Rev. Brasil. Genet. X, 4, 709-727 (1987)
(Brazil. J. Genetics)

INHERITANCE OF RESISTANCE TO *Xanthomonas campestris* PV *phaseoli* (SMITH) DYE IN *Phaseolus vulgaris* L.*

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ABSTRACT

Sixty populations from 10 crosses including parents were studied for their disease reaction to isolate Xp CNF 15 of *Xanthomonas campestris* pv *phaseoli* (Xp). Bean cultivars showing different degrees of susceptibility were selected as parents based on inoculations in the field and greenhouse with isolate Xp CNF 15.

Leaf reaction data for the 10 crosses and pod reaction data for six crosses were submitted to weighted generation mean analysis. Gene action models showed that additive gene action for resistance was significant for leaf and pod reaction in all cases. Estimates of gene action were not biased by linkage.

Heritability estimates in the broad and narrow sense were obtained for leaf reaction of the crosses. These estimates were generally high and evaluations by the maximum value showed to be more convenient except in three crosses in which GN Jules was the resistant parent.

Finally, correlation coefficients between leaf and pod reaction were also calculated for the F_2 populations of eight crosses. Leaf reaction was correlated with pod reaction only in crosses where PI 207.262 and Mexico 168 were included. In all other cases the two traits segregated independently.

INTRODUCTION

Chemical control of common bacterial blight, induced by *Xanthomonas campestris* pv. *phaseoli* (Smith) dye, has generally been of low efficiency. Cultural

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* Part of a Doctoral Thesis presented by Carlos Agustin Rava to the Universide Federal de Viçosa.

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control practices, including adequate crop rotation and use of disease-free seeds are not practical in regions where subsistence cropping is prevalent. In these areas, resistant cultivars would be the most viable method of disease control (Webster *et al.*, 1980).

The goal of breeding programs is to develop resistant cultivars with specific agronomic characteristics. Thus, the inheritance of resistance of the new sources should be elucidated (Saettler, 1977).

Several authors have reported that resistance of the dry bean (*Phaseolus vulgaris* L.) to *Xanthomonas campestris* pv. *phaseoli* is genetically complex (Honma, 1956; Coyne *et al.*, 1965, 1966, 1973; Pompeu and Crowder, 1972; Coyne and Schuster, 1974b,c; Valladares-Sanchez *et al.*, 1979; Webster *et al.*, 1980). In the cross between Tendergreen and GN Nebraska 1 Sel 27, the distribution of degrees of infection of the F_2 population showed that resistance was quantitatively inherited and that susceptibility was partially dominant over resistance (Coyne *et al.*, 1966). Later, Pompeu and Crowder (1972), using the resistant lines 7272-1 and 7299-2 (the first derived from GN Nebraska 1 Sel 27), verified that resistance was determined by a few partially dominant genes. The trait was quantitative and highly heritable, with transgressive segregation appearing in all crosses; thus, the level of resistance could be increased by crosses among resistant lines or between resistant and susceptible cultivars.

Gene linkage was found between resistance and late maturity when GN Nebraska 1 Sel 27 was used as the resistant parent (Coyne *et al.*, 1973). No linkage was observed when the resistant parent was PI 207.262 which showed dominance of the tolerant reaction in F_1 (Coyne and Schuster, 1974b). However, Mohan (1981), in the State of Paraná, found that crosses between GN Nebraska 1 Sel 27 and commercial cultivars included plants with higher levels of resistance than their parents in the segregating populations, in which the flowering period continued to be similar to that of the commercial cultivars, with no gene linkage being present.

Cultivar PI 207.262 showed a high level of resistance in leaves and low susceptibility in pods, GN 1140 showed high susceptibility in leaves and moderate in pods, and Bush Roma No. 4 showed moderate and high susceptibility in leaves and pods, respectively (Coyne and Schuster, 1974a). Based on these results, they suggested that the disease reaction of these cultivars may be due to the recombination of genes that control the reaction of different plant parts to the bacterial infection. Later reports confirmed that inheritance of leaf and pod reaction was determined by different genes or groups of genes (Valladares-Sanchez *et al.*, 1979, 1983), which had to be taken into consideration for selection (Vieira, 1983).

The objectives of the present study were: 1) to determine models of gene action that permit the evaluation of main genetic effects in the crosses studied; 2) to estimate the heritability of resistance to predict selection gains; and 3) to determine the relationship between leaf and pod reaction.

MATERIAL AND METHODS

The experiments were conducted in the laboratory, experimental field, greenhouse, and screenhouse of CNPAF (Centro Nacional de Pesquisa de Arroz e Feijão) EMBRAPA, in Goiânia, Goiás, Brazil, from September 1981 to May 1984.

Parents were chosen on the basis of diasease reaction to inoculation with isolate Xp CNF 15 in the greenhouse and field (Rava, 1985) and the crosses shown in Table I were obtained from the selected parents. Due to the insufficient number of seeds in some backcrosses, F_2 seeds of the backcrosses were obtained in the case of crosses numbers 2, 3, 6, and 10 (Table I).

Table I - Type of cross, parents and populations of 10 crosses between *P. vulgaris* cultivars that were studied for their disease reaction to *X. campestris* pv. *phaseoli*.

Number	Crosses		Populations[2]
	Type of cross[1]	Parents	
1	R x R	GN Jules x Feijão 60 Dias	$P_1, P_2, F_1, F_2, RC_1, RC_2$
2	R x MR	PI 207.262 x México 29	$P_1, P_2, F_1, F_2, F_2RC_1, F_2RC_2$
3	R x MR	México 168 x México 29	$P_1, P_2, F_1, F_2, F_2RC_1, F_2RC_2$
4	R x S	GN Jules x Ricopardo 896	$P_1, P_2, F_1, F_2, RC_1, RC_2$
5	R x S	PI 207.262 x Aroana	$P_1, P_2, F_1, F_2, RC_1, RC_2$
6	R x S	GN Jules x CNF 0010	$P_1, P_2, F_1, F_2, RC_1, RC_2$
7	R x S	México 168 x Cornell 49-242	$P_1, P_2, F_1, F_2, F_2RC_1, F_2RC_2$
8	S x MR	Rosinha G-2 x México 29	$P_1, P_2, F_1, F_2, RC_1, RC_2$
9	S x S	Bico de Ouro x Aroana	$P_1, P_2, F_1, F_2, RC_1, RC_2$
10	S x S	CNF 0010 x Cornell 49-242	$P_1, P_2, F_1, F_2, F_2RC_1, F_2RC_2$

[1] R = resistant; MR = moderately resistant; S = susceptible.

[2] P_1 = female parent; P_2 = male parent; F_1 = first hybrid generation; F_2 = first selfed generation after hybridization; RC_1 = backcross generation ($P_1 \times F_1$); RC_2 = backcross generation ($P_2 \times F_1$); F_2RC_1 = selfed generation of RC_1; F_2RC_2 = selfed generation of RC_2.

Disease reaction in primary leaves

Six progenies of each cross were sown, and the plants identified. Bacterial suspensions of the highly pathogenic isolate Xp CNF 15 (Rava, 1984) were obtained

from bacterial cultures grown in PDA (potato-dextrose-agar) for 48 hours at 28°C and adjusted with the spectrophotometer to a concentration of 5 x 10^7 cfu/ml (colony forming units/ml). Inoculation was made 11 days after sowing by clipping the primary leaves during the late afternoon (Webster, 1978; Sartorato and Rava Seijas, 1981; Rava, 1984). Ten plants of the susceptible cultivar Rosinha G-2, used as control, were also inoculated.

Disease evaluation of the crosses was done when the plants of Rosinha G-2 showed high-intensity symptoms, which occurred 8-10 days after inoculation depending on air temperature. During this period, greenhouse temperature oscillated between 28 and 30°C after midday and between 20 and 22°C at night. All observations were made in the early morning or late afternoon by using a 0-6 grade scale described by Rava (1984). The fours halves of the two primary leaves in each plant were evaluated. Average as well as maximum ratings per plant were included in the analysis.

Weighted generation mean analysis for the crosses (average and maximum values) were done based on a digenic model (Mather and Jinks, 1982). A separate equation was utilized for each generation. Equations 1 to 6 were used when there were enough backcross seeds and equations 1 to 4 and 7 and 8 when it was necessary to obtain F_2 generations from the backcrossed populations.

$$P_1 = m + a + aa \quad (1)$$
$$P_2 = m - a + aa \quad (2)$$
$$F_1 = m + d + dd \quad (3)$$
$$F_2 = m + 1/2d + 1/4dd \quad (4)$$
$$RC_1 = m + 1/2a + 1/2d + 1/4aa + 1/4ad + 1/4dd \quad (5)$$
$$RC_2 = m - 1/2a + 1/2d + 1/4aa - 1/4ad + 1/4dd \quad (6)$$
$$F_2RC_1 = m + 1/2a + 1/4d + 1/4aa + 1/8ad + 1/16dd \quad (7)$$
$$F_2RC_2 = m - 1/2a + 1/4d + 1/4aa - 1/8ad + 1/16dd \quad (8)$$

Six equations with six unknowns were formed for each cross. A two-parameter model was initially fitted including the midparent mean (m) and the additive effect (a). The dominance effect (d) and nonallelic interactions (additive by additive epistasis = aa; additive by dominant epistasis = ad; and dominant by dominant epistasis = dd) were sequentially included according to their contribution to the reduction of the residual mean squares, until all unknowns but one were considered. The solution was achieved by the least squares method. All estimates of parameters which showed values equal to or above twice the value of their standard errors were included in the models of gene action (Zimmermann, 1983; Zimmermann *et al.*, 1985). Goodness-of-fit of the models was estimated according to the residual mean squares, minimized in the sequential inclusion of parameters. The residual mean square has a χ^2 distribution with residual degrees of freedom (Mather and Jinks, 1982).

To evaluate whether the results were biased by linkage the following estimates were obtained from the nonsegregating generations.

$$a = 1/2 (P_1 - P_2) \quad (9)$$
$$m + aa = 1/2 (P_1 + P_2) \quad (10)$$
$$d + dd - aa = F_1 - 1/2 (P_1 + P_2) \quad (11)$$

When such estimates differ from those obtained through the analysis of all generations, there is evidence of linkage bias (Zimmermann, 1983; Zimmermann *et al.*, 1985).

For the estimates of broad- and narrow-sense heritabilities, additive (A), dominant (D), and environmental variances (E) were calculated for the crosses and backcrosses from the following equations (Mather and Jinks, 1982).

$$VF_2 = 1/2A + 1/4D + E \quad (12)$$
$$VRC_1 + VRC_2 = 1/2A + 1/2D + 2E \quad (13)$$
$$VP_1 = E \quad (14)$$
$$VP_2 = E \quad (15)$$
$$VF_1 = E \quad (16)$$

When the F_2 generations of the backcrosses were used, equation 13 was changed to the following equation:

$$VF_2RC_1 + VF_2RC_2 = A + 3/8D + 2E \quad (17)$$

The system of five equations with three unknowns was solved by the least squares method.

Disease reaction in pods

After evaluation of primary leaves, plants from the six progrenies of each cross were identified and transplanted to the field. Two flowers of each plant were labelled two to three days after pollination to reduce the errors caused by pod age in the evaluation of their reaction to the inoculation with Xp CNF 15. One pod from the labelled flowers was harvested 20 days after labelling from each plant, washed in tap water and disinfested by successive immersions in 70°GL alcohol, followed by commercial 20% hypochlorite and three washings in sterile distilled water. Pods were placed in previously disinfected plastic gerboxes with three to a box.

The inoculum consisted of a bacterial suspension obtained as described earlier at a concentration of 10^8 cfu/ml. Each pod was inoculated by injection of 2 μl of the

inoculum at three points between the seeds. The injection was made with an adjustable hypodermic syringe previously disinfected with 96°GL alcohol. The gerboxes were placed on a table in the laboratory, where they received additional lighting during the day (fluorescent 40 W lamps at a 50 cm distance) for a 12-hour period. Room temperature was maintained at 28°C ± 2°C during the 3 days of incubation.

Evaluations were done by taking vernier caliper measurements of two perpendicular diameters of each lesion. The averages of the diameters of the three lesions (average value) and the average diameter of the larger lesion (maximum value) in each pod were considered in the analysis.

Weighted generation mean analysis was done using the method described for leaf reaction. For crosses numbers 2 and 4 (Table I) only four generations were considered due to the loss of backcross plants during transplanting. Due to the small number of plants of all generations, it was not possible to calculate heritabilities of reactions to Zp CNF 15 in pods. Presence of bias due to linkage was investigated in the same way as described for disease reaction in leaves.

Correlation coefficients between leaf and pod reaction of F_2 populations from eight crosses that survived transplanting were determined for average and maximum values.

RESULTS

Disease reaction in primary leaves and pods

Tables II and III include the estimates of the coefficients of significant parameters (b) and their standard deviations, genetic models, coefficients of determination (r^2), goodness-of-fit of the models (χ^2), and their probability levels (P) obtained from the weighted generation mean analysis for average and maximum values, respectively. Table IV includes the estimates obtained by equations 9, 10, and 11 for comparison with those from the weighted generation mean analysis, which were not significantly different from each other by the *t*-test at the 5% probability level. Average heritability estimates varied from 98 to 63% for broad-sense heritability and from 90 to 0% for narrow-sense heritability and are included in Table V. For maximum values, heritability varied between 93 and 41% and between 93 and 9% for broad and narrow sense, respectively.

An adequate number of adult plants for measuring pod reaction to Xp CNF 15 could be obtained from only six crosses in all generations. Only four generations were considered in crosses 2 and 4, due to the loss of individuals from the backcrosses after transplanting.

Significant parameters with their respective coefficients and standard deviations, genetic models, coefficients of determination (r^2), goodness-of-fit of the models

Table II - Estimates and standard deviations (S.D.) of genetic parameters included in the gene-action models obtained by generation mean analysis, coefficients of determination (r^2), goodness of fit of the models (X^2 and P) for reactions in leaves of 10 crosses of *P. vulgaris* evaluated by average values.

Cross	Estimate S.D.	Model	r^2	X^2 (df)	P
Jules x F. 60 dias (R x R)	m = 1.87 a = -0.79 ±0.1445	Y = m + a	0.8811	0.1575 (4)	>0.99
PI 207.262 x Mex. 29 (R x MR)	m = 2.12 a = -0.41 ±0.0587 d = 0.46 ±0.1137	Y = m + a + d	0.9538	0.0331 (3)	>0.99
Mex. 168 x Mex. 29 (R x MR)	m = 2.02 a = -0.52 ±0.0440 aa = -0.54 ±0.0795	Y = m + a + aa	0.9883	0.0193 (3)	>0.99
Jules x Ricopardo (R x S)	m = 2.18 a = -0.80 ±0.0897	Y = m + a	0.9526	0.0466 (4)	>0.99
PI 207.262 x Aroana (R x S)	m = 2.87 a = -0.79 ±0.0690 d = 0.44 ±0.1507	Y = m + a + d	0.9841	0.0546 (3)	>0.99
Jules x CNF 0010 (R x S)	m = 2.88 a = -1.43 ±0.1455 dd = -0.52 ±0.2378	Y = m + a + dd	0.9699	0.1614 (3)	0.95
Mex. 168 x Cornell (R x S)	m = 2.42 a = -1.31 ±0.0451 d = 0.58 ±0.0432	Y = m + a + d	0.9970	0.0228 (3)	>0.99
Ros. G-2 x Mex. 29 (S x MR)	m = 3.12 a = 0.71 ±0.0412 d = -0.23 ±0.0773 ad = 1.30 ±0.1740	Y = m + a + d + ad	0.9973	0.0121 (2)	>0.99
B. Ouro x Aroana (S x S)	m = 3.39 a = -0.14 ±0.0362 ad = 0.45 ±0.1892 dd = 0.18 ±0.0566	Y = m + a + ad + dd	0.9257	0.0150 (2)	>0.99
CNF 0010 x Cornell (S x S)	m = 4.29 a = 0.07 ±0.179 ad = -0.70 ±0.2232	Y = m + a + ad	0.8639	0.0033 (3)	>0.99

m = Mid parent mean; a = additive effect; d = dominant effect; aa = additive by additive effect; ad = additive by dominant effect; dd = dominant by dominant effect; (df) = degrees of freedom.

Table III - Estimates and standard deviations (S.D.) of genetic parameters included in the gene-action models obtained by generation mean analysis, coefficients of determination (r^2), goodness of fit of the models (χ^2 and P) for reactions in leaves of 10 crosses of *P. vulgaris* evaluated by maximum values.

Cross	Estimate S.D.	Model	r^2	χ^2 (df)	P
Jules x F. 60 dias (R x R)	m = 2.58 a = -0.87 ± 0.1522	Y = m + a	0.8901	0.1201 (4)	>0.99
PI 207.262 x Mex. 29 (R x MR)	m = 2.70 a = -0.49 ± 0.0774 dd = 0.72 ± 0.1550	Y = m + a + dd	0.9556	0.0293 (3)	>0.99
Mex. 168 x Mex. 29 (R x MR)	m = 2.34 a = -0.65 ± 0.0014 d = 0.45 ± 0.0052 aa = -0.09 ± 0.0041 ad = -0.14 ± 0.0017	Y = m + a + d + aa + ad	0.9999	0.0000 (1)	>0.99
Jules x Ricopardo (R x S)	m = 2.85 a = -0.95 ± 0.0283 d = 0.32 ± 0.0533 ad = 0.43 ± 0.1091	Y = m + a + d + ad	0.9986	0.0019 (2)	>0.99
PI 207.262 x Aroana (R x S)	m = 3.58 a = -0.81 ± 0.0162 ad = 0.50 ± 0.0906 dd = 0.33 ± 0.0295	Y = m + a + ad + dd	0.9993	0.0012 (2)	>0.99
Jules x CNF 0010 (R x S)	m = 3.29 a = -1.38 ± 0.2229	Y = m + a	0.9059	0.1836 (4)	0.99
Mex. 168 x Cornell (R x S)	m = 3.02 a = -1.29 ± 0.0897 d = 1.78 ± 0.6402 dd = -1.80 ± 0.5939	Y = m + a + d + dd	0.9935	0.0384 (2)	0.95
Ros. G-2 x Mex. 29 (S x MR)	m = 3.82 a = 0.58 ± 0.0522 aa = 0.21 ± 0.0824 ad = 1.23 ± 0.2345	Y = m + a + aa + ad	0.9933	0.0178 (2)	>0.99
B. Ouro x Aroana (S x S)	m = 3.97 a = -0.06 ± 0.0118 aa = 0.09 ± 0.0340 dd = 0.16 ± 0.0390	Y = m + a + aa + dd	0.9631	0.0031 (2)	>0.99
CNF 0010 x Cornell (S x S)	m = 4.79 a = 0.04 ± 0.0062 aa = 0.02 ± 0.0088 ad = -0.02 ± 0.0076	Y = m + a + aa + ad	0.9644	0.0004 (2)	>0.99

m = Midparent mean; a = additive effect; d = dominant effect; aa = additive by additive effect; ad = additive by dominant effect; dd = dominant by dominant effect; (df) = degrees of freedom.

Table IV -Estimates of dominant effect plus dominant by dominant epistasis minus additive by additive epistasis (d + dd - aa), of midparent mean plus additive by additive epistasis (m + aa) and of additive effect (a), from the nonsegregating populations (P's & F_1) and from generation mean analysis (G) for reaction in leaves to *X. campestris* pv. *phaseoli* from 10 crosses of *P. vulgaris*.

Crosses	Estimates	Average values		Maximum values	
		P's & F_1	G	P's & F_1	G
Jules x F. 60 Dias	d + dd - aa	-0.17	0.00	-0.13	0.00
	m + aa	1.85	1.87	2.52	2.58
	a	-0.78	-0.79	-0.88	-0.87
PI 207.262 x Mex. 29	d + dd - aa	0.44	0.46	0.67	0.71
	m + aa	2.14	2.12	2.74	2.70
	a	-0.42	-0.41	-0.51	-0.49
Mex. 168 x Mex. 29	d + dd - aa	0.60	0.54	0.54	0.54
	m + aa	1.47	1.48	2.25	2.25
	a	-0.50	-0.52	-0.65	-0.65
Jules x Ricopardo	d + dd - aa	-0.02	0.00	0.34	0.32
	m + aa	2.06	2.18	2.86	2.85
	a	-0.78	-0.80	-0.95	-0.95
PI 207.262 x Aroana	d + dd - aa	0.39	0.44	0.33	0.33
	m + aa	2.87	2.87	3.58	3.58
	a	-0.80	-0.79	-0.81	-0.81
Jules x CNF 0010	d + dd - aa	-0.46	-0.52	-0.35	0.00
	m + aa	2.77	2.88	3.32	3.29
	a	-1.36	-1.43	-1.32	-1.38
Méx. 168 x Cornell	d + dd - aa	0.58	0.58	-0.04	-0.02
	m + aa	2.42	2.42	3.04	3.03
	a	-1.30	-1.31	-1.28	-1.29
Ros. G-2 x Mex. 29	d + dd - aa	-0.23	-0.23	-0.19	-0.21
	m + aa	3.12	3.12	4.04	4.03
	a	0.71	0.71	0.58	0.58
B. Ouro x Aroana	d + dd - aa	0.17	0.18	0.07	0.07
	m + aa	3.40	3.39	4.06	4.06
	a	-0.14	-0.14	-0.06	-0.06
CNF 0010 x Cornell	d + dd - aa	-0.02	0.00	-0.02	-0.02
	m + aa	4.30	4.28	4.81	4.81
	a	0.07	0.07	0.04	0.04

Table V - Heritability (h^2) estimates of disease reaction in leaves (average and maximum values) to Xp CNF 15 isolate of *X. campestris* pv. *phaseoli* in 10 crosses of *P. vulgaris*.

Crosses	Broad sense h^2 (%)		Narrow sense h^2 (%)	
	Average	Maximum	Average	Maximum
GN Jules x F. 60 Dias	86	85[1]	58	85
PI 207.262 x Mex. 29	82	75[1]	55	75
Mex. 168 x Mex. 29	80	58	75	52
GN Jules x Ricopardo	63[1]	68[1]	63	68
PI 207.262 x Aroana	85	72	19	51
GN Jules x CNF 0010	80[1]	41	80	9
Mex. 168 x Cornell	98[2]	85[1]	0	85
Ros. G-2 x Mex. 29	90[1]	86[1]	90	86
B. Ouro x Aroana	89[1]	93[1]	89	93
CNF 0010 x Cornell	81[1]	82[1]	81	82

[1]Estimate of dominant variance (D) = 0.

[2]Estimate of additive variance (A) = 0.

(χ^2) and probability levels (P) obtained from the weighted generation mean analysis are presented in Tables VI and VII. Estimates obtained from the nonsegregating populations and from the analysis of all generations did not differ significantly by the *t*-test at the 5% probability level (Table VIII).

Table IX includes the correlation coefficients between primary leaf reaction and pod reaction (for average and maximum values) for the F_2 plants of the eight crosses.

DISCUSSION

Disease reaction in primary leaves

Weighted generation mean analysis detected significant gene effects for the two evaluation criteria used (average and maximum values). The differences in the models obtained by the two criteria indicated the existence of a scale effect similar to that described by Mather and Jinks (1982). Despite these differences, significance of additive effects for higher resistance was detected in all models. This is especially important because dry beans are an autogamous crop in which homozygous plants are the regular components of any population. Additive gene effects indicate that re-

Table VI - Estimates and standard deviations (S.D.) of genetic parameters included in the gene-action models obtained by generation mean analysis as well as coefficients of determination (r^2), goodness of fit of the models (χ^2 and P) for reaction in pods of 6 crosses of *P. vulgaris* evaluated by average values.

Crosses	Estimate	Model	r^2	χ^2(df)	P
PI 207.262 x Mex. 29	m = 1.16 a = -0.77 ± 0.0438 dd = 0.97 ± 0.0496	Y = m + a + dd	0.9975	0.1060 (1)	0.90
Mex. 168 x Mex. 29	m = 2.27 a = -0.70 ± 0.0714 aa = -0.61 ± 0.0922	Y = m + a + aa	0.9705	0.0373 (3)	>0.99
Jules x Ricopardo	m = 1.12 a = -0.69 ± 0.0169 d = 0.57 ± 0.0327	Y = m + a + d	0.9995	0.0018 (1)	0.95
PI 207.262 x Aroana	m = 1.39 a = -1.12 ± 0.0114 aa = -0.06 ± 0.0263 dd = 0.79 ± 0.0276	Y = m + a + aa + dd	0.9999	0.0017 (2)	>0.99
Mex. 168 x Cornell	m = 1.33 a = -0.60 ± 0.0441 d = 0.12 ± 0.0573	Y = m + a + d	0.9842	0.0126 (3)	>0.99
B. Ouro x Aroana	No model adjusted				

m = Midparent mean; a = additive effect; d = dominant effect; aa = additive b additive effect; ad = additive by dominant effect; dd = dominant by dominant effect; (df) = degrees of freedom.

sistance can be fixed in the homozygous individuals of advanced generations. The importance of additive gene effects has been pointed out by Valladares-Sanchez *et al.* (1983). Although independent of the evaluation criterion used, additive effects were significant in all crosses, and their expression was more evident in the crosses between resistant and susceptible cultivars.

When the resistant parent was GN Jules, especially in cross number 4 for maximum values (Table VIII), dominance for susceptibility was significant. These

Table VII - Estimates and standard deviations (S.D.) of genetic parameters included in the gene-action models obtained by generation mean analysis as well as coefficients of determination (r^2), goodness of fit of the models (χ^2 and P) for reaction in pods of 6 crosses of *P. vulgaris* evaluated by maximum values.

Crosses	Estimate	Model	r^2	X^2 (df)	P
PI 207.262 x Mex. 29	m = 1.43 a = -0.89 ± 0.0493 dd = 1.04 ± 0.0579	Y = m + a + dd	0.9443	0.0099 (1)	0.90
Mex. 168 x Mex. 29	m = 2.59 a = -0.60 ± 0.0845 aa = -0.42 ± 0.1096	Y = m + a + aa	0.9443	0.0308 (3)	>0.99
Jules x Ricopardo	m = 1.44 a = -0.75 ± 0.0377 d = 0.44 ± 0.0636	Y = m + a + d	0.9975	0.0050 (1)	0.90
PI 207.262 x Aroana	m = 1.68 a = -1.27 ± 0.0554 ad = 0.47 ± 0.2025 dd = 0.84 ± 0.0752	Y = m + a + ad + dd	0.9969	0.0125 (2)	>0.99
Mex. 168 x Cornell	m = 1.78 a = -0.56 ± 0.0829 dd = 0.43 ± 0.1192	Y = m + a + dd	0.9405	0.0261 (3)	>0.99
B. Ouro x Aroana	No model adjusted				

m = Midparent mean; a = additive effect; d = dominant effect; aa = additive by additive effect; ad = additive by dominant effect; dd = dominant by dominant effect; (dd) = degrees of freedom.

results are similar to those obtained by Coyne *et al.* (1966) but are different from those reported by Pompeu and Crowder (1972). In both cases the resistant parent was GN Nebraska 1 Sel 27 from which GN Jules was derived (Coyne and Schuster, 1970). Due to the quantitative nature of the trait, marked environmental influence is expected. Also, the expected influence of the inoculation procedure, evaluation criterion, plant age, and susceptible parent may be responsible for the discrepancies of some of the results observed.

Table VIII -Estimates of dominant effect plus dominant by dominant epistasis minus additive by additive epistasis (d + dd - aa), of midparent mean plus additive by additive epistasis (m + aa) and of additive effect (a), from the non segregating populations (P's & F_1) and from generation mean analysis (G) for reaction in pods to *X. campestris* pv. *phaseoli* from five crosses of *P. vulgaris*.

Crosses	Reaction in leaves	Average values		Maximum values	
		P's & F_1	G	P's & F_1	G
PI 207.262 x Mex. 29	d + dd - aa	0.99	0.97	1.06	1.04
	m + aa	1.15	1.16	1.41	1.43
	a	-0.78	-0.77	-0.90	-0.89
Mex. 168 x Mex. 29	d + dd - aa	0.66	0.61	0.45	0.42
	m + aa	1.65	1.58	2.19	2.18
	a	-0.71	-0.70	-0.58	-0.60
GN Jules x Ricopardo	d + dd - aa	0.56	0.57	0.43	0.44
	m + aa	1.11	1.12	1.43	1.44
	a	-0.69	-0.69	-0.75	-0.75
PI 207.262 x Aroana	d + dd - aa	0.85	0.85	0.83	0.84
	m + aa	1.32	1.32	1.70	1.68
	a	-1.12	-1.12	-1.27	-1.27
CNF 0010 x Cornell	d + dd - aa	0.11	0.12	0.36	0.43
	m + aa	1.33	1.33	1.84	1.78
	a	-0.61	-0.60	-0.52	-0.56

With GN Jules, additive effects for resistance were also detected without interference of dominance or of additive by dominant and dominant by dominant epistasis for average values in crosses number 1 and 4 (Table II) and for maximum values in crosses numbers 1 and 6 (Table III). However, when comparing the models for maximum and average values, the advantage of the second model is evident. Although both criteria had shown additive effects in two crosses, evaluations by the average value in cross number 6 showed dominant by dominant epistasis for resistance. Therefore, in this cross and in the other two, rigorous selection can be applied in the early segregating generations without the risk of eliminating resistant lines. The

Table IX - Correlation coefficients between disease reaction on leaves and disease reaction on pods of F_2 plants of eight crosses of *P. vulgaris* inoculated with the isolate Xp CNF 15 of *X. campestris* pv. *phaseoli*.

Cross	Reaction in leaves	Reaction in pods	
		Average	Maximum
PI 207.262 x Mex. 29	Average	0.49**	0.47**
N = 197	Maximum	0.48**	0.45**
Mex. 168 x Mex. 29	Average	0.38**	0.36**
N = 107	Maximum	0.31**	0.30**
GN Jules x Ricopardo	Average	-0.05	-0.07
N = 162	Maximum	-0.06	-0.08
PI 207.262 x Aroana	Average	0.30**	0.28**
N = 192	Maximum	0.19**	0.20**
GN Jules x CNF 0010	Average	0.08	0.06
N = 95	Maximum	0.12	0.08
Mex. 168 x Cornell	Average	0.45**	0.29
N = 41	Maximum	0.35*	0.22
Ros. G-2 x Mex. 29	Average	0.12	0.07
N = 54	Maximum	0.12	0.09
B. de Ouro x Aroana	Average	0.02	0.02
N = 286	Maximum	0.03	0.06

*Significant at the 5% level; **Significant at the 1% level.

interactions disappear with increasing homozygosity. The same did not occur with maximum values because cross number 4 included dominance and additive by dominant epistatic effects for susceptibility. Thus, F_2 plants that may yield some resistant lines in more advanced generations could be eliminated.

Considering the average values (Table II) of the two crosses in which PI 207.262 was the resistant parent, significant positive dominance effects for suscepti-

bility were also detected. These results did not agree with those obtained by Coyne and Schuster (1974b) in studies of parental and F_2 distributions. However, with maximum values (Table III), gene action models also showed nonallelic interaction effects for susceptibility.

Mexico 29, although classified as moderately resistant, appears to be a very good parent. There were additive by additive epistatic effects for resistance in two of three crosses for maximum values (Table III) and in one for average values (Table II). This suggests transgressive segregation for higher resistance in advanced generations especially when evaluations are done by maximum values.

In the two crosses with Mexico 168, there were additive gene effects for resistance (Tables II and III) and additive by additive epistasis in the cross with Mexico 29. Therefore, Mexico 168 is also a valuable source of resistance to Xp CNF 15, especially for tropical regions in which it does not present limiting adaptation problems such as those of GN Jules and PI 207.262.

Nonfixable dominance and epistatic effects of variable magnitude in the direction of higher susceptibility were detected by both evaluation criteria in the crosses with PI 207.262 and Mexico 168 (resistant), Bico de Ouro, Cornell 49-242, and Rosinha G-2 (susceptible). These results indicate the convenience of only applying rigorous selection in advanced generations. In crosses including GN Jules (resistant) and Rico Pardo 896 or CNF 0010 (susceptible) there was interference of dominance effects or of epistatic effects for susceptibility by both evaluation procedures. In the cross between CNF 0010 and Cornell 49-242, there was additive by additive epistasis of low magnitude for maximum values (Table III). Due to its small, although significant value, such epistatic effects may be of little importance in selection for resistance. This was expected because both cultivars are susceptible to Xp CNF 15.

In addition to the additive effects, the models shown in Tables II and III also permitted the identification of other types of gene action in the crosses studied. Additive by additive epistasis for higher resistance was evident in four crosses for maximum values (Table III) and only in one case for average values (Table II). Additive by additive epistasic effects are the only ones that can be fixable by selection, and, thus, they are potentially useful for a pure line selection program. Also, these four crosses are promising for obtaining transgressive segregants with higher resistance in advanced generations (Zimmermann, 1983; Zimmermann *et al.*, 1985).

Evaluations by the maximum values permitted the detection of larger numbers of gene effects, are easier to calculate, and permit the formation of a smaller number of classes. In conclusion, evaluations by the maximum value of each plant proved to be generally more convenient, except for the crosses in which GN Jules was the resistant parent.

The existence of different gene action models in the crosses studied makes it difficult to draw general conclusions about the inheritance of resistance to Xp CNF 15

in *P. vulgaris,* which seems to be determined not only by the parental genotypes, but also by the evaluation criterion and by the environment.

In generation mean analysis, estimates of epistatic effects are unique, but estimates of additive and dominance effects may be biased by the presence of epistasis and by linkage disequilibrium (Hallauer and Miranda F°, 1981). In the present study, the whole set of generations that would be necessary to study the significance of linkage was not obtained. The total set would require four more double backcrosses besides the generations that were obtained (Mather and Jinks, 1982). However, since the epistatic effects were significant, estimates of parameters from the nonsegregating generations were compared to the ones obtained by generation mean analysis (Table IV). Since they did not show significant differences, there was an indication that linkage did not bias the estimates.

In comparing generation mean analysis and determination of variance components, Hallauer and Miranda F° (1981) pointed out that generation mean analysis is particularly useful for autogamous crops because of the limited number of hand pollinations that are required to obtain the different generations. Besides, averages are first order statistics and as such, their errors are inherently smaller than the error of variances (second order statistics). The same authors emphasized that generation mean analysis does not permit the estimation of heritability, and, therefore, does not permit the estimation of selection gains. Also, gene effects of several loci, when of opposite sign may cancel each other and may be nondetectable. Therefore, broad- and narrow-sense heritability estimates by variance components were obtained in the present study (Table V).

Except for average values in crosses numbers 5 and 7 and for maximum value in cross number 6, narrow- and broad-sense heritability estimates were higher and similar to those obtained by Pompeu and Crowder (1972). Thus, it is possible to predict a good efficiency of selection for resistance to Xp CNF 15 under similar conditions to those described in the present study.

For all crosses except the three where GN Jules was the resistant parent, maximum value estimates seem to be better. Two of these three crosses showed small differences for the two evaluation procedures, but in cross number 6, narrow-sense heritability was much greater when average values were used. In the remaining crosses, there was no estimate equal to zero for the additive variance when maximum values were used. Furthermore, the easier procedure and the smaller number of classes obtained by maximum values than by average values indicated the superiority of the first criterion for evaluation.

In comparing the estimates of variance components with estimates of gene effects for average values, in 4 of 5 crosses where dominant variance was equal to zero (Table V), the presence of dominant gene effects was not detected (Table III). For maximum values (Tables III and V) the results were similar in 5 of 7 crosses. The

discrepancy between the two statistical procedures in some cases may be explained by the larger error of the analysis of variance compared to generation mean analysis (Hallauer and Miranda F°, 1981).

Disease reaction in pods

In crosses 5 and 7 some differences in the detected gene effects between the two evaluation procedures (average and maximum values) were noticed (Tables VI and VII). However, in all models and by both criteria, additive gene effects for resistance were significant, similarly to what was observed for leaf reaction.

For average values of crosses numbers 3 and 5 (Table VI) and for maximum values of cross number 3 (Table VII), there were additive by additive epistatic effects. The fact that cross number 3 was the only one that showed such effects for leaf reaction by the two evaluation procedures supports the convenience of using Mexico 29 and Mexico 168 in breeding programs for resistance to Xp CNF 15. The importance of these genetic effects was discussed for the reaction on leaves.

All crosses, except number 3, showed dominant gene effects as well as large additive by dominant and dominant by dominant epistatic effects for susceptibility.

No model gave a good fit for the evaluation by the average or maximum values for cross number 9, which possibly indicates the genetic similarity of both susceptible parents.

Similarly to what was reported for the disease reaction in leaves, Table VIII shows that gene linkage did not significantly bias the estimates of genetic effects in pods (Zimmermann, 1983; Zimmermann *et al.*, 1985).

Correlation between the disease reaction in leaves and the disease reaction in pods

The low association between leaves and pod disease reaction in most crosses is in accordance with the results of Valladares-Sanchez *et al.* (1983) (Table IX). Similar results were obtained for F_2 plants of the two crosses in which GN Jules was the resistant parent. They disagree with those reported by Webster (1978) who found a highly significant correlation coefficient ($r = 0.72$) between disease reaction in leaves and in pods in only 15 F_4 families from the cross GN Jules x Porrillo Sintético.

A highly significant correlation coefficient was obtained between disease reaction in leaves and disease reaction in pods of the F_2 plants from crosses in which PI 207.262 and Mexico 168 were the resistant parents. The low coefficients of determination rules out indirect selection. Another reason to recommend the use of these cultivars for breeding programs for resistance to Xp CNF 15 is the existence of an association of favorable traits in these two parents.

ACKNOWLEDGMENTS

The authors are very grateful to Dr. Francisco Zimmermann for statistical analysis, to the technicians João D. Purissimo and Elcio de O. Alves, to the laboratory assistants Edilamar E. de Souza and Maria de Lourdes Soares and to Mr. Juracy de Oliveira for their valuable assistance.

RESUMO

Foram estudadas 60 populações (incluindo os progenitores) de 10 cruzamentos entre cultivares de feijão com diferentes graus de suscetibilidade a *Xanthomonas campestris* pv. *phaseoli* (Xp), selecionadas com base nas reações exibidas ao serem inoculadas com o isolamento Xp CNF 15 em casa-de-vegetação e no campo.

Foi realizada a análise ponderada de médias de gerações dos 10 cruzamentos, no caso da reação foliar, e de seis cruzamentos, no caso da reação em vagens. Os modelos de ação gênica permitiram que se constatasse a existência de efeito gênico aditivo para resistência, da reação em folhas e em vagens, em todos os casos estudados. As estimativas dos efeitos gênicos não foram afetadas pelo desequilíbrio ocasionado pela ligação gênica.

Também foram obtidas estimativas da herdabilidade no sentido amplo e no sentido restrito em relação à reação foliar dos 10 cruzamentos estudados, as quais, em geral, foram altas, sendo mais conveniente a avaliação pela nota máxima, com exceção dos três cruzamentos nos quais 'GN Jules' foi o progenitor resistente.

Finalmente, foram calculados os coeficientes de correlação entre a avaliação em folhas primárias e em vagens das plantas das progênies F_2 de oito cruzamentos. As plantas das progênies F_2, dos cruzamentos nos quais os cultivares 'PI 207.262' e 'Mexico 168' foram os progenitores resistentes, apresentaram associação entre a reação em folhas e em vagens, ao passo que, nos casos restantes, houve segregação independente desses caracteres.

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(Received April 27, 1987)