultivars and, therefore, chemical control is used to reduce losses caused by Asian rust. However, the use of fungicides has also resulted in lower economic returns due to an increase in total production costs of approximately 3.5% for each application in 2004–2005.

Soybean breeding for resistance to Asian rust has been concentrated on qualitative genes named *Rpp1, Rpp2, Rpp3* and *Rpp4* (Hartwig 1995; Hartman et al. 2004) since 2001. Other major genes conferring (monogenic) resistance have been identified and introduced in the breeding programs since 2004. In spite of the well-known stability limitations of single gene resistance for controlling plant diseases, several important soybean diseases were controlled by introducing single genetic resistance factors into commercial cultivars. Monogenic resistance to frog-eye leaf spot, stem canker, bacterial pustule and oidium, to name a few, have proved to be efficient and durable. However, resistance expressed by soybean lines carrying *Rpp1* and *Rpp3* has already been broken in MT State, which suggested high genetic variability of the Asian rust fungus and left no great expectations of longevity for the other resistance single genes.

Therefore, studies of quantitative genes expressing resistance to Asian rust in soybean genotypes are welcome to support the development of effective longer lasting resistance in new cultivars. This study reports on quantitative genetic control of rust resistance in a sample of Brazilian soybean cultivars.

Material and methods

Six parents (FT-2, EMBRAPA 48, BRS 154, BRS 184, BRS 214 and BRS 231) were crossed in diallel to obtain 30 sets of F2 and 30 sets of F3 generations including reciprocals. The parents were chosen for the following characteristics: (a) FT-2 carries a single gene, probably *Rpp1* or *Rpp3*, for resistance to the Asian rust (*P. pachyrhizi*) strain prevalent in Brazil in 2001, but is not resistant to the MT State strain, which was detected in the 2002/03 season. It is a 6.4 maturity group cultivar; (b) Embrapa 48 is susceptible to rust and was chosen because it was the second most extensively cropped cultivar in Paraná State in the 2004/2005 growing season. It is a 6.8 maturity group cultivar; (c) BRS 154 is the check cultivar for susceptibility to Asian rust in most of the Brazilian experiments and is 7.4 maturity group cultivar; (d) BRS 184 and BRS 214 are both moderately susceptible to Asian rust, but BRS 214 carries minor genes for resistance. They are, respectively, 6.7 and 7.4 maturity group cultivars; (e) BRS 231 carries quantitative genes for Asian rust resistance. It is a 7.6 maturity group cultivar. Cultivars of similar maturity groups were purposefully chosen to minimize growth effects on the Asian rust infection cycle.

The experiment was carried out at the Embrapa Soybean Research Center (at 23°11′ S and 51°10′ W) in Londrina, PR, in a completely randomized design with 5,700 single plant hill plots. Each parent was represented by 50 individuals, each F2 and RF2 by 80 individuals and each F3 and RF3 by 20 families of five individuals.

The single plant hill plots were spaced at 0.20 m within row and 1.5 m between rows. Two border rows of remnant seeds were sown between the useful experimental rows and around the experiments. The final row spacing was 0.50 m resulting in an average population of 250,000 plants/ha. Irrigation was used twice a week to guarantee favorable Asian rust development conditions.

Starting at the V3 stage (Fehr 1981) the experiment was spray-inoculated twice, at a six-day interval, on the borders and on the experimental rows, respectively, with a 10^4 spores/ml distilled H_2O suspension of the MT State isolate. The inoculum was prepared with spores obtained from leaves of the BRS Bacuri cultivar grown in a green house previously inoculated with the fungus isolate. 'BRS Bacuri' is derived from 'FT-2 and carries the same gene (*Rpp1* or *Rpp3*) for rust resistance, therefore functioning as filter for the isolate.

Initial assessments of the experiment were performed to detect the presence of Asian rust infection, which was confirmed on Dec 20, 2004. Seven and 39 days after detection, disease severity was scored on the first trifoliate leaf following the cotyledonal leaves and on the third leaf from the top of each plant, respectively, using a diagrammatic scale of disease severity scores (Canteri and Godoy 2003). The assessments were not performed on the same leaf because of extensive first leaf fall due to the rust infection.

Means and variances of severity scores of the parents and their derived F2, RF2, F3 and RF3 generations were obtained. Means and variances of the reciprocal generations were pooled after checking for non-significance of reciprocal effects.

Analyses of generation means and variances provided complementary information on the genetic and environmental expression of Asian rust in soybeans. Genetic models were fitted to these data to obtain estimates of the additive, dominance, epistatic, and environmental components (Mather and Jinks 1982). Goodness of fit tests for each adjusted model were performed. Predictions of the cross potential to generate inbred lines with greater resistance to Asian rust were carried out whenever possible (Triller et al. 1996).

Results and discussion

The means and variances of severity scores on leaves of the F2 and RF2 and the F3 and RF3 generations indicated absence of reciprocal differences for Asian rust (*P. pachyrhizi*) infection in all the soybean crosses. Consequently, the respective scores of the F2 and RF2 and the F3 and RF3 in the complete diallel were pooled to obtain a total of 15 F2 and 15 F3 parental combinations.

The frequency distributions of the F2 and F3 severity scores of each cross in the first and second assessments did not show well-defined phenotypic classes, which indicated absence of major genes controlling Asian rust resistance in these germplasms. Indeed, the frequency distributions of the severity scores suggested the presence of quantitative inheritance controlling resistance in most of the analyzed crosses, since transgressive F2 individuals and F3 family means towards resistance and susceptibility were observed in all crosses and in 11 out of 15 crosses, respectively. Figure 1 shows the distributions of rust infection scores of F3 families from eight crosses obtained in the second assessment.

Table 1 shows the means and variances of parents, F2 and F3 generations in the first and second assessments. Disease severity increased from the first to the second assessment and the average parental scores were 4.52 and 22.19%, respectively. This result was expected since the 22-day period between evaluations allowed greater disease development. The Tukey test ($P \leq 5\%$) discriminated two parental score groups in the first assessment: BRS 184 (3.5%) and BRS 214 (5.4%) were the highest and lowest resistance parents, respectively. The Tukey ($P \leq 5\%$) test discriminated three groups in the second assessment. The most resistant and susceptible parents were BRS 231 and FT-2, which showed severity scores of 14.58 and 32.24%, respectively. These results confirmed previous observations that the MT State Asian rust isolate had broken the resistance of the cultivar FT-2. It also broke resistance of several plant introductions (PIs) that are carriers of the *Rpp1* and *Rpp3* resistance genes described by Hartwig (1995) and Hartman et al. (2004). It is worth noting that BRS 231, which showed a considerably lower severity score than the other parents in later developmental stages, did not express a comparatively significant resistance at earlier stages. On the other hand, Embrapa 48, which showed the second lowest score in the first assessment, presented a high severity score in the second assessment.

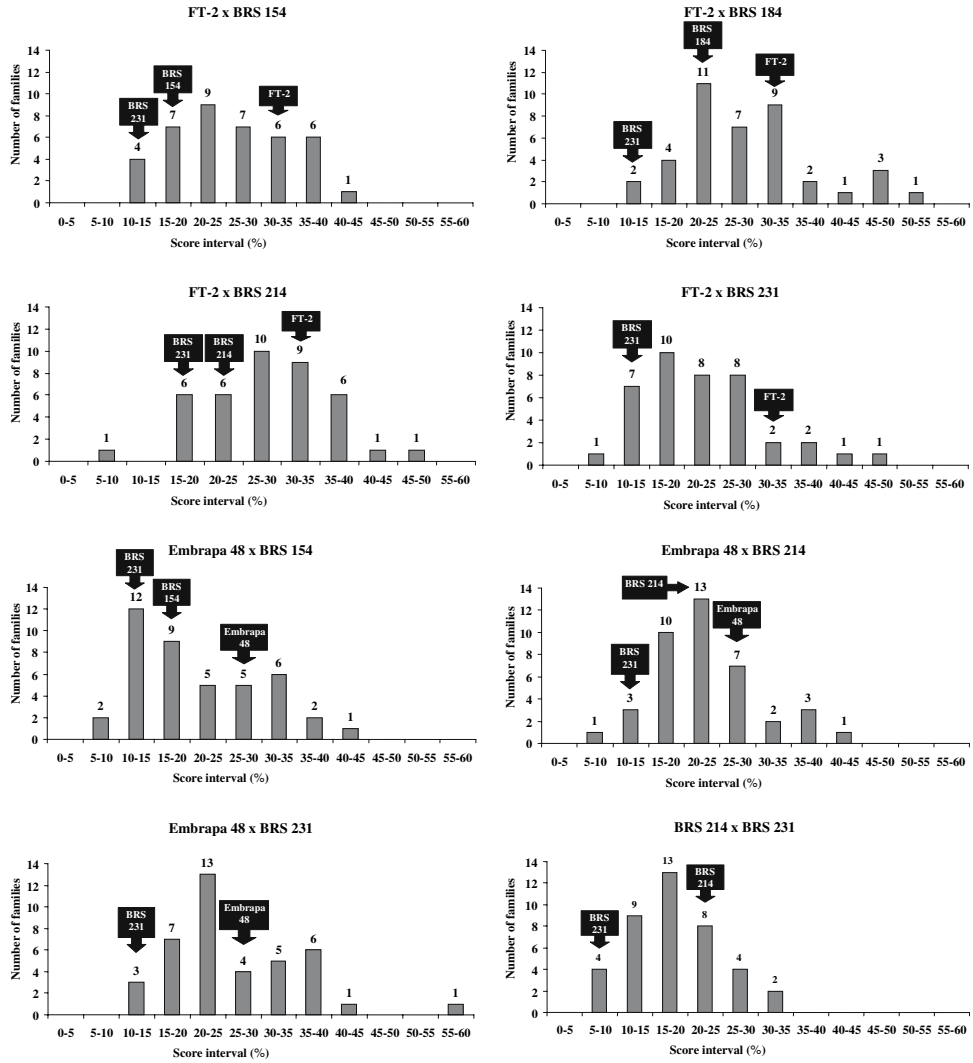

Fig. 1 Frequency distribution of rust infection scores of F3 families derived from eight crosses in the second assessment. Arrows indicate respective cross parents and BRS 231 scores

According to Brogin (2001), the capacity of soybean genotypes to express resistance to Asian rust varies over time. This has important consequences for the breeder, as early assessments may prove misleading for breeding purposes. The mean data from the first assessment also showed prevalence of low severity scores in most individuals, which made it difficult to screen among susceptible or resistant genotypes. Therefore, analyses were focused on the second assessment. However, breeders may consider combining early stage resistance with later stage resistance by carrying out assessments at different developmental stages.

Analysis of severity scores of the 40 F3 families of each of the 15 crosses in the second assessment indicated that all the cross combinations produced at least one F3 family displaying rust infection score smaller than BRS 231 (14.56%). In all, 111 F3 families, equivalent to 18.5% of all the F3 families tested, expressed greater resistance to rust than BRS 231. Six crosses produced 82 of these 111 F3 families: BRS 184 × BRS 231 (17 families), BRS 154 × BRS 184 (16 families), BRS 154 × BRS 214 (13 families), BRS 154 × Embrapa 48 (13 families), BRS 214 × BRS 231 (12 families) and BRS 154 × BRS 231 (11 families). The tested cultivars

Table 1 Degrees of freedom (df), means and variances of parents and their respective cross-derived F2 and F3 generations of severity scores of Asian rust in soybeans

	1st assessment				2nd assessment			
	df	Means	Tukey 5%	Variances	df	Means	Tukey 5%	Variances
Parents								
FT-2	49	4.74	ab	7.75	49	32.24	a	365.41
Emb 48	48	4.05	ab	3.75	48	26.94	ab	316.68
BRS 154	49	4.74	ab	6.76	49	18.02	bc	218.26
BRS 184	49	3.50	b	1.89	49	20.62	bc	356.61
BRS 214	49	5.40	a	13.43	49	20.74	bc	219.01
BRS 231	48	4.67	ab	7.06	47	14.58	c	140.63
Crosses								
FT-2 × Emb 48								
F2	158	4.74		7.36	158	30.52		322.38
F3	198	4.27		5.45	198	32.29		374.72
F3 between (families)	39			4.82	39			480.03
F3 within (families)	159			5.61	159			348.19
FT-2 × BRS 154								
F2	159	5.08		11.29	159	24.92		287.93
F3	198	4.66		8.69	197	25.92		338.42
F3 between (families)	39			8.70	39			304.80
F3 within (families)	159			8.69	158			346.95
FT-2 × BRS 184								
F2	159	4.38		5.28	158	26.86		265.48
F3	199	4.57		7.26	198	28.34		322.89
F3 between (families)	39			7.55	39			468.49
F3 within (families)	160			7.19	159			286.21
FT-2 × BRS 214								
F2	158	5.21		11.18	158	30.65		332.93
F3	197	5.01		18.28	197	27.64		287.53
F3 between (families)	39			19.12	39			322.21
F3 within (families)	158			18.07	158			278.73
FT-2 × BRS 231								
F2	158	5.54		14.50	158	18.69		195.19
F3	199	4.62		7.95	199	22.15		298.21
F3 between (families)	39			6.05	39			363.23
F3 within (families)	160			8.43	160			281.95
Emb 48 × BRS 154								
F2	159	4.42		6.23	159	20.03		216.23
F3	198	4.40		6.72	198	20.43		232.00
F3 between (families)	39			5.26	39			331.96
F3 within (families)	159			7.09	159			206.82
Emb 48 × BRS 184								
F2	159	3.76		4.14	159	25.33		263.75
F3	198	3.82		4.70	197	23.76		257.78
F3 between (families)	39			4.53	39			321.27
F3 within (families)	159			4.75	158			241.67
Emb 48 × BRS 214								
F2	157	4.35		6.50	156	27.25		301.38
F3	198	4.16		6.49	198	23.26		257.11
F3 between (families)	39			4.82	39			281.59
F3 within (families)	159			6.91	159			250.94
Emb 48 × BRS 231								
F2	159	4.65		8.80	159	16.24		186.83
F3	197	4.21		7.74	197	25.90		315.38
F3 between (families)	39			8.23	39			409.07
F3 within (families)	158			7.61	158			291.60

Table 1 continued

	1st assessment				2nd assessment			
	df	Means	Tukey 5%	Variances	df	Means	Tukey 5%	Variances
BRS 154 × BRS 184								
F2	158	4.71		10.18	157	17.96		236.60
F3	198	4.56		7.50	198	18.14		238.00
F3 between (families)	39			7.79	39			266.72
F3 within (families)	159			7.43	159			230.76
BRS 154 × BRS 214								
F2	159	5.56		13.99	159	20.55		271.14
F3	199	5.00		17.38	199	20.20		290.58
F3 between (families)	39			14.05	39			406.58
F3 within (families)	160			18.21	160			261.58
BRS 154 × BRS 231								
F2	159	5.62		13.90	159	15.47		161.40
F3	198	5.22		8.86	197	18.62		212.59
F3 between (families)	39			10.98	39			224.67
F3 within (families)	159			8.33	158			209.52
BRS 184 × BRS 214								
F2	159	4.59		5.30	159	24.48		336.15
F3	199	4.18		7.96	199	23.39		322.24
F3 between (families)	39			5.75	39			366.25
F3 within (families)	160			8.51	160			311.24
BRS 184 × BRS 231								
F2	159	4.61		6.62	159	17.10		222.15
F3	199	4.25		7.04	198	16.65		216.76
F3 between (families)	39			6.23	39			190.38
F3 within (families)	160			7.24	159			223.40
BRS 214 × BRS 231								
F2	158	4.86		8.14	158	16.63		169.07
F3	197	5.67		11.92	197	18.10		222.75
F3 between (families)	39			15.53	39			203.13
F3 within (families)	158			11.00	158			227.73

Data from 15 crosses scored 7 days (1st assessment) and 39 days (2nd assessment) after two sequential (6-day interval) inoculations

appeared as parents of the 111 resistant F3 families at the following frequencies: BRS 154 (49.5%), BRS 231 (45.0%), BRS 184 (39.6%), BRS 214 (31.5%), Embrapa 48 (22.5%) and FT-2 (11.7%). Listing cultivar BRS 154 as most frequent parent of the resistant F3 families was not expected, since it is presently used as susceptibility standard in experiments with Asian rust. These results were a strong indication that quantitative genes with small effects for resistance to Asian rust are dispersed in the parents of this study. Further, these genes are probably located in different loci since they proved capable of expressing significant levels of resistance when combined in different progenies.

Tables 2 and 3 show the genetic parameters fitted to the means and variances of the parents, F2 and F3 generations in the two assessments. Thirty models (15 for means and 15 for variances) per assessment were fitted to each cross generations (parents and their derived F2 and F3). Of these, 10 (six in the first and four in the second assessment) had only *m* as mean parameter and *E* (or *E*1 and *E*2) (Mather and Jinks 1982) as variance parameter, which indicated absence of significant genetic variability for Asian rust in the cross-concerned. The crosses that did not show genetic variability for resistance were: (a) FT-2 × Embrapa 48, FT-2 × BRS 154, Embrapa 48 × BRS 154, Embrapa 48 × BRS 184, Embrapa 48 × BRS 214, Embrapa 48 × BRS 231 in the first assessment; and, (b) FT-2 × Embrapa 48, BRS 154 × BRS 184, BRS 154 × BRS 231, BRS 184 × BRS 214 in the second assessment.

Table 2 Genetic parameters fitted to means and variances of Asian rust severity scores of parents and respective F2 and F3 generations. Data from the first assessment

Crosses/Parameters	*m*	[*d*]	[*h*]	[*l*]	χ^2	df	*P*	*D*	*H*	*E* or *E*1 and *E*2	χ^2	df	*P*
FT 2 × EMBRAPA 48	4.85 ± 0.14	–	–	–	6.85	3	0.08	–	–	8.47 ± 1.00 3.91 ± 0.74	4.67	3	0.20
FT 2 × BRS 154	4.80 ± 0.14	–	–	–	1.61	3	0.66	–	–	9.29 ± 0.62	6.58	4	0.16
FT 2 × BRS 184	4.34 ± 0.11	0.76 ± 0.19	–	–	1.52	2	0.47	–	–	10.28 ± 0.90 1.98 ± 0.39	6.49	3	0.09
FT2 × BRS 214	5.08 ± 0.17	–	–	–	1.42	3	0.70	14.65[a] ± 4.16	–	10.95 ± 0.97	3.43	3	0.33
FT2 × BRS 231	4.84 ± 0.14	–	–	–	6.75	3	0.08	–	24.73[b] ± 7.26 –6.33[c] ± 2.34	6.58 ± 0.89	6.55	2	0.04[d]
EMBRAPA 48 × BRS 154	4.38 ± 0.11	–	–	–	2.43	3	0.49	–	–	6.16 ± 0.41	6.21	4	0.18
EMBRAPA 48 × BRS 184	3.76 ± 0.09	–	–	–	3.04	3	0.39	–	–	4.08 ± 0.27	9.62	4	0.05
EMBRAPA 48 × BRS 214	4.27 ± 0.12	–	–	–	5.91	3	0.12	–	–	3.41 ± 0.67 10.36 ± 1.07	4.35	3	0.23
EMBRAPA 48 × BRS 231	4.35 ± 0.13	–	–	–	4.02	3	0.26	–	–	7.60 ± 0.51	8.31	4	0.08
BRS 154 × BRS 184	4.44 ± 0.12	0.80 ± 0.19	–	–	3.89	2	0.14	–	18.49 ± 4.49	5.66 ± 1.45 1.95 ± 0.39	4.18	2	0.12
BRS 154 × BRS 214	5.17 ± 0.17	–	–	–	3.64	3	0.30	9.84[a] ± 4.16	–	17.45 ± 2.36 7.50 ± 1.44	3.26	2	0.20
BRS 154 × BRS 231	4.73 ± 0.23	–	1.84 ± 0.79	–	0.06	2	0.97	–	19.41 ± 5.47	6.77 ± 0.87	4.09	3	0.25
BRS 184 × BRS 214	4.41 ± 0.12	0.92 ± 0.20	–	–	2.33	2	0.31	3.87[a] ± 1.96	–	10.28 ± 1.16 1.79 ± 0.36	5.25	2	0.07
BRS 184 × BRS 231	4.32 ± 0.12	0.72 ± 0.19	–	–	3.39	2	0.19	–	–	2.02 ± 0.40 10.96 ± 0.95	4.27	3	0.23
BRS 214 × BRS 231	4.92 ± 0.31	–	6.09 ± 2.73	–12.44 ± 4.97	1.29	1	0.26	5.97[a] ± 2.88	–	11.41 ± 1.66 6.41 ± 1.20	3.87	2	0.14

[a] *D* was calculated as 0.5(*D*1 + *D*2), where *D*1 and *D*2 are estimates of *D* in the presence of linkage—Mather and Jinks (1982)

[b] *H* was calculated as 0. 5(*H*1 + *H*2), where *H*1 and *H*2 are estimates of *H* in the presence of linkage—Mather and Jinks (1982)

[c] This values corresponds to the cross products estimated according to Oliveira 1994

[d] Best model found

*E*1 and *E*2 are the additive environmental component of *P*1 and *P*2, respectively, in the presence of genotype × micro-environmental interaction—Mather and Jinks (1982)

Note: All estimates are significant the 5% level of probability unless otherwise specified

Table 3 Genetic parameters fitted to means and variances of Asian rust severity scores of parents and respective F2 and F3 generations. Data from the second assessment

Crosses/Parameters	*m*	[*d*]	[*h*]	[*l*]	χ^2	df	*P*	*D*	*H*	*E* or E_1 and E_2	χ^2	df	*P*
FT 2 × EMBRAPA 48	30.98 ± 0.87	–	–	–	3.81	3	0.28	–	–	350.36 ± 23.31	3.32	4	0.51
FT 2 × BRS 154	25.36 ± 0.82	7.17 ± 1.67	–	–	0.31	2	0.86	–	–	311.65 ± 20.71	4.32	4	0.36
FT 2 × BRS 184	27.39 ± 0.82	5.82 ± 1.90	–	–	0.98	2	0.61	–	–	311.64 ± 20.71	8.27	4	0.08
FT2 × BRS 214	25.99 ± 1.46	5.62 ± 1.69	8.61 ± 4.45	–	0,31	1	0.57	–	–	303.10 ± 20.16	4.11	4	0.39
FT2 × BRS 231	23.99 ± 1.37	9.08 ± 1.56	–10.03 ± 3.81	–	0.49	1	0.48	161.95[a] ± 71.63	–	304.59 ± 41.31 129.22 ± 25.25	2.95	2	0.23
EMBRAPA 48 × BRS 154	20.66 ± 0.71	4.11 ± 1.62	–	–	1.56	2	0.46	–	–	233.58 ± 15.52	8.11	4	0.09
EMBRAPA 48 × BRS 184	24.33 ± 0.77	3.13[b] ± 1.84	–	–	0.94	2	0.62	–	–	276.07 ± 18.36	4.56	4	0.34
EMBRAPA 48 × BRS 214	24.65 ± 0.77	3.26 ± 1.62	–	–	5.33	2	0.07	–	–	271.89 ± 18.13	2.58	4	0.63
EMBRAPA 48 × BRS 231	35.56 ± 2.75	6.18 ± 1.53	–38.64 ± 6.66	–14.80 ± 3.15	[c]	–	–	225.88[a] ± 73.28	–	273.65 ± 38.40 130.62 ± 25.23	3.30	2	0.19
BRS 154 × BRS 184	18.24 ± 0.73	–	–	–	0.86	3	0.83	–	–	249.09 ± 16.57	5.59	4	0.23
BRS 154 × BRS 214	20.10 ± 0.76	–	–	–	1.21	3	0.75	68.71 ± 36.01	–	234.87 ± 21.31	0.40	3	0.94
BRS 154 × BRS 231	16.76 ± 0.63	–	–	–	6.85	3	0.08	–	–	187.78 ± 12.50	5.49	4	0.24
BRS 184 × BRS 214	23.07 ± 0.83	–	–	–	3.09	3	0.38	–	–	318.93 ± 21.14	3.41	4	0.49
BRS 184 × BRS 231	16.99 ± 0.70	2.77 ± 1.47	–	–	0.26	2	0.88	–	–	315.39 ± 35.86 132.98 ± 25.64	0.96	3	0.81
BRS 214 × BRS 231	17.42 ± 0.65	3.03 ± 1.33	–	–	1.02	2	0.60	–	–	195.73 ± 13.05	5.61	4	0.23

[a] *D* calculated as 0.5(*D*1 + *D*2), where *D*1 and *D*2 are estimates of *D* in the presence of linkage—Mather and Jinks (1982)

[b] [*d*] is significant at the 10% level of probability

[c] Perfect fit solution

*E*1 and *E*2 are the additive environmental component of *P*1 and *P*2, respectively, in the presence of genotype × micro-environmental interaction—Mather and Jinks (1982)

Note: All estimates are significant the 5% level of probability unless otherwise specified

Additive genetic effects were significant in seven and 11 of fifteen crosses in the first and second assessment, respectively, as detected by the mean ([*d*]) and variance (*D*) genetic components of the models fitted to the parents and F2 and F3 generations (Mather and Jinks 1982 and Jinks and Pooni 1982). The dominance mean component ([*h*]) was detected two and three times in the first and second assessments, respectively, being directional towards susceptibility in the first and bi-directional in the second assessment. Non-allelic interaction mean components of the duplicate and complementary type in the first and second assessment, respectively, were detected in two crosses. Considering the detection of bi-directional dominance and complementary and duplicate non-allelic interaction, it appears that neither effect played significant roles in the evolution of Asian rust resistance in soybeans.

The detection of a greater amount of significant genetic models in the second assessment confirmed results from analyses performed directly on the F2 and F3 score distributions, i.e., evaluation of rust resistance in soybean genotypes at later plant developmental stages under severe rust infection proved useful for breeding screening purposes, since the genotypes fully expressed their resistance or susceptibility characteristics and were better discriminated.

The non-significance of additive mean effects coupled with significant dominance mean and/or additive or dominance variance components was an indication of dispersed resistance genes among the parents, as in crosses FT-2 × BRS 214, FT2 × BRS 231, BRS 154 × BRS 214, BRS 154 × BRS 231 and BRS 214 × BRS 231 in the first assessment and BRS 154 × BRS 214 in the second assessment. Dispersion was also the probable cause behind detection of dominance components of means larger than the additive components (in absolute values) in crosses FT-2 × BRS 214, FT-2 × BRS 231 and Embrapa 48 × BRS 231 in the second assessment. Further, the occasional significance of *D*2 and non-significance of *D*1 components of the variance model fitting (indicated but not explicitly shown in Tables 2 and 3) point to the presence of repulsion phase linkage (Jinks and Pooni 1982) among the genes for resistance to Asian rust. The predominance of non-significant *H* effects in the variance model fitting, especially in the second assessment, confirmed the mean analysis indications that dominance was less important than additive effects in the control of Asian rust resistance in soybean. These results provided the general picture that Asian rust resistance is controlled predominantly by genes expressing additive effects that are dispersed in the parental genotypes. Combining these genes in the descendant genotypes may prove laborious, especially if they are in fact mostly linked in repulsion phase as our data indicated. Recurrent selection for resistance may prove, therefore, to be the most efficient tool to develop resistant cultivars.

The estimates of the additive environmental components of variance showed that several crosses in the first assessment presented significant genotype × micro-environment interaction (significant *E*1 and *E*2 estimates), while only three out of 15 crosses in the second assessment showed significant interaction. This indicated that the Asian rust pathogen depended on specific combinations of host and micro-environmental conditions to manifest early infection severity. These first assessment *E*1 and *E*2 estimates, however, could also have arisen from spraying the useful plot area directly. In future genetic studies of Asian rust resistance, the experiment border rows should be inoculated and the disease progression into the useful plot area should occur by natural spore dissemination. Inoculations made directly on experimental rows may have caused non-uniformity of disease progress, especially at early stages of plant development. In any case, the data supports the view that delaying evaluation to later soybean developmental stages should result in more reliable assessments.

Narrow sense heritabilities based on F3 family means $\{h_r^2 = [0.5D/(0.5D + 0.0625H + E/5)]\}$ estimated from crosses with significant D genetic parameters were moderate to high, ranging from 0.42 to 0.74 in the second assessment, respectively (Table 4). These heritability values suggested that selection is likely to result in genetic progress.

Estimates of the genetic parameters were also used for predicting the genetic potential of the crosses to generate inbred lines displaying greater resistance than the best parent, cultivar BRS 231

Table 4 Estimates of narrow sense heritability and cross potential togenerate inbred lines showing higher resistance to Asian rust than BRS 231

Crosses	Heritability 2nd assessment	Genetic potential 2nd assessment
FT-2 × BRS 231	0.65	22.96
Embrapa 48 × BRS 231	0.74	8.08
BRS 154 × BRS 214	0.42	25.14

(Jinks and Pooni 1976; Triller et al 1996). Due to the non-significance of D, only three estimates were obtained in the second assessment (Table 4). Crosses FT-2 × BRS 231, Embrapa 48 × BRS 231 and BRS 154 × BRS 214 have potential, respectively, to generate 22.96, 8.08 and 25.14% of inbred lines with greater Asian rust resistance than BRS 231.

The small numbers of heritabilities and cross potential estimates were due to the low number of significant *D* estimates. This was partially expected because estimates of variance components usually have large standard errors and are non-significant a number of times. However, it also portrayed the general scenario of complicated genetic control of soybean resistance to rust.

In brief, the analyses of the data suggested that development of quantitative resistance to Asian rust in soybean cultivars is feasible. However, it will require an elaborate strategy of combining minor genes from several parents and recurrent selection. This type of resistance is likely to be more durable than that provided by major genes such as *Rpp1*, *Rpp2*, *Rpp3* and *Rpp4* and others already identified [Hartwig 1995; Hartman et al. 2004; Miles et al. 2006 and Arias (personal communication)].

Conclusions

1. Early soybean genotype evaluation and selection for Asian rust resistance do not ensure resistant adult plants.
2. Quantitative genes expressing additive gene action for resistance to Asian rust are dispersed among soybean cultivars.
3. Selection for Asian rust resistance based on F3 or later family means is likely to result in satisfactory genetic progress.

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