Bean Common Mosaic Virus - BCMV
&
Bean Common Mosaic Necrosis Virus - BCMNV

Bean common mosaic virus (BCMV) and *Bean common mosaic necrosis virus* (BCMNV) are aphid-transmitted potyviruses that can cause significant bean yield loss (Gálvez and Morales, 1989). This seed-borne virus can persist in infected seed lots.

Isolates, or pathotypes, of BCMV and BCMNV are assigned to one of eight different pathogenicity groups based on a differential bean host reaction as described by Drijfhout (1978). Each pathotype within a pathogenicity group corresponds to a known set of resistance genes. There are two major forms of resistance: recessive isolate-specific genes consisting of five alleles *bc-1*, *bc-1*^{2}, *bc-2*, *bc-2*^{2}, and *bc-3* at three loci that require *bc-u* for expression, and the dominant *I* gene. Drijfhout's (1978) tables 6 and 31 provide information on isolate – host compatibility reactions. With the exception of *bc-3*, isolate-specific genes provide resistance to some but not all pathogenicity groups of these two viruses. Moreover, the *I*, *bc-1*^{2}, and *bc-3* gene loci have been associated with resistance to other potyviruses (Fisher and Kyle, 1994; Larsen et al, 2008; Larsen and Miklas, 2010).

Figure 1. Susceptible mosaic symptoms on bean leaf infected with BCMV or BCMNV. (Photo from M. Silbernagel collection)

[Updated by Phil Miklas and Richard Larsen, USDA-ARS, Prosser, WA; May 20I0]

Cultivars susceptible to BCMV and BCMNV show vein banding and mosaic or mottle symptoms on leaves, and may allow seed transmission (Fig. 1). Plants with resistance to a specific isolate generally remain symptomless, although some host – pathogen combinations will produce necrotic local lesions after mechanical inoculation with the virus. The *I* gene gives immunity to BCMV isolates at temperatures less than 30C. For some BCMV isolates at temperatures greater than 30C and for all BCMNV susceptible cultivars infected with BCMNV and held at any growing temperature, plants will first exhibit pinpoint necrotic local lesions on inoculated primary leaves, followed by veinal necrosis and eventually top necrosis in the trifoliolate leaves which eventually kills the plant (Fig 2).

Figure 2. Pinpoint lesions and veinal necrosis on primary leaves of plants with *I* gene that were inoculated with a strain of BCMNV. Note darkened stems and top necrosis resulting in death of the emerging trifoliolate leaves. Soon these young plants will die (photo by P. Miklas).

This symptom is called “black root” (which should not be confused with the black root rot fungal disease caused by *Thielaviopsis basicola*) because of the blackened necrotic veins apparent when stems, pods and roots are cross-sectioned. When the isolate-specific genes are combined with the *I* gene, they will “protect” the *I* gene from necrosis-inducing isolates for which they provide specific resistance (Fig. 3 and 4). It is interesting that *bc-1*, *bc-1*^{2}, and *bc-3* will protect *I* gene without *bc-u* present but *bc-2*^{2} requires presence of *bc-u* to protect *I* gene. The mechanism of *bc-2* on protection of *I* gene is unknown. The viruses are never seed-transmitted in cultivars possessing the *I* gene, but fields of an unprotected *I* gene cultivar may suffer total crop failure if seed of a susceptible cultivar contaminated with any strain of BCMNV is planted in close enough proximity for aphids to transport the virus from the susceptible to another unprotected *I* gene plant.

Figure 3. Plants inoculated with BCMNV NL-3 strain that possess *I* gene only (left) and *I* gene protected by *bc-1*^{2} gene (right). The plants on the right show restricted-veinal necrosis symptoms which occur only on the inoculated primary leaves (photo by P. Miklas).

Figure 4. Primary leaf inoculated with the NL-3 strain showing restricted local pinpoint necrotic lesions which indicates presence of the *I* gene protected by the *bc-2*^{2} gene (photo from M. Silbernagel collection).

Mills and Silbernagel (1992) described a protocol for preparation of inoculum and subsequent inoculation of bean plants with BCMNV. The strain is maintained on a susceptible cultivar such as 'Sutter Pink', 'Dubble Witte', or BT-II. . Approximately 25% of the seed harvested from BCMNV infected Sutter Pink plants are often infected with the virus (J. Kelly, personal communication). However, percent of seed transmission is affected by time of infection. Infection occurring from emergence to bloom will result in higher seed transmission. Seed transmission will be very low or nonexistent for infection occurring after flowering. Infected seed is ideal for short and long-term storage of the virus. Inoculum can also be stored in infected leaf tissue that has been lyophilized, or in fresh collected tissue stored in a -80C freezer. However, rejuvenation of the virus from such tissues stored for extended periods can be difficult.

Inoculum is prepared by grinding 1 g BCMNV or BCMV-infected leaves in cold 0.01-0.05 M phosphate buffer, pH 7 at approximately 1:10 w/v. Primary leaves are inoculated when about ¾ expanded which is usually about 7 days after planting. Leaves are dusted with 600 mesh carborundum and the inoculum is gently rubbed on the entire surface of the primary leaf. Alternatively, carborundum can be incorporated into the virus-buffer mixture.

With unprotected *I* gene and BCMNV isolates, necrotic local lesions may appear in as few as 3 days with top necrosis ensuing within a week (the reaction will take longer in a cool environment). Some isolate – host cultivar combinations such as NL-3 and a plant with *I* + *bc-1*^{2} will show delayed symptoms. A plant absent the *I* gene but with *bc-1*^{2} gene inoculated by NL-3 will take 21 to 28 days to exhibit mild mosaic symptoms.

Large scale field inoculations can be done with an airless electric paint sprayer. Inoculum is made up in liter batches with the buffer solution as described above. It is critical to use chilled buffer for tissue maceration, and the inoculum should be kept on ice until use to avoid oxidation of the macerated tissue. Approximately 100 g of infected leaf tissue is placed in an industrial blender with a liter of buffer. Tissue is macerated on the high setting for three minutes, and then strained through three layers of cheesecloth. The inoculum is applied through the paint sprayer without carborundum. Depending on the type of sprayer, it may be necessary to remove the nozzle guard to allow the nozzle of the sprayer close enough to the plants to achieve a water-soaked lesion when the sprayer is activated. This method allows the inoculation of a hundred meters of row in about a half hour with about 95% of susceptible inoculated plants showing symptoms in four weeks. Aphids will generally continue the spread of the virus in the field such that all susceptible plants will eventually show symptoms.

Mills and Silbernagel (1992) proposed a 1-9 evaluation scale in accordance with the scale proposed by the CIAT Bean Research Program where 1-3 is considered resistant, 4-6 intermediate and 7-9 are susceptible. An alternative 0-10 evaluation scale for BCMNV reactions was proposed by Strausbaugh et al. (2003a).

The NL-3 strain of BCMNV can be used to screen bean lines for resistance to both BCMV and BCMNV. Kelly (1997) described the differential host reaction of eight varieties of common bean inoculated with the NL-3 strain of BCMNV (Table 1).

Table 1. The differential host reaction of eight varieties of common bean inoculated with the NL3 strain of BCMNV.

Variety	Genotype[1]	Host symptoms
Sutter Pink	*ii*	M – Mosaic
Black Turtle Soup 1	*II*	TN – Top necrosis
Olathe*	*ii bc-1^{2}bc-1^2*	MM – Mild mosaic
Beryl	*II bc-1^{2}bc-1^2*	VN – Vein necrosis
Othello*	*ii bc-2^{2}bc-2^2*	NR – No reaction
92US-1006*	*II bc-2^{2}bc-2^2*	NLL – Necrotic local lesions
G94574*	*ii bc-3bc-3*	NR – No reaction
Raven	*II bc-3bc-3*	NR – No reaction

[1] The presence of the *bc-u* is assumed in those varieties marked by an asterisk *, Source: Kelly (1997).

Care must be taken when evaluating the NL-3 – *bc-1^2* host group reactions because some symptomless plants will have high virus titer in their tissues (detectable by ELISA) and are capable of seed transmission (Strausbaugh et al., 2003b).

Enzyme-linked immunosorbent assay (ELISA) provides a means of detecting infection by BCMV and BCMNV. A broad-spectrum polyclonal antibody that detects most potyviruses. ELISA kits are available commercially from Agdia (http://www.agdia.com/). There are PCR-based probes that can be used to distinguish isolates between BCMV and BCMNV (Abdallah, 1995).

Cultivars such as 'Raven' that combine the *I* and *bc-3* genes are resistant to all known strains of BCMNV and BCMV (Kelly et al., 1994) except NL 3-K in some cases (Larsen et al, 2005). Most commercial seed types of common bean have at least one line with the *I* and *bc-3* resistance genes (Table 2).

Table 2. Sources of *bc-3* resistance to BCMV and BCMNV in different seed classes.

Name or number	Seed color / type	Resistance genes	Reference
Raven	9 / Black	*I, bc-3*	Kelly et. al (1994)
BelMiDak RMR 10-12	1 / Navy	*I, bc-3*	Pastor-Corrales (2003)
TARS-VR-1, 7, 8			Miklas et al. (1997)
BelDakMi RMR 19-23	2M / Pinto	*I, bc-3*	Pastor-Corrales (2003)
BelMiNeb RMR 9-13	1 / Great Northern	*I, bc-3*	Pastor-Corrales (2003)
PR9357-107	6 / Small red	*I, bc-3*	Beaver et al. (1998)
USCR 7 and 9	2R / Cranberry	*I, bc-3*	Miklas & Kelly (2002)
BRB 198	6M / Red mottled	*I, bc-3*	
USDK-4	6K / Dark red kidney	*I, bc-3*	Miklas et al. (2002)
USLK-2	5K / Light red kidney		
USWK-6	1 / White kidney		

References

Abdallah, R. 1995. An immunocapture/reverse transcription/polymerase chain reaction technique for detection and molecular characterization of bean common mosaic virus in mixed infections. Ph.D Thesis, Washington State University, Pullman.

Beaver, J.S., P.N. Miklas, J.D. Kelly, J.R. Steadman and J.C. Rosas. 1998. Registration of PR9357-107 small red germplasm resistant to BCMV, BCMNV and rust. Crop Sci. 38:1406-1407.

Drijfhout, E. 1978. Genetic interaction between *Phaseolus vulgaris* and bean common mosaic virus with implications for strain identification and breeding for resistance. (Doctoral thesis, Wageningen) Agric. Res. Rep. 872. 98 pp.

Fisher, M.L., and M.M. Kyle. 1994. Inheritance of resistance to potyviruses in *Phaseolus vulgaris* L. III. Cosegregation of phenotypically similar dominant responses to nine potyviruses. Theor. Appl. Genet. 89:818-823.

Gálvez, G.E. and F.J. Morales. 1989. Aphid-transmitted viruses. p. 333-362. *In* H.F. Schwartz and M.A. Pastor-Corrales (eds.). Bean production problems in the tropics. Centro Internacional de Agricultura Tropical (CIAT). Cali, Colombia.

Hosfield, G.L., G.V. Varner, M.A. Uebersax and J.D. Kelly. 2004. Registration of 'Merlot' small red bean. Crop Sci. 44:351-352.

Kelly, J.D. 1997. A review of varietal response to bean common mosaic potyvirus in *Phaseolus vulgaris*. Plant Varieties and Seeds 10:1-6.

Kelly, J.D., G.L. Hosfield, G.V. Varner, M.A. Uebersax, L.K. Afanador and J. Taylor. 1994. Registration of Raven black bean. Crop Sci. 34:1406-1407.

Larsen, R.C and P.N. Miklas. 2010. Mapping resistance to *Peanut mottle* virus in common bean. Annu. Rep. Bean Improv. Coop. 53: 50-51.

Larsen, R.C., P.N. Miklas, K. Eastwell, and C.R. Grau. 2008. A strain of *Clover yellow vein virus* that causes severe pod necrosis disease in snap bean. Plant Dis. 92:1026-1032.

Larsen, R.C., P.N. Miklas, K.L. Druffel, S.D. and Wyatt. 2005. The NL-3 K strain is a stable and naturally occurring interspecific recombinant derived from *Bean common mosaic necrosis virus* and *Bean common mosaic virus*. Phytopathology 95:1037-1042.

Miklas, P.N., J.S. Beaver, J.R. Steadman, M.J. Silbernagel, and G.F. Freytag. 1997. Registration of three bean common mosaic virus-resistant navy bean germplasms. Crop Sci. 37:1025.

Miklas, P.N., and J.D. Kelly. 2002. Registration of two cranberry bean germplasm lines resistant to bean common mosaic and necrosis potyviruses: USCR-7 and USCR-9. Crop Sci. 42:673-674.

Miklas, P.N., A.N. Hang, J.D. Kelly, C.A. Strausbaugh, and R.L. Forster. 2002. Registration of three kidney bean germplasm lines resistant to bean common mosaic and necrosis potyviruses: USLK-2 light red kidney, USDK-4 dark red kidney, and USWK-6 white kidney. Crop Sci. 42:674-675.

Mills, L.J. and M.J. Silbernagel. 1992. A rapid screening technique to combine resistance to halo blight and bean common mosaic virus in Phaseolus vulgaris L. Euphytica 58:201-208.

Pastor-Corrales, M.A. 2003. Sources, genes for resistance and pedigrees of 52 rust and mosaic resistant dry bean germplasm lines released by the USDA Beltsville bean project in collaboration with Michigan, Nebraska and North Dakota Agricultural Experiment Stations. Annu. Rep. of the Bean Improv. Coop. 46:235-241.

Strasbaugh, C.A., J.R. Myers, R.L. Forster, and P.E. McClean. 2003a. A quantitative method to screen common bean plants for resistance to *Bean common mosaic necrosis virus*. Phytopathology 93:1430-1436.

Strausbaugh, C.A., P.N. Miklas, S.P. Singh, J.R. Myers, and R.L. Forster. 2003b. Genetic characterization of differential reactions among host group 3 common bean cultivars to NL-3 K strain of bean common mosaic necrosis virus. Phytopathology 93:683-690.