Inheritance of Resistance to Races 69 and 453 of *Colletotrichum lindemuthianum* in the Common Bean

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ABSTRACT

The cultivars, AB 136 and G 2333 both resistant to Colletotrichum lindemuthianum *races 69 and 453, were crossed with the cultivars Michelite and Perry Marrow (susceptible to both races), with Dark Red Kidney and Cornell 49242 (resistant to both races) and F_1 and F_2 generations were obtained. Plants were inoculated using a spore suspension at 1.2 x 10^6 concentration. The reaction of F_1 and F_2 populations showed that Dark Red Kidney, Cornell 49242 and AB 136 cultivars had the dominant genes A (Co-1), Are (Co-2) and Co-6, respectively, was conferring resistance to races 69 and 453. The segregation data obtained from F_2 populations indicated that G 2333 carried two dominant resistance genes Co-5 gene and another one Co-7 for 69 and 453 races. The dominant genes in G 2333 and its resistance to C. lindemuthianum race could be transferred to provide anthracnose resistance to susceptible cultivars relatively easy.*

Key Words: *Phaseolus vulgaris, Colletotrichum lindemuthianum,* anthracnose, resistance

INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) anthracnose, caused by the *Colletotrichum lindemuthianum* (Sacc. et Magn.) Scrib. fungus is one of the most damaging seed-borne diseases. The use of resistant cultivars is considered to be the most efficient and economic control method for the disease. It is widely used in Brazil where a considerable proportion of bean producers are small farmers who use low technology level, including the use of their own seeds, no crop rotation and no fungicide treatment. Sources of resistance for the different races have been sought in breeding programs (Paradela Filho *et al.*, 1981; Menezes and Dianese, 1988), but the development of resistant cultivars is hindered because there are various physiological races of the agent which causes the disease. Later studies concluded that the genetic base for resistance to anthracnose in common bean varied from simple to complex, depending on the varieties involved and the physiological races considered (Del Peloso *et al.*, 1989). There are some sources of resistance such as Dark Red Kidney, Cornell 49242, Mexico 222, TO, TU, AB 136 and G 2333, which carry the genes *A* (*Co-1*), *Are* (*Co-2*), *Mexique 1* (*Co-3*), *Mexique 2* (*Co-4*), *Mexique 3* (*Co-5*), *Q* (*Co-6*) and *Co-5, Co-7*, respectively.

McRostie (1919) reported the first gene (named *A*) for resistance to anthracnose, present in the Dark Red Kidney variety and expressing resistance to the alpha race. Mastenbroek (1960) found a dominant resistant gene in the Venezuelan variety Cornell 49242 and several breeding programs have been based on this gene. The *Are* (*Co-2*) gene gives resistance to alpha, beta, gama, delta, epsilon and lambda races. The kappa, iota and alpha-brazil races, however, break this resistance (Fouilloux, 1979; Menezes, 1985). Three dominant resistant *Mexique* genes were identified by Fouilloux (1979) in a collection of Mexican germplasm in Europe. Allelism tests showed that these genes segregate independently amongst each other and in relation to the *Are* (*Co-2*) gene. Vieira (1983) reported that the use of the *Are* (*Co-2*) gene in breeding studies was successful, but led to a dangerous situation because this resistance is

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conferred by a single gene and could be easily broken by the appearance of a new race.

The dominant resistance gene present in the AB 136 variety was first described by Schwartz *et al.* (1982). Inheritance studies showed that only the *Q* (*Co-6*) gene, which was independent of others previously characterized, was present in this cultivar (Gonçalves-Vidigal 1994; Gonçalves-Vidigal *et al.*, 1997; Young and Kelly, 1996a). Pastor-Corrales *et al.* (1994) showed that only the G 2333 line was resistant to 380 isolates of *C. lindemuthianum.* This line was resistant to all the Brazilian isolates and all the European and North American races (Balardin and Pastor-Corrales, 1990; Balardin *et al.*, 1990; CIAT, 1990; Pastor-Corrales and Tu, 1989). Thus, line G 2333, which has wide adaptation, high seed yield in many environments and tolerance to low fertility soils, can positively contribute to breeding programs, especially those for areas where *C. lindemuthianum* is highly variable. Resistance in G 2333 is controlled by two independent dominant genes with equivalent effects (Pastor-Corrales *et al.* 1994). Young and Kelly (1996b) showed that G 2333 also carries the *Co-5* gene, which was not detected by the Pastor-Corrales *et al.* (1994) since race 521 is virulent to the *Co-5* gene. According to Young *et al.* (1998), the presence of more than one gene in G 2333 may account for its broad resistance to all known races of *C. lindemuthianum* (Balardin *et al.* 1997; Pastor-Corrales *et al.* 1994; Schwartz *et al.* 1982). If resistance in G 2333 is conditioned by three resistance genes (Young and Kelly, 1996b), one of which has been previously described (*Co-5*), the two other genes need to be characterized, one of them, named Co-7, in this present study and according to Young *et al.* (1998). In view of this, the inheritance of common bean resistance to the physiological races *69* and *453* of *C. lindemuthianum* (Sacc. and Magn.) was studied in crosses using the AB 136 and G 2333 cultivars.

MATERIAL AND METHODS

Plant Material: The seeds of the cultivars were supplied by Embrapa-CNPAF (Brazilian Agricultural Research Corporation - National Research Center for Bean and Rice at Goiania). The crosses were obtained in March, 1997, with the related avaliations being conducted in the same year in a green house and at a phytopatolgy laboratory of the Universidade Estadual de Maringá, respectively. Table 1 shows the characterization of the cultivars. Nine F_1 hybrids derived from a partial diallel among the AB 136 and G 2333 lines and the Michelite, Perry Marrow, Dark Red Kidney and Cornell 49242 cultivars were obtained. The F_1 plants were left in the greenhouse to obtain the F_2 generation. Fifteen days old seedlings of the parents and F_1 and F_2 generations were tested and assessed for their resistance/susceptibility reaction to the physiological races *69* and *453* of *C. lindemuthianum.*

Preparation of *C. lindemuthianum* isolates: The physiological races *69* and *453* were supplied by the fungi collection at the Federal University of Viçosa and Embrapa-CNPAF. These races were chosen because of their most frequent presence in Paraná State but more information on the inheritance of resistance is needed. The numeric designation of each race, followed the binary system proposed by Habgood's specifications (1970) in the case of *C. lindemuthianum*, 12 differentiating varieties were recommended by the Centro International de Agricultural Tropical (CIAT) in 1990. Each variety received a value, 2^n, where 2 was the number of classes of reactions considered (resistant or susceptible) and n was the function of the order of the differentiators. The resistance reaction or susceptibility shown by the varieties received values zero and one, respectively.

Monosporic cultures of each *Colletotrichum lindemuthianum* race were transferred to test tubes containing Mathur *et al.* (1950) culture medium. They were incubated at 22°C for eight to ten days. After sporulation, the pathogen cultures were kept in a refrigerator at 5°C and used as a culture stock for the later experiments. The isolates were inoculated in the set of 12 differential cultivars for anthracnose to confirm their phenotypes (Pastor-Corrales, 1988).

Table 1 - Common bean (*Phaseolus vulgaris* L.) cultivars evaluated for resistance to races 69 and 453 of Colletotrichum lindemunthianum in 1997

Cultivar	Phenotype	Growth habit [a]
Dark Red Kidney	Resistant to races 69 and 453	I
Perry Marrow	Susceptible to race 69 and 453	II
Michelite	Susceptible to race 69 and 453	III
Cornell 49242	Resistant to races 69 and 453	III
AB 136	Resistant to races 69 and 453	IV
G 2333	Resistant to races 69 and 453	IV

[a] I = determinate; II = indeterminate, erect bush; III = indeterminate, weak-stemmed, semiclimber; IV = indeterminate, weak-stemmed, climber (Singh, 1982).

Resistance tests: Plants with their first trifoliate leaf completely developed (15th day) old were transferred to a humid chamber at approximately 22° ± 2°C. Inoculation of the parents and the F_1 and F_2 generations of each one of the nine crosses was carried out separately for each race to prevent contamination. Spore suspensions were prepared by flooding 14-day-old fungus cultures with distilled water. This process was carried out using a brush previously moistened in a spore suspension, at 1.2 x 10^6 concentration, using an adaptation of the method used by Cárdenas *et al.* (1964). The seedlings were kept in the same chamber for 96 hours after inoculation, at 20° ± 2°C, controlled light (12 hours with 689 lux illumination alternated with 12 hours of darkness) and approximately 100% relative humidity. Four replications of the parents and their F_1 and F_2 generation from the 10 crosses were evaluated for each physiological race.

The first trifoliate leaves of individual plants were scored on a 1-5 scale to assess the symptoms induced by the physiological races. Plants scored as 1 and 2 were considered resistant (R) and scored as 3-5, susceptible (S). This system was used by Cárdenas *et al.* (1964), Muhalet *et al.* (1981), Fukuda (1982), Del Peloso *et al.* (1989) and Gonçalves-Vidigal (1994).

The chi-square test was used to compare the fit goodness of the observed distribution to the expected ratios. This statistic was calculated by using Genes Program (Cruz, 1997), which offered the probabilities for a chi-square distribution in each cross.

RESULTS AND DISCUSSION

Race 69: Four of the nine crosses belonged to the R x S combination and five to the R x R combination. All F_1 plants behaved as resistant, indicating that resistance was dominant (Table 2). In the case of R x S combination, two crosses had the segregation ratio of 3R:1S in the F_2 generation, indicating that a single dominant gene controled the resistance reaction in each cross. When the AB 136 resistant cultivar was crossed with the susceptible Michelite and Perry Marrow cultivars, the ratio of 3R:1S was observed, showing the action of the *Q* (*Co-6*) gene, present in AB 136 and reported by Gonçalves-Vidigal *et al.* (1993). Resistance in AB 136 cultivar was described by Schwartz *et al.* (1982). This gene has been reported as being independent from all the others previously characterized. Young and Kelly (1996b) confirmed the presence of a single dominant gene in the Catrachita genotype, which is the same of AB 136.

Table 2 - Cross, number of plants evaluated and expected phenotypic ratios in the F_2 population for resistance to race *69* of *Colletotrichum lindemuthianum* [(b)].

Cross		F_1	Number of Plants R	Number of Plants S	Expected ratio R:S	X^2	Probability
AB 136 x Michelite	(R x S)	R	172	59	3:1	0.0360	0.85
AB 136 x Perry Marrow	(R x S)	R	158	44	3:1	1.1155	0.30
AB 136 x Dark Red Kidney	(R x R)	R	266	19	15:1	0.0844	0.77
AB 136 x Cornell 49242	(R x R)	R	105	8	15:1	0.1327	0.72
G 2333 x Michelite	(Rx S)	R	241	17	15:1	0.0506	0.82
G 2333 x Perry Marrow	(Rx S)	R	206	15	15:1	0.1088	0.74
G 2333 x Dark Red Kidney	(R x R)	R	229	4	63:1	0.0360	0.85
G 2333 x Cornell 49242	(R x R)	R	238	4	63:1	0.0128	0.91
G 2333 x AB 136	(R x R)	R	236	4	63:1	0.0169	0.90

[b] R = Resistance; S = Susceptibility

The data obtained in the G 2333 x Michelite and G 2333 x Perry Marrow crosses fitted the ratio of 15R:1S explained by the segregation of the two dominant and independent genes for resistance with equivalent effects, present in the G 2333 variety (Pastor-Corrales *et al.*, 1994). One of these genes was denominated *Co-5* following latest gene nomenclature proposing symbols for anthracnose resistance genes (Kelly and Young, 1996), whereas the second gene was assigned the temporary name *Co-7* until a complete characterization with other known resistance genes could be conducted (Young *et al.*, 1998). The segregation data obtained from five different F_2 populations indicated that G 2333 carried two dominant resistance genes for 69 and 453 races, *Co-5* gene and another one *Co-7*; Co for *Colletotrichum* and *7* because it was the seventh greatest resistance gene to anthracnose characterized and reported in the literature. Within the crosses involving two resistant cultivars (R x R), two fitted the ratio 15R:1S and three fitted the ratio of 63R:1S.

The ratio 15R:1S obtained in the F_2 generation of the AB 136 x Dark Red Kidney cross indicated that the resistance in these crosses was explained by the segregation of the *A* (*Co-1*) in Dark Red Kidney, and *Q* (*Co-6*) in AB 136 genes. As previously stated, all these genes were independent amongst each other, and any one of them might give resistance in the dominant condition. The 15R:1S segregation, involving the crosses AB 136 x Cornell 49242 and AB 136 x Dark Red Kidney indicated that a single dominant gene gave resistance in AB 136 *Q* (*Co-6*), which segregated independently from the *Are* (*Co-2*) and *A* (*Co-1*) genes present in Cornell 49242 and in Dark Red Kidney, respectively.

When the G 2333 cultivar was crossed with the Dark Red Kidney, Cornell 49242, and AB 136, the data from the F_2 generation fitted the 63R:1S ratio, indicating that resistance was controlled by the action of three dominant genes. In the first cross, the action of the *A* (*Co-1*) gene from Dark Red Kidney and of the *Co-5* (Kelly and Young, 1996) and *Co-7* genes from G 2333 became evident. In the cross G 2333 x Cornell 49242 the action of the *Co-5* and *Co-7* genes from G 2333 and of *Are* (*Co-2*) from Cornell 49242 was observed. Regarding the cross G 2333 x AB 136, where AB 136 cultivar carried the *Q* (*Co-6*) gene, it was observed that they gave resistance when placed together in a single genotype with the independent and dominant genes from G 2333.

Race 453: The data from the F_2 generation from the nine crosses showed that four belonged to the R x S combination and five to the R x R combination. All the F_1 plants behaved as resistant, indicating that resistance was dominant (Table 3). In the case of the R x S, two crosses showed the ratio of 3R:1S in the F_2 generation, indicating that a single dominant gene controlled

the resistance reaction. The results from the AB 136 x Michelite and AB 136 x Perry Marrow crosses fitted the 3R:1S ratio, showing that the action of the *Q* (*Co-6*) gene present in the first cultivar was independent and dominant, as also shown by Gonçalves-Vidigal (1994). The gene from the AB 136 cultivar which provides dominant resistance to alpha, delta, and kappa (and other) races was named Co-6 by Young and Kelly (1996b).

When the susceptible varieties Michelite and Perry Marrow were crossed with the resistant line G 2333, all the three crosses fitted the 15R:1S ratio. This demonstrated that the resistant variety had two independent dominant genes with equivalent effects, as quoted by Pastor-Corrales *et al*. (1994). The first was named Co-5 according to the latest nomenclature suggested by Kelly and Young (1996) and the other one was named Co-7 in this study, following the same designation of Young *et al*. (1998).

Table 3 - Cross, number of plants evaluated and expected phenotypic ratios in the F_2 population for resistance to race 453 of *Colletotrichum lindemuthianum* [(b)].

Cross		F_1	Number of plants		Expected ratio	X^2	Probability
			R	S	R:S		
AB 136 x Michelite	(R x S)	R	170	52	3:1	0.2942	0.59
AB 136 x Perry Marrow	(R x S)	R	167	60	3:1	0.2481	0.62
AB 136 x Dark Red Kidney	(R x R)	R	137	10	15:1	0.0766	0.78
AB 136 x Cornell 49242	(R x R)	R	222	17	15:1	0.3037	0.58
G 2333 x Michelite	(Rx S)	R	215	16	15:1	0.1803	0.67
G 2333 x Perry Marrow	(Rx S)	R	219	17	15:1	0.3661	0.54
G 2333 x Dark Red Kidney	(R x R)	R	214	4	63:1	0.1051	0.74
G 2333 x Cornell 49242	(R x R)	R	210	4	63:1	0.1308	0.72
G 2333 x AB 136	(R x R)	R	232	4	63:1	0.0269	0.87

[b] R = Resistance; S = Susceptibility

The segregation in the crosses AB 136 x Dark Red Kidney and AB 136 x Cornell 49242 fitted the ratio 15R:1S. These could be explained by the action of the independent dominant *A* (*Co-1*) genes, from Dark Red Kidney, *Are* (*Co-2*) from Cornell 49242 and *Q* (*Co-6)* from AB 136.The data obtained in the F_2 generation from crosses G 2333 x Dark Red Kidney, G 2333 x Cornell 49242 and G 2333 x AB 136 fitted the 63R:1S ratio, showing that there was joint segregation of the *A* (*Co-1*), *Are* (*Co-2*) and *Q* (*Co-6*) genes. The resistance present in G 2333 was controlled by two independent and dominant genes, named *Co-5* and *Co-7*. In each one of these crosses, the resistance genes were located in independent loci and any one of them could give full resistance, even in the presence of the recessive allele of the other two, behaving, therefore, as triplicate dominance resistance factors.

The allele tests carried out for race *69* and for race *453* indicated that all the genes present in the resistant cultivars were independent from each other. The *Co-5* and *Co-7* genes, present in G 2333 behaved as dominant resistant genes with equivalent effects, independent from those present in the other cultivars. Dominant genes in G 2333 and its resistance to *C. lindemuthianum* races, could transferred anthracnose resistance with relative easy to susceptible cultivars.

ACKNOWLEDGMENTS

This research was supported by a grant from Capes/CNPq. The authors would like to express their gratitude to Dr. Aloísio Sartorato for the isolate concerning. We thank the Embrapa-CNPAF for providing seed of differential cultivars.

RESUMO

Os cultivares AB 136 e G 2333 ambos resistentes às raças 69 e 453 de *Colletotrichum lindemuthianum* foram cruzados com os cultivares Michelite e Perry Marrow (suscetíveis à ambas as raças) e com Dark Red Kidney e Cornell 49242 (resistentes à ambas as raças) e, obtidas as gerações F_1 e F_2. As plantas foram inoculadas com uma suspensão de esporos, utilizando-se uma concentração de 1,2 x 10^6 esporos/ml de água. As reações das populações F_1 e F_2 evidenciaram que os cultivares Dark Red Kidney; Cornell 49242 e AB 136 possuem, respectivamente, os genes dominantes *A* (*Co-1*) *Are* (*Co-2*) e *Co-6*, os quais conferiram a resistência às raças 69 e 453. Os dados de segregação obtidos nas populações F_2 indicaram que G 2333 carrega os genes dominantes de resistência *Co-5* e *Co-7*. Os genes dominantes presentes em G 2333 e sua resistência às raças de *C. lindemuthianum*, poderão serem transferidos para cultivares suscetíveis com relativa facilidade.

REFERENCES

Balardin,- R. S.; Pastor-Corrales, M. A. (1990), Reação de germoplasma de *Phaseolus vulgaris* a nove raças de *Colletotrichum lindemuthianum. Fitopatologia Brasileira*, **15**, 269-273.

Balardin, R. S.; Pastor-Corrales, M. A. & Otoya, M. M. (1990), Variabilidade patogênica de *Colletotrichum lindemuthianum* no Estado de Santa Catarina. *Fitopatologia Brasileira*, **15**, 243-245.

Balardin, R. S.; Jarosz, A. M. & Kelly, J. D. (1997), Virulence and molecular diversity in *Colletotrichum lindemuthianum* from South, Central and North America. *Phytopathology*, **87**, 1184-1191.

Cárdenas, F.; Adams, M. W. & Andersen, A. (1964), The genetic system for reaction of field beans (*Phaseolus vulgaris* L.) to infection by three physiologic races of *Colletotrichum lindemuthianum.* Euphytica, 13, 178-186.

Centro Internacional de Agricultura Tropical - CIAT (1990), *Annual Report. Bean Program.* CIAT: Cali, 70-125.

Cruz, C.D. (1997), Programa Genes: aplicativo computacional em genética e estatística. UFV, Viçosa. 442p.

Del Peloso, M. J.; Cardoso, A. A.; Vieira, C.; Saraiva, L. S & Zimmermann, M. J. de O. (1989), Genetic system for the reaction of *Phaseolus vulgaris* to the BA-2 (alpha) race of *Colletotrichum lindemuthianum. Revista Brasileira de Genética*, **12**, 313-318.

Fouilloux, G. (1979), New races of bean anthracnose and consequences on our breding programs. Paper presented at International Symposium on Diseases of Tropical Food Crops, 221-235, Universite Catholique de Louvain, Louvain la Neuve.

Fukuda, W. M. G. (1982), Herança da resistência a três raças fisiológicas de *Colletotrichum lindemuthianum* (Sacc. et Magn.) Scrib. em feijoeiro comum (*Phaseolus vulgaris* L.). Master's thesis, Universidade Federal de Viçosa, Viçosa, MG.

Gonçalves-Vidigal, M. C.; Cardoso, A. A.; Vieira, C.; Zambolim, L.; Saraiva, L. S.; Cruz, C. D. & Vidigal-Filho, P. S. (1993), Resistance inheritance of beans to race kappa of *C. lindemuthianum.* Paper presented at 17th International Congress of Genetics, 119, School of Plant Sciences, Birmingham.

Gonçalves-Vidigal, M. C. (1994), Herança da resistência às raças alfa, delta e capa de *Colletotrichum lindemuthianum* (Sacc. et Magn.) Scrib. no feijoeiro (*Phaseolus vulgaris* L.). Doctor's thesis, Universidade Federal de Viçosa, Viçosa, MG.

Gonçalves-Vidigal, M. C.; Cardoso, A. A.; Vieira, C. & Saraiva, L. S. (1997), Inheritance of anthracnose resistance in common bean genotypes P.I. 207262 and AB 136. *Brazilian Journal of Genetics*, **20**, 59-62.

Habgood, H. (1970), Designation of physiological races of plant pathogens. *Nature*, **227**, 1267-1269.

Kelly, J. D. & Young, R. A. (1996), Proposed symbols for anthracnose resistance genes. *Annual Report of the Bean Improvement Cooperative*, **39**, 20-24.

Mastenbroek, C. (1960), A breeding programme for resistance to anthracnose in dry shell haricot beans, based on a new gene. *Euphytica*, **9**, 177-184.

Mathur, R. S.; Barnett, H. L.; Lilly, V. G. (1950), Sporulation of *Colletotrichum lindemuthianum* in culture. *Phytopathology*, **40**, 104-114.

McRostie, G. P. (1919), Inheritance of anthracnose resistance as indicated by a cross between a resistant and a susceptible bean. *Phytopathology*, **9**, 141-148.

Menezes, J. R. (1985), Variabilidade patogênica de *Colletotrichum lindemuthianum* (Sacc. et Magn.) Scrib. em *Phaseolus vulgaris* L.. Master's thesis, Universidade de Brasília, Brasília, DF.

Menezes, J. R. & Dianese, J. C. (1988), Race characterization of Brazilian isolates of *Colletotrichum lindemuthianum* and detection of resistance to anthracnose in *Phaseolus vulgaris*. *Phytopathology*, **78**, 650-655.

Muhalet, C. S.; Adams, M. W.; Saettler, A. W. & Ghaderi, A. (1981), Genetic system for the reaction of field beans to beta, gamma, and delta races of *Colletotrichum lindemuthianum. J. Amer. Soc. Hort. Sci*., **106**, 601-604.

Paradela Filho, O.; Ito, M. F. & Pompeu, A. S. (1981), Novas raças fisiológicas de *Colletotrichum lindemuthianum* (Sacc. et Magn.) Scrib. *Summa Phytopathologica*, **7**, 20.

Pastor-Corrales, M. A. (1988), Variación patogénica de *Colletotrichum lindemuthianum*, el agente causal de la antracnosis del frijol y una propuesta para su estandarización. In- *La antracnosis del frijol común, Phaseolus vulgaris, en América Latina*, ed. M. A. Pastor-Corrales. CIAT, Cali, pp. 212-239.

Pastor-Corrales, M. A. & TU, J. C. (1989), Anthracnose. In- *Bean production problems in the tropics*, ed. H. F. Schwartz & M. A. Pastor-Corrales. CIAT, Cali, pp. 77-104.

Pastor-Corrales, M. A.; Erazo, O. A.; Estreda, E. I. & Singh, S. P. (1994), Inheritance of anthracnose resistance in common bean accession G 2333. *Plant Disease*, **78**, 959-962.

Schwartz, H. F.; Pastor-Corrales, M. A & Singh, S. P. (1982), New sources of resistance to anthracnose and angular leaf spot of beans (*Phaseolus vulgaris* L.). *Euphytica*, **31**, 741-754.

Vieira, C. (1983), Doenças e pragas do feijoeiro. Imprensa Universitária, Viçosa. 231p.

Young, R. A. & Kelly, J. D. (1996a), RAPD markers flanking the Are gene for anthracnose resistance in common bean. *J. Amer. Soc. Hort. Sci*., **121**, 37-41.

Young, R. A. & Kelly, J. D. (1996b), Characterization of the genetic resistance to *Colletotrichum lindemuthianum* in common bean differential cultivars. *Plant Disease*, **80**, 650-654.

Young, R. A.; Melotto, M.; Nodari, R. O. & Kelly, J. D. (1998), Marker - assisted dissection of the oligogenic anthracnose resistance in the common bean cultivar, "G 2333". Theor. Appl. Genet., **96**, 87-94.

Received: June 28, 1999;
Revised: June 06, 2000;
Accepted: July 25, 2000.