

Review

Bean Common Mosaic Disease: Etiology, Resistance Resource, and Future Prospects

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Abstract: Bean common mosaic disease is one of the most destructive diseases of the common bean, which is one of the most important legumes worldwide. It is caused by two closely related potyviruses: bean common mosaic virus (BCMV) and bean common mosaic necrosis virus (BCMNV). Both viruses have spread to all the common bean-growing areas worldwide and have become a major challenge in bean production. In this review, we summarized the biology and diversity of BCMV and BCMNV, discussed the current knowledge on the resistance genes of BCMV, and finally pointed out the future prospects for the control of bean common mosaic disease.

Keywords: bean common mosaic disease; common bean; biology; diversity; resistance genes; control

1. Introduction

The common bean (*Phaseolus vulgaris* L.) is one of the most important legumes for human consumption worldwide and is an important source of vegetable protein, minerals, antioxidants, and bioactive compounds [1–3]. Bean common mosaic virus (BCMV) and bean common mosaic necrosis virus (BCMNV) are aphid-transmitted viruses that can also be seed-transmissible in common beans with an efficiency of up to 80%, depending on the bean cultivar and viral strain [4]. An infection by phytopathogens, including plant viruses, can cause significant losses to the common bean in terms of both yield and quality. BCMV and the closely related BCMNV are the most destructive viruses of the common bean, which cause the common bean mosaic disease and the so-called “black root” disease. Since their discovery in 1917, BCMV and BCMNV have been reported in 57 countries and regions and have become two of the most important biological factors affecting common bean production [5–9]. Both BCMV and BCMNV belong to the genus *Potyvirus* in the family *Potyviridae*, the largest family of known plant RNA viruses, which are classified into 12 genera (*Arepavirus*, *Bevemovirus*, *Brambyvirus*, *Bymovirus*, *Celavirus*, *Ipomovirus*, *Macluravirus*, *Poacevirus*, *Potyvirus*, *Roymovirus*, *Rymovirus*, and *Tritimovirus*) [10–13].

Understanding the molecular biology of BCMV and BCMNV and the plant antiviral mechanism is a prerequisite for the development of effective management strategies. Over the last two decades, several advances have been made in this field. For instance, the strain composition of BCMV has been well documented, infectious cDNA has been successfully developed for analyzing the pathogenicity of BCMV, and several resistance genes in the common bean have been successfully identified and utilized in breeding practices [14–24]. In this short review, we summarize the current knowledge on BCMV and BCMNV, including genome organization, strain composition, evolution, and resistance in the common bean. Moreover, we highlight the areas that are particularly worthy of further investigation and the potential methods for developing novel antiviral strategies with broad-spectrum, efficient, sustainable, and environmentally friendly resistance.



Citation: Tang, M.; Feng, X. Bean Common Mosaic Disease: Etiology, Resistance Resource, and Future Prospects. *Agronomy* **2023**, *13*, 58. <https://doi.org/10.3390/agronomy13010058>

Academic Editor: Xiaofei Cheng

Received: 20 November 2022

Revised: 19 December 2022

Accepted: 21 December 2022

Published: 24 December 2022



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2. Biological Characteristics of BCMV

2.1. Genome Structure

Both BCMV and BCMNV have a positive-sense single-stranded RNA (+ssRNA) genome of approximately 9600 nucleotides (nt) that is encapsulated in non-enveloped, flexuous, and filamentous virions of 750 nm long and 11–13 nm in diameter [25–27]. The 3' end of the genome is polyadenylated, whereas the 5' end is covalently linked to the viral genome-linked protein (VPg) [28]. Similar to other potyviruses, the genomes of BCMV and BCMNV encode a large open reading frame (ORF), which is translated into a polypeptide of approximately 350 kDa [29]. This polypeptide is hydrolyzed by three viral proteinases into ten mature proteins; from the N- to C-termini are protein 1 (P1), a helper component-protease (HC-Pro), protein 3 (P3), 6-kilodalton protein 1 (6K1), a cylindrical inclusion (CI), 6-kilodalton protein 2 (6K2), viral protein genome-linked (VPg), nuclear inclusion a-protease (NIa-Pro), nuclear inclusion b (NIb), and a coat protein (CP) [30]. Additionally, a polymerase slippage motif (GAAAAAA) within the P3 cistron allows the expression of an additional short polypeptide, which is hydrolyzed into three mature proteins, namely P1, HC-Pro, and P3 N-terminal fused with the pretty interesting *Potyviridae* ORF (P3N-PIPO), which shares the same N-terminal domain as P3, but has a different C-terminal domain [31–34]. These proteins function similarly to their counterparts in the genus *Potyvirus* [35]. In addition, precursors such as P1-HcPro, 6K2-VPg, and P3-6K1 also exist in virus-infected cells via partial hydrolysis [36,37]. These precursors may play important roles in virus proliferation [38–61]. The cleavage of P1 may play dual roles in viral infectivity [62,63], and HC-Pro has been shown to play a role in virus movement and aphid transmission [64,65], while P3 has been associated with virus replication, systemic infection, and cell-to-cell movement [66–68]. Moreover, the 6K2 protein has been shown to play a pivotal role in the formation of the virus replication complex [69,70], VPg has been shown to bind eukaryotic translation initiation factor 4E (eIF4E) and its isoform eIF(iso)4E and to enhance viral translation in plants [29], and 6K1 binding to the membrane is involved in replication and viral movement between cells [61].

2.2. Transmission

BCMV and BCMNV can be transmitted through seeds, pollen, aphids, and mechanical friction [71,72]. The seed transmission rate of BCMV is between 3% and 95%, depending on the viral strain and host variety [73,74]. The high seed transmission rate is one of the major reasons for the global BCMV epidemic and is the primary virus resource in the field. BCMV is limited to the seed coat of bean varieties with a low transmission rate; however, it can invade the seeds (embryos) of bean varieties with high transmission rates. Generally, BCMV infects seeds via the endosperm [75]. However, it can also infect seeds through pollen. BCMV can survive for more than 30 years in seeds grown under suitable conditions. However, the gene(s) involved in seed transmission and the molecular mechanisms of seed transmission remain elusive [75]. It has been reported that 36 aphids in 21 genera, including *Acyrtosiphon pisum*, *Aphis fabae*, *Myzus persica*, and *Aphis craccivora*, effectively transmit BCMV [76–79]. Studies have shown that BCMV-infected plants are more attractive to aphids than healthy plants [80], indicating that BCMV may modulate the jasmonic acid pathway to reduce anti-insect defenses to benefit the aphid vector. BCMV can also be transmitted to new plants via mechanical inoculation [80,81]. Pathogen infection can induce changes in plant morphology and traits, such as wrinkled, curled, and yellowed leaves, increased or decreased glandular trichome density, callose deposition, and increased or decreased leaf thickness. These changes may affect the feeding and transfer of vector insects to host plants. Most viruses can cause yellowing or light-green symptoms in the host leaves. Vector insects are more inclined to select yellow and light green plant tissues, which may have been formed by the long-term evolution of virus–host plant–vector insects [82–86]. However, mechanical inoculation may not contribute significantly to the natural spread of BCMV.

2.3. Host Range and Symptoms

Potyrus collectively exhibit a broad host range; however, the host range of their individual members is usually limited to a few plants. For instance, the host range of the soybean mosaic virus (SMV), a phylogenetically related potyvirus, is restricted to two plant species of a single genus: cultivated soybean (*Glycine max*) and wild soybean (*G. soja*) [87]. Interestingly, BCMV has a much wider host range, including over 100 plants in 44 genera, such as the common bean, peanut (*Arachis hypogaea* Linn.), pigeonpea (*Cajanuscajan* (Linn.) Millsp), jack bean (*Phaseolus lunatus* L.), sickle senna (*Cassia tora* Linn.), chickpea (*Cicer arietinum* Linn.), showy croton (*Crotalaria spectabilis* Roth), common lentil (*Lens culinaris* Medic.), white lupine (*Lupinus albus* L.), soybean (*Glycine max*), white sweet clover (*Melilotus albus* Medic. ex Desr), broad bean (*Vicia faba* L.), bingdou (*Lens culinaris* Medic.), and sesame (*Sesamum indicum* L.). It is one of the most widespread plant viruses infecting legumes under natural conditions, with the common bean being the most affected host [76,88–90].

The symptoms induced by BCMV in the common bean include leaf mosaic, chlorosis, deformation and etiolation, plant retardation, local or systematic necrosis, and even whole-plant death [91–96]. The symptoms are mainly affected by three factors: host plants, environmental conditions, and viral strains [97–100] (Figure 1). BCMV can seriously affect the common bean's yield reduction or even total failure. Even mild or symptomless infections can decrease crop yield by 50% [101]. An infection by the BCMV strains NL2 and NL6 on common bean cultivars carrying the *I* gene at a high temperature will result in systemic necrosis symptoms that are typically known as “black root” disease. However, all BCMNV strains cause systemic necrosis symptoms in *I*-gene-containing bean cultivars, regardless of the temperature [91,92,96].

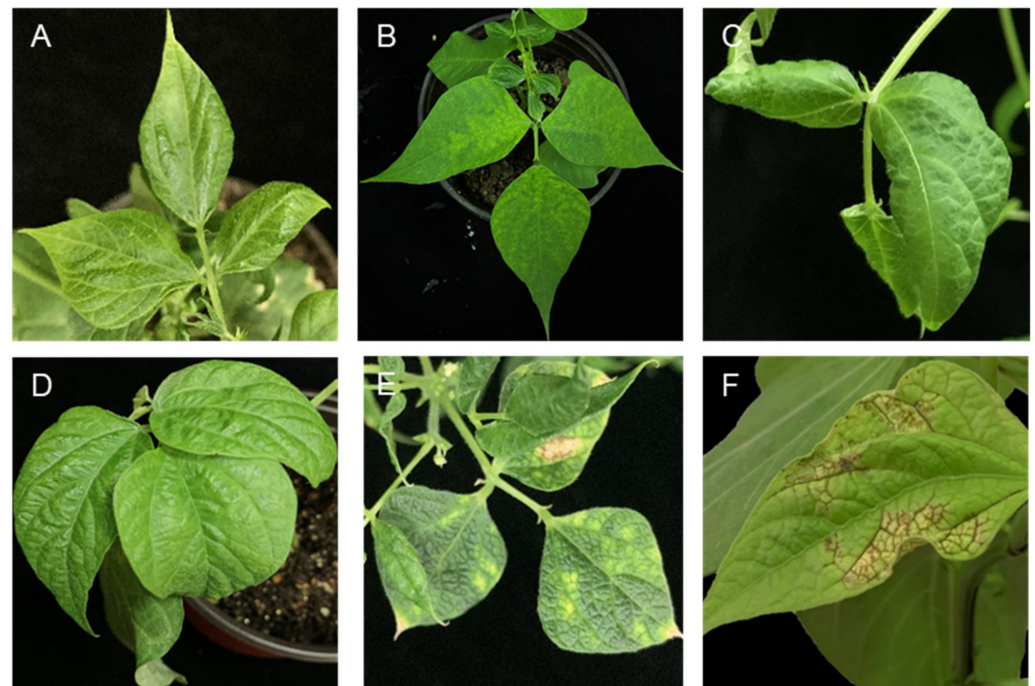


Figure 1. Symptoms of bean common mosaic virus (BCMV) in different bean cultivars. (A) Crinkling and leaf rolling of Dubbele Witte seedlings; (B) mild mottling of Stringless Green Refugee seedlings; (C) leaf deformation and rolling of IVT-7214 seedlings; (D) crinkling of Redland's Greenleaf “C” seedlings; (E) leaf rolling, chlorotic spots, and leaf deformation of Sanilac seedlings; and (F) leaf vein necrosis of SGR seedlings.

3. Diversity and Evolution of BCMV and BCMNV

3.1. Strain Composition

According to the symptoms of BCMV and BCMNV on eleven bean cultivars (Dubbele Witte (DW), Stringless Green Refugee (SGR), Redland's Greenleaf "C" (RGLC), Redland's Greenleaf "B" (RGLB), Sanilac, UI35, IVT-7214, Jubila, Amamda, US1006, and IVT7233), Drifhout divided BCMV into seven pathotypes, namely I–VII [18,19,91–95]. Recently, Feng discovered a new pathotype, VIII [102]. The symptoms caused by strains of the same pathogenic type may also differ. For example, the BCMV strains 3PF and A1 are both pathogenic type PG I, but 3PF causes mild mosaic on "DW", while the A1 strain can cause systemic necrosis on "DW", causing plant death [103,104]. Clearly, not all pathotypes have been characterized, and new pathotypes may be discovered in the future [102–105].

Historically, BCMV isolates have been divided into two serotypes, serotypes A and B, based on serological reactions [106]. Serotype A includes pathotypes III and VI, which are strains that cause temperature-sensitive necrotic spots on bean plants carrying the *I* gene, including TN1, NL3, NL5, and NL8, whereas serotype B includes all isolates of other pathotypes, such as CH2, NL1, NL4, NL6, NL7, PR1, RU1, and US1-US10. Subsequent high-performance liquid chromatography (HPLC) and gene sequence analyses demonstrated that the serotypes A and B belong to different virus species [107]. McKern et al. [106] initially named members of the serotype A bean necrosis mosaic virus, which was later renamed the bean common mosaic necrosis virus (BCMNV) based on its typical symptoms and serological characteristics by the International Committee on Taxonomy of Viruses (ICTV). Recently, some soybean-infecting potyviruses, such as the blackeye cowpea mosaic virus (BICMV), azuki bean mosaic virus (AzMV), peanut strip virus (PStV), and cowpea-aphid-borne mosaic virus (CABMV), have been classified as strains of BCMV based on their serological characteristics and genome sequences [107].

There are many BCMV and BCMNV isolates; however, the identification of the BCMV strains in China and abroad has not been unified. Currently, there are eighty-six full-length genomes of BCMV in the GenBank database, including seven from cowpeas (*Vigna unguiculata*), one from yam beans (*Pachyrhizus erosus*), one from phaseolus riukiensis (*Vigna riukiensis*), twenty-eight from common beans (*Phaseolus vulgaris*), one from the common nandina (*Nandina domestica*), two from mung beans (*Vigna radiata*), one from sesame (*Sesamum indicum* L.), one from azuki beans (*Abrus precatorius* L.), one from tricuspid cudrania (*Cudrania tricuspidata*), thirty-two from soybeans (*Glycine max*), two from peanuts (*Arachis hypogaea*), one from hyacinth beans (*Lablab purpureus*), and one from purple bushbeans (*Macroptilium atropurpureum*) (Figures 2 and 3). A phylogenetic analysis showed that Chinese isolates of BCMV derived from soybeans, cowpeas, peanuts, sesame, and yam beans clustered into the same clade. Four cowpea isolates from China clustered with one soybean isolate, and one mung bean isolate clustered with another clade. The common bean isolates from India, South Korea, Colombia, Iran, Tanzania, and the United States clustered in the same clade as one cowpea strain from Iran. Another clade contained BCMV isolates from the common bean, cowpea, azuki bean, mung bean, purple bushbean, common nandina, and tricuspid cudrania. Members of this clade are widely distributed in the USA, Mexico, Australia, Germany, Iran, Tanzania, and Asian countries such as China, Japan, South Korea, and India. There are twenty-two full-length genomes of BCMNV in the GenBank database, including nineteen from the common bean (*Phaseolus vulgaris*), one from Fabaceae (*Fabaceae* Lindl.), and one from the benth (*Nicotiana benthamiana*). A phylogenetic analysis showed that these BCMNV isolates were clustered into two major clades: one clade contained the Indian isolate from the common bean, and the other contained the remaining BCMNV isolate. The BCMV lineage probably originated in South and East Asia. The BCMV sequence was first provided by Spain in 1992 [108–111], and the BCMNV sequence was first provided by the USA in 1995 [112]. In general, BCMV isolates from the same host exhibit a close genetic relationship. At the same time, the reported clustering between BCMV strains is closely related to the region, indicating a high frequency of gene communication between strains in the same country or region and vice versa.

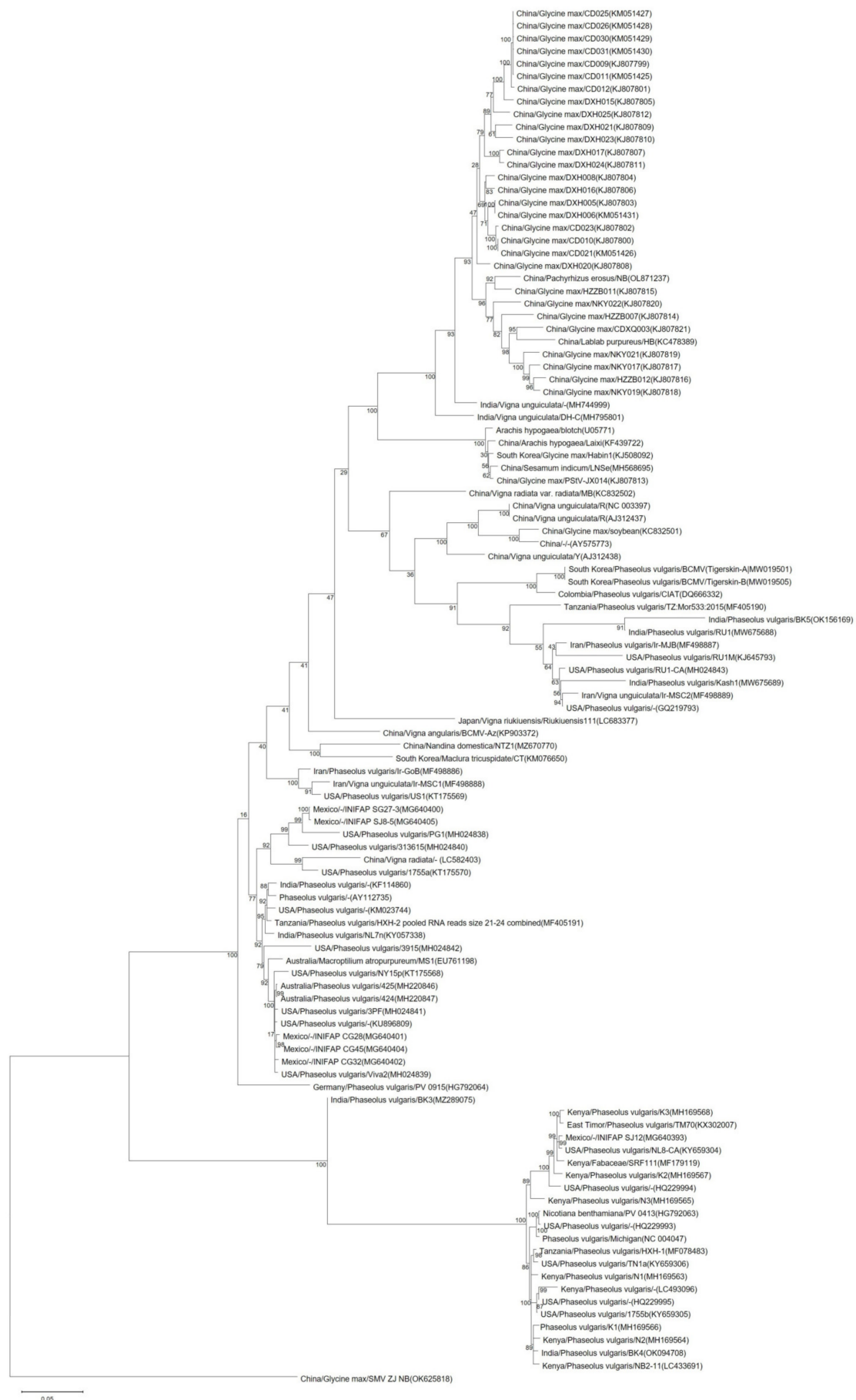


Figure 2. Maximum-likelihood phylogenetic tree for BCMV and BCMNV whole-genome sequences.

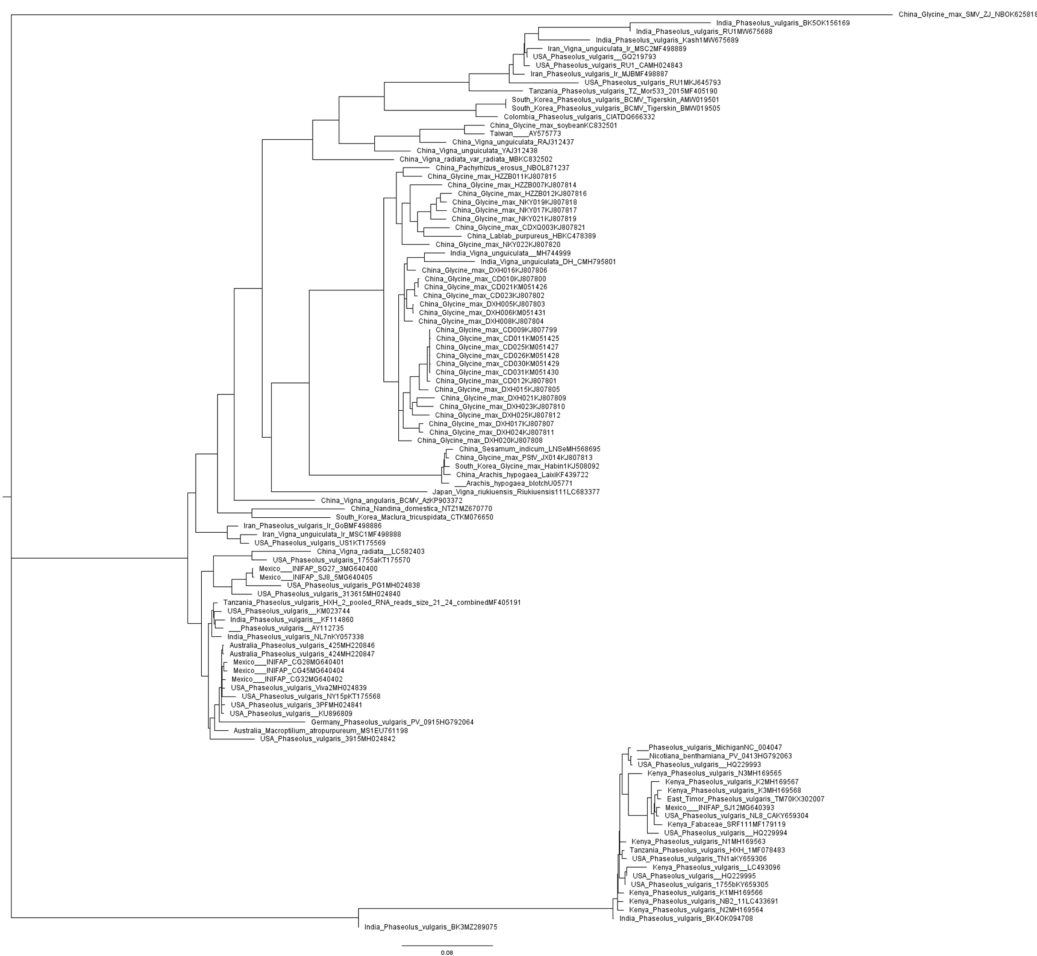


Figure 3. Phylogenetic tree produced through Bayes methods for BCMV and BCMNV whole-genome sequences.

3.2. Evolution

It is well known that mutation is the key factor driving the rapid evolution of RNA viruses and generating genetic diversity to adopt different hosts and environments for survival and transmission [113–118]. The situation is the same as that of BCMV: it has been shown that mutation is the major factor allowing BCMV to quickly overcome multiple resistance genes in the common bean [109,119,120]. Recombination is another major source of plant virus evolution, as it benefits viruses by allowing them to obtain new genetic information and optimize their existing biological characteristics through gene expression, increasing the possibility of breaking through host resistance to cause a successful infection [121]. Studies have shown that recombination between different BCMV or BCMNV strains is common [113,122,123]. For example, US10 is a recombinant isolate containing segments derived from the strains US1 or RU-1. Thus, interspecific recombination with other potyviruses is possible. For example, NL-3K is a natural recombinant strain of the BCMV strain RU-1 with the BCMNV strain NL-3D's derived sequence at the 5' end [22,23]. RU1-OR is a new strain produced by the recombination of BCMV RU-1 with another unknown member of the *Potyvirus* genus [124]. The current hotspots of BCMV recombination are mainly centered on the 5' end of the genome, including the 5' non-coding region (5'-UTR) and its adjacent P1 and HC-Pro protein-coding regions [22,23,124]. It has been shown that P1 is associated with the pathogenicity and host specificity of potyviruses, such as the plum pox virus (PPV) and SMV [125,126]. For instance, a recombinant SMV isolate containing a watermelon mosaic virus (WMV)'s 5'-UTR was able to infect *Nicotiana*

benthamiana, a non-host for normal SMV isolates [127]. However, the effect of recombination on the biological characteristics of BCMV and BCMNV requires further investigation.

4. Resistance Resources

4.1. Dominate Resistance

Hitherto, only one dominant resistance gene has been identified in common bean germplasm resources. The *I* gene, which was initially described in 1950 by Ali, confers a high resistance or even complete immunity to most strains of BCMV at temperatures below 30 °C [128]. Consequently, it has been rapidly used for breeding, and most commercially available common bean cultivars contain this gene. However, when the temperature exceeds 30 °C, BCMV induces temperature-dependent systemic necrosis symptoms. Therefore, the use of common bean cultivars harboring the *I* gene often leads to plant death in high-temperature areas, such as Africa and South America [91,92,96]. Studies have shown that BCMV can replicate and move intercellularly in *I*-gene-containing common bean cultivars without causing the plant to express any viral symptoms [91]. However, the molecular mechanisms underlying the temperature-induced necrosis induced by BCMV remain elusive. It has been proposed that the expression of the *I* gene is negatively regulated by temperature. As a result, the amount of *I* protein is too low to confer reliable resistance at high temperatures. Alternatively, immune responses induced by the *I* gene may be attenuated at high temperatures [91,92,96]. Moreover, an infection with BCMNV stimulates systemic necrosis in common bean cultivars harboring the *I* gene. Several closely linked molecular markers have been successfully developed in the BeanCAP diversity panel for breeding practices [15,16,21]. Additionally, six single nucleotide polymorphisms (SNPs) that are closely associated with the *I* gene were identified, and two kompetitive allele-specific PCR(KASP) and CAPS molecular markers were developed to accurately detect the *I* gene (Table 1). The *I* gene likely encodes a nucleotide-binding domain leucine-rich repeat-containing protein (NLR), which has been mapped to the end of the arm of chromosome 2. A total number of seven potential NLR genes were found in the resistance locus [21]. However, further studies are needed to clone the *I* gene and elucidate the mechanism against BCMV [15,21,129].

Moreover, several dominant resistance genes have been identified in several soybean cultivars [130–132]. In the soybean cultivars Suweon 97 and Raiden, the BCMV resistance locus was mapped to a region within the complex SMV-resistance locus Rsv1 on chromosome 13 [130,131]. However, the resistance gene in the soybean cultivar V94-5152 has been mapped to a genetic region in proximity to the SMV-resistance locus Rsv4. Rsv4 on chromosome 2 encodes an RNase H family protein with substrate specificity for double-stranded RNA, which confers resistance to all SMV isolates [133]. Therefore, Rsv4 confers resistance against both SMV and BCMV. Breeding resistant common bean cultivars using soybean-derived dominant genes via horizontal transfer is a promising direction for future breeding research.

4.2. Recessive Resistance

A total number of five strain-specific recessive resistance genes have been identified, namely *bc-1*, *bc-2*, *bc-3*, *bc-4*, and *bc-u^d* [18,92]. Scientists have long believed that the *Bc-1* locus contains a pair of highly linked alleles (*bc-1* and *bc-1²*). However, a recent study suggests that *bc-1* and *bc-1²* are actually the same resistance gene, and that different resistance phenotypes are caused by the *bc-u^d* gene [19]. The *bc-1* gene was mapped to chromosome 3 using a genome-wide association analysis, and three candidate, receptor-like kinases (RLKs) (Phus.003G038700, Phus.003G038700, and Phus.003G038800 1) have been proposed (Table 2). *bc-1* alone confers resistance to the NL-8 strain of BCMNV, but requires other recessive resistance genes, such as *bc-2* and *bc-u*, to resist other strains [19,134]. It has been proposed that the symptom induction and systematic movement, but not replication, of BCMV and BCMNV are limited in *bc-1* genotype common bean cultivars [103,105]. However, the identity of *bc-1* is still unknown at the present.

Genome-wide associations have mapped *bc-2* on chromosome 11, and several specific molecular markers have been developed to identify *bc-2* (Table 1). A sequence alignment between the common bean genome of G19833 v2.1 and UI 111 v1.1 showed that UI 111 v1.1 has a 10 kb deletion in the *Bc-2* locus [18]. A common bean with the mesoamerican genotype has a frameshift mutation at the *Bc-2* locus [18]. Both mutations involve the same gene, Phvul.011G092700, which encodes vacuolar protein sorting 4 (Vps4) AAA + ATPase required for the endosomal sorting complexes used for transport (ESCRT) (Table 2). ESCRT plays a critical role in many important physiological processes in plants, including endosomal trafficking to lysosomes, organelle biogenesis, DNA replication, protein folding, and proteolysis [135]. Although the *bc-2* gene has a minor influence on BCMV replication and intercellular movement, it can significantly inhibit the efficacy of the systemic movement of the virus [103]. However, *bc-2* alone cannot confer resistance to BCMV; it can confer resistance to most BCMV strains except for PG-V in the presence of *bc-4*, or resistance to all strains of BCMV (except PG-VI) and BCMNV in the presence of *bc-u^d* [18]. The cucumber homolog of the Vps4 AAA + ATPase was identified as a candidate gene for zymA192-18, providing recessive resistance against the zucchini yellow mosaic virus (ZYMV), a potyvirus infecting cucurbitaceous plants [136]. Interestingly, the host's ESCRT-related mechanism is also required for the replication of tomato bushy stunt virus (TBSV), an +ssRNA virus from the genus *Tombusvirus* of the family *Tombusviridae* [137]. Thus, it is possible that the ESCRT, including Vps4 AAA + ATPase, plays an essential role in the proliferation of varied RNA viruses.

The *bc-3* gene is the most well-studied recessive gene, and it encodes the eukaryotic translation initiation factor 4E (eIF4E) on chromosome 6 [17]. Several KASPar allele-specific SNP and CAPS molecular markers have been developed for the rapid identification of it in the common bean [93,95]. The eIF4E and its isomer, eIF(iso)4E, are essential for normal protein translation in all eukaryotes by binding to the 5' terminal cap of the mRNA [138]. In potyviruses, the 5' termini are covalently linked to the viral VPg protein, which responds to eIF4E or eIF(iso)4E binding for protein translation [139]. A mutation in eIF4E that blocks VPg interaction will result in the impediment of viral protein translation and the complete loss of infectivity [70,140–145]. Given its obvious application value in breeding, the interaction between VPg and eIF4E has been extensively studied, and the structure of the complex has been successfully determined recently [140–142,146–149]. Indeed, it is the main source of potyvirus resistance in many crops [150]. In the common bean, non-silent mutations in codons 53, 65, 76, and 111 of eIF4E have been identified in common bean cultivars carrying the *bc-3* gene, indicating that this region is required for the interaction between eIF4E and the VPg protein [93,95,151,152]. The combination of *bc-3* and *bc-u^d* can protect the common bean from infection by all BCMV strains, except BCMV-1755a [92,102]. However, potyviruses can overcome eIF4E-mediated resistance by introducing mutations in the VPg coding regions [139].

The *bc-4* gene was mapped on chromosome 5 of the common bean using a genome-wide association study [18]. Specific molecular markers have been developed to identify *bc-4* (Table 1). Phus.005G125100 is the best candidate gene for *bc-4*, which also encodes Vps4 AAA + ATPase. Similar to *bc-2*, *bc-4* cannot act alone, but requires other recessive resistance genes for resistance to BCMV. For instance, *bc-4* and *bc-3* are required for the bean variety IVT7214 to inhibit C1YVV, and *bc-1* and *bc-3* are required for the common bean cultivar USCR-8 to resist infection by C1YY, BCMV, and BCMNV (except for the strain NL-3K) [22,153]. Thus, *bc-4* may negatively regulate *bc-3* resistance through an unknown molecular mechanism [18].

The *bc-u* gene has always been recognized as a nonspecific assistant gene required for other recessive resistance genes [92,94]. Recently, Soler et al. remapped *bc-u* on chromosome 5 in a genome-wide association study (GWAS). Furthermore, this gene has been confirmed in the common bean varieties host group (HG)-3, -6, -10, and -11, but not HG-2, -4, -5, or -7, using specific molecular markers (Tables 1 and 3). After amending the genetic background of each HG cultivar, the *bc-u* gene was renamed *bc-u^d* [19]. Phvul.005G124100 was identified

as the candidate gene of *bc-u^d*, which encodes a basic Leucine Zipper (bZIP) transcription factor [19]. The *bc-u^d* gene in the Durango Diversity Panel (DDP) carries a non-synonymous mutation at codon 14 of the first exon (Pv05: 36,114,516 bases), which results in premature termination [19]. Reports have shown that bZIP is involved in regulating many important plant physiological processes and is a crucial component of the plant regulatory network in response to biotic stresses [154–157]. For instance, AtbZIP10 in *Arabidopsis thaliana* is a positive regulator of pathogen-induced hypersensitive response (HR) [154]. bZIP can also interact with the non-expressor of pathogenesis-related 1 (NPR1) in tobacco (*Nicotiana tabacum*), which is the receptor of salicylic acid and is required for resistance to the tobacco mosaic virus (TMV) in tobacco [158]. Moreover, *bc-u^d* can improve the resistance of the *bc-1* or *I* gene against BCMNV-NL3 and NL-8 in the common bean [19].

5. Future Prospects

The most cost-effective method for controlling BCMV infections is breeding resistant cultivars [159–163]. Indeed, the dominant *I* gene and recessive genes have been extensively used to breed resistant varieties [164–166]. However, pyramiding all the resistance into one cultivar is extremely time-consuming. The development of rapid DNA molecular markers, including Random Amplified Polymorphic DNA (RAPD), Restriction Fragment Length Polymorphism (RFLP), Amplified Fragment Length Polymorphism (AFLP), and Simple sequence repeat (SSR), will accelerate the identification of resistance genes in common bean populations for breeding purposes [167]. Viruses tend to escape resistance through the accumulation of mutations. Therefore, it is necessary to identify new dominant or recessive resistance genes in the common bean and related legumes. For instance, the translationally controlled tumor protein (TCTP) is required for the proliferation of several potyviruses [168,169]. Therefore, the counterpart in the common bean may also be a suitable candidate for resisting BCMV and BCMNV.

BCMV is naturally spread by aphids, and the control of the aphid population by chemical spraying is another method for controlling the outbreak of BCMV or BCMNV in the common bean [170]. However, special attention should be paid to the potential contamination of beans with pesticides and their influence on the environment. Recent developments in the biosafety of pesticides have shed new light on the control of aphids. Worrall et al. [171] developed a BioClayTM as the carrier of dsRNA that is a homolog to BCMV CP, which can effectively inhibit the spread of BCMV. Another study showed that 0.9% chitosan could effectively inhibit the population quality and change the feeding preference of *Aphis craccivora*, reducing BCMV transmission efficiency [172]. Biological prevention can effectively reduce the incidence and severity of viral diseases [27,173–175]. Seed transmission capacity is a key survival strategy for many potyviruses, including BCMV and BCMNV. However, the molecular mechanisms underlying seed transmission are largely unknown at the present. Therefore, it is necessary to investigate the molecular mechanisms of seed transmission and develop an efficient tissue culture method to obtain virus-free common bean seeds.

Table 1. List of resistance gene markers.

BCMV Resistance Alleles	ID Marker		Sequence	Reference
<i>I</i>	SW-13	F	CACAGCGACATTAATTTTCCTTTC	[176]
		R	CACAGCGACAGGAGGAGCTTATTA	
	S02_48908259	Fa	gcgggcCAAAGTGCTAGAGGCATGATCA	[19]
		R	TGGTATCATTATTGTGAAGTCAATG	
	BCMV_48289723_CAPS	Fb	gcgggcaggcggcAATCTTATGCTTGAAGCAGTGAAGC	
		F	AGGAGGAAGAACGGTGGTC	[15]
		R	TTTGGTGGTAATTTGAAATGG	
	OW13690	RAPD marker	CACAGCGACA	[177]

Table 1. Cont.

BCMV Resistance Alleles	ID Marker		Sequence	Reference
<i>bc-1</i>	SBD-5	F	GTGCGGAGAGGCCATCCATTGGTG	[134]
		R	GTGCGGAGAGTTTCAGTGTTGACA	
<i>bc-2</i>	S03_4203361	Fa	gcgggcTGGTCAGTTTGTCTTCCCTAACT	[19]
		R	TGCAGAAAGAGCTCAACTCGAAG	
	Pvmps4_del	Fb	gcgggcagggcgccGGTCAGTTTGTCTTCCCTAACA	[18]
		Fa	AGACCGTTTGCTAGGTTCAACA	
	Pvmit-2_C_del	R	TGTAGGCAATAAGGCGACGTTT	[18]
		Fb	AAATTATAAACATGTGTTGGCGAGC	
<i>bc-3</i>	ROC11	Fa	gcgggcagggccATTCTGCGTGATTGCCTCT	[18]
		R	CTTCAAAACGCACCTCAAGTATGA	
	eIF4E	Fb	gcgggcTCTGCGTGATTGCCTCC	[177]
		F	CCAATTCTCTTCACTTGTAACC	
		R	GCATGTTCCAGCAAACC	[17]
		F	ACCGATGAGCAAAACCCTA	
	OAD19690	R	CAACCAACTGGTATCGGATT	[178]
	OS13660	RAPD marker	CTTGGCACGA	
<i>bc-4</i>	Pvmit1_T_G	RAPD marker	GTCGGTTCCTG	[178]
		Fa	gcgggcCAATCTTATGCTTGAAGCAGTGAAAGT	
		R	ATTACAATAACATTCAACACCCGAGCAA	[19]
		Fb	gcgggcagggcgccAATCTTATGCTTGAAGCAGTGAAAGC	
		Fa	gcgggcCGAAAGCGTTCCTCTCTACAT	[18]
		R	GCGTGATGGCTTCCTTGATCTT	
<i>bc-u^d</i>	Pvbzip1_A_C	Fb	gcgggcagggcgccCGAAAGCGTTCCTCTCTACAG	[19]
		Fa	gcgggcTAGGAGAACTTGGTTTGTCTGAGTA	
		R	GCACTCCATAAGGGATGTGGT	
		Fb	gcgggcagggcgccGGAGAACTTGGTTTGTCTGAGTC	

Note: Fa and Fb: allele specific positive primers; F: forward primer; R: reverse primer.

Table 2. Candidate BCMV resistance genes in common bean.

BCMV Resistance Alleles	Location	Candidate Genes	Reference
<i>I</i>	Pv02: 48,908,259	Phvul.002G323000~Phvul.002G323,500 Phvul.002G323800	[15,21]
<i>bc-1</i>	Pv03: 4,203,361	Side-by-side receptor-like protein kinases (RLKs), Phvul.003G038700, Phvul.003G038800	
<i>bc-2</i>	Pv11: 9,272,542 -9,262,459	Phus.011G092700	[18]
<i>bc-3</i>	Pv06: 27,204,768	Phvul.006G168400	[176]
<i>bc-4</i>	Pv05: 36,225,550	Phus.005G125100	[18]
<i>bc-u^d</i>	Pv05: 36,114,516	Phvul.005G124100	[19]

Table 3. List of BCMV and BCMNV pathotypes.

Bean Varieties	Resistance Genes	PG-I	PG-II	PG-III	PG-IV	PG-V	PG-VI	PG-VII	PG-VIII
DW	/	+/+	+/+	+/+	+/+	+/+	+/+	+/+	+/+
Poncho (DDP041)	<i>bc-u^d</i>	+/+	+/+	+/+	+/+	+/+	+/+	+/+	+/+
RGLC	<i>bc-1</i>	-/-	+/+	-/-	+/+	+/+	+/+	+/+	-/-
RGLB	<i>bc-1, bc-?</i>	-/-	-/-	-/-	+/+	-/-	+/+	+/+	-/-
Sanilac	<i>bc-2, bc-4</i>	-/-	-/-	+/+	-/-	+/+	+/+	-/-	+/+
UI35	<i>bc-u^d, bc-1, bc-2</i>	-/-	-/-	-/-	-/-	-/-	-/-	+/+	-/-
IVT7214	<i>bc-3, bc-4, bc-2?</i>	-/-	-/-	-/-	-/-	-/-	-/-	-/-	+/+
Jubila	<i>I, bc-1</i>	-/-	-/-	-/-	-/-	-/-	-/-	-/-	-/-
Amanda	<i>I, bc-1, bc-?</i>	-/-	-/-	-/-	-/-	-/-	-/-	-/-	-/-
US1006	<i>I, bc-u^d, bc-2</i>	-/-	-/-	-/-	-/-	-/-	-/-	-/-	-/-
IVT7233	<i>I, bc-u^d, bc-2</i>	-/-	-/-	-/-	-/-	-/-	-/-	-/-	-/-

Note: Pathotype determination criterion: symptoms appear after each strain inoculation/TAS-ELISA test result. For example, if a symptom is presented and tested as BCMV-positive, it is marked as +/+, and vice versa, it is marked as -/-. *bc-?* indicates a helper gene other than *bc-u^d*, and work characterizing these other genes is in progress.

Author Contributions: M.T.: Writing original draft preparation; Literature search and chart making; X.F.: Writing—review and editing; funding acquisition. All authors made direct and intellectual contributions to the manuscript. All authors have read and agreed to the published version of the manuscript.

Funding: This research was funded by the Shanxi Province Returned Overseas Talents Program, grant number 2020-063, the National Natural Science Foundation of China, grant number 32202247, and the Natural Science Foundation of Shanxi of China, grant number 20210302123367.

Data Availability Statement: Not applicable.

Acknowledgments: We would like to thank KetengEdit (www.ketengedit.com) for its linguistic assistance during the preparation of this manuscript.

Conflicts of Interest: The authors declare no conflict of interest.

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