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# Virus diseases and resistance to Bean common mosaic and Bean common mosaic necrosis potyvirus in common bean (*Phaseolus vulgaris* L.)

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**ABSTRACT**

*Bean common mosaic potyvirus* (BCMV) and *Bean common mosaic necrosis potyvirus* (BCMNV) are economically the most important viruses of common bean. They can reduce yield and quality of harvested product. High percentage of seed transmission (up to 83%) is one of the reasons for its distribution worldwide. They represent 7 pathogenicity groups and 10 strains. Pathogenicity groups can be determined by reactions of differential bean cultivars. BCMV and BCMNV can be differentiated on serological and molecular level but all strains cause similar symptoms in bean genotypes lacking resistance genes. The development of different reactions to virus infections depends on virus strain, bean cultivar and temperature. The host resistance genes and virus pathogenicity genes interact in development of different responses of bean plants after infection. Different combinations of dominant and recessive strain unspecific and recessive strain specific resistance genes confer more stable resistance to wider spectrum of virus strains. The causal viruses, interactions of host resistance and virus pathogenicity genes, *in vivo* recombinations of both viruses and methods for selecting the desired host genotype are reviewed.

**Key words:** BCMV, BCMNV, resistance genes, pathogenicity genes, BCMV/BCMNV resistance, *Phaseolus vulgaris*

**IZVLEČEK**

**VIRUSNE BOLEZNI IN ODPORNOST NA VIRUS NAVADNEGA MOZAIKA FIŽOLA IN VIRUS NAVADNEGA NEKROTIČNEGA MOZAIKA FIŽOLA PRI FIŽOLU (*Phaseolus vulgaris* L.)**

Virus navadnega mozaika fižola (BCMV) in virus navadnega mozaika in nekroze fižola (BCMNV) sta ekonomsko najpomembnejša potivirusa, ki okužujeta fižol. Povzročata veliko ekonomsko škodo pri pridelavi fižola, tako z zmanjševanjem pridelka, kot tudi s slabšo kakovostjo pridelanega produkta. Virusa sta v svetu splošno razširjena, k temu pa je pripomogla tudi njuna lastnost, da se v veliki meri prenašata s semenom. Prenos s semenom

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lahko doseže do 83%. Virusne izolate obeh virusov uvrščamo v 7 patogrup in 10 različkov. Uvrstitev v patogrupe in različke nam omogoča inokulacija diferencialnih sort fižola in opazovanje njihove reakcije na virusno okužbo. Oba virusa se razlikujeta po seroloških in molekulsko bioloških lastnostih, vendar pa na rastlinah brez genov za odpornost povzročata zelo podobna bolezenska znamenja. Razvoj bolezenskih znamenj je odvisen od virusnega različka, sorte gostiteljske rastline in temperature. Interakcije med produkti genov za odpornost gostitelja in genov za patogenost virusa vplivajo na tip reakcije, ki se pojavi na fižolu po okužbi z virusom. Kombinacije dominantnega gena za odpornost z recesivnimi geni za odpornost omogočajo razvoj stabilnejše odpornosti na več različkov virusa, zato želje žlahtniteljev po genotipu fižola, ki bi bil odporen na vse znane različke obeh virusov, niso neosnovane. V prispevku sta predstavljena oba virusa, interakcije med produkti genov za odpornost gostitelja in genov za patogenost virusa, rekombinacije obeh virusov ter metode za selekcijo željenega genotipa gostitelja, odpornega na posamezne ali vse znane različke BCMV in BCMNV.

**Ključne besede:** BCMV, BCMNV, geni za odpornost, geni za patogenost, odpornost na BCMV/BCMNV, *Phaseolus vulgaris*

## 1 INTRODUCTION

Common bean (*Phaseolus vulgaris* L.) is one of the major food legumes produced. Its production is very important in North, Central and South America, eastern Africa, eastern Asia and south eastern Europe. Virus diseases are a major yield reduction factor in bean production. Most of the viruses infecting common bean are insect transmitted, some of them are also seed transmitted. Insect transmission is very important for virus spread on short distances while seed transmission is the most important factor in spread of viruses around the world. Even low seed transmission produces infected plants at the most suitable time for vector transmission and this way spreading in the field can be very fast. The list of viruses infecting beans is very long (Brunt *et al*., 1996 onwards; Kumar *et al*., 1994) but economically the most important ones are potyviruses, *Bean common mosaic potyvirus* (BCMV), *Bean common mosaic necrosis potyvirus* (BCMNV) and *Bean yellow mosaic potyvirus* (BYMV); *Cucumber mosaic cucumovirus* (CMV), *Southern bean mosaic sobemovirus* (SBMV), *Tobacco streak ilarvirus* (TSV) and *Tomato aspermy cucumovirus* (TAV). Because of high seed transmission the most important ones are BCMV and BCMNV.

## 2 BEAN COMMON MOSAIC POTYVIRUS (BCMV) AND BEAN COMMON MOSAIC NECROSIS POTYVIRUS (BCMNV)

BCMV was first reported in *P. vulgaris* in USA in 1917 and was called bean mosaic virus. To distinguish it from BYMV, epithet common was added later. The virus is now distributed worldwide (Brunt *et al*., 1996 onwards; Jeyandandarajah and Brunt, 1993; Klein *et al*., 1988; Mavrič *et al*., 2002 and 2003; Omunyin *et al*, 1995; Ravnikar *et al*., 1996; Sáiz *et al*., 1995) and causes a big economic damage by reducing yield (as much as 80%) and quality of harvested product (Drijfhout, 1991). It is transmitted in non-persistent manner by several aphid species including *Acyrthosiphon pisum*, *Aphis craccivora*, *A. fabae* and *Myzus persicae*. It is also transmitted by mechanical inoculation, up to 83% by seed. It was found that the virus could be transmitted to offspring from healthy plant through the pollen of infected plant. Seed transmission is irregular and depends on plant age at time of infection,

cultivar and virus strain. Flower buds, infected just before or after fertilisation, never produce infected seeds. Virions are 847-886 nm long filamentous, usually flexuous particles 12-15 nm wide. They contain 5% nucleic acids and 95% protein of 32-35kDa. 17 potyviruses are serologically related to BCMV, including *Potato virus Y*, *Watermelon mosaic virus 2*, BYMV, *Blackeye cowpea mosaic virus* (BlCMV) and *Soybean mosaic virus* (SMV) (Brunt *et al*., 1996 onwards, Drijfhout, 1978). The last two are now considered to be strains of BCMV (Mink *et al*., 1994). The symptoms induced on bean cultivars are severe and represent light and dark green mosaic, leaf roll, malformation of leaves and pods, rugosity of lower leaves or yellow dots, often causing growth reduction. In some cultivars severe vascular necrosis may occur and infected plants may die. This phenomenon is known as 'black root'. The type of symptoms induced depends on the interaction between host resistance genes and virus pathogenicity genes (Brunt *et al*., 1996 onwards; Drijfhout, 1991; Frison *et al*., 1990).

There used to be two serotypes of the virus, serotype A and B, which are now considered two distinct viruses (McKern *et al*., 1992). BCMV, formerly BCMV serotype B, has many different strains, some of them, like Azuki bean mosaic virus, Blackeye cowpea mosaic virus, Cowpea aphid-borne mosaic virus, Cowpea (blackeye) mosaic virus, Cowpea vein-banding mosaic virus, Peanut blotch virus, Peanut stripe virus and some others, were once considered to be different viruses (Brunt *et al*., 1996 onwards; Higgins *et al*., 1998; McKern *et al*., 1992a; Mink *et al*., 1994). On the basis of new sequence data, serology and biological properties they were proved to be strains of one virus. While sequence data are the major criteria for discriminating between different viruses, biological and biophysical properties are the major criteria for discriminating between virus strains. BCMV usually causes mosaic symptoms on bean cultivars and only some strains can cause systemic lethal necrosis on sensitive cultivars at higher temperatures. The BCMNV isolates, formerly BCMV serotype A, cause systemic lethal necrosis on bean genotypes possessing dominant resistance gene *I* at lower and higher temperatures (Silbernagel *et al*., 2001). However, all known BCMV and BCMNV strains cause similar symptoms in bean genotypes lacking resistance genes (Morales and Bos, 1988).

Additional differences were observed between serotype A and serotype B isolates or between BCMNV and BCMV. Apparent molecular mass of serotype A isolates capsid protein obtained by SDS polyacrylamide gel electrophoresis and Western blot serology was lower (about 33 kDa) than that of the serotype B isolates (34.5 – 35 kDa). Also the normal lengths of the particles were different, 810-818 nm for serotype A particles and 847-886 nm for serotype B. All isolates studied induced pinwheel and scroll inclusions in cytoplasm, but only in cells infected with serotype A isolates a specific type of proliferated endoplasmic reticulum was observed. The comparison of 3'-end of the genome of two serologically different isolates, representing both viruses, showed considerable differences in the N-terminal part of the coat protein (CP) and 3' non-coding region, while core region and C-terminal part of the CP appeared to be highly conserved (Vetten *et al*., 1992).

Table 1: Differences between BCMV and BCMNV

| BCMV | BCMNV |
|---|---|
| mosaic symptoms, only some strains can cause systemic lethal necrosis on sensitive cultivars at higher temperatures | systemic lethal necrosis on bean genotypes possessing dominant resistance gene *I* at lower and higher temperatures |
| serotype B | serotype A |
| CP of 34.5-35 kDa | CP of about 33 kDa |
| normal length 847-886 nm | normal length 810-818 nm |
| only typical potyvirus inclusions | proliferation of ER |
| sequence differences in CP and 3' non-coding region | sequence differences in CP and 3' non-coding region |

Both viruses can be found on the same area and sometimes even infecting the same plant (Silbernagel *et al*., 2001). Although *P. vulgaris* originates from Latin America and BCMV is found there, BCMNV is not common in that part of the world. However, BCMNV is widespread in Africa and in some parts the problems are so serious that cultivars possessing the *I* gene resistance cannot be grown successfully (Kelly *et al*., 1995; Omunyin *et al*., 1995).

For BCMV and BCMNV the first level of biological subdivision is the patogenicity group or pathogroup, which is determined with ability of virus isolates to systemically infect specific cultivars. Isolates inducing symptom variations (either intensity or type) on specific hosts within a pathogroup are defined as virus strains (Silbernagel *et al*., 2001).

## 3 PATHOGENICITY AND RESISTANCE GENES

According to the presence of pathogenicity genes (P0, P1, $P1^2$, P2, $P2^2$) and the reactions on the differential bean cultivars, BCMV and BCMNV strains are grouped into 7 pathogenicity groups (PGs) (I – VII) and 10 strains or subgroups with two subgroups in each of PGs IV, V and VI (Drijfhout *et al*., 1978). They differ by the number of pathogenicity genes present. PGs I, II and III have only one pathogenicity gene, P0, P1 or P2, respectively. PGs IV and V have two genes each, P1, $P1^2$ and P1, P2. The highest numbers of pathogenicity genes are in PG VI and PG VII. They both have P1 and $P1^2$ in combination with P2 in PG VI and $P2^2$ in PG VII. All of the BCMV strains used in the study of Drijfhout (1978) belong to one of PGs I, II, IV, V or VII and all the BCMNV strains were classified as either PG III or PG VI (Silbernagel *et al*., 2001). PGs are determined by the ability of virus isolate to systemically infect a set of differential bean cultivars possessing defined combinations of recessive and dominant resistance genes.

The host resistance genes involved in these interactions form two groups, strain unspecific and strain specific genes. The dominant strain unspecific gene is gene *I* and the recessive strain unspecific gene is *bc-u*. All strain specific genes, *bc-1*, $bc\text{-}1^2$, *bc-2*, $bc\text{-}2^2$ and *bc-3*, are recessive and are independently inherited except for the allelic pairs *bc-1*, $bc\text{-}1^2$ and *bc-2*, $bc\text{-}2^2$. In the absence of the *I* gene, the *bc-u* gene is required for the expression of all strain specific recessive resistance genes. Gene combinations of *bc-u* with any of the strain specific recessive resistance genes but *bc-*

*3*, confer strain-specific recessive resistance. Only the combination *bc-u*, *bc-3* gives recessive resistance to all strains of both viruses (Drijfhout, 1978 and 1991).

The dominant *I* gene is known to inhibit all known strains of both viruses, but can be overcome by necrosis-inducing strains, the BCMNV. They stimulate systemic hypersensitive response and infected plants develop systemic lethal necrosis. This reaction is a big disadvantage of resistance by unprotected *I* gene. However, dominant *I* gene can be protected by combining it with recessive resistance genes. These combinations can restrict, prevent or delay extreme hypersensitive response. In plants infected with BCMV unprotected *I* gene conditions immune or temperature sensitive response (Drijfhout, 1991; Miklas *et al*., 2000). Another disadvantage of resistance induced using *I* gene is that it has a darkening effect on red and yellow coloured seed. If bright red or yellow colour is desired, the *bc-3* gene can be used in breeding for resistance (Drijfhout, 1991). Kelly *et al*. (1995) demonstrated that in the presence of *I* gene, *bc-3* and *bc-1*$^2$ do not require *bc-u* for expression of activity while *bc-2*$^2$ does.

The recognition of gene combinations in bean genotypes is not always possible because of the epistatic masking of the weaker recessive genes. This way *bc-3* masks *bc-2*$^2$ and *bc-1*$^2$, and *bc-2*$^2$ masks *bc-1*$^2$ when inoculated with NL-3 strain of BCMNV. However, all combinations of *I* gene with recessive resistance genes can be recognized when inoculated with BCMNV. The exception is the *bc-3* gene which is epistatic to the *I* gene while all other recessive resistance genes are hypostatic to the *I* gene. In the *I*, *bc-3* gene combination the discrimination between genotypes *I*, *bc-3* and *i*, *bc-3* is possible only by test-crossing or marker assisted selection (Kelly *et al*., 1995).

A gene-for-gene relationship has been found between strain specific resistance genes *bc-1*, *bc-1*$^2$, *bc-2*, *bc-2*$^2$, and virus pathogenicity genes with the same numerical codes. According to this theory, each strain specific, host resistance gene can be overcome (plant can be infected and systemically invaded) if the infecting virus strain contains the appropriate pathogenicity gene(s). The resistance gene *bc-3* has no corresponding pathogenicity gene known so far and bean genotypes with this gene are resistant to all know strains of BCMV and BCMNV. Strains possessing P0 can infect only bean genotypes without resistance genes. (Drijfhout, 1978 and 1991).

## 4 EFFECT OF TEMPERATURE ON HOST PLANT REACTIONS

The differential bean cultivars used for determination of BCMV and BCMNV PGs and strains are divided into two main groups according to the presence of dominant *I* gene. Inside these groups different combinations of recessive strain specific resistance gene combinations are representing each host resistance group (Table 2). For determining PGs of BCMV and BCMNV at least one cultivar from each group should be used for testing, and for precise strain identification at least two should be used, where possible (Drijfhout *et al*., 1978).

BCMNV induces a temperature insensitive, hypersensitive and often lethal necrosis in bean cultivars possessing the dominant *I* gene, while BCMV causes only mosaic symptoms in susceptible bean cultivars but can induce temperature-sensitive necrosis

in *I* gene cultivars at temperatures over 30°C. Cultivars from host groups 7 and 11 are resistant to all known strains of both viruses (Drijfhout, 1978; Sengooba *et al*., 1997).

Table 2: Groups of host differentials on the basis of their genotype

| host resistance group | resistance genes |
|---|---|
| 1 | |
| 2 | *bc-u*, *bc-1* |
| 3 | *bc-u*, *bc-1*$^2$ |
| 4 | *bc-u*, *bc-2* |
| 5 | *bc-u*, *bc-1*, *bc-2* |
| 6 | *bc-u*, *bc-1*$^2$, *bc-2*$^2$ |
| 7 | *bc-u*, *bc-2*, *bc-3* |
| 8 | *I* |
| 9 | *bc-1*, *I* |
| 10 | *bc-1*$^2$, *I* |
| 11 | *bc-u*, *bc-1*$^2$, *bc-2*$^2$, *I* |

Bean cv. Black Turtle 1 (BT1) represents host group 8 possessing only dominant resistance gene *I*. All strains of BCMNV induce systemic hypersensitive vascular necrosis and dieback on plants of BT1 grown at constant temperatures of either 22°C or 32°C. This phenomenon is called temperature insensitive necrosis (TIN). Some, but not all strains of BCMV cause systemic vascular necrosis and death only when BT1 plants are grown at constant 32°C. These strains can not infect BT1 plants grown at 22°C – phenomenon called temperature sensitive necrosis (TSN). A few BCMV strains will not infect BT1 at either temperature. These are called non-necrotic (NN) strains (Silbernagel *et al*., 2001).

A significant aspect of BCMV/BCMNV resistance is that all virus strains are capable of infecting the primary leaves of all bean cvs. in host groups 1 through 6 whereas no strains are capable of infecting the primary leaves of host group 7 which possesses recessive resistance gene *bc-3*. This type of resistance appears to involve a form of immunity rather than the strain specific resistance to systemic movement determined by the other recessive genes (Silbernagel *et al*., 2001).

## 5 *IN VIVO* RECOMBINATIONS OF BCMV AND BCMNV

Since BCMV and BCMNV can sometimes be found on the same area and even infecting the same plant Silbernagel *et al*. (2001) suspected that both viruses can somehow be capable of recombination *in vivo* to create new strains or even new pathotypes. In their experiments they used US-5 strain of BCMV and NL-8 strain of BCMNV representing two different PGs. NL-8 represents PG III, has only one pathogenicity gene P2 and induces TIN on BT1 but not on other *I* gene cultivars. US-5 from pathogenicity group IV has pathogenicity genes P1 and P1$^2$, is a non-necrotic strain and can systemically infect cultivars from host groups 1, 2 and 3. During cultivation of single strains on susceptible or resistant cultivars, no isolates with unusual pathogenicity or serological characteristics were detected. This indicates that spontaneous mutations in these characteristics are either extremely rare or do not

occur under used conditions. The same situation was observed when both strains were used in the same inoculum for inoculation of bean cultivars which were either susceptible or resistant to both strains. When inoculating plants, resistant to one strain but susceptible to the other they obtained some isolates causing systemic infection in resistant plant. These isolates were inoculated on differential cultivars and their characteristics remained stable through serial transfers made over a period of at least two years. That justified their classification as distinct virus strains. Three strains of PG IV and serotype A were found, what is the first finding of this combination. All serotype A isolates before represented PGs III or VI. They also obtained unusual serotype B isolates representing PGs III and VI, which were not found before. According to authors experience *in vivo* recombination between BCMV and BCMNV can occur only when both strains are able to infect and replicate in the primary leaves of the resistant plants and when plants are resistant to systemic movement of one of the parental strains. The results of the study suggest that the recombinant virus strains will retain the coat protein gene of the original incompatible strain while obtaining a portion of the compatible strain genome which controls systemic movement. This in turn suggests that the strain-specific recessive resistance host genes function in a way as to modify the effects of virus-induced proteins. In this case the proteins that regulate systemic movement of BCMV and BCMNV in beans are likely to be highly strain-specific.

Only the serotype A recombinant strains obtained in this study produced TIN on cultivars with the dominant *I* gene, the same as in nature. This indicates a close relationship between serotype A and ability to induce TIN. In contrast the association of serotype B epitope and ability to induce TSN was not linked.

According to the authors the results of this study also suggest that serotype and pathogenicity gene determinants are independent, pathogenicity and symptomatology have different determinants and under the conditions in experiment multiple exchanges can occur between virus strains within a single plant. New pathotypes of BCMV are known to occur in bean growing areas after the introduction of cultivars having resistance controlled only by combinations of the strain-specific recessive resistance genes. In the US, breeders now incorporate the non-strain specific dominant *I* gene along with various recessive genes to reduce the potential development of new pathotypes (Silbernagel *et al*., 2001).

## 6 SELECTION OF SPECIFIC GENOTYPES

The *I* gene was effectively used in breeding for resistance to BCMV until necrosis inducing strains of BCMNV appeared. After that attempts to protect *I* gene with recessive resistance genes (primarily *bc-2*$^2$ and *bc-3*) were made. The cultivars with multiple resistance genes are known to possess more stable resistance against a broader spectrum of virus strains (Miklas *et al*., 2000). The recognition of specific resistance gene combinations in a single bean genotype is not always possible because of the epistatic masking of expression of the weaker recessive genes. However, all combinations of *I* gene with strain specific recessive resistance genes are recognizable, when inoculated with BCMNV strains. The exception is the *bc-3* gene which is epistatic to the *I* gene while all the other recessive genes are hypostatic to it in their mode of action. It is also known that the *bc-3* gene alone conditions resistance

to all BCMV and BCMNV strains, so it is not possible to detect accompanying resistance sources without a test cross (Haley *et al*., 1994). The alternative to test-crossing would be marker assisted selection of desired genotypes. This method would enable breeders to discriminate also between *I*, *bc-3* genotypes and *i*, *bc-3* genotypes when needed and also to select for specific resistance genes in the absence of the virus. Molecular markers linked to some of bean resistance genes were developed in past years (Haley *et al*., 1994; Melotto *et al*., 1996; Miklas et al., 2000; Strausbaugh *et al*., 1999).

The most resistant bean genotype suggested by Drijfhout would be *I*, *bc-u*, *bc-1*$^2$, *bc-2*$^2$, *bc-3*. He also clearly demonstrated that, in the absence of the *I* gene, the *bc-u* gene was required for the expression of all other recessive strain specific resistance genes. However, in plants with the *I* gene, *bc-u* is not always necessary to be present. The *bc-u* gene is not required for expression of resistance, for instance in *I*, *bc-3* genotypes but would be needed for expression of the *bc-2*$^2$ resistance genotype *I*, *bc-2*$^2$, *bc-3*. In this case a marker linked to the *bc-u* gene would be needed to assure that all recessive resistance genes are present and active.

## 7 CONCLUSIONS

BCMV and BCMNV are two economically very important bean viruses transmitted by several aphid species and by seed. Seed transmission is the major mode of transmission of both viruses on long distances. BCMNV induces a temperature insensitive, hypersensitive and often lethal necrosis in bean cultivars possessing the dominant *I* gene, while BCMV causes only mosaic symptoms in susceptible bean cultivars but can induce temperature-sensitive necrosis in *I* gene cultivars at temperatures over 30°C. Since both viruses can be found on the same area and even in the same plant, recombinations between them are possible and can lead to creation of new strains or even new pathotypes. The *I* gene was effectively used in breeding for resistance to BCMV until necrosis inducing strains of BCMNV appeared. Later, attempts were made to protect *I* gene with recessive resistance genes. It is now known that the cultivars with multiple resistance genes possess more stable resistance against a broader spectrum of virus strains.

Viruses are known to greatly reduce bean yield. BCMV can reduce yield up to 24%, and BYMV up to 40% (Kumar *et al*., 1994). 40% increase of yield was observed by Benedičič and Berljak (1996) after pathogen elimination from bean cv. Zorin. However, it is important for the breeders to use every available methods and tools for breeding for resistance to BCMV and BCMNV to increase the yield and improve the quality of beans.

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