# Resistance and Tolerance to Bean Common Mosaic Virus (BCMV) and Bean Common Mosaic Necrosis Virus (BCMNV) in Bean

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Resistance to BCMV and BCMNV (3) is controlled at five independent loci (1). The dominant *I* gene confers immunity or a hypersensitive response depending on interactions with temperature and virus pathogroup Genes at other loci are recessive, and one (*bc-u*) is required for expression of the others (*bc-1*, *bc-2*, *bc-3*) For *bc-1* and *bc-2*, a second allele is present (*bc-1*$^2$ and *bc-2*$^2$, respectively) Having molecular markers associated with the BCMV resistance genes increases the efficiency of gene pyramiding. To date, only *I* and *bc-3* have been tagged with RAPD and RFLP markers Since it is required for expression of the other *bc* genes, tagging the *bc-u* gene is particularly important In this study, we characterized resistance of Sierra x Olathe recombinant inbred (RI) lines to BCMV and BCMNV, and identified RAPD markers for the resistance genes carried in this population.

**Materials and Methods:** Seventy $F_{2:5}$ or $F_{2:7}$ RI lines from the cross 'Sierra' x 'Olathe' were evaluated for resistance using the following isolates: NL-8 (ID), NL-3, NY-15 (Z), and BlCMV (GA). The inoculation procedures of Forster et al , (2) were used. Previous tests demonstrated that Olathe possesses *bc-u bc-1*$^2$ whereas Sierra had no resistance genes (2). Four plants per line were inoculated with an isolate. Treatments were replicated four times in a randomized complete block design Data were collected on visual symptoms, vigor ratings, and single plant dry weight, and ELISA absorbance values. The latter was performed by the ELISA Laboratory, Prosser WA using the 197 monoclonal. RAPD mapping procedures are reported elsewhere (4).

**Results and Discussion:** *Tolerance to NL-3* All families inoculated with NL-3 became infected, but some were highly susceptible, while others showed tolerance. Susceptible families exhibited severe stunting, strong mosaic mottle, epinasty, chlorosis, and were positive by ELISA, while plants in tolerant families were nearly normal for plant vigor, had few if any visual symptoms, and varied from negative to positive by ELISA (a temperature-dependent reaction). Of the parents, Olathe was tolerant and Sierra was susceptible. Families segregated in a 1 1 ratio for tolerance and susceptibility to NL-3 as would be expected for a single gene (Table 1).

Within the tolerant group, families segregated in approximately a 1:1 ratio for resistance and tolerance/susceptibility to NL-8 and NY-15 (Table 1). Such a result would be obtained

**Table 1.** Number of families from $F_{2:5}$ or $F_{2:7}$ RI lines from the cross Sierra x Olathe showing disease reaction to five strains of BCMV, and predicted genotypes for resistance.

| Phenotypic | | | | | No. of Families[1] | | Putative |
|---|---|---|---|---|---|---|---|
| Group | NL-3 | NL-8 | NY-15 | BlCMV | Obs. | Exp. | Genotypes |
| A | S[2] | S | S | S | 6 | 8.75 | *Bc-1*$^2$ *Bc-u R* |
| B | S | S/T | S/T | R | 29 | 26 25 | *Bc-1*$^2$ *Bc-u r* |
| | | | | | | | *Bc-1*$^2$ *bc-u R* |
| | | | | | | | *Bc-1*$^2$ *bc-u r* |
| C | T | S/T | S/T | R | 17 | 17.5 | *bc-1*$^2$ *Bc-u R* |
| | | | | | | | *bc-1*$^2$ *Bc-u r* |
| D | T | R | R | R | 18 | 17 5 | *bc-1*$^2$ *bc-u R* |
| | | | | | | | *bc-1*$^2$*bc-u r* |

[1] $\chi^2$ = 1 18, Prob = 0 76 [2]S susceptible, T=tolerant, R=resistant

where two genes are required for resistance, and is in agreement with our knowledge of BCMV resistance (1) and the phenotypes for Olathe and Sierra (2). Because the gene conferring tolerance to NL-3 interacts with a second gene to confer resistance to NL-8 and NY-15, it maybe *bc-u* or *bc-1²*. Although the evidence is not conclusive, we believe *bc-1²* confers tolerance to NL-3 because this gene can protect *I* from systemic necrosis when inoculated with NL-3 even when *bc-u* is not present (1) Probably, the *bc-1²* gene product incompletely inhibits viral replication and/or movement when alone, but when combined with *I*, this weak activity is sufficient to inhibit systemic necrosis. Combined with *bc-u*, viral inhibition is greatly increased to provide resistance to pathogroups 1, 2, 3, and 5

When RI lines were inoculated with BlCMV, few susceptible families were observed Other studies (5) suggest that *bc-u* confers resistance to BlCMV. In addition, *bc-1²* and a third factor ("r") apparently confer resistance as well. Thus, a 7 1 ratio was observed, requiring a three gene model (Table 1) We hypothesize that *bc-1²*, *bc-u*, and "r" can act independently to produce resistance to BlCMV. The third resistance gene has been designated "r" until further characterized Such a model requires a change in our concept of BCMV resistance from *bc* genes acting only in concert, to these genes having independent biological activity.

*Linkage Relationships* Using groupings produced by the four virus isolates, the putative *bc-1²* and *bc-u* genes could be mapped. Putative *bc-1²* was mapped to linkage group 1 (Figure 1) which also contains *Ur-3*. In preliminary tests, two RAPD markers were linked to the putative *bc-u* gene (Figure 1).

**Figure 1.** RAPD markers and linkage distances (cM) associated with putative *bc-1²* on linkage group 1 and putative *bc-u* with associated markers from analysis of RI lines of the cross Sierra x Olathe

C12/800--J16/1300--Y6/1200--*bc-1²*--H19/1000--H2/1500--C4/1100
3.4 11.9 26.8 16.3 2.5 8.6

G15/1600--*bc-u*--C4/1300
29.4 31.6

Further research needed to verify the identity of the BCMV resistance genes includes locating associated RAPD markers in the appropriate BCMV differentials, and using tests of allelism with differentials possessing known resistance genes. Efforts are also underway to screen additional RI lines to increase the accuracy of linkage mapping.

**References:**


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