Bean Common Mosaic Virus

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Bean common mosaic virus (BCMV) causes one of the world's most serious diseases of beans. The virus is seed-borne and easily spread by aphid vectors. It occurs in all parts of the world where susceptible bean varieties are grown.

Economic losses vary greatly. During severe epidemics, yields of susceptible varieties may be reduced by one-third or more. Because the virus is seed-borne, infected plants being raised for seed may be disqualified for certification, eliminating the price premium seed producers normally receive.

The disease was first observed nearly 100 years ago in Russia. It has been in Idaho for at least the past 70 years. In the 1920s, before resistant varieties were available, BCMV was widespread and common in southern Idaho bean fields. Since the development of resistant varieties in the 1930s, epidemics have become sporadic. The last major epidemic occurred in 1977.

During the 1989 growing season, an outbreak of BCMV affected many bean fields in southcentral and southwestern Idaho. Affected varieties included pintos UI 114, UI 126 and NW 410 and great northern UI 60, all of which are resistant to strains of the virus commonly found in Idaho. The Idaho Crop Improvement Association rejected about 650 acres of beans due to excessive levels of BCMV. Almost one-half of those acres were planted with UI 114. Subsequent testing of seed stocks revealed infection of two foundation seed lots of UI 114 produced in 1987 and 1988 and of one foundation lot of UI 60 produced in 1982.

Cause of bean common mosaic

Bean common mosaic virus is a member of the potyvirus (potato virus Y) group. Potyviruses are the largest single group of plant viruses and one of the most important economically. They are spread in nature by aphids, and a few, including BCMV, can be seed-borne and pollen-borne. In addition, they can be transmitted experimentally by mechanical means, for example, by rubbing sap from an infected plant onto a healthy plant. A closely related virus, bean yellow mosaic virus, is *not* seed-borne.

In nature, BCMV is found primarily in beans (*Phaseolus* spp.) and occasionally in wild legumes. It can be transmitted experimentally to other hosts in the legume family.

Disease diagnosis

Disease diagnosis based solely on symptoms usually is unreliable. Several viruses including alfalfa mosaic virus, tobacco streak virus and beet curly top virus infect beans and commonly occur in the Pacific Northwest. Bean common mosaic (BCM) symptoms may be confused with those of bean yellow mosaic or alfalfa mosaic in some bean varieties, or they may be very subtle and overlooked.

Serological assays are the most reliable and sensitive methods for detecting BCMV and confirming diagnoses. These assays use antibodies derived from animals such as mice or rabbits that have been injected previously with the virus. Antisera are available that can readily detect all known strains of BCMV in infected plant tissue and seed. Certain specific polyclonal antisera and/or monoclonal antibodies can differentiate strains of BCMV. A number of different serological assays are available, but the best suited is enzyme-linked immunosorbent assay (ELISA).

Strains of BCMV

The virus that causes BCM is quite variable. At least 22 strains of BCMV have been identified. They have been divided into eight pathogenicity groups on the basis of disease reactions in a series of differential bean varieties in which a particular variety may be susceptible to one virus strain but resistant to a second. BCMV strains have also been subdivided into two serogroups (A and B), which can be differentiated with specific antisera.

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Fig. 1. Mosaic is the symptom most commonly associated with bean common mosaic virus. Distinct or diffuse zones of light- and dark-green tissue are typical.

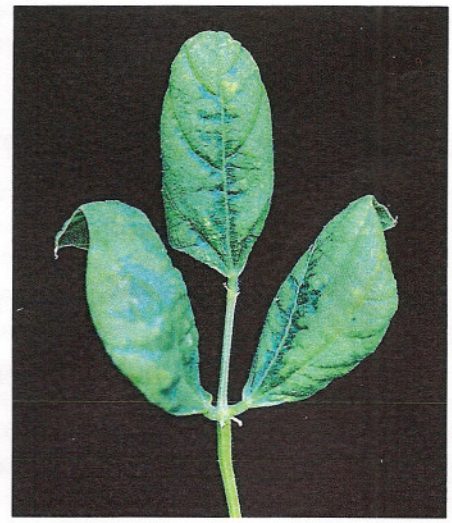

Fig. 2. Vein banding and leaf cupping and rolling are commonly seen in bean common mosaic virus infections.

Fig. 3A. Black root occurs when certain strains of bean common mosaic virus infect beans containing the *I* gene. All plant stages from seedling to mature plant may be affected. Plants die soon after infection.

Figs. 3B and 3C. Cross sections of a bean pod (B) and root (C) reveal darkened vascular tissue in the black root reaction.

Symptoms

Symptoms of BCM vary with bean variety, virus strain, environmental conditions and stage of plant growth at the time of infection. Infected plants may be symptomless, or they may be stunted and have foliar symptoms from barely visible to severe. The three principal foliar symptoms associated with BCMV are mosaic (Figs. 1 and 2), systemic necrosis (black root) (Figs. 3A, 3B and 3C) and local leaf lesions (Fig. 4). In addition, leaves may be curled or malformed.

Mosaic appears in systemically infected varieties and consists of light- and dark-green leaf tissue in a mosaic pattern or, more commonly in Idaho, dark-green bands along the main veins and lighter green interveinal tissue (''vein banding'') (Fig. 2). Some strains of the virus initially cause a translucence or clearing of the veins (''vein clearing'') while the interveinal tissue remains dark green. Later, leaves may exhibit typical mosaic symptoms. Leaflets frequently curl downwards and appear to be longer and narrower than healthy ones.

Table 1. Dry bean varieties grown in the Pacific Northwest and their types of genetic resistance to bean common mosaic virus.

Variety	Type of resistance	Variety	Type of resistance
Black Turtle		**Great Northern**	
Black Magic	I	Harris	bc‡
Midnight	I	Tara	none
UI 906	I	UI 425	bc‡
Cranberry		UI 31	bc§
Michigan Improved	none	UI 59	bc‡
Taylor	none	UI 60	bc‡
UI 50	I	US 1140	bc‡
UI 686	I	Valley	bc‡
Navy and Small White		**Pink**	
Aurora	I	Harold	bc§?
C-20	I	Roza	bc§?
Fleetwood	I	Sutter	none
Mayflower	I	Victor	bc§?
Sanilac	bc†	Viva	bc‡?
Snow Flake	bc†	**Pinto**	
UI 125	I	Nodak	bc‡
UI 158	I	NW-410	bc‡
Upland	bc†	NW-590	bc§?
Red Kidney		Olathe	bc‡
Isabella	I	Othello	bc§
Kamiakin	I	Ouray	bc‡
Kardinal	I	Pindak	bc‡?
Montcalm	I	Sierra	none
Red Kloud	I	UI 111	bc†
Royal Red	I	UI 114	bc†
UI 722	I	UI 126	bc‡
Red Mexican		UI 129	bc‡
UI 35	bc§	UI 196	bc§
NW-59	bc§	**Yellow Eye**	
NW-63	bc§	Steuben	none
UI 36	bc‡		

Note: I = dominant *I* gene resistance, bc = *bc* gene resistance
† = resistance only to the common strain of BCMV
‡ = resistance to strains of BCMV commonly found in Idaho
§ = resistance to all but the NL-4 strain of BCMV
? = resistance inferred from variety release notices and pedigree information, but testing with differential virus strains has not been reported

In varieties that carry the dominant *I* gene for resistance to BCMV (Table 1), systemic necrosis (black root) may occur (Figs. 3A, 3B and 3C). This is a reaction to the virus in which the vascular tissue of the roots, stems and pods turns black and dies, leading to the rapid death of leaves, branches and sometimes the whole plant.

Strains of BCMV commonly found in the United States are referred to as "temperature sensitive, necrosis-inducing" strains because they cause black root in *I* gene varieties only at temperatures above 86°F. Strains of the virus that are common in Africa are referred to as "temperature *in*sensitive, necrosis-inducing" strains because they can cause black root at cooler temperatures as well. Both types have been found in several states including Idaho and Washington. Several other viruses, including a necrotic strain of bean yellow mosaic virus (or clover yellow vein virus), also may induce black root symptoms.

Local lesions, the third principal foliar symptom, is sometimes referred to as "road mapping" (Fig. 4). This symptom occurs at the point of inoculation, typically by

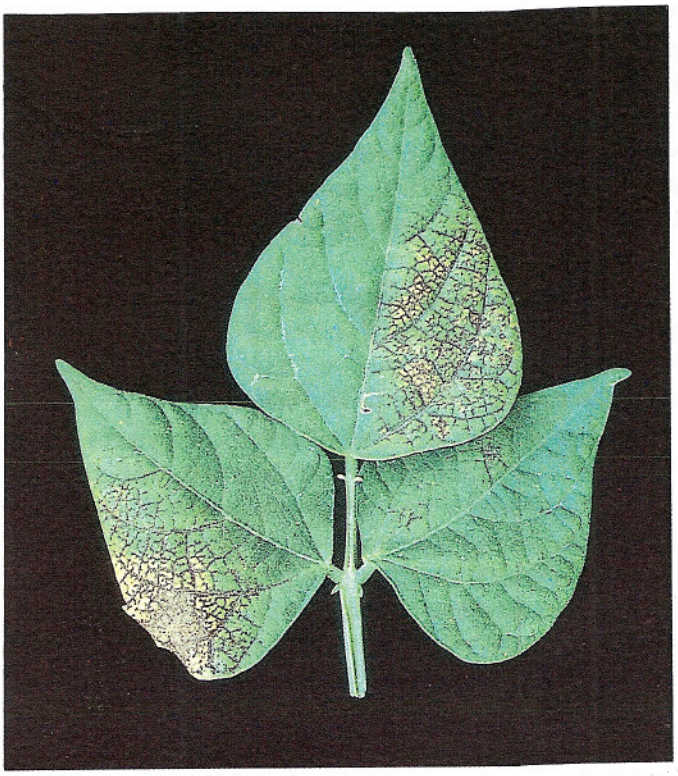

Fig. 4. Local lesions, also known as road mapping or veinal necrosis, may develop at the inoculation site of bean common mosiac virus.

aphid transmission. It appears initially as a chlorotic zone with reddish brown to black lines (veins), and it may expand from small areas to cover the leaflet or whole leaf. In varieties containing the recessive *i* gene, these areas may cover only a portion of a leaflet.

False symptoms

Stunting, leaf rolling and cupping are common symptoms of BCM, but they may also be caused by several other factors. Bean yellow mosaic virus and beet curly top virus may cause leaf cupping. Cupping is generally more pronounced in older leaves infected with bean yellow mosaic virus; younger leaves are more severely cupped in beet curly top virus infections.

Hot, dry winds may cause drying and death of cells at the margins of expanding leaflets, resulting in a cupping of the leaflet as it grows. Phenoxy herbicides may also cause plant distortion that may be confused with BCM symptoms.

Spread of BCMV

BCMV can be spread in nature via seed, pollen or aphids. Seed transmission is the primary means of long-distance spread and of the spread of exotic strains between regions and continents. Seed transmission is not known to occur in *I* gene varieties.

The rate of seed transmission (i.e., the percentage of seeds that produce infected plants) depends on the stage

of plant growth at the time of infection, the virus strain and the bean variety. Seed transmission rates range from 0 to 83 percent but seldom exceed 50 percent. Plants that become infected after flowering typically develop little or no seed infection. Seed transmission has occurred in seed after storage for 30 years, hence, prolonged storage of BCMV-infected seed does not appear to affect viability of the virus.

The rate of seed transmission from parents infected through pollen is generally very low under field conditions. This is because beans are self-fertile and usually are pollinated before the flower opens.

Dispersal within and between fields occurs primarily via aphid feeding and movement. Numerous aphid species can transmit the virus in a "nonpersistent" manner. This means that an aphid can acquire the virus from an infected plant and transmit it to a healthy plant in a matter of seconds and that *feeding* aphids will not retain the ability to transmit virus for more than a few hours. However, nonfeeding aphids, such as winged aphids that are flying or moving with wind currents, may be able to retain the ability to transmit the virus for much longer periods.

The most notable aphid vectors of BCMV are the pea aphid (*Acyrthosiphon pisum*), bean aphid (*Aphis fabae*) and green peach aphid (*Myzus persicae*). The aphid vector need not be capable of colonizing beans; various transient or noncolonizing aphids also can transmit BCMV. **Growers and seed dealers should be aware that low levels of infected seed can provide sufficient inoculum for an epidemic when large numbers of aphids are present.**

Genetic resistance to BCMV

Two basic mechanisms confer genetic resistance to BCMV. One is the dominant *I* gene; the second is a series of five recessive *bc* genes, *bc-1, bc-1*^{2}, bc-2, bc-2^2 and *bc-3*. Each recessive gene confers resistance to a subset of BCMV strains: *bc-2*^{2} confers resistance to most of the known strains, and *bc-3* confers resistance to all known strains. All of these recessive genes require the presence of a *bc-u* gene in order to activate resistance gene expression.

The best combination for broad-spectrum, stable resistance is the dominant *I* gene paired with the recessive *bc-2*^{2} or *bc-3* gene. This is the "protected" *I* gene form of resistance. The recessive *bc* gene prevents the black root reaction that may occur in plants with an "unprotected" *I* gene (*I* gene only, without *bc* genes). The *I* gene decreases the likelihood of new virus strains developing through mutations that could overcome the resistance of the *bc* genes.

Status of resistance in bean varieties

Many older varieties have limited or no resistance to BCMV. Most recently released garden bean, navy, black, kidney and cranberry varieties carry unprotected *I* gene resistance. In the great northern, red Mexican, pink and pinto market classes, resistance is presently conferred by genes of the *bc* series. Most of these varieties have *bc-1, bc-1*^{2} or *bc-2* resistance, separately or in various combinations. Only a few varieties, such as the Othello pinto, have the more desirable *bc-2*^{2} resistance, and no commercially grown U.S. varieties have *bc-3* resistance.

Control

Use virus-free "clean" seed and resistant varieties. However, no currently available varieties have resistance to all strains. Seed certified by the Idaho Crop Improvement Association has passed field standards for BCMV (foundation seed − 0 percent tolerance, registered seed − 0.5 percent tolerance, certified seed − 1.0 percent tolerance) and is recommended for seeding, when available.

Growers who cannot obtain certified seed should request proof that the seed lot has been assayed and determined to be free of BCMV before purchasing it. Seed assays for BCMV are helpful in identifying infected seed lots, but the level of seed transmission of BCMV may be lower than the level of BCMV detected in the seed lot. Seed assays for BCMV are available through the ELISA Lab, Irrigated Agriculture Research and Extension Center (IAREC) at Washington State University, Prosser, Washington.

Insecticide applications to control the aphid vectors are not recommended for BCMV because aphids can transmit the virus before they are repelled or killed by the insecticide. Indeed, insecticide applications may increase BCMV spread by causing the aphids to be restless and to move more often.

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