Increased Disease Severity Induced by Some Comoviruses in Bean Genotypes Possessing Monogenic Dominant Resistance to Bean Common Mosaic Potyvirus

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ABSTRACT

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Three comoviruses isolated from diseased bean plants in Brazil (CV-BZ), Venezuela (CV-VZ), and Honduras (CV-HD) were inoculated to 53 bean genotypes possessing or lacking the dominant alleles of the necrosis (*I*) gene, which conditions resistance to bean common mosaic virus (BCMV). These comoviruses systemically infected all bean genotypes tested, predominantly inducing mosaic and plant malformation. Symptom expression was considerably enhanced in bean cultivars possessing the dominant *I* gene, particularly when infected by CV-VZ and CV-HD. The latter comovirus also induced necrotic symptoms in dominant *I* gene bean cultivars, regardless of the absence or presence of specific recessive genes, which protect the *I* gene against necrotic BCMV strains. The test comoviruses also induced top necrosis in two bean genotypes, Arbolito Retinto and Honduras 46, which do not possess the dominant alleles of the necrosis gene. The implications of these findings are important considering that most of the improved bean cultivars grown in Latin America, the United States, and Europe possess monogenic dominant resistance to BCMV and that some of the chrysomelid vectors of these comoviruses also are present in temperate countries.

Various comoviruses are natural pathogens of the common bean (*Phaseolus vulgaris* L.) in North, Central, and South America. The main comoviruses reported to date include bean rugose mosaic virus (BRMV), bean curly dwarf mosaic virus (BCDMV), and cowpea severe mosaic virus (CpSMV) in Latin America and bean pod mottle virus (BPMV) in the United States (5,11-13). Each of these comoviruses belongs to a different but related serogroup (1,10). The endemic nature of comoviruses in common bean-producing regions of Central and South America probably is related to the traditional association of bean and maize plantings because the latter species also supports large populations of some of the main beetle vectors of comoviruses (2).

Since 1985, severe epidemics of comoviruses have taken place in important Central American (El Salvador, Guatemala, and Honduras) and South American (Venezuela) bean-growing regions. Moreover, we reported in 1985 (3) that disease severity was greater in improved bean cultivars possessing monogenic dominant resistance to BCMV (7-9). Thus, this investigation was conducted to confirm the above observation, considering the wide distribution of bean cultivars carrying the dominant necrosis gene (*I*), in North, Central, and South America. The genetics of resistance to BCMV have been summarized recently by Drijfhout (8).

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MATERIALS AND METHODS

Virus isolates, maintenance, and transmission tests. The viruses used in this study were isolated from symptomatic leaf samples collected in bean fields in the States of Aragua (Venezuela) and Goias (Brazil) and in the Province of Francisco Morazán (Honduras). These virus isolates will be referred to as the Venezuelan (CV-VZ), Brazilian (CV-BZ), and Honduran (CV-HD) isolates. Under field conditions, symptom expression in systemically infected bean plants varied from mosaic and rugosity for CV-BZ and CV-VZ to mosaic, plant malformation, and necrosis for CV-HD. These isolates were maintained in the bean cultivar Bountiful, inside separate insect-proof cages in a glasshouse with a maximum light intensity of 1,100 $\mu E \cdot m^{-2} \cdot s^{-1}$, average temperature of 27 C, and 75% RH. All mechanical inoculation tests were conducted in insect-proof facilities, using extracts from infected plants diluted fivefold in sterile distilled water. The inoculation materials and the test plants were incinerated after their use and final evaluation.

A local isolate of bean southern mosaic virus (BSMV) also was used in this study for comparative purposes.

Test plants. A total of 53 common bean genotypes (six test plants minimum per genotype) were mechanically inoculated as described above, with the three selected virus isolates. The bean genotypes Dubbele Witte, Stringless Green Refugee, Redlands Greenleaf-C, Puregold Wax, Imuna, Redlands Greenleaf-B, Great Northern 123, Sanilac, Michelite 62, Red Mexican 34, Pinto 114, Monroe, Great Northern 31, Red Mexican 35, and IVT 7214 possess the recessive alleles (I^+I^+) of the necrosis gene described for BCMV (7-9). Conversely, cultivars Widusa, Black Turtle Soup, Jubila, Topcrop, Improved Tendergreen, and Amanda possess the dominant alleles (*II*) of the necrosis gene (7). Three bean genotypes, IVT 7233, Orfeo-INIA, and MCM 5001, which possess specific recessive genes (*bc-2*^{2} or *bc-3*) that protect the dominant *I* gene against necrotic strains of BCMV (7), also were tested. All of the above bean cultivars, with the exception of Orfeo-INIA and MCM 5001, are used to differentiate BCMV strains (7-9) and are maintained at the Virology Research Unit of CIAT.

Cultivars Bountiful (G 06374), Commodore (G 06716), Mexico 309 (G 05652), Potomac (G 05692), Tendergreen (G 07476 and 09391), Tenderlong 15 (G 05497), Beka (G 01594, 08666, and 10067), Columbia Pinto (G 11211), Great Northern 59 (G 05050), Guatemala 416 (G 08501), ICA-Tui (G 03717 and 04454), La Vega (G 05728), Plentiful (G 03769 and 09292), Porrillo 1 (G 01741 and 04461), Sensutepeque (G 03090), Surecrop Wax (G 03773), Sutter Pink (G 09384), and Rico 23 (G 02944, 04469, and 03827), previously tested (12) with BCDMV, also were inoculated with the three selected comoviruses. These accessions are maintained at the germ plasm bank of the Genetic Resources Unit of CIAT. As indicated above, some of these cultivars are represented by more than one accession in the germ plasm bank, some of which may be genetically different.

A set of nine cultivars including Alubia-Cerrillos (G 07930), Diacol-Calima (G 01853), Red Kidney (G 06054), ICA-Pijao (G 05773), Porrillo Sintetico (G 04495), Bayo Gordo (G 13707), Cacahuate 72 (G 05481), Arbolito Retinto (G 18247), and Honduras 46 (G 04791) also were included for evaluation with the test viruses to increase the number of different bean genotypes evaluated.

Finally, the bean cultivars Kentucky Wonder (G 00148), Iguaçu (G 04120), Red Mexican (G 05507), Cornell 49-242 (G 05694), and Moruna 80 (G 17703) were tested as reported local lesion assay

genotypes for cowpea severe mosaic and bean southern mosaic viruses (4).

Serological tests. The three virus isolates were serologically assayed by immunosorbent electron microscopy (ISEM) as described by Derrick (6). Antisera to cowpea mosaic virus (CpMV), CpSMV, BRMV, BPMV, and quail pea mosaic virus (QPMV), are maintained at the Virology Research Unit of CIAT. The antisera to BPMV and QPMV were originally obtained from the Department of Plant Pathology, University of Arkansas, and the antisera to CpMV and CpSMV from the Department of Plant Pathology, University of Florida. The antiserum to BRMV was produced at CIAT.

RESULTS

As the data in Table 1 indicate, none of the first group of 24 bean genotypes evaluated was immune to any of the three viruses inoculated. With few exceptions, these viruses induced chlorotic local lesions (Fig. 1A) and, less frequently, ring spots (Fig. 1B) on the inoculated primary leaves. Symptom severity in I^+I^+ genotypes varied from mild to severe mosaic and foliar malformation (Fig. 2), according to the virus isolate. The most virulent virus was CV-HD, followed by CV-VZ and CV-BZ. Symptom severity was clearly greater in bean genotypes possessing monogenic dominant resistance to BCMV, particularly when inoculated with CV-HD or CV-VZ. CV-HD consistently induced necrosis on the leaves and stems of *I* gene cultivars, as previously observed under field conditions in Honduras and other Central American countries. CV-VZ also induced necrosis in cultivars Widusa and Topcrop but not in the rest of the *I* gene cultivars tested. CV-BZ did not induce necrosis in any of the bean genotypes. The presence of BCMV strain-specific (7) recessive genes, such as *bc-2*^{2} (IVT 7233 and Orfeo-INIA) and *bc-3* (MCM 5001), further increased symptom severity (Fig. 3), as determined by the more severe malformation, defoliation, and necrosis observed in plants inoculated with CV-VZ and, particularly, CV-HD.

Table 2 shows the comparative response of 18 bean genotypes to CV-HD and, as previously reported, to BCDMV from El Salvador. In general terms, the results of this evaluation demonstrate that although there are similarities, there are notable differences among the symptoms reported for BCDMV from El Salvador

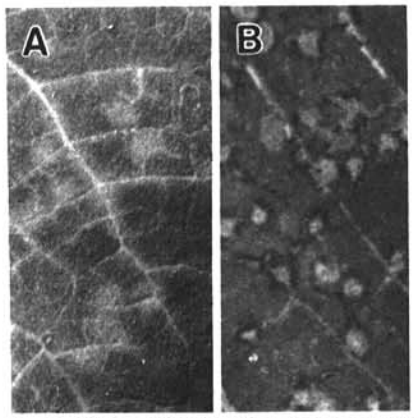

Fig. 1. (A) Local chlorotic lesions and **(B)** ring spots induced on the manually inoculated primary leaves of Topcrop and Red Mexican 35 bean plants, respectively, by a bean comovirus isolated in Venezuela.

Fig. 2. Mosaic and foliar malformation symptoms observed on IVT 7233 bean plants systemically infected by a bean comovirus isolated in Honduras.

Fig. 3. Severe mosaic and plant malformation symptoms expressed by MCM 5001 bean plants inoculated with a bean comovirus from Honduras.

Table 1. Reaction[a] of three groups of common bean cultivars possessing the recessive or dominant alleles of the necrosis gene[b], or the necrosis gene protected against necrotic strains of bean common mosaic virus (BCMV) by specific recessive genes, to the inoculation of selected comovirus isolates from Brazil, Honduras, and Venezuela

Common bean cultivar	BCMV strain-specific recessive genes[c]	Comovirus isolate[d]		
		CV-BZ	CV-VZ	CV-HD
Group 1[e]				
Dubbele Witte	None	cl/ep,m	cl/m	−/ep,m,fm
Stringless Green Refugee	None	cl/m	cl/m,fm	cl/m,fm
Redlands Greenleaf-C	*bc-1*	cl/m	cl/m,fm	−/m,fm
Puregold Wax	*bc-1*	cl/m	cl/m	ep/m
Imuna	*bc-1*	cl/m	cl/m,fm	cl/ep,m
Redlands Greenleaf-B	*bc-1*^{2}	cl/ep,m	cl/m,fm	cl/m,fm
Great Northern 123	*bc-1*^{2}	cl/mm	cl/mm	cl/ep,m,fm
Sanilac	*bc-2*	cl/m	cl/m,fm	−/m,fm
Michelite 62	*bc-2*	cl/m	cl/m,fm	cl/ep,M,FM
Red Mexican 34	*bc-2*	rs/m	rs/mm	cl/mm
Pinto 114	*bc-1,bc-2*	rs/ep,m	rs/mm	−/mm
Monroe	*bc-1*^{2},*bc-2*^{2}	cl/m	cl/m,fm	cl,ep,n/m,clf
Great Northern 31	*bc-1*^{2},*bc-2*^{2}	rs/m	rs/m	cl/mm
Red Mexican 35	*bc-1*^{2},*bc-2*^{2}	rs/m	rs/m	cl/mm
IVT 7214	*bc-3*	cl/mm	cl/mm	cl/mm
Group 2[f]				
Widusa	None	cl/m	cl/ep,N,fm	cl/M,FM,N
Black Turtle Soup	None	cl/im	cl/M,FM	cl/M,FM,N
Jubila	*bc-1*	cl/m	cl/M,FM	cl/M,FM,N
Topcrop	*bc-1*	cl/m	cl/m,fm,m	cl/M,FM,N
Improved Tendergreen	*bc-1*	cl/m	cl/M,FM	cl/M,FM,N
Amanda	*bc-1*^{2}	cl/m	cl/M,FM	cl/M,FM,N
Group 3[g]				
IVT 7233	*bc-2*^{2}	cl/m	cl/M,FM,n	cl/M,FM,N
Orfeo-INIA	*bc-2*^{2}	cl/m	cl/M,FM	rs/ep,M,N
MCM 5001	*bc-3*	rl/ep,m	rl/M,FM	rs/ep,M,N

[a] Predominant (local/systemic) symptoms: cl = chlorotic lesions, rl = reddish lesions, rs = ring spots, ep = epinasty, m = mosaic, mm = mild mosaic, fm = mild moderate foliar malformation, M = intense mosaic, FM = severe foliar malformation, n = moderate necrosis, and N = severe necrosis.
[b] According to E. Drijfhout (7,8).
[c] According to E. Drijfhout (7,8).
[d] Comovirus (CV) isolates from Brazil (BZ), Venezuela (VZ), and Honduras (HD).
[e] Cultivars with recessive alleles (I^+I^+) of the necrosis gene.
[f] Cultivars with dominant alleles (*II*) of the necrosis gene.
[g] Cultivars with dominant alleles (*II*) of the necrosis gene protected against necrotic BCMV strains by recessive genes.

and those caused in this by the test CV-HD virus from neighboring Honduras. The main difference observed was the lack of a consistent interaction between BCDMV and the dominant alleles of the necrosis gene. On the contrary, only the *I* gene cultivars Commodore, Potomac, and accession G 09391 of Tendergreen reacted with leaf and stem necrosis to CV-HD. The rest of the inoculated bean genotypes, which possess the recessive alleles (I^+I^+) of the necrosis gene, exhibited only mosaic and foliar malformation, with the exception of one Kentucky Wonder accession (G 05513), which reacted with top necrosis in one test plant.

The results of the inoculation of the nine additional bean genotypes, including cranberry, red kidney, and white- and black-seeded cultivars, further confirmed the enhanced symptom expression (severe mosaic, rugosity, and plant malformation) induced by the three test viruses on the dominant *I* gene cultivars ICA-Pijao and Porrillo Sintetico. As observed above for other *I* gene bean genotypes, only CV-HD induced foliar and stem necrosis in ICA-Pijao and Porrillo Sintetico. Bean cultivars Alubia Cerrillos, Diacol-Calima, Red Kidney, Bayo Gordo, and Cacahuate 72, devoid of the dominant alleles of the necrosis gene, expressed only mosaic symptoms. The remaining I^+I^+ bean genotypes, Arbolito Retinto and Honduras 46, reacted with top necrosis to the mechanical inoculation of the three test viruses (Fig. 4).

The five bean cultivars reported to possess hypersensitive genes to BSMV and CpSMV reacted to BSMV with necrotic ring spot lesions, 1–2 mm in diameter, on the manually inoculated primary leaves (Fig. 5A), confirming their hypersensitivity to this sobemovirus. However, these bean cultivars became systemically infected by the three test viruses. Arbolito Retinto, included for comparative purposes, did not show any local lesions on the BSMV-inoculated primary leaves but developed severe mosaic and rugosity. This bean genotype reacts with concentric ring-shaped lesions (Fig. 5B) to the inoculation of the viruses selected in this investigation, suggesting its potential use as a diagnostic host.

Serological tests. The three test viruses were shown to be comoviruses serogically related to CpSMV, QPMV, and BRMV, in that order of relatedness, as determined by a nine-, seven-, and two-fold increase in the number of virus particles trapped in ISEM tests, respectively, relative to the untreated control (approximately 700 particles per 1,000 μm^2). The CpSMV and QPMV antisera, and the antiserum previously produced at CIAT against a bean comovirus from El Salvador, reacted more strongly with CV-HD than with CV-VZ or CV-BZ.

DISCUSSION

Besides the extreme pathogenic range of the three test comoviruses in *P. vulgaris*, it was clear from this investigation that two of these comoviruses, CV-VZ and CV-HD, interact with the dominant alleles of the necrosis gene. These comoviruses cause severe mosaic, plant malformation, and, particularly in the case of CV-HD, necrosis of the stem and leaves of dominant *I* gene plants. Although cultivars Great Northern 31, Red Mexican 35, and Pinto 114 and the line IVT-7214, which possess recessive genes against necrotic BCMV strains, developed only mild mosaic when inoculated with the test comoviruses, these genes failed to protect *II* bean genotypes IVT 7233, Orfeo-INIA, and MCM

Table 2. Comparative reaction of selected bean genotypes previously evaluated for their reaction to bean curly dwarf mosaic comovirus (BCDMV) from El Salvador to a bean comovirus isolate (CV-HD) from Honduras

Bean cultivar	G accession[a]	Alleles of necrosis gene[b]	Reaction to CV-HD[c]
Group 1[d]			
Bountiful	6374	I^+I^+	M
Commodore	6716	*II*	M,D,N
Mexico 309	5652	I^+I^+	M,FM,N
Potomac	5692	*II*	M,FM,N
Tendergreen	7476	I^+I^+	m
Tendergreen	9391	*II*	M
Tenderlong 15	5497	*II*	N
Kentucky Wonder	5513	*II*	M,TN
Kentucky Wonder	0148	*II*	M,N
Group 2[e]			
Beka	1594	I^+I^+	M
Beka	8666	I^+I^+	M
Beka	10067	I^+I^+	M
Columbia Pinto	11211	I^+I^+	M
Great Northern 59	5050	I^+I^+	m
Guatemala 416	8501	I^+I^+	m
ICA-Tui	3717	*II*	M,D,N
ICA-Tui	4454	*II*	M,D,N
La Vega	5728	*II*	M,D,N
Plentiful	3769	I^+I^+	m
Plentiful	9292	I^+I^+	m
Porrillo 1	1741	*II*	M,D,N
Porrillo 1	4461	*II*	M,D,N
Sensutepeque	3090	I^+I^+	m
Surecrop Wax	3773	II^+	M,D,N
Sutter Pink	9384	I^+I^+	m

[a] G = identification number given by the Genetic Resources Unit of CIAT.
[b] Evaluation previously conducted by the Virology Research Unit of CIAT.
[c] Main symptoms induced by the Honduran comovirus: m = mild mosaic, M = intermediate to severe mosaic, D = dwarfing, N = necrosis, FM = foliar malformation, and TN = top necrosis.
[d] Cultivars reported to react with top necrosis to BCDMV according to J. P. Meiners et al (12).
[e] Cultivars reported to react with mosaic, dwarfing, and vein necrosis to BCDMV.

Fig. 4. Systemic necrosis in an Arbolito Retinto bean plant inoculated with a bean comovirus from Brazil.

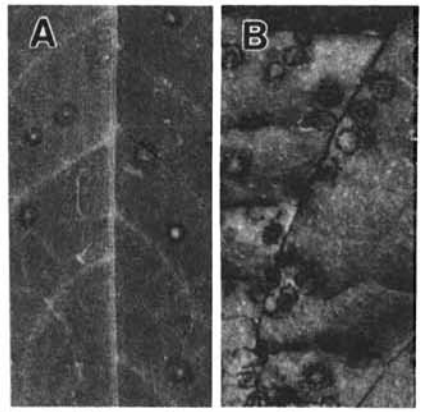

Fig. 5. (A) Necrotic ring spot lesions caused by bean southern mosaic virus on the manually inoculated primary leaves of a Red Mexican bean plant and **(B)** concentric annular local lesions induced by a bean comovirus from Brazil on the primary leaves of an Arbolito Retinto bean plant.

5001 against the severe symptoms induced by the three comoviruses tested.

Regarding the recent report by Costa (4) on the hypersensitive response of certain *I* gene bean cultivars to CpSMV and BCMV, we found notable differences in this investigation. First, the CpSMV isolate used by Costa only induced pinpoint local lesions on inoculated leaves but no systemic infection of the bean cultivars he listed as carriers or possible carriers of the dominant alleles of the necrosis gene. Second, some of the hypersensitive bean genotypes tested by Costa, such as Columbia Pinto, Pinto UI 115, Red Mexican, Red Mexican UI 37, and Sanilac, do not possess the dominant alleles of the necrosis gene. And third, the five cultivars that reacted with local lesions to CpSMV in Brazil (Kentucky Wonder, Iguaçu, Red Mexican, Cornell 49-242, and Moruna 80) were systemically infected by the three comovirus isolates selected for this study. In fact, none of the dominant *I* gene bean cultivars inoculated in this investigation with CV-BZ, CV-HD, or CV-VZ developed pinpoint local lesions on the inoculated primary leaves. Instead, they reacted with enlarged local lesions (unlike those in Costa's study) associated with dominant factors, which condition hypersensitivity to both BCMV and BSMV. Therefore, we conclude that although Costa's CpSMV isolate also interacts with the dominant alleles of the necrosis gene, it cannot infect dominant *I* gene bean cultivars systemically, as is the case with the three comoviruses investigated here.

It seems unlikely that any of the three comoviruses tested in this investigation are the same as the BCDMV isolate described in El Salvador as a strain of QPMV, although CV-HD is closely serologically related to BCDMV. Also, it is not clear whether the three comoviruses selected in this investigation belong to the QPMV or CpSMV serogroups. The high affinity of the CpSMV antiserum for the three bean comoviruses tested by ISEM suggests these isolates may belong to the CpSMV serogroup. Moreover, from an examination of the literature, it is apparent that most of the Latin American comoviruses belong to the CpSMV serogroup (10). Unfortunately, when BCDMV was originally reported as a strain of QPMV, its serological relationship with CpSMV was not determined (12).

In Latin America, we are increasingly encountering some comoviruses capable of infecting all common bean cultivars tested to date, causing severe mosaic, plant malformation, stunting, and, sometimes necrosis and plant death in cultivars possessing dominant hypersensitive resistance to BCMV. The implications of this pathogenic interaction are significant, considering that the majority of the improved common bean cultivars grown in Latin America, the United States, and Europe possess monogenic dominant resistance to BCMV and that some of the chrysomelid vectors of these comoviruses also are found in bean-growing regions in temperate countries.

We have been collectively referring to the mosaic, plant malformation, and necrosis syndrome observed in Latin America as bean severe mosaic and to the comoviruses that induce them as bean severe mosaic virus strains.

LITERATURE CITED

1. Bruening, G. 1978. Comovirus group. No. 199 in: Descriptions of Plant Viruses. Commonw. Mycol. Inst./Assoc. Appl. Biol., Kew, England. 5 pp.
2. Cardona, C. 1989. Insects and other invertebrate bean pests in Latin America. Pages 505-570 in: Bean Production Problems in the Tropics. 2nd ed. H. F. Schwartz and M. A. Pastor-Corrales, eds. CIAT, Cali, Colombia.
3. Centro Internacional de Agricultura Tropical 1986. Bean Program. 1985 CIAT Annu. Rep. 331 pp.
4. Costa, A. S. 1989. Hipersensibilidade do feijoeiro ao comovirus do mosaico severo do caupi e ao potyvirus do mosaico comum são condicionadas pelo mesmo fator genetico. Summa Phytopathol. 15:73-82.
5. Cupertino, F. P., Costa, C. L., Lin, M. T., and Kitajima, E. W. 1981. Infecção natural do feijoeiro pelo virus do mosaico severo do caupi no Centro Oeste do Brasil. (Abstr.) Fitopatol. Bras. 6:529.
6. Derrick, K. S. 1973. Quantitative assay for plant viruses using serologically specific electron microscopy. Virology 56:652-653.
7. Drijfhout, E. 1978. Genetic interaction between *Phaseolus vulgaris* and bean common mosaic virus with implications for strain identification and breeding for resistance. Agric. Res. Rep. Wageningen. 98 pp.
8. Drijfhout, E. 1991, Bean common mosaic. Pages 37-39 in: Compendium of Bean Diseases. R. Hall, ed. American Phytopathological Society, St. Paul, MN.
9. Drijfhout, E., Sibernagel, M. J., and Burke, D. W. 1978. Differentiation of strains of bean common mosaic virus. Neth. J. Plant Pathol. 84:13-26.
10. Fulton, J. P., and Scott, H. A. 1979. A serogrouping concept for legume comoviruses. Phytopathology 69:305-306.
11. Gamez, R. 1972. Los virus del frijol en Centroamérica. II. Algunas propiedades y transmisión por crisomélidos del virus del mosaico rugoso del frijol. Turrialba 22:249-257.
12. Meiners, J. P., Waterworth, H. E., Lawson, R. H., and Smith, F. F. 1977. Curly dwarf mosaic disease of beans from El Salvador. Phytopathology 67:163-168.
13. Zaumeyer, W. J., and Thomas, H. R. 1948. Pod mottle, a virus disease of beans. J. Agric. Res. 77:81-96.