***Bc-1* and *Bc-u*—Two Loci Controlling Bean Common Mosaic Virus Resistance in Common Bean are Linked**

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ABSTRACT. Resistance to bean common mosaic virus (BCMV) strain NY15 (Zaumeyer) and bean common mosaic necrosis virus (BCMNV) strain NL-3 (Drijfhout) was assessed in 98 F_{5:6} recombinant inbred lines (RILs) derived from a cross between pinto bean (*Phaseolus vulgaris* L.) cultivars ‘Olathe’ and ‘Sierra’. ‘Olathe’ has *bc-u* and *bc-1*² whereas ‘Sierra’ has no known resistance genes. The differentiation of resistant and susceptible lines was based on visual symptoms, virus titer, and top dry weight. Forty-seven RILs were moderately resistant to NL-3, while 51 RILs were susceptible. This segregation fits a 1 susceptible : 1 resistant ratio characteristic of a single gene. Sixty-nine RILs were susceptible to NY15, while 29 RILs were resistant, which fits a 3 susceptible : 1 resistant ratio characteristic of a two-gene model. Moderate resistance to NL-3 was conferred by *bc-1*² with or without *bc-u* present. Bulk segregant and two point linkage analysis identified randomly amplified polymorphic DNA (RAPD) markers linked in coupling to the *Bc-1* and *Bc-u* alleles. The OH14₁₁₀₀ RAPD marker was 4.5 cM from the *Bc-1* locus. The OC16₁₀₀₀ RAPD marker was linked at a distance of 10.9 cM from the *Bc-u* locus. Multipoint analysis, using segregation data for *bc-1*², *bc-u*, and the two markers, estimated the distance between the *Bc-1* and *Bc-u* loci as 22.8 cM.

Bean common mosaic virus (BCMV), known since the early 1900s, is one of the world’s most serious bean viral pathogens (Drijfhout et al., 1978; Klein et al., 1988; Morales and Castaño, 1987; Pierce and Hungerford, 1929). In nature, BCMV infects primarily *Phaseolus* sp., with *P. vulgaris* as the predominant host. It is spread through seed and pollen and by numerous aphid species in a nonpersistent manner (Morales and Bos, 1988). Recently, serotype A strains were separated into a new species called bean common mosaic necrosis virus (BCMNV) (Mink et al., 1994).

Drijfhout (1978) and Drijfhout et al. (1978) presented a system of seven pathogenicity groups of BCMV with 11 host groups of common bean that differentiate the virus strains. Resistance to different strains of the pathogen is conditioned by the dominant *I* gene and/or specific combinations of recessive genes at three loci (*bc-1*, *bc-2*, and *bc-3*). In the absence of the *I* gene, resistance to specific virus strains is fully expressed only when the recessive allele *bc-u* is present in addition to one of the recessive alleles at *bc-1*, *bc-2*, and *bc-3* loci. The *bc-1* and *bc-2* loci each have two alleles (*bc-1* vs. *bc-1*², and *bc-2* vs. *bc-2*²) (in addition to the wild type allele) that confer resistance to different pathogroups (Drijfhout, 1978). Recently, molecular markers linked to *I* and *bc-3* were identified (Haley et al., 1994a, 1994b; Johnson et al., 1997; Kelly et al., 1995). Preliminary reports were published describing markers for the *Bc-u* and *Bc-1* loci (Myers et al., 1996a; Strausbaugh et al., 1995). Although markers linked to individual BCMV resistance genes

were reported, linkage among resistance genes has not been determined. In this paper we demonstrate that *bc-u* and *bc-1*² are linked.

Materials and Methods

INHERITANCE OF RESISTANCE. A recombinant population of 98 lines was derived from a cross between pinto bean cultivars ‘Olathe’ (*bc-u bc-u bc-1*² *bc-1*²) and ‘Sierra’ (*Bc-u Bc-u Bc-1 Bc-1*) via single-seed descent of F₂ plants through the F₅ generation. Eight seeds of each RIL per treatment were planted into potting mix (EKO compost: 80% compost + 20% vermiculite by volume; Organix Supply, Inc., Missoula, Mont.) in 1 L plastic pots (two seeds per pot). The pots were arranged in a randomized complete block design with four blocks and three inoculation treatments. Throughout the experiments, plants were grown on a greenhouse bench with mean temperatures between 23 and 28 °C (minimum of 20 °C and maximum of 33 °C). Natural light was supplemented during the day with lighting from sodium halide lamps to maintain an irradiance of at least 1000 mmol·m⁻²·s⁻¹ and a photoperiod of 13 h.

The treatments were plants inoculated with BCMV strain NY15 (Z), BCMNV strain NL-3 (D), or left uninoculated. Virus strains were maintained in ‘Dubbele Witte’ plants in aphid-proof cages. The virus strains were also inoculated into plants of differential cultivars (‘Stringless Green Refugee’, ‘Dubbele Witte’, ‘Black Turtle Soup I’, ‘Monroe’, and ‘Redlands Greenleaf B’) and the parental cultivars. One primary leaf per plant was inoculated mechanically with a triturate of infected tissue (1 g of tissue per 10 mL of 50 mM sodium phosphate buffer, pH 7) with a small amount of 600 mesh carborundum powder when primary leaves were 50% to 75% expanded (7 to 10 d after planting). A separate sterile cotton-tipped stick was used to inoculate each plant. Plants were evaluated 21 d after inoculation for visual symptoms and top dry weight. At least half the plants were sampled by collecting the second trifoliolate leaf above the inoculated leaf 21 d after inoculation and testing with enzyme-linked immunosorbent assays (ELISA). G. Mink (Washington State Univ., Prosser) conducted the ELISA assays using the

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Table 1. Reaction of a 'Sierra' x 'Olathe' recombinant inbred population to the NL-3 strain of bean common mosaic necrosis virus and the NY-15 strain of bean common mosaic virus; segregation ratios and putative genotypes.

Virus strain	Virus reaction phenotypic group ^z			Expected ratio	χ^2	P
	A	B	C			
NL-3	S	MR	MR	1S:1MR	0.16	0.69
NY-15	S	S	R	3S:1R	1.10	0.31
	No. of lines					
Total lines	51	18	29	2:1:1	2.63	0.28
Putative genotypes	<i>Bc-uBc-u Bc-1Bc-1</i> <i>bc-ubc-u Bc-1Bc-1</i>	<i>Bc-uBc-u bc-1²bc-1²</i>	<i>bc-ubc-u bc-1²bc-1²</i>			

^zS = susceptible (systemic virus movement, severe symptoms, and strongly positive ELISA OD), MR = moderately resistant (limited systemic virus movement, mild symptoms, and positive but relatively low ELISA OD), and R = no systemic virus movement, symptomless, and negative ELISA OD.

broad-spectrum monoclonal antibody bc-197 (Wang et al., 1985). Percent reduction of top dry weight, visual reading, and vigor reading were determined by comparing the treated plants with the uninoculated checks. Statistical analyses were performed using SAS procedures (1990, SAS Institute, Inc., Cary, N.C.).

DNA ISOLATION, POLYMERASE CHAIN REACTION ANALYSIS, AND LINKAGE ESTIMATION. Plants were grown in the greenhouse for 2 weeks, and 1 to 2 g of tissue from each of four uninfected plants was pooled for DNA extraction (Doyle and Doyle, 1990). DNA concentration was determined by fluorometry. Oligonucleotide primers were obtained from Operon Technologies, Inc. (Alameda, Calif.). The RAPD protocol reported by Williams et al. (1990) was followed with minor modifications. The volume for each amplification was 10 mL. Taq DNA polymerase and reaction buffer were purchased from Promega Corp. (Madison, Wis.). The total reaction mixture contained 10× buffer (50 mM KCl, 10 mM Tris-HCl pH 9.0, 0.1% Triton X-100, 1.5 mM MgCl₂), 200 mM each of dATP, dCTP, dGTP, and dTTP, 0.2 mM primer, 1 unit DNA polymerase, and 50 ng of template DNA. Polymerase chain reaction (PCR) amplifications were performed with a Perkin Elmer Model 480 thermal cycler (Perkin Elmer Corp., Norwalk, Conn.) programmed for 45 cycles at 94 °C for 1 min, 35 °C for 1 min, and 72 °C for 2 min, for denaturing, annealing, and primer extension, respectively. An additional 7-min extension at 72 °C followed the completion of the 45 cycles. The samples were run on a 2% agarose gel in 1× TAE at 65 V. Sizes of the amplification products were estimated by comparison to a 1-kb ladder (Life Technologies Inc., Rockville, Md.). The linkage between the RAPD markers and the loci of the resistance genes was

determined with the program Mapmaker 3.0 (Lander et al., 1987; Lincoln et al., 1992). Linkage distances were calculated as two-point data. Linkage groups were then defined by the Group command with a criterion of LOD > 3.0 and distance < 30 cM. Map order was determined using the Compare (criteria: LOD > 3.0) command, and the robustness of the map was measured by the Ripple (criteria: LOD > 3.0) command.

Results and Discussion

INHERITANCE OF RESISTANCE. When the virus reaction data to NL-3 and NY-15 were combined, three phenotypic classes were observed: lines susceptible to both virus strains (Group A); lines susceptible to NY-15 and moderately resistant to NL-3 (Group B); and lines resistant to NY-15 and moderately resistant to NL-3 (Group C) (Table 1). Two classes were discernible following inoculation with NL-3. The susceptible class (Group A) showed extensive systemic virus movement, severe symptoms, low plant vigor (quantified by top dry weight), and high ELISA optical density (OD) readings (Table 2). The moderately resistant class (Groups B and C) exhibited limited systemic virus movement, very mild visual symptoms, nearly normal vigor, and relatively low ELISA OD values (Table 2). Inoculations with NY-15 produced the expected reaction where susceptible lines (Groups A and B) showed systemic virus movement, mosaic mottle symptoms, and high ELISA OD readings, and resistant plants (Group C) had no systemic virus movement, no symptoms, and very low or near zero ELISA OD values (Table 2).

Table 2. Summary of disease reaction of 'Olathe' x 'Sierra' recombinant inbred lines inoculated with the NL-3 strain of bean common mosaic necrosis virus and NY-15 strain of bean common mosaic virus.

Virus strain	Phenotype group ^z	Symptoms	ELISA OD ^y	Percent reduction in top dry wt ^x
NL-3	A	Severe, reduced growth	1.15	50
	B, C	Mild, near normal growth	0.56	16
	$P > t$		0.0001	0.0001
NY-15	A, B	Severe, reduced growth	0.64	20
	C	None, normal growth	0.11	-2
	$P > t$		0.0001	0.0244

^zValues in the table represent the means for the recombinant inbred lines placed in that phenotype group (see Table 1).

^yELISA OD = Enzyme-linked immunosorbent assay optical density using the bc-197 monoclonal antibody.

^xPercent reduction in comparison to the uninoculated checks.

Fig. 1. Segregation of RAPD primer OH14₁₁₀₀ (arrow) linked to *Bc-1* in a recombinant inbred population from the cross of 'Sierra' x 'Olathe'. The first two lanes are amplification products of 'Sierra' (S) and 'Olathe' (O) DNA. The remainder of the lanes represents amplification of individual RI lines. Lane R represents a recombinant between *bc-1²* and the marker.

Fig. 2. Segregation of the RAPD primer OC16₁₀₀₀ linked to *Bc-u* in a recombinant inbred population from the cross 'Sierra' x 'Olathe'. The first two lanes are the amplification products of 'Sierra' (S) and 'Olathe' (O) DNA. The remainder of the lanes represents amplification of individual RI lines. The arrow points to the segregating band that is in close proximity to an adjacent monomorphic band.

Lines inoculated with NL-3 segregated into two nearly equal classes, approximating a 1 susceptible:1 moderately resistant ratio (Table 1). For a recombinant inbred population at the F₅ level of inbreeding, a 1:1 ratio is consistent with the presence of a single major gene conditioning reaction to NL-3. The segregation ratio for lines when inoculated with NY-15 was 3 susceptible: 1 resistant (Table 1). This is the expected segregation pattern with a two-gene model where both factors are required for expression of resistance and agrees with the results observed by Drijfhout (1978) for *bc-u* and *bc-1²*. For all lines, the data fit a 2:1:1 ratio when the three phenotypic classes were considered together (Table 1).

Based on the reported genotypes of 'Olathe' and 'Sierra' (Forster et al., 1994) and the reaction to NY-15 as shown in Table 1, *bc-u* and *bc-1²* must have been segregating in this population. In addition, one gene independent of the second gene conditioned a discrete differential reaction to NL-3. We believe that this gene is *bc-1²* because of the mild symptoms that developed when *bc-ubc-ubc-1²bc-1²* genotypes were inoculated with NL-3 (Drijfhout, 1978; Forster et al., 1994) and the ability of *bc-1²*, in the absence of *bc-u*, to suppress systemic necrosis in lines with *I* gene when inoculated with NL-3 (Drijfhout, 1978; Kelly, 1997). From the latter observation, Drijfhout (1978) suggested that *bc-1²* alone has weak antiviral activity to NL-3. When combined with the *I* gene, this activity is sufficient to inhibit virion movement that leads to systemic necrosis.

From observations of other researchers and our present results, we propose a model for recessive gene resistance to BCMV and BCMNV where the *bc-* numbered genes have some degree of antiviral activity, but when combined with *bc-u*, resistance is enhanced against specific virus strains. Except for *bc-3*, inoculating any virus strain into a differential containing the recessive resistant genes causes infection with replication in the inoculated leaf. Virus can be detected by serology in the inoculated leaf, but not from other parts of resistant plants (G. Mink, personal communication). The *bc-3* gene prevents replication at the site of infection because virus cannot be detected in the inoculated leaf. If the *Bc-1* and *Bc-2* loci

prevent systemic virus movement (as opposed to inhibiting viral replication), then a candidate mechanism for resistance would be for the resistance genes to code for and modify surveillance translocation proteins (Jorgensen et al., 1998) that facilitate long distance transport of host and viral RNA.

LINKAGE BETWEEN *bc-u*, *bc-1²* AND MOLECULAR MARKERS. RILs representing the genotypes from the classes listed in Table 1 were used to create bulk DNA samples to search for RAPD markers linked to *bc-u* and *bc-1²*. To screen for putative markers linked to *bc-1²*, DNAs from eight RILs susceptible to NL-3 (Group A) were combined to create a *Bc-1* pool. Likewise, DNAs from 8 RILs with moderate resistance to NL-3 (Groups B and C) were combined to create a *bc-1²* pool. Amplification with Operon primer H14 generated a 1100 bp fragment (OH14₁₁₀₀) present in the *Bc-1* bulk and absent in the *bc-1²* bulk. This fragment was also amplified from 'Sierra' (*Bc-1*), but not 'Olathe' (*bc-1²*) DNA (Fig. 1). The RIL population was screened, and two-point linkage analysis determined that OH14₁₁₀₀ and *Bc-1* were linked in coupling at a Kosambi (1944) distance of 4.5 cM (recombination fraction = 0.045).

A similar approach was used to identify a RAPD marker linked to *bc-u*. For this locus, the *bc-u* DNA pool was derived from eight RILs resistant to NY-15 and moderately resistant to NL-3 (Group C), and the *Bc-u* pool was developed from eight RILs susceptible to NY-15 and moderately resistant to NL-3 (Group B). Amplification with Operon primer C16 generated a 1000 bp fragment (OC16₁₀₀₀) from the *Bc-u* pool that was absent in the *bc-u* pool. This fragment was also amplified from 'Sierra' (*Bc-u*), but not 'Olathe' (*bc-u*) DNA (Fig. 2). Screening the population with Operon primer C16 revealed that OC16₁₀₀₀ was linked in coupling to *Bc-u* at a Kosambi (1944) distance of 10.9 cM (recombination fraction = 0.107).

Two point linkage analysis estimated the Kosambi (1944) distance between *bc-1²* and *bc-u* to be 31.6 cM. In turn, Mapmaker multilocus analysis using the same segregation data estimated this distance to be 22.8 cM (Table 3). The difference between the two-point and multilocus analyses is a direct result of the robustness of the multilocus analysis, which takes into account double crossovers within the interval. In our case, the two point analysis of *bc-1²* and *bc-u* cannot uncover double crossover events between these two genes. But the two additional markers that we have discovered adds important double crossover information relevant to the distance estimate. By considering all genotype data simultaneously, multilocus analysis generates map distances that are more consistent with all of the segregation data in the genetic interval being analyzed than two-point analysis (Lander et al., 1987; Liu, 1998). Therefore, we consider the 22.8 cM distance the better estimate of the distance between *bc-1²* and *bc-u*.

Our finding of a loose linkage between *bc-1²* and *bc-u* differs from Drijfhout's conclusion that the "...strain-unspecific gene is independent of the strain-specific genes..." (Drijfhout, 1978, p. 52). To investigate this further, we reanalyzed Table 13 from that paper ['Dubbele Witte' (*Bc-uBc-u Bc-1Bc-1*) x 'Redlands Greenleaf B'

Table 3. Recombination fraction and two point linkage and multilocus distances between *bc-1²* and *bc-u* and RAPD markers.

Linkage comparison	Recombination fraction	Two-point distance (cM) ^a	Multilocus distance (cM)
<i>bc-1²</i> -OH14 ₁₁₀₀	0.045	4.5	4.6
OH14 ₁₁₀₀ -OC16 ₁₀₀₀	0.069	6.9	7.0
OC16 ₁₀₀₀ - <i>bc-u</i>	0.107	10.9	11.2
<i>bc-1²</i> - <i>bc-u</i>	0.279	31.6	22.8

^aBased on Kosambi (1944) mapping function.

Table 4. Association of RAPD markers OC16₁₀₀₀ for *Bc-u* and OH14₁₁₀ for *Bc-l* with BCMV resistance genes in common bean cultivars.

Cultivar	Race ^z	Allele at <i>Bc-u</i> locus	OC16 ₁₀₀₀	Allele at <i>Bc-l</i> locus	OH14 ₁₁₀
Sierra	D (M) ^y	<i>Bc-u</i>	+	<i>Bc-l</i>	+
Olathe	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Stringless Green Refugee	NG	<i>bc-u</i>	—	<i>Bc-l</i>	+
Redlands Greenleaf B	NG	<i>bc-u</i>	—	<i>bc-l</i> ²	+
Redlands Greenleaf C	NG	<i>bc-u</i>	—	<i>bc-l</i>	+
Isabella	NG	<i>Bc-u</i>	—	<i>bc-l</i> ²	+
Amanda	NG	<i>Bc-u</i>	—	<i>bc-l</i> ²	+
Montcalm	NG	?	—	?	+
Linden	NG	?	—	?	+
California Dark Red Kidney 82	NG	?	—	?	+
California Early Light Red Kidney	NG	?	—	?	+
Sutter	D	<i>Bc-u</i>	—	<i>Bc-l</i>	—
UI 537	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Viva	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
UI 114	D	<i>bc-u</i>	—	<i>bc-l</i>	—
UI 129	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Fiesta	D	<i>bc-u</i>	—	? ^w	—
Topaz	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Othello	D	<i>bc-u</i>	—	? ^w	+
UI 59	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Harris	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Emerson	D	<i>bc-u</i>	—	<i>bc-l</i> ²	—
Beryl	D	?	—	<i>bc-l</i> ²	—
Dubbele Witte	M	<i>Bc-u</i>	—	<i>Bc-l</i>	+
Aurora	M	?	—	<i>Bc-l</i>	+
Domino	M	?	+	<i>Bc-l</i>	+
Mayflower	M	?	+	<i>Bc-l</i>	+
Seafarer	M	?	—	<i>Bc-l</i>	+
ICA-Bunsi	M	?	—	<i>Bc-l</i>	+
C-20	M	?	+	<i>Bc-l</i>	+

^zM = Mesoamerican race, and D = Durango race for cultivars of Mesoamerican origin; NG = Nueva Granada race from the Andean center of origin as defined by Singh et al., 1991.

^y'Sierra' represents a special case where actual pedigree is unknown, but has had major mixing of race Mesoamerica and race Durango germplasm. 'Sierra' has seed characteristics of race Durango, but shows agronomic performance and adaptation more similar to race Mesoamerica.

^x(+) Presence and (–) absence of a fragment for the RAPD marker.

^wCultivar possesses *bc-2*² that masks expression at the *Bc-l* locus.

(*bc-ubc-u bc-l*²*bc-l*²)] (Myers et al., 1999). In the six tests where the cross segregated for resistance, all fit a 15:1 S:R ratio. The data were pooled and chi-square tests of homogeneity and goodness of fit to a 15:1 ratio were performed. The pooled data were homogeneous but deviated from the expected segregation ratio ($\chi^2 = 5.08$, $P = 0.02$). More resistant and fewer susceptible individuals than expected were observed, consistent with the hypothesis of loose coupling linkage. Our multilocus analysis demonstrating that the two genes were linked supports that conclusion.

Clustering of disease resistance genes is common in plants. In many cases, genes in clusters are tightly linked. However, clusters of downy mildew (*Bremia lactucae* Regel) resistance genes occur in lettuce (*Lactuca sativa* L.) at distances of 20–25 cM (Hulbert and Michelmore 1985; Farrara et al. 1987). Drijfhout (1978) indicated that some deviations in his segregation data might have been caused by weak linkage among the strain-specific resistance genes. In particular, data from the cross 'Dubbele Witte' × 'Great Northern UI 31' suggested that *bc-l*² and *bc-2*² may be loosely linked. To date, the *Bc-2* locus has not been mapped. If the *bc* resistance genes are loosely clustered, a search of the chromosome region in the vicinity of the loci studied here might pinpoint the *Bc-2* locus.

DISTRIBUTION OF *bc-l*² AND *bc-u* MARKERS AMONG COMMON BEAN GENOTYPES. A number of different cultivars with and without the relevant resistance genes, and representing Mesoamerican and Andean centers of origin (Singh et al., 1991), were tested for the presence of the two RAPD markers (Table 4). For OC16₁₀₀₀, the presence of a fragment was associated with the *Bc-u* allele in some but not all cultivars. Cultivars of Andean origin appeared to be monomorphic, however, many of these possess *I* gene resistance to BCMV. Without test crosses, it would be impossible to know what allele is present at the *Bc-u* locus. From Drijfhout's (1978) work, we know that 'Amanda' has the *Bc-u* allele while 'Redlands Greenleaf B' and 'Redlands Greenleaf C' possess *bc-u*. In all cases, these cultivars lacked the fragment. Within the Mesoamerican center, 'Sutter', and 'Dubbele Witte' should have contained the fragment whereas they did not. The cultivars with the fragment originated from the Michigan State University breeding program (as did 'Sierra'), thus the marker may be program-specific rather than representative of a particular center of origin, or race.

There is a high degree of correspondence between OH14₁₀₀₀ and *Bc-l* for cultivars of Mesoamerican origin (Table 4). 'Sutter' is the only exception where linkage appears to be broken. We could

hypothesize that 'Sutter' has the genotype *Bc-uBc-ubc-I²bc-I²*, but tests of an F₂ population of the cross 'Sutter' × 'Stringless Green Refugee' with blackeye cowpea mosaic virus indicate that 'Sutter' does not have any resistance genes (Myers et al., 1996b). Cultivars of Andean origin appear to be monomorphic for this marker (both 'Isabella' and 'Amanda' should carry *bc-I²*, but also possess the fragment).

'Redlands Greenleaf C' and 'UI 114' were included in this analysis because they possessed the *bc-I* allele. 'Redlands Greenleaf C' is of Andean origin and has the OH14₁₀₀₀ fragment, consistent with the hypothesis that this marker is monomorphic in an Andean background. 'UI 114', of Mesoamerican background, does not contain the fragment. This is consistent with the fragment being linked with the wild type (dominant) allele, and implies that while OH14₁₀₀₀ distinguishes between the dominant allele and all others; it cannot distinguish between the two recessive alleles.

Both 'Fiesta' and 'Othello' have *bc-2²* that is epistatic to alleles at the *Bc-I* locus. These data suggest that 'Othello' has the dominant allele at the *Bc-I* locus whereas 'Fiesta' has one of the recessive alleles. Test crosses would be necessary to verify these predictions.

A traditional genetic analysis of the recessive BCMV resistance genes does not permit linkage mapping because the expression of a strain-specific resistance gene is dependent on the allele at the strain-unspecific locus, and can only be determined in some, but not all progenies from a segregating F₂ population. However, this problem can be solved with the use of molecular markers, and the development of appropriate recombinant inbred populations. The recombinant inbred population used herein allowed specific analysis of the *Bc-ubc-I²* mild mosaic interactions with NL-3 that Drijfhout (1978) was not able to study in detail in F₂ populations. The finding that the *Bc-u* and *Bc-I* loci are loosely linked is significant to breeding efforts for BCMV resistance, and to an understanding of the coevolution of common bean and BCMV.

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