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REVIEW ARTICLE

Viral diseases in *Vigna* species - Impacts, management opportunities and future perspectives: A review

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Abstract

Pulses are the second-largest class of food crops worldwide, with around 1.58 million hectares under cultivation. represent. They are also excellent sources of protein. The major cultivated *Vigna* species in India include Blackgram [*Vigna mungo* (L.) Hepper], Greengram [*Vigna radiata* (L.) Wilczek], Cowpea [*Vigna unguiculata* (L.) Walp.], Moth bean [*Vigna aconitifolia* (Jacq.) Maréchal] and Adzuki bean [*Vigna angularis* (Willd.) Ohwi & H. Ohashi]. However, the yield of these crops is significantly reduced by viral diseases caused by a diverse range of viral strains. Notable viral diseases affecting *Vigna* species include yellow mosaic, cowpea severe mosaic, cowpea yellow mosaic, cowpea aphid-borne mosaic, cowpea golden yellow mosaic, bean common mosaic, leaf crinkle and leaf curl. The primary challenge in managing these viral diseases lies in effectively integrating the substantial knowledge accumulated, which is essential for developing genotypes with durable resistance to viral infections. Molecular markers and QTL (Quantitative Trait Locus) mapping are valuable tools for identifying genomic regions associated with viral disease resistance, aiding future breeding programs. This abstract provides an overview of each *Vigna* species, the viral diseases affecting them, and recent advancements in developing resistant genotypes. It also highlights systematic screening efforts within *Vigna* germplasm to identify various sources of viral resistance in *Vigna* species.

Keywords

management; molecular breeding approaches; transgenic breeding approaches; Vigna species; viral diseases

Introduction

Pulses are the world's second-largest class of food crops, grown on approximately 1.58 million hectares and producing 23.4 million tonnes of protein annually. India is among the leading producers and consumers of pulses, contributing 0.23 million tonnes from 0.66 million hectares (1). Within the Papilionaceae family, *Vigna* is one of the most significant genera, comprising up to 150 species primarily found in Africa and Asia (2). In India, the key *Vigna* species cultivated include black gram (*Vigna mungo* [L.] Hepper), green gram (*Vigna radiata* [L.] Wilczek), cowpea (*Vigna unguiculata* [L.] Walp.), moth bean (*Vigna aconitifolia* [Jacq.] Maréchal) and adzuki bean (*Vigna angularis* [Willd.] Ohwi & H. Ohashi).

In India, black gram is cultivated on 4.63 million hectares, yielding 600 kg/ha and producing 2.78 million tonnes (3). Asia contributes to 90% of the world's mung bean production (4), a crop known for its high protein content (25g/100g) (5). Green gram (mung bean), native to India, is grown on approximately 4.03 million hectares with a production of 3.01 million tonnes and a productivity of 783 kg/ha. It contains around 20-50% protein (5). Cowpea, primarily cultivated as a *kharif* crop, can also be grown as a *rabi* crop in peninsular India, with a protein content of 22-24% (5). Moth bean thrives in warm, dry climates, particularly in India's semi-arid and desert regions. It is widely found across India, from the north-eastern Himalayan foothills to Saurashtra in the west and from the north-western Himalayas down to Karnataka in the south (6). Adzuki bean, with a protein content ranging from 16.33% to 29.2% (7), is primarily grown in China, Korea, Japan and India, with an annual cultivation area of about 0.70 million hectares (8). These *Vigna* species are essential to the Indian diet.

India exported 775,024.48 metric tonnes of pulses globally, valued at USD 672.31 million during the 2022-23 fiscal year (9). Viral diseases are a significant biotic factor contributing to both production and economic losses (10). Table 1 lists notable viral diseases affecting *Vigna* species, including their genus, family and genome structure. Although various disease management strategies have been developed and implemented, complete resistance has not yet been achieved. Researchers must focus on advanced breeding techniques to improve resistance and yields against these viral diseases. This review highlights potential management strategies for viral diseases in different *Vigna* species, encouraging further research (Fig. 1).

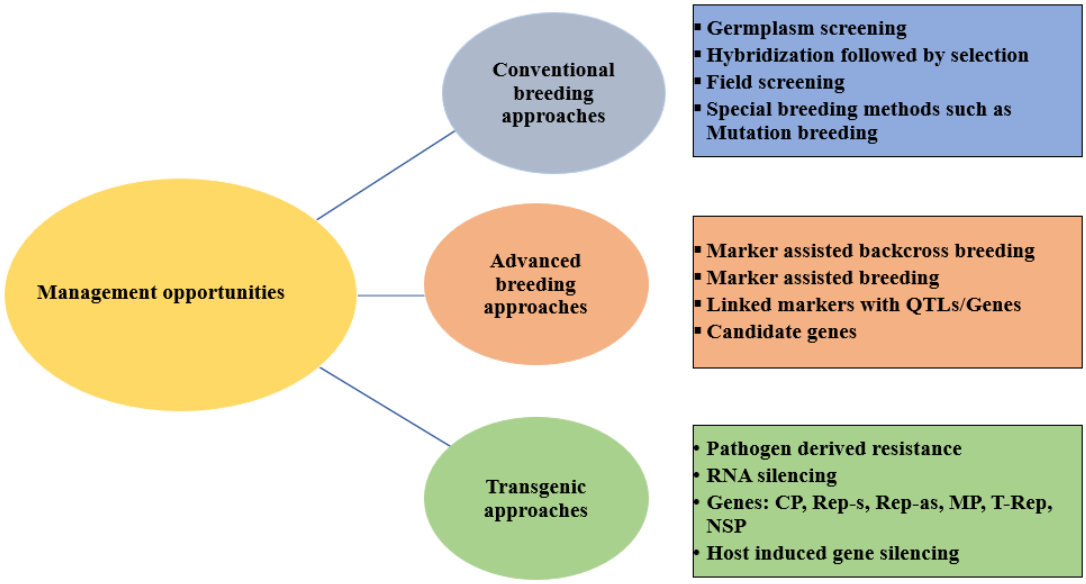

Fig. 1. Outline of management opportunities against viral diseases in *Vigna* species.

(CP-Coat protein, Rep- Replication protein, NSP-Nuclear shuttle protein, MP-Movement protein, QTL-Quantitative trait loci)

Table 1. Genus, family and genome structure of notable viral diseases of *Vigna* species

Crop	Viral Diseases	Viral species	Genus and Family	Genome	Reference
Blackgram and Green gram	Mung bean yellow mosaic	Mung bean yellow mosaic virus (MYMV)	Begomovirus, Geminiviridae	ssDNA	(82)
		Mung bean yellow mosaic India virus (MYMIV)	Begomovirus, Geminiviridae	ssDNA and β-satellite	
	Leaf crinkle	Urdbean leaf crinkle virus (ULCV)	Begomovirus, Geminiviridae	ssDNA	
	Leaf curl	Groundnut bud necrosis virus (GBNV)	Begomovirus,	ssDNA	
Cowpea	Cowpea mosaic	Cowpea mosaic virus (CPMV)	Comovirus,	ssRNA	
		Cowpea severe mosaic virus (CPSMV)		ssRNA	
		Cowpea aphid borne mosaic virus (CABMV)	Potyvirus, Potyviridae	ssRNA	
		Cowpea golden yellow mosaic virus (CGYMV)	Begomovirus,	ssDNA	
Moth bean	Yellow Mosaic	Mung bean yellow mosaic virus (MYMV)	Begomovirus,	ssDNA	
Adzuki bean	Mosaic	Bean common mosaic virus (BCMV)	Potyvirus, Potyviridae	ssRNA	

Occurrence of yellow mosaic virus diseases in black gram, green gram and moth bean

In 1960, yellow mosaic disease was first identified in mungbean and the virus responsible was named mungbean yellow mosaic virus (MYMV) (11). Losses due to the MYMIV strain range from 60% to 100% in Northern India, while MYMV has caused significant yield reductions in Southern India. MYMV and MYMIV strains are distinguishable based on nucleotide sequence identity (12) and in India, MYMV is readily transmitted by *Bemisia tabaci* (13). MYMIV also infects moth bean, initially discovered in Pakistan (14).

Genetics of yellow mosaic virus resistance and various resistance sources in black gram, green gram and moth bean

The main challenges in the genetic improvement of black gram include limited genetic variability, the absence of an ideal genotype suited for diverse cropping systems, susceptibility to biotic stresses and a shortage of high-quality improved seeds (15). Since the 1970s, plant breeders and virologists have worked on breeding for resistance to yellow mosaic virus (YMV) through host resistance. However, reports differ on the inheritance pattern of yellow mosaic disease (YMD) resistance. In black gram, YMV resistance has been described as monogenic dominant (16), governed by complementary recessive genes (17), monogenic recessive (18) and digenic recessive (19). To improve YMD resistance in blackgram and greengram, it is important to research and use the available genetic diversity (20). Resistant genotypes identified for various mosaic diseases in black gram and green gram are listed in Table 2.

Mutation breeding for yellow mosaic virus disease resistance in black gram and green gram

Mutations serve as a valuable tool for introducing variability and aiding in selecting mutants with desirable traits, including YMD resistance in crop plants, which can be achieved through mutation breeding (21). In the M3 generation, five green gram mutants (M5, M18, M26, M70 and M71) were identified as resistant to YMD (22). Prasad, Sarla and Vamban 2 are three mutant black gram varieties derived from the popular variety T9, known for their resistance to mung bean yellow mosaic disease (MYMD) (Table 3).

Molecular breeding approaches for YMV resistance in *Vigna* species

Marker-assisted selection is a vital tool in breeding programs, especially for traits that are difficult to evaluate phenotypically. The effectiveness of breeding programs that provide resistance to MYMV has significantly increased through studies on germplasm diversity, identification of markers associated with resistant genes and development of QTL maps using molecular markers (23). Simple Sequence Repeat (SSR) markers, known for their effectiveness in identifying varieties, hold substantial potential in genetic and breeding studies (24). Various SSR markers linked to YMV resistance, as identified in previous studies, include CEDG180 (25), CEDG141 (26), CEDG228, CEDG044, Vrd1 and STSbr1 (27) for MYMIV and CEDG185 (28) for MYMV. Additionally, SCAR markers such as CM 815, CM 9 (29), ISSR 8111357, YMV1 FR (30) and MYMVR-583 (31) are associated with MYMV resistance.

Table 2. Resistant genotypes identified in black gram, green gram and moth bean for various mosaic diseases

S. No	Crop	Diseases	Resistant genotypes	References
1.	Black gram	MYMV	IC 27026, IC 06088, UL 2, HPU 4, HPU 188, UH 80-26, IP 99-127 and PLU 62	(83)
			Vamban 7, Ujala (OBG-17), Pant U-31, Ujala (OBG-17), Prasad (B 3-8-8), Pant U-40, Pant U 84, Vamban 2, Prasad (B 3-8-8) and UPU 2	(84)
			Pant U-84 and UPU-2	(19)
			VBN-6, VBN-8, VBN-9 and VBN-10	(85)
			KKB 05011 (KKM 1)	(86)
		MYMIV	NP 16, PLU 62, PLU 63, PLU 131, PLU 158, PLU 277, IPU12-19, IPU13-5, K 66-110, K 66-188, DPU 88-31, IPU13-6, NP 19, NP 21, NDU 88-8 and VMR 84	(87)
2.	Green gram	MYMV	NM 94, ML 1628, CO-GG 930, CO-27, VPM 50, MH-565, VBN (Gg) 3, VBN (Gg) 4, Pusa Vishal, Bing Mung-2 and Bing Mung-1	(88)
		MYMIV	PDM 139, PDM 143, ML Nos. 131, 267, 337, NDM 88-14, PBM 14, PBM 27, IPM02-03, IPM410-9, IPM205-9, Pant M 4, MH 303, IPM409-4 and IPM205-7	(42)
3.	Moth bean	MYMV	PLMO 12, IC 36392, IC 129177, IC 129177, IC 129208, IC 36467, IC 129194, PLMO 30, IC 36096, IC 415152, IC 36573 and RMO 40	(89)

Table 3. Resistant mutant genotypes for MYMD in black gram and green gram

S. No	Crop	Diseases	Resistant genotypes	Reference
1.	Black gram	MYMD	Prasad and Sarla	(83)
			TU-94-2	(90)
			Vamban 2	
2.	Green gram	MYMV	Pant Moong 2, BM 4, Dhauli, ML 26-10-3, MUM-2	

CEL-I nuclease-based genotyping has been utilized to identify MYMV-associated SNPs on mungbean chromosomes 2, 5, 7, 9 and 10 (32) mapping resources are limited, bulked segregant analysis (BSA) becomes a crucial tool for labeling genes that lack a linkage map (32). For example, the SSR marker VR9 effectively differentiated resistant and susceptible bulks in a black gram F2 population from the T9 × LBG-759 cross (33). QTLs identified for MYMIV and MYMV resistance through composite interval mapping (CIM) and composite interval epistasis mapping (CIM-EPI) in black gram and green gram are detailed in Table 4.

QTL studies are critical in marker-assisted backcrossing (MABC), a precise method to introduce targeted genes into elite cultivars to enhance specific traits (34). Foreground selection helps confirm the transfer of desired QTLs, while background selection evaluates the recovery of the recipient parent genome. SSR markers showing polymorphism between parents are used at each backcross generation to calculate the recurrent parent genome (RPG) recovery percentage in the background selection (35). In black gram, foreground selection for MYMV resistance was conducted using six SSR markers from two key QTL regions found in linkage groups 2 (*qmymv2_60*) and 10 (*qmymv10_60*) (36).

Occurrence of urd bean leaf crinkle virus (ULCV) and urd bean leaf curl virus in black gram and green gram

Leaf crinkle disease, prevalent in Pakistan and India, can cause crop losses of up to 100%, depending on the season and variety affected (37). ULCV disease was first reported in Delhi in 1966 (38) and is transmitted by aphids (39). In India, mung bean and urd bean leaf curl diseases were initially observed in 1968 in Pantnagar, Uttar Pradesh (40). According to Nene, mungbean leaf curl disease has historically caused significant yield losses, reaching up to 40% in 33 districts of Uttar Pradesh. In southern regions like Andhra Pradesh, the disease has already become a serious concern (41).

Resistance sources of ULCV and urd bean leaf curl virus in black gram and green gram

In India, resistant sources for leaf crinkle and leaf curl diseases are limited. However, black gram genotypes IUP 11-02 and IUP 110-26 show resistance to ULCV (42). In Tamil Nadu, varieties VBN (Bg) 9 and VBN (Bg) 10 are also highly resistant to ULCV (43). Two black gram varieties, Mash 391 and Mash 479, developed through the pedigree method, demonstrate high tolerance against multiple diseases, including leaf crinkle virus (LCV) (44). Among greengram varieties, MLT-GG R-16-007, RME-16-12, RME-16-3, MLT-GG R-16-009 and COGG 1319 are resistant to ULCV (45).

The black gram genotype PU-31 showed the lowest incidence of leaf curl, with GBG-1 following closely (46). In a study 500 seeds were collected from PBNV-GG (peanut bud necrosis virus-green gram) and PBNV-BG (peanut bud necrosis virus-black gram) infected green gram cv. K-851 and black gram cv. LBG-20 revealed no seed transmission of the leaf curl virus in these crops, as seedlings showed neither typical PBNV symptoms nor tested positive in DAC-ELISA tests (47). Additionally, black gram varieties VBN (Bg) 9, VBN (Bg) 10 and VBN (Bg) 11, released by the National Pulse Research Centre in Vamban, exhibit resistance to urd bean leaf curl virus (43).

Table 4. QTLs detected for MYMIV and MYMV resistance by composite interval mapping (CIM) and composite interval epistasis mapping (CIM-EPI) in blackgram and greengram

S.No	Resistance	Crop	Population	QTLs	LOD score	LG	Marker interval	PVE (%)	References
1.	MYMV	Black gram	F_2	qMYMVD_60	4.56	10	CEDG180-CEDG116	21	(91)
2.	MYMV	Black gram x rice bean	F_9 and RIL	qMYMV4-1	6.07	4	VgSNP_04_32 - VgSNP_04_36	20.04	(92)
				qMYMV5-1	5.02	5	VgSNP_05_18 - VgSNP_05_19	15.02	
				qMYMV6-1	3.32	6	VgSNP_06_32 - VgSNP_06_33	10.11	
				qMYMV10-1	3.48	10	VgSNP_10_02 - VgSNP_10_03	11.24	
3.	MYMV	Black gram	$F_{2:3}$ and RIL	qmymv2_60	5.71	2	CEDAAG002-CEDG225-GMES4236	20.90	(93)
				qmymv10_60	6.98	10	cp05325-CEDG180-GMES4431	24.90	
4.	MYMIV	Green gram	$F_{2:3}$	qYMIV2.1	44.60	2	CEDG275–CEDG006	44.60	(94)
				qYMIV7.1	37.65	7	CEDG041–VES503	37.65	
5.	MYMIV	Green gram	F_{12}	MYMIVr7_104	4.7	7	v02a7	0.23	(95)
				MYMIVr8_48.8	4.6	8	mg3pat423	0.22	
				MYMIVr9_6.4	8.2	9	m4pcc585	0.36	
				MYMIVr9_25	16.4	9	9DMB158	0.59	
6.	MYMIV	Green gram	F_8 and RIL	qYMIV1	2.62	2	CEDG100–cp02662	9.33	(96)
				qYMIV2	2.54	4	DMB-SSR008–VR113	10.67	
				qYMIV3	3.42	9A	CEDG166–CEDG304	12.55	
				qYMIV4	10.00	2	CEDG100–cp02662	27.93	
				qYMIV5	2.55	6	CEDG121–CEDG191	6.24	
7.	MYMV	Black gram	RIL	qYMV2.1	7.44	2	CEDG020 – CEDG264	2.98	(97)
				qYMV2.2	6.96	2	CEDG 264 – CEDG008	2.96	
				qYMV5.1	13.83	5	CEDG 264 – CEDG008	1.64	
				qYMV8.1	10.56	8	CEDG 186 – CEDG271	1.63	
				qYMV9.1	10.30	9	CEDG 022 – CEDG166	1.51	

(LOD - Logarithm of the Odds, LG- Linkage Group, PVE- Phenotypic Variation Explained)

Occurrence of mosaic diseases in cowpea

Figure 2 shows a list of the most common viruses that reduce cowpea yield, as well as their vectors. Cowpea severe mosaic virus (CPSMV) possesses a single-stranded RNA genome. Distinguished from cowpea mosaic virus (CPMV) by (48) in 1964, CPSMV belongs to the family *Comoviridae*, genus *Comovirus* and is spread by chrysomelid beetles (*Cerotoma ruficornis* and *C. trifurcata*) (49). CPSMV is also seed-transmitted (50) and may cause up to 85% yield loss in seeds (51). Cowpea yellow mosaic virus, primarily an African virus, has occasionally been reported in the Americas (Suriname, U.S.A.) (52). An isolate from Suriname was identified as CPMV (48) but was previously known as cowpea yellow mosaic virus (CYMV) (53). CYMV, belonging to the family *Potyviridae*, genus *Potyvirus*, has a single-stranded RNA genome and is seed-borne at low levels (1-5%), easily transmitted by sap, with its primary vector being *Ootheca mutabilis* (52). CYMV can sometimes cause 5-10% yield losses (54).

Cowpea aphid-borne mosaic virus (CABMV), also from the family *Potyviridae*, genus *Potyvirus*, has a single-stranded RNA genome and is spread through the sap, seed (0-40%) and aphids (*Aphis craccivora*) (55). Cowpea aphid-borne mosaic virus can cause yield losses ranging from 13 to 87% in field settings (55). Cowpea golden yellow mosaic virus (CGMV) has a single-stranded DNA genome. Researchers have identified CGMV-like diseases in at least seven African countries (56). It remains unclear how similar diseases in Niger, Kenya, Tanzania, India and Pakistan relate to each other. Cowpea golden yellow mosaic disease (CGMD) is limited to northern India and is transmitted by whiteflies (57).

Genetics of mosaic resistance in cowpea and various resistance sources

CYMV resistance is controlled by a single dominant gene identified in *Dixielee Sel*, designated as the yellow mosaic resistance (YMR) genotype (58). Only when the resistance gene is homozygous recessive (*ymr ymr*) are tolerant and susceptible plants observed, as the dominant allele masks the effects of the three additive loci. CABMV resistance is managed by either a single dominant or recessive gene, with studies concluding that a single recessive gene confers CABMV resistance (59). In India, cowpea genotypes have shown resistance to CABMV under field conditions through sap inoculation, confirmed by DAS-ELISA testing (60). The resistant genotypes identified in cowpea for various mosaic diseases are listed in Table 5.

Molecular breeding approaches for mosaic disease resistance in cowpea

The current cowpea genetic map comprises 11 linkage groups (LGs) totalling 2,670 cM, with an average marker spacing of approximately 6 cM. This map contains 242 AFLP and 17 RFLP markers related to disease and pest resistance (61), as well as 133 RAPD, 39 RFLP and 25 AFLP markers from the original map (62). CPSMV resistance has been mapped to LG3 in cowpea (61). Identified QTLs linked to SSR markers AG1/AF48383 (AGB1), VM31 and VM1 show resistance to cowpea yellow mosaic virus (63). These three SSR loci are closely linked to a QTL on linkage group 2, with the VM31 locus associated with the QTL within a 95 % confidence interval covering 19 cM. CPMV resistance was found to be strongly correlated with six SNP markers (C35069548_1883, scaffold66293_6549,

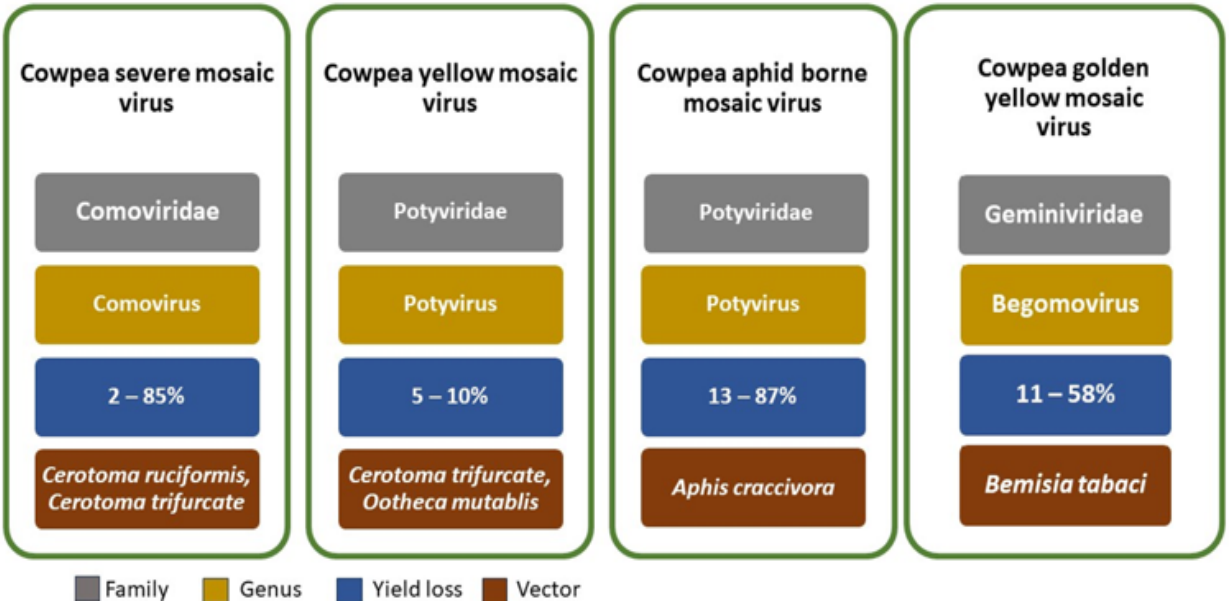

Fig. 2. Mosaic diseases and their vectors in cowpea.

Table 5. Resistant genotypes identified in cowpea for various mosaic diseases

Crop	Viral Diseases	Resistant genotypes	Reference
Cowpea	CYMV	TPTC 29, DC-15, JCPL-11, JCPL-18, JCPL-44, JCPL-45, JCPL-87, Pant Lobia 3, Pant Lobia 4, Pant Lobia 5, UPC 622, UPC 628, and KBC-9	(98)
	CABMV	IT86F-2014-1, IT86F-20895-1, TC1-6-10-1, CO6, IC521495, TC1-6-9-E, TC501-1-4, TC503, TC605, TCM418SDT, PGCP12, RC101 and TC99-1	(60)
	CGYMV	VRCP-4, VRCP-6	(99)

scaffold65342_6794, scaffold95805_2175, C35081948_540 and scaffold17319_4417). The first three markers are linked to immune response, while the remaining three are related to hypersensitive response (64). For CGMV resistance, the gene was linked to three AFLP markers: E.AAC/M.CCC515 at 4.3 cM, E.AAA/M.CAG352 at 16.8 cM and E.AGG/M.CTT280 at 14.2 cM, with LOD scores of 50.4, 24.4 and 28.7, respectively. Additionally, AFLP markers E.AAA/M.CAG352 and E.AAC/M.CCC515 were used to develop eight SCAR markers (65).

Occurrence of bean common mosaic virus (BCMV) in adzuki bean

Bean common mosaic virus (BCMV) was first reported in the United States in 1917 and the associated disease was initially referred to as bean mosaic. In 1934, it was renamed Bean Common Mosaic to differentiate it from Bean Yellow Mosaic, which was caused by the Bean Yellow Mosaic virus (BYMV) (66). BCMV consists of two serotypes, both of which were identified by (67). Reports have documented BCMV infections in *Vigna angularis* plants from China, Korea and India (68, 69). The virus has been shown to cause severe symptoms in adzuki beans, and even mild or symptomless infections can reduce crop yield by up to 50% (70). The absence of resistance sources and QTLs against BCMV in adzuki beans underscores the need for advanced breeding approaches.

Expression of RNA silencing genes against BCMV in adzuki bean

Plants utilize RNA silencing as a defense mechanism against viruses, with miRNAs playing a significant role in regulating target gene expression in response to various stresses (71). Essential for the biogenesis of miRNAs are regulatory proteins such as Argonaute (AGO), Dicer-like (DCL) and RNA-dependent RNA polymerase (RDR). DCL, an endoribonuclease -active member of the RNase III family, cleaves double-stranded RNAs (dsRNAs) into small RNA duplexes containing 21-24 nucleotides. Small RNAs guide AGOs to their targets, leading to the cleavage of target mRNA, chromatin modifications like histone and/or cytosine methylation, or heterochromatin formation (72). Initiating and amplifying the silencing signal requires the conserved RDR catalytic domain (73).

Additionally, the expression patterns of DCL, AGO and RDR genes under biotic (*Podosphaera xanthii* and BCMV infection) and abiotic (drought) stress were analyzed using quantitative real-time reverse transcription PCR (qRT-PCR) to investigate their role in post-transcriptional regulation of gene expression. In BCMV-infected plants, the genes VaDCL2a/2b/2d/4 and VaRDR1b/1c/2/3 were downregulated, while VaDCL2c was upregulated (74). The varying expression patterns of these genes in response to BCMV infection suggest that RNA silencing plays complex roles in regulating adzuki beans ability to withstand BCMV.

Candidate-resistant genes and gene silencing techniques against viral diseases in *Vigna* species

The traits' QTLs have been mapped to known genes using the candidate gene approach. Resistance gene analog (RGA) markers, specifically YR4 and CYR1, are fully associated with MYMIV resistance in *Vigna mungo* and *Vigna radiata*, suggesting that CYR1 may be a candidate gene for disease resistance (75). In mungbean, CYR1 is found to be partially, but not completely, linked to MYMIV resistance, indicating that multiple loci, rather than a single gene, contribute to the transmission of resistance. Selected candidate differentially expressed genes (DEGs) associated with MYMV defense mechanisms-Vradi06g11500, Vradi09g06830, Vradi04g07450, Vradi08g04110, Vradi06g13520 and Vradi01g04820-were functionally validated through qRT-PCR analysis in green gram. The results revealed essentially identical expression patterns for each of the studied DEGs, consistent with the RNA-Seq findings and demonstrated that these DEGs were expressed in both susceptible and resistant genotypes (76).

Pathogenic derived resistance through transgenic approaches in *Vigna* species

Various transgenic approaches have been developed to confer virus resistance in crops, with pathogen-derived resistance (PDR) playing a crucial role in crop protection. Additionally, gene silencing technologies can be employed to express different functional or dysfunctional YMV genes in mung bean, such as coat protein (CP), protease, membrane protein (MP) and replicase (4). In one study, MYMIV clones were inoculated with a complementary-sense gene (AC1) encoding Rep (Replication) in mung bean plants, resulting in a 64% infection rate (77). However, the severity of symptoms and the percentage of infections decreased when co-inoculation was performed with the Anti-Rep construct. It was discovered that deletions of 75 and 150 amino acids at the N-terminal of the CP of MYMIV affect both pathogenicity and systemic spread (78). In mung bean, agro-inoculation of the CP hairpin construct (Cphp) has been shown to inhibit viral pathogenesis (79). An RNAi (hairpin) constructed under d35S, containing the coat protein gene of CABMV and the proteinase cofactor gene of CPSMV, was introduced via particle bombardment (80). In another study, researchers demonstrated that RNAi-derived resistance to MYMIV in cowpeas led to nearly complete resistance in plants where agro-infection of transgenic lines expressing AC2-hp and AC2+AC4-hp RNA was performed (81). Given the rapid advancements in biotechnology, PDR for managing YMD in *Vigna* holds great promise for the near future.

Prospects

The pursuit of resistance breeding and the use of modern breeding techniques to treat viral infections in *Vigna* are critical strategies for guaranteeing the sustainable and resilient production of these key plants . In addition to traditional resistance breeding, the use of advanced techniques such as Marker-Assisted Selection (MAS), Pathogen-Derived Resistance (PDR), Host-Induced Gene Silencing (HIGS) and Virus-Induced Gene Silencing (VIGS) has significantly accelerated the pace and precision of developing resistant genotypes within *Vigna* species. Furthermore, functional genomics has facilitated the identification of novel candidate genes that confer disease resistance, along with elucidating the molecular mechanisms underlying host plant resistance. In this era of rapid biotechnological advancement, PDR holds great potential for managing yellow mosaic diseases (YMD) in *Vigna* in days to come.

Conclusion

Climate change presents significant challenges to agroecosystems and global food security. To sustainably meet the growing demand for food, food production must be doubled. *Vigna* species represent an economically important group of crop plants, but the increasing incidence of various viral diseases is threatening their yield. Therefore, utilizing the genetic diversity within *Vigna* species is essential for developing resistance to these viral diseases. As technology advances and our understanding of plant-virus interactions deepen, gene silencing, gene editing and omics technologies are poised to play a pivotal role in shaping the future of crop protection and ensuring food security.

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Authors' contributions

GP and SD conceived the concept and wrote the manuscript. SD and AP gave ideas for the design of the diagrams and tables. GP designed the diagrams and tables. SD, SJ, SS, KP, AS and JS revised and finalized the manuscript. All authors read and approved the final manuscript.

Compliance with ethical standards

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