Brazilian Journal of Genetics, 20, 1, 51-58 (1997)
(Revista Brasileira de Genética)

Genetic control of characters associated with bean golden mosaic geminivirus resistance in *Phaseolus vulgaris* L.*

Luis Alberto Pessoni[1], Maria José de O. Zimmermann[2] and Josias Corrêa de Faria[2]

ABSTRACT

Bean golden mosaic is the most important viral disease of the bean crop (*Phaseolus vulgaris* L.) in Latin America. The genetics of resistance to a Brazilian strain of bean golden mosaic virus (BGMV), was studied in a 4 x 4 diallel cross without reciprocals, among the parental genotypes DOR 303, EMGOPA 201 Ouro, Carnaval, and Redlands Greenleaf C. Seedlings of the four parents, six F_1 hybrids, 12 backcrosses, and F_2 generations for each combination were inoculated on the eighth day after sowing by exposure to a viruliferous whitefly (*Bemisia tabaci* Genn.) population for 24 h, in a glasshouse, prior to transplantation to field conditions. The full set of two parents, F_1, F_2 and respective backcrosses for each combination was considered to be a family. Data were recorded and analyzed for foliar yellowing, plant dwarfing, and pod malformation, using a randomized block design, with two replications. Weighted generation mean analysis was performed for each of the six families. An additive gene action model was significant for the three characteristics evaluated. On the other hand, non-additive gene action had greater absolute value in most cases. Resistance to foliar yellowing conferred by genes from DRO 303 was highly heritable and was expressed equally well in the different genetic backgrounds evaluated. Such resistance may be oligogenic. Broad- and narrow-sense heritabilities were relatively high for all response traits. The three traits studied were all positively correlated, indicating that they can be simultaneously selected for enhancement. The highest correlation coefficient was obtained for dwarfing x pod malformation.

INTRODUCTION

Bean golden mosaic was first described in Brazil by Costa (1965), who found that the causal agent was transmitted by the whitefly *Bemisia tabaci* Genn. (Homoptera: Aleyrodidae). This disease has produced high yield losses on the crop sown in the "second planting season" in some regions of Brazil (February-March), traditionally done by small and medium income growers (Costa *et al.*, 1973; De Fazio, 1985), and throughout Central America and the Caribbean Basin (Brown and Bird, 1992).

Subsequent work by Gálvez and Castaño (1976) has led to the conclusion that the new disease was caused by a virus containing geminate particles, which was named bean golden mosaic virus (BGMV). BGMV is classified in the geminivirus group, characterized by twinned quasi-isometrical particles containing single stranded DNA (Goodman, 1977). Isolates from Brazil (Costa, 1976; Figueira, 1980) and Argentina (Gilbertson

* Part of a thesis presented by L.A.P. to the Universidade Federal de Goiás in partial fulfillment of the requirements for the Master's degree.

[1] Universidade Federal de Goiás, Goiânia, GO, Brasil.

[2] EMBRAPA, Centro Nacional de Pesquisa de Arroz e Feijão (CNPAF), Caixa Postal 179, 74001-970 Goiânia, GO, Brasil. Send correspondence to M.J.O.Z.

et al., 1991a,b) are not mechanically transmissible as opposed to those from Mexico, Central America and the Caribbean Basin. DNA sequences of virus isolates from the different regions have confirmed the previously observed biological differences (Gilbertson *et al.*, 1993; Faria *et al.*, 1994). These authors have shown that geminiviruses causing bean golden mosaic belong to two distinct phylogenetic groups, named type I (Brazilian isolate) and type II (Central American and Caribbean).

Resistance to BGMV in *Phaseolus* spp. has been sought since the disease became epidemic in the early 70's (Pompeu and Kranz, 1977; Hohmann and Carvalho, 1982; Alberini, 1982). However, promising sources of disease tolerance have only been identified recently, in races and gene pools different from those cultivated in Brazil (Morales and Niessen, 1988; Singh *et al.*, 1991; Beebe and Corrales, 1991).

The objective of the present research was to investigate models of gene action and the heritability of characteristics associated with the resistance to a Brazilian strain of BGMV, under early and uniform inoculation conditions.

MATERIAL AND METHODS

The experiments were carried out at the National Research Center for Rice and Bean (CNPAF) of the Brazilian Enterprise for Agricultural Research (EMBRAPA), located in Goiânia, Goiás, Brazil.

Data were collected from a diallel cross, without the reciprocals, among four parents representing distinct reaction types to BGMV, as follows: when seedlings are submitted to inoculations with BGMV, DOR 303 plants show no yellowing and a very high degree of plant dwarfing and pod malformation; EMGOPA 201 Ouro plants show an intermediate degree of yellowing and dwarfing and a high degree of pod malformation; Redlands Greenleaf C plants show a high degree of foliar yellowing, intermediate to low plant dwarfing, and little or no pod malformation, and Carnaval plants show a high degree of yellowing, little dwarfing and medium to high pod malformation.

Each group, composed of parent 1 (P_1), parent 2 (P_2), F_1, F_2, backcross 1 (BC1), and backcross 2 (BC2), was designated as a family (Pereira *et al.*, 1989).

Sowing, inoculation and transplantation of bean seedlings to the field followed the methodology described by Faria *et al.*, 1991. The field experiment was set up in a complete randomized block design with the treatments in a split plot. Families were placed in the main plots, and the different generations from each family in the sub-plots (Pereira *et al.*, 1989). Plant rows were three meters long, with 0.20 m between plants and 0.50 m between rows. The size of each sub-plot varied, with parents and F1's being represented by a row each, the BC's by two rows, and the F2 by nine rows.

Individual plant data were taken for foliar yellowing (typical golden mosaic), plant dwarfing/deformation, and pod malformation at 35, 40, and 65 days after transplantation, respectively. A general evaluation rating scale varying from 1 to 9 was used for each trait (1 indicates complete absence of symptoms, and 9 indicates high symptom expression. Separate analyses of variance were performed for each variable and generation, within families, taking into account data from those plants that showed any evidence of infection by BGMV geminivirus. This was due to the fact that no immunity to infection by the virus is known. Therefore the complete lack of symptoms was taken as an indication of escape from infection.

Weighted generation mean analysis was done for the six generations available, according to the digenic model of Mather and Jinks (1982). A separate equation was utilized for each generation, as follows:

$$\overline{P}_1 = m + a + aa$$

$$\overline{P}_2 = m - a + aa$$

$$\overline{F}_1 = m + d + dd$$

$$\overline{F}_2 = m + \frac{1}{2} d + \frac{1}{4} dd$$

$$\overline{BC}_1 = m + \frac{1}{2} a + \frac{1}{2} d + \frac{1}{4} aa + \frac{1}{4} ad + \frac{1}{4} dd$$

$$\overline{BC}_2 = m - \frac{1}{2} a + \frac{1}{2} d + \frac{1}{4} aa - \frac{1}{4} ad + \frac{1}{4} dd$$

An initial model was established, including only the mean component (m). Then, the additive effects [a], dominance effects [d], pooled digenic epistatic additive x additive effect [aa], pooled digenic epistatic additive x dominance effect [ad], and pooled digenic epistatic dominance x dominance effect [dd] were included in a sequential manner, according to their contribution in order to reduce the residual mean squares, until all unknowns but one were considered (Hallauer and Miranda Filho, 1981). The solution was reached by the least squares method. The adjustment of each model was verified by using a goodness-of-fit test proposed by Cavalli (1952), which consists of a chi-square for the residual mean square of the six populations available, weighted by the reciprocal of

their variances, and according to the residual degree of freedom. Once an adjustment was met (non-significant χ^2), no other component was added to the model (Zimmermann *et al.*, 1985).

Broad (H^2) and narrow-sense heritabilities (h^2) were calculated from the estimated variances, solving the following system of equations:

$$VP_1 = VE$$

$$VP_2 = VE$$

$$VF_1 = VE$$

$$VF_2 = VA + VD + VE$$

$$VBC_1 + VBC_2 = VA + 2\,VD + 2\,VE$$

where VE is the environmental variance, VA is the additive genetic variance, and VD is the dominant genetic variance. The five-equation system with three unknowns was resolved by the weighted least squares method. The weighting factor was the inverse of the estimated variances of each generation, as proposed by Peternelli (1992). The errors associated with estimated heritabilities were also obtained using the equations of Vello and Vencovsky (1974).

The correlation coefficients between pairs of characters were estimated according to Falconer (1976), using the equations:

$$r_P = \frac{COV_P(X,Y)}{\sqrt{V_P X . V_P Y}}$$

$$r_G = \frac{COV_G(X,Y)}{\sqrt{V_G X . V_G Y}}$$

$$r_E = \frac{COV_E(X,Y)}{\sqrt{V_E X . V_E Y}}$$

where r_P, r_G and r_E are the phenotypic, genotypic, and environmental correlation coefficients, respectively. Accordingly, $COV_P(X,Y)$, $COV_G(X,Y)$, and $COV_E(X,Y)$ are the phenotypic, genotypic and environmental covariances for the characters X and Y, estimated according to Weber and Moorthy (1952). Thus, the phenotypic covariances were estimated between pairs of characters in generation F_2, and the environmental covariances by the arithmetic mean of covariances between pairs of characters of the parental and F_1 generations, and the genotypic covariance by the difference between phenotypic and environmental covariances. V_P, V_G, and V_E are the phenotypic, genotypic and environmental variances of X and Y, respectively, and were calculated as described previously.

RESULTS AND DISCUSSION

The genetic effects were variable, according to the family and the trait considered (Tables I, II and III). The signal of [d] would depend upon the direction of dominance (Mather and Jinks, 1982). The presence of a negative value for [a] is attributed to the fact that the parental named P_1, as well as its respective BC_1 does not always refer to the genotype with the maximum expression of the variable, which in this case would be the parental with the greatest susceptibility to disease.

As dry bean is an autogamous species, the objective of a breeding program is to create pure lines. Thus, only the additive genetic effect which is expressed in homozygous genotypes ([a] and [aa]) is of practical interest to a plant breeder. In the present work, the additive effects, quantified by the [a] component, were present in every case but one, though with variable magnitude and relative importance, according to the cross and/or variable studied. The presence of additive x additive [aa] effects indicates the possibility of transgressive segregants in the later selfed generations, besides being the only epistatic effect, at least theoretically, which could be effectively utilized in a selection process (Zimmermann *et al.*, 1985).

The [aa] effect was observed for the cross 'DOR 303 x Redlands' for the variable 'yellowing' and in all models for the crosses 'Redlands x EMGOPA' and 'EMGOPA x Carnaval' (Table I, II and III). Thus, the greatest frequency of transgressive segregants is expected in the progeny of crosses involving such parentals, which are of divergent genetic origins, these being, Mesoamerican (EMGOPA), Andean (Redlands and Carnaval). Even though DOR 303 was considered to belong to the Andean type by Blair (1992), it resulted from a cross between genotypes of Andean and Mesoamerican origin. Its high degree of resistance to yellowing is attributed to transgressive segregation, since none of the parentals shows this characteristic (Morales and Niessen, 1988). On the other hand, the same type of effect [aa] in crosses in which parentals had a low level of resistance, such as 'EMGOPA x Carnaval' can be of little or no value in breeding programs since the magnitude of the value is small.

For the cross 'Carnaval x Redlands' the model for 'yellowing' was adjusted solely with the [a] component (Table I). However, the magnitude of the [a]

Table I - Estimates of genetic parameters, their standard errors, determination coefficients (r^2), goodness of fit of the models (χ^2 and P), for the yellowing reaction.

Cross	Estimate ± SE	Model	χ^2 (d.f.)	P	r^2
DOR 303 x Redlands	m = 4.685 ± 0.020 a = -2.349 ± 0.006 d = 1.211 ± 0.026 aa = -0.775 ± 0.021 ad = 1.589 ± 0.054	Y = m + a + d + aa + ad	0.0094 (1)	> 0.99	0.9999
Carnaval x DOR 303	m = 4.377 ± 0.037 a = 2.823 ± 0.037 d = 2.117 ± 0.075	Y = m + a + d	1.2363 (3)	> 0.99	0.9996
EMGOPA x DOR 303	m = 3.854 ± 0.028 a = 2.314 ± 0.028 d = 1.622 ± 0.080	Y = m + a + d	0.7380 (3)	0.94	0.8530
Carnaval x Redlands	m = 6.715 ± 0.020 a = 0.472 ± 0.031	Y = m + a	2.5909 (4)	> 0.99	0.9835
Redlands x EMGOPA	m = 6.532 ± 0.070 a = 0.058 ± 0.029 aa = -0.316 ± 0.077 dd = -0.430 ± 0.169	Y = m + a + aa + dd	1.3322 (2)	0.88	0.9197
EMGOPA x Carnaval	m = 7.596 ± 0.359 a = -0.508 ± 0.031 d = -3.591 ± 0.973 aa = -0.915 ± 0.357 dd = 2.745 ± 0.638	Y = m + a + d + aa + dd	0.6965 (1)	0.91	0.9968

(m) = Mean; [a] = additive effect; [aa] = additive x additive effect; [ad] = additive x dominant effect; [d] = dominant effect; [dd] = dominant x dominant effect; (d.f.) = degrees of freedom.

Table II - Estimates of genetic parameters, standard errors, determination coefficients (r^2), goodness of fit of the models (χ^2 and P), for the dwarfing reaction.

Cross	Estimate ± SE	Model	χ^2 (d.f.)	P	r^2
DOR 303 x Redlands	m = 6.663 ± 0.073 a = 1.486 ± 0.080 dd = -2.099 ± 0.192	Y = m + a + dd	1.8980 (3)	> 0.99	0.9914
Carnaval x DOR 303	m = 7.348 ± 0.099 a = -0.408 ± 0.111 dd = -1.260 ± 0.195	Y = m + a + dd	5.0643 (3)	0.98	0.9330
EMGOPA x DOR 303	m = 7.449 ± 0.093 a = -0.510 ± 0.099 dd = -0.900 ± 0.255	Y = m + a + dd	3.3030 (3)	0.97	0.8983
Carnaval x Redlands	m = 5.931 ± 0.069 a = 0.889 ± 0.062 aa = 0.134 ± 0.095	Y = m + a + aa	7.1855 (3)	> 0.99	0.9866
Redlands x EMGOPA	m = 7.196 ± 0.111 a = -0.867 ± 0.051 aa = -1.130 ± 0.125 dd = -1.753 ± 0.211	Y = m + a + aa + dd	3.4986 (2)	> 0.99	0.9945
EMGOPA x Carnaval	m = 7.285 ± 0.066 aa = -0.327 ± 0.072 dd = -0.941 ± 0.119	Y = m + aa + dd	1.6959 (3)	> 0.99	0.9571

Abbreviations as in Table I.

Table III - Estimates of genetic parameters, standard errors, determination coefficients (r^2), goodness of fit of the models (χ^2 and P), for the pod malformation reaction.

Cross	Estimate ± SE	Model	χ^2 (d.f.)	P	r^2
DOR 303 x Redlands	m = 6.956 ± 0.080 a = 1.587 ± 0.095 dd = -0.743 ± 0.165	Y = m + a + dd	2.6773 (3)	> 0.99	0.9899
Carnaval x DOR 303	m = 7.836 ± 0.031 a = -0.777 ± 0.037 dd = -1.601 ± 0.084	Y = m + a + dd	0.4180 (3)	> 0.99	0.9949
EMGOPA x DOR 303	m = 8.269 ± 0.054 a = -0.341 ± 0.061 dd = -1.803 ± 0.181	Y = m + a + dd	0.9257 (3)	> 0.99	0.9710
Carnaval x Redlands	m = 6.196 ± 0.054 a = 0.830 ± 0.054 d = -0.465 ± 0.121	Y = m + a + d	2.4476 (3)	> 0.99	0.9876
Redlands x EMGOPA	m = 9.195 ± 0.041 a = -1.278 ± 0.013 d = -3.305 ± 0.062 aa = -2.575 ± 0.043 ad = 0.913 ± 0.100	Y = m + a + d + aa + ad	0.0390 (1)	0.99	0.9999
EMGOPA x Carnaval	m = 10.827 ± 0.429 a = 0.401 ± 0.052 d = -6.944 ± 1.182 aa = -3.347 ± 0.426 dd = 2.752 ± 0.797	Y = m + a + d + aa + dd	0.6239 (1)	0.90	0.9950

Abbreviations as in Table I.

component was relatively small, probably reflecting the small divergence in the parentals regarding this variable. For the crosses 'Carnaval x DOR 303' and 'EMGOPA x DOR 303', the models were adjusted to include the dominance component besides the additive one, with [a] of higher magnitude and also highly significant. For the other crosses the models were adjusted to include epistatic components, besides [a] and [d] (Table I). As discussed before, [a] effects bring the possibility of obtaining transgressive segregants. This would be a highly desirable situation, mainly in the cross 'DOR 303 x Redlands', for which there was a significant additive gene effect for resistance associated with a low mean value (m) for the parentals. The combination of these factors could lead to the selection of lines highly resistant to 'yellowing'. Generally, genetic effect ([a] and [aa]) controlling resistance to yellowing was evident whenever 'DOR 303' was present. Also, the [a] value of the models was of similar magnitudes, indicating that the gene(s) from DOR 303 which control this trait are expressed in different genetic backgrounds with the same intensity.

For the variable 'plant dwarfing/deformation' (Table II), only the additive [a] and the epistatic dominance x dominance [dd] effects were included in the models for the crosses involving DOR 303. The absolute values for these components were two to three times higher when Redlands was the second parent. The epistatic component had a higher magnitude in every case, however. The negative value for [dd] indicates the epistatic effect in the direction of resistance. Non-fixable effects, shown by a high specific combining ability, were also shown by Morales and Singh (1991), when studying the resistance to stunting caused by a BGMV strain from Guatemala.

DOR 303 had the highest degree of dwarfing after ealy inoculation. The data of Table II suggest that resistance to dwarfing could be easily bred for, through strong early selection in the segregating population. In the case of the cross 'Carnaval x Redlands', only additive [a] and additive x additive [aa] epistatic components were included for a perfect model adjustment (χ^2 non-significant), however, the [aa] value was not significant (estimated value less than twice the value of its standard error). This leads to the conclusion that the model of gene action is similar to that for yellowing.

In all crosses involving DOR 303, the models for gene action for pod malformation were adjusted including the [a] and the [dd] components (Table III), as happened in the case of dwarfing (Table II). For the cross 'Carnaval x Redlands' the model had [a] and [d]

components, but the additive component had a magnitude twice as high as the dominance one.

For most crosses, even though the additive or the interaction additive x additive genetic effect significantly controlled the characteristic, the non-fixable genetic effects (dominance and respective epistatic components) had greater magnitudes.

Several estimates of additive or dominance variances were negative, resulting in negative values for the narrow sense heritability estimates, and therefore invalidating them (Table IV). The experimental errors, and possibly the existence of non-estimated epistatic variance contributed to obtaining negative values. Broad sense (H^2) heritability presented small standard deviations, indicating higher confidence than the narrow sense heritabilities (h^2) with higher standard deviations, even in the cases without negative values. The greatest estimates for H^2, in the case of yellowing, were obtained in crosses involving DOR 303. The values found for h^2 were comparable to those calculated by Morales and Singh (1994), and by Vizgarra (1991) who found 36 ± 13%, and 33 ± 17%, respectively.

In the case of plant dwarfing, taking into account only the cases with positive additive variances, the values found for h^2 were inferior to those found by Blair (1992), ranging from 60 to 87%. In his studies the plants were naturally infected at any age in the field, while in the current research there was a uniform and early inoculation. According to Costa (1975), plant age at inoculation is a fundamental factor affecting the severity of the symptoms. Also, beans are more susceptible in the young seedling stage (Morales and Niessen, 1988).

There were several negative variance estimates for the variable pod malformation. In the case of DOR 303 x Redlands the heritabilities were 72.2 and 23.5% for H^2 and h^2, respectively. Blair (1992) found values ranging from 35 to 85% for h^2. He also found values not significantly differing from zero when the average for the parentals was not as divergent.

Most of the phenotypic, environmental and genotypic correlation coefficients were positive for the pair of variables analyzed (Table V), and the only two negative correlations were non-significant at P = 0.05. The correlation between plant dwarfism and pod malformaton was highly significant in all combinations. The general conclusion is that the selection for improvement of any of the variables involved in resistance to BGMV will not negatively affect selection for another variable. Negative associations for dwarfing x yellowing and dwarfing x pod malformation were detected by Blair (1992) in certain crosses involving DOR 303, in the Dominican Republic. The contrasting results obtained in these two cases may be due to the use of different genotypes as well as different viral strains. More detailed work may be needed to clarify these differences.

This investigation led to interesting general conclusions. Considerable additive genetic effects were observed for the variables analyzed. However, in most cases, the non-additive effects had higher absolute values. Resistance to yellowing conferred by DOR 303 was highly heritable and expressed in different genetic backgrounds. It may be oligogenic. Generally, all of the evaluated variables were positively correlated, indicating that they can be simultaneously selected. However, the phenotypic and genotypic correlation coefficients were not high enough to guarantee the exclusive use of indirect selection in a breeding program.

Table IV - Broad- (H^2) and narrow-sense (h^2) heritabilities for three characters associated with resistance to bean golden mosaic virus.

Character	Leaf yellowing		Dwarfing		Pod malformation	
Cross	H^2 ± SD	h^2 ± SD	H^2 ± SD	h^2 ± SD	H^2 ± SD	h^2 ± SD
DOR 303 x Redlands	88.14 ± 5.48	-42.45 ± 38.75[1]	73.75 ± 10.87	40.97 ± 23.77	72.24 ± 9.13	23.49 ± 26.65
Carnaval x DOR 303	89.80 ± 5.67	44.34 ± 26.90	81.88 ± 12.43	56.74 ± 21.52	45.20 ± 11.08	68.69 ± 18.26[2]
EMGOPA x DOR 303	93.37 ± 6.02	66.40 ± 22.20	83.27 ± 13.48	-37.94 ± 35.81[1]	44.31 ± 13.90	50.59 ± 21.74[2]
Carnaval x Redlands	40.04 ± 8.05	-19.97 ± 30.40[1]	61.98 ± 7.21	-47.73 ± 33.29[1]	46.96 ± 8.29	47.29 ± 20.50[2]
Redlands x EMGOPA	68.78 ± 8.11	110.62 ± 11.94[2]	74.31 ± 6.48	53.81 ± 19.57	58.99 ± 7.40	-35.89 ± 31.90[1]
EMGOPA x Carnaval	71.95 ± 6.95	10.03 ± 25.81	79.52 ± 6.15	43.26 ± 22.08	29.87 ± 9.41	-24.49 ± 31.55[1]

[1]Additive genetic variance estimate was negative.
[2]Dominant genetic variance estimate was negative.

Table V - Phenotypic, environmental, and genotypic correlation coefficients for the three characters associated with resistance to bean golden mosaic virus.

Cross		Yellowing x dwarfing	Yellowing x pod malformation	Dwarfing x pod malformation
DOR 303	r_P	0.16198**	0.09058	0.64865**
x	r_E	0.66744	0.35780	0.55685
Redlands	r_G	0.05483	0.03214	0.68445
Carnaval	r_P	0.14103*	-0.04206	0.48411**
x	r_E	0.80362	0.39305	0.62385
DOR 303	r_G	0.03742	-0.18314	0.47265
EMGOPA	r_P	-0.10439	0.22590**	0.47294**
x	r_E	1.00980	0.26222	0.77066
DOR 303	r_G	-0.23376	0.27198	0.40059
Carnaval	r_P	0.59449**	0.47801**	0.48192**
x	r_E	0.33220	0.20748	0.45216
Redlands	r_G	0.87482	0.83240	0.51690
Redlands	r_P	0.36179**	0.28105**	0.50662**
x	r_E	0.54897	0.10647	0.39578
EMGOPA	r_G	0.28861	0.34601	0.57116
EMGOPA	r_P	0.24175**	0.28564**	0.61790**
x	r_E	0.39930	0.39661	0.46187
Carnaval	r_G	0.19309	0.23675	0.90873

*, **Significant at the 5% and 1% levels, respectively, based on *t*-test.

Broad sense heritability estimates were high in most cases. On the other hand, the narrow sense heritabilities were associated with high standard deviation values, which indicates that the number of plants evaluated in the BC generations was insufficient. Several estimates of variances and narrow sense heritabilities were negative. This suggests that number of generations (higher number of generations would make possible the inclusion of a larger number of genetic effects in the models), and in some cases number of plants per generation, was insufficient to estimate the parameters.

RESUMO

O mosaico dourado é a principal doença virótica da cultura do feijoeiro (*Phaseolus vulgaris* L.) na América Latina. A genética da resistência ao vírus do mosaico dourado do feijoeiro (BGMV), isolado do Brasil, foi conduzida em um dialelo 4 x 4 sem os recíprocos, entre os parentais DOR 303, EMGOPA 201 Ouro, Carnaval e Redlands Greenleaf C. Plântulas com oito dias de idade, dos pais, híbridos F_1, retrocruzamentos e geração F_2, foram inoculadas através da exposição a uma população virulífera de moscas brancas (*Bemisia tabaci* Genn.) por 24 h, antes do transplantio para condição de campo. O grupo completo de dois parentais, F_1, F_2 e respectivos retrocruzamentos foi considerado uma família. Foram coletados e analisados os dados de amarelecimento foliar, nanismo e de deformação de vagens, usando o delineamento experimental de blocos ao acaso com três repetições.

Análise ponderada de médias de gerações foi executada para cada uma das seis famílias. Foram detectados efeitos gênicos aditivos significantes controlando todos os caracteres avaliados. Por outro lado, na maioria dos casos, houve predominância da ação gênica não aditiva. A resistência ao amarelecimento foliar, conferida por genes de DOR 303, foi altamente herdável e se expressou igualmente bem nos diferentes 'backgrounds' genéticos avaliados. Ela pode ser oligogênica. Estimativas das herdabilidades no sentido amplo e restrito foram relativamente altas para as três características. O maior coeficiente de correlação foi obtido entre nanismo e deformação de vagens. As três características estudadas foram todas positivamente correlacionadas, indicando que podem ser selecionadas simultaneamente.

REFERENCES

Alberini, J.L. (1982). Avanços no melhoramento do feijoeiro (*Phaseolus vulgaris* L.) para resistência ao mosaico dourado e "superbrotamento". In: *Reunião Nacional de Pesquisa de Feijão, 1. Anais da RENAFE*. EMBRAPA-CNPAF, Goiânia, pp. 49-52.

Beebe, S.E. and **Corrales, M.P.** (1991). Breeding for disease resistance. In: *Common Beans Research for Crop Improvement* (Schoonhoven, A. van. and Voysest, O., eds.). Centro Internacional de Agricultura Tropical, Cali, pp. 561-618.

Blair, M.W. (1992). Heritability of field resistance to bean golden mosaic virus and the sweetpotato whitefly (*Bemisia tabaci* Genn.) in dry bean (*Phaseolus vulgaris* L.). Doctoral thesis, University of Puerto Rico, Puerto Rico.

Brown, J.K. and **Bird, J.** (1992). Whitefly-transmitted geminiviruses and associated disorders in the Americas and the Caribbean Basin. *Plant Dis. 76*: 220-225.

Cavalli, L.L. (1952). An analysis of linkage in quantitative inheritance. In: *Quantitative Inheritance* (Rieve, E.C. and Waddington, C.H., eds.). HMSO, London, pp. 135-144.

Costa, A.S. (1965). Three whitefly-transmitted virus diseases of beans in São Paulo, Brazil. *FAO Plant Protection Bulletin 13*: 3-12.

Costa, A.S. (1975). Increase in the population density of *Bemisia tabaci*, a threat of widespread virus infection of legume crops in Brazil. In: *Tropical Disease of Legumes* (Bird, J. and Maramorosch, K., eds.). Academic Press, New York, pp. 27-49.

Costa, A.S. (1976). Whitefly-transmitted plant diseases. *Annu. Rev. Phytopathol. 14*: 429-449.

Costa, A.S., Costa, C.L. and **Sauer, H.F.G.** (1973). Surto da mosca branca em culturas do Paraná e São Paulo. *Anais da Sociedade Entomológica do Brasil*, 2: 20-30.

De Fazio, G. (1985). O mosaico dourado do feijoeiro no Brasil. *Fitopatol. Bras. 10*: 41-48.

Falconer, D.S. (1976). *Introduction to Quantitative Genetics*. 2nd edn. Longman, London, pp. 279.

Faria, J.C., Zimmermann, M.J.O. and **Yokoyama, M.** (1991). Development of *Phaseolus vulgaris* L. tolerant to bean golden mosaic virus. *Annual Report of the Bean Improvement Cooperative 34*: 11-12.

Faria, J.C., Gilbertson, R.L., Hanson, S.F., Morales, F.J., Ahlquist, P., Loniello, A.O. and **Maxwell, D.P.** (1994). Bean golden mosaic geminivirus type II isolates from the Dominican Republic and Guatemala: Nucleotide sequence, infectious pseudorecombinants, and phylogenetic relationships. *Phytopathol. 84*: 321-329.

Figueira, A.R. (1980). Estudos realizados com o vírus do mosaico dourado do feijoeiro do Brasil visando sua transmissão por métodos mecânicos. Master's thesis, UNICAMP, Campinas, SP.

Gálvez, G.E. and **Castaño, M.J.** (1976). Purification of the whitefly-transmitted bean golden mosaic virus. *Turrialba 26*: 205-207.

Gilbertson, R.L., Hidayat, S.H., Martinez, R.T., Leong, S.A., Faria, J.C., Morales, F. and **Maxwell, D.P.** (1991a). Differentiation of bean-infecting geminiviruses by nucleic acid hybridization probes and aspects of bean golden mosaic in Brazil. *Plant Disease 75*: 336-342.

Gilbertson, R.L., Faria, J.C., Hanson, S.F., Morales, F.J., Ahlquist, P.G., Maxwell, D.P. and **Russell, D.R.** (1991b). Cloning of the complete DNA genomes of four bean-infecting geminiviruses and determining their infectivity by electric discharge particle acceleration. *Phytopathology 81*: 980-985.

Gilbertson, R.L., Faria, J.C., Ahlquist, P. and **Maxwell, D.P.** (1993). Genetic diversity in geminiviruses causing bean golden mosaic disease: The nucleotide sequence of the infectious cloned DNA components of a Brazilian isolate of bean golden mosaic geminivirus. *Phytopathology 83*: 709-715.

Goodman, R.M. (1977). Infectious DNA from a whitefly transmitted virus of *Phaseolus vulgaris*. *Nature*: 266 (No. 5597): 54-55.

Hallauer, A.R. and **Miranda Filho, J.B.** (1981). *Quantitative Genetics in Maize Breeding*. Iowa State University Press, Ames, pp. 468.

Hohmann, C.L. and **Carvalho, S.M.** (1982). Avaliação de cultivares de feijoeiro (*Phaseolus vulgaris* L., 1753) quanto à resistência à mosca branca, *Bemisia tabaci* (Gennadius 1889) e incidência do mosaico dourado. In: *Reunião Nacional de Pesquisa de Feijão*, 1. *Anais*. EMBRAPA-CNPAF, Goiânia, pp. 23-24.

Mather, S.K. and **Jinks, J.L.** (1982). *Biometrical Genetics*. University Press, Cambridge, pp. 396.

Morales, F.J. and **Niessen, A.I.** (1988). Comparative responses of selected *Phaseolus vulgaris* germ plasm inoculated artificially and naturally with Bean Golden Mosaic Virus in *Phaseolus vulgaris*. *Plant Dis. 72*: 1020-1023.

Morales, F.J. and **Singh, S.P.** (1991). Genetics of resistance to bean golden mosaic virus in *Phaseolus vulgaris* L. *Euphytica 52*: 113-117.

Morales, F.J. and **Singh, S.P.** (1994). Mejoramento por la resistencia al virus del mosaico dorado del frijol (BGMV) en problaciones inter-raciales de *Phaseolus vulgaris* L. In: *Bean Golden Mosaic: Research Advances* (Morales, F.J., eds.). Centro Internacional de Agricultural Tropical, Cali, pp. 156-159.

Pereira, O.A.P., Balmer, E. and **Miranda Filho, J.B.** (1989). Inheritance of resistance to stalk rot caused by *Colletotrichum graminicola* (CES) WILS, in maize. *Rev. Bras. Genet. 12*: 53-65.

Peternelli, L.A. (1992). Herdabilidades, ganhos devidos à seleção e correlações do rendimento de feijão com seus componentes primários, no monocultivo e no consórcio com milho. Master's thesis, Universidade Federal de Viçosa, Viçosa, MG.

Pompeu, A.S. and **Kranz, W.M.** (1977). Linhagens de feijoeiro (*Phaseolus vulgaris* L.) resistentes ao vírus do mosaico dourado. *Summa Phytopathol. 3*: 162-163.

Singh, S.P., Gepts, P. and **Debouck, D.G.** (1991). Races of common bean (*Phaseolus vulgaris*, Fabaceae). *Econ. Bot. 45*: 379-396.

Vello, N.A. and **Vencovsky, R.** (1974). Erros associados às estimativas de variâncias e coeficientes de herdabilidades. Relatório Científico do Instituto de Genética, 8. ESALQ, Piracicaba, pp. 238-348.

Vizgarra, O.N. (1991). Capacidade de combinação de algumas cultivares de feijão (*Phaseolus vulgaris*) com diferentes mecanismos de resistência ao vírus do mosaico dourado. Master's thesis, ESAL, Lavras, MG.

Weber, C.R. and **Moorthy, B.R.** (1952). Heritable and nonheritable relationships and variability of oil content and agronomic characters in the F_2 generations of soybean crosses. *Agron. J. 44*: 202-209.

Zimmermann, M.J. de O., Rosielle, A., Foster, K.W. and **Waines, J.G.** (1985). Gene action for grain yield and harvest index of common bean grown as sole crop and in intercrop with maize. *Field Crops Res. 12*: 319-329.

(Received February 2, 1996)