



Review

Breeding for Enhancing Legumovirus Resistance in Mungbean: Current Understanding and Future Directions

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Abstract: Yellow mosaic disease (YMD) affects several types of leguminous crops, including the *Vigna* species, which comprises a number of commercially important pulse crops. YMD is characterized by the formation of a bright yellow mosaic pattern on the leaves; in severe forms, this pattern can also be seen on stems and pods. This disease leads to tremendous yield losses, even up to 100%, in addition to deterioration in seed quality. Symptoms of this disease are similar among affected plants; YMD is not limited to mungbean (*Vigna radiata* L. Wilczek) and also affects other collateral and alternate hosts. In the last decade, rapid advancements in molecular detection techniques have been made, leading to an improved understanding of YMD-causing viruses. Three distinct bipartite begomoviruses, namely, *Mungbean Yellow Mosaic India Virus* (MYMIV), *Mungbean Yellow Mosaic Virus* (MYMV), and *Horsegram Yellow Mosaic Virus* (HgYMV), are known to cause YMD in *Vigna* spp. *Vigna* crops serve as an excellent protein source for vegetarians worldwide; moreover, they aid in improving soil health by fixing atmospheric nitrogen through a symbiotic association with *Rhizobium* bacteria. The loss in the yield of these short-duration crops due to YMD, thus, needs to be checked. This review highlights the discoveries that have been made regarding various aspects of YMD affecting mungbean, including the determination of YMD-causing viruses and strategies used to develop high-yielding YMD-resistant mungbean varieties that harness the potential of related *Vigna* species through the use of different omics approaches.

Keywords: Begomovirus; DoYMV; HgYMV; inter-specific gene flow; MYMIV; MYMV; omics approaches; YMD; *Vigna*

1. Introduction

Mungbean, or green gram (*Vigna radiata* L. Wilczek), is one of the most important commercial *Vigna* species, and its production has been steadily increasing in South and Southeast Asia. It is a very popular legume due to its short life cycle, high growth rate, and its use in numerous food recipes [1]. It has wide adaptability and low input requirements; moreover, it has the ability to fix atmospheric nitrogen into the soil through a symbiotic association with *Rhizobium*, which improves the soil health and enhances the yield of subsequently planted crops [2]. This crop has valuable nutritional and health benefits, especially in developing countries where malnutrition is a major issue. It represents a cheap source of vegetable proteins. Aside from its nutritional value, certain characteristics of mungbean have led to its consideration as a model leguminous plant. These include its small genome size [3], short life cycle, capacity to self-pollinate, and its close genetic relationship to other legumes. After the recent release of its assembled sequence, its genomic data has been widely used [3]. Various types of biotic and abiotic stresses cause severe yield loss to this crop [4]. Among biotic stresses, more than a dozen viruses like *Yellow Mosaic Virus* (YMV), *Urdbean Leaf Crinkle Virus* (ULCV), *Alfalfa Mosaic Virus* (AMV), *Bean Golden Yellow Mosaic Virus* (BGYMV), *Cowpea Yellow Mosaic Virus* (CPMV), and *Dolichos Yellow Mosaic Virus* (DoYMV) affect the pulse crops [5]. Among these, Yellow Mosaic Disease (YMD) caused by different viruses is a major economic significance. YMD caused by Begomoviruses (family *Geminiviridae*, genus *Begomovirus*) has emerged as the major threat. This disease currently is a major hazard to the flourishing production of mungbean in India, Sri Lanka, Pakistan, Bangladesh, Papua New Guinea, Philippines, and Thailand [6–9]. YMD was first reported in lima bean (*Phaseolus lunatus*) in 1940 [10] and was subsequently discovered in other plants, such as *Dolichos* bean (*Dolichos lablab*) [11,12] and mungbean [13]. It creates malformations in mungbean plants, which in turn inhibit photosynthetic efficiency and plant growth, ultimately leading to significant yield losses [7,14,15]. Khattak et al. [16] reported that YMD might lead to up to 32%–78% reduction in grain yield of mungbean. However, yield loss is more severe when the disease appears at early growth stages and may even lead up to 100% yield loss [17]. Several approaches are adopted to manage YMD through the control of its vector, whitefly, such as the use of insecticide sprays and the application of different plant extracts; however, achieving 100% disease management remains very difficult. The wide host range, virus variation, and quantitative inheritance make it more challenging to breed YMD-resistant mungbean cultivars. Deploying resistant genotypes is an effective way to mitigate this disease. As such, there is a dire need to develop mungbean genotypes that are highly resistant to the specific viruses that cause YMD in them. In 2013, Panigrahi et al. [18] reviewed the recent development of molecular markers linked to YMD in *Vigna* species, identifying *Yellow Mosaic Virus* (YMV)-linked markers that are useful for urdbean (also known as black gram, *Vigna mungo*). Recently, Naimuddin et al. [19] reviewed the current status of YMD in both mungbean and urdbean. They summarized the information on virus history, disease transmission, Polymerase Chain Reaction (PCR)-based virus species detection, and the management of YMV through various approaches, such as cultural practices, integrated nutrient management (INM), integrated disease management (IDM), integrated pest management (IPM), and deployment of resistant genotypes.

2. Genome Organization of YMD-Causing Legumovirus

Based on their genomic organization, begomoviruses can be classified as either bipartite (having two DNA components, DNA-A and DNA-B) or monopartite (with a single-stranded DNA-A-like component) [20]. Through the molecular analysis of YMD-causing viruses, four bipartite viruses, viz., *Mungbean Yellow Mosaic India Virus* (MYMIV), *Mungbean Yellow Mosaic Virus* (MYMV), *Horsegram Yellow Mosaic Virus* (HgYMV), and *Dolichos Yellow Mosaic Virus* (DoYMV), were found as having close relationships with each other. Among them, MYMIV, MYMV, and HgYMV are known to cause YMD in mungbean and other *Vigna* crops in the Indian subcontinent [21,22], and they also affect the majority of legume crops [23], whereas infection of DoYMV is still limited to only *Dolichos* bean [19,24]. Begomoviruses multiply membrane-based barriers, including midgut epithelial cells,

the apical plasmalemma of these cells, and the basal lamina of primary salivary glands [25] of whiteflies. They have geminate (twin) particles measuring 18–20 nm in diameter, apparently consisting of two incomplete icosahedra joined together in a structure consisting of 22 pentameric capsomeres and 110 identical protein subunits. These viruses are bipartite, having DNA-A and DNA-B components, and have genomes of about 2.7 Kb in size [26,27]. Both of their DNA components encode genes involved in replication, transcription activation, movement, and yellow mosaic symptom development [28–32]. Before 2015, it was believed that *DoYMV* is a monopartite virus, but recently, it was proven that it is also a bipartite virus [33]. Now, *MYMIV*, *MYMV*, *HgYMV*, and *DoYMV* are collectively termed as legume-infecting begomoviruses, i.e., “legumoviruses” [20], and can cause YMD in various pulses, including *Vigna* crops. However, *MYMV*, *MYMIV*, and *HgYMV* lead to YMD symptoms that are similar to those in mungbean; therefore, there is a need to confirm the specific virus(es) that lead to YMD development in mungbean, in order to accelerate the breeding of YMD-resistant genotypes [34]. Several DNA markers have been reported to be useful in detecting the presence of the specific virus leading to YMD development [35–40].

3. Host–Virus–Vector Interaction

YMD of mungbean is caused by different Begomovirus species (*Legume Yellow Mosaic Viruses*; *LYMVs*), transmitted by the polyphagous pest *Bemisia tabaci* in a persistent (circulative) manner, but not through the sap, seed, or soil. The insect vector and virus survive on different collateral and alternate hosts, including the main crop, which serves as the primary source of inoculum for yellow mosaic disease throughout the year (Figure 1).

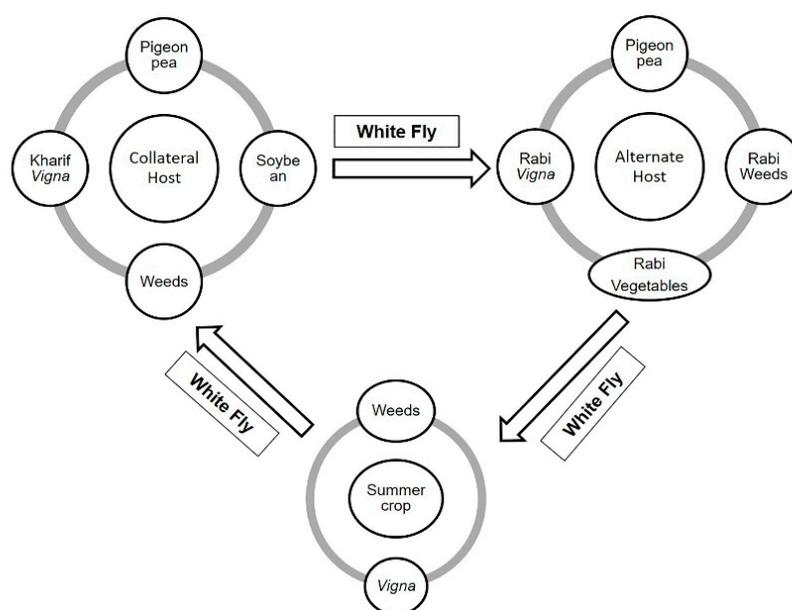


Figure 1. Presence of vector-virus at collateral and alternate hosts over the year.

The indigenous *B. tabaci* cryptic species Asia II-1 was predominantly found in Northern India, whereas Asia II-8 was predominantly found in southern India [41]. As whitefly has a latent period of less than four hours, the virus can be transmitted efficiently to the host by whitefly [13,42,43]. A single viruliferous adult can transmit the virus with an acquisition and inoculation access period of 24 h. The insect can obtain the virus after a single probe, and its transmission efficiency increases as it spends more time on the source plant of the virus as well as on the healthy mungbean plant [22]. The most efficient female and male adults in a population can retain their viruliferous nature for 10 and 3 days, respectively. Neither female nor male adults can retain infectivity throughout their life span. Nymphs of *B. tabaci* can acquire the virus from diseased leaves, but the virus does not pass through eggs of

B. tabaci. The viral particles penetrate the gut membranes and invade the hemolymph by crossing the epithelial cells of the whitefly digestive tract, which serves as a bridge between the gut lumen and the hemolymph that circulates around the body cavity surrounding the various insect organs. Viral particles reach the salivary glands and finally enter the salivary duct, from where they are ingested together with the saliva. Translocation of Begomoviruses from the digestive tract to the hemolymph and from the hemolymph to the salivary gland is thought to be mediated by still-unidentified receptors. The findings of Wei et al. [44] demonstrated that whitefly primary salivary glands, in particular, have cells around the secretory region that control the specificity of Begomovirus transmission. Extensive studies on host–virus–vector interaction have been carried out by several groups [45–47]. Some of these reports suggest the seed-borne nature of MYMV in urdbean [48] and mungbean [6], but the transmission of MYMIV and HgYMV through seeds has not yet been reported. Naimuddin et al. [19] performed a study on seed transmission of MYMV using the infected seeds of a mungbean cultivar, T44; however, they did not detect any yellow mosaic symptoms and were also unable to detect the presence of virus using PCR primers. They observed that YMD couldn't be transmitted by seeds because of the active release and high concentration of endonucleases in seeds during the germination period, which are unfavorable to the YMD-causing DNA viruses, as they cannot survive at high concentrations of endonucleases. Likewise, sap inoculation as a means of YMD transmission had been ruled out by Nair [45].

4. Host Range and Disease Symptoms

Aside from mungbean, these YMD-causing viruses have adopted a wider range of hosts and consequently are able to survive on various alternate and collateral hosts. The alternate and collateral hosts of LYMVs are listed below (Table 1).

Table 1. Collateral and alternate hosts of *Legume Yellow Mosaic Viruses* (LYMVs).

Viruses	Hosts	References
MYMV	Pigeon pea (<i>Cajanus cajan</i>)	[49–51]
	Urdbean (<i>V. mungo</i>)	[49,51]
	Soybean (<i>Glycine max</i>)	[49,51,52]
	Common bean (<i>Phaseolus vulgaris</i>)	[49,51]
	Horse gram (<i>Macrotyloma uniflorum</i>)	[49,50]
	Cowpea (<i>Vigna unguiculata</i>)	[50]
	<i>Nicotiana benthamiana</i>	[50]
	<i>Croton bonplandianum</i>	[50]
	<i>Euphorbia geniculata</i>	[50]
	<i>Parthenium hysterophorus</i>	[50]
	<i>Malvestrum cormandelianum</i>	[50]
<i>Acalypha indica</i>	[50]	
<i>Alternanthera sessilis</i>	[50]	
MYMIV	Common bean (<i>P. vulgaris</i>)	[53]
	Lima bean (<i>P. lunatus</i>)	[53]
	Pigeon pea (<i>C. cajan</i>)	[54]
	Soybean (<i>G. max</i>)	[55–57]
	Urdbean (<i>V. mungo</i>)	[58–60]
	Wild mungbean (<i>V. radiata</i> var. <i>sublobata</i>)	[36]
	Wild urdbean (<i>V. mungo</i> var. <i>silvestris</i>)	[35]
	Cucumber (<i>Cucumis sativus</i>)	[61]
<i>Ageratum conyzoides</i>	[37,60,62]	
<i>Corchorus olitorius</i>	[37,60,62]	
<i>A. sessilis</i>	[37,60,62]	
HgYMV	Common bean (<i>P. vulgaris</i>)	[63]
	Pole bean (<i>Phaseolus coccineus</i>)	[63]
	Soybean (<i>G. max</i>)	[63]
	Lima bean (<i>P. lunatus</i>)	[63]
	Rice bean (<i>Vigna umbellata</i>)	[63]
Moth bean (<i>Vigna aconitifolia</i>)	[21,63]	

The first visible symptom of YMD is the appearance of yellow and green irregularly shaped areas on the infected leaves of the host plants. The leaf size is gradually reduced, which is followed by complete yellowing and drying (Figure 2). The number of flowers and pods in severely infected plants also become drastically reduced. Another symptom is that infected plants produce fewer pods with small and shriveled seeds [64].

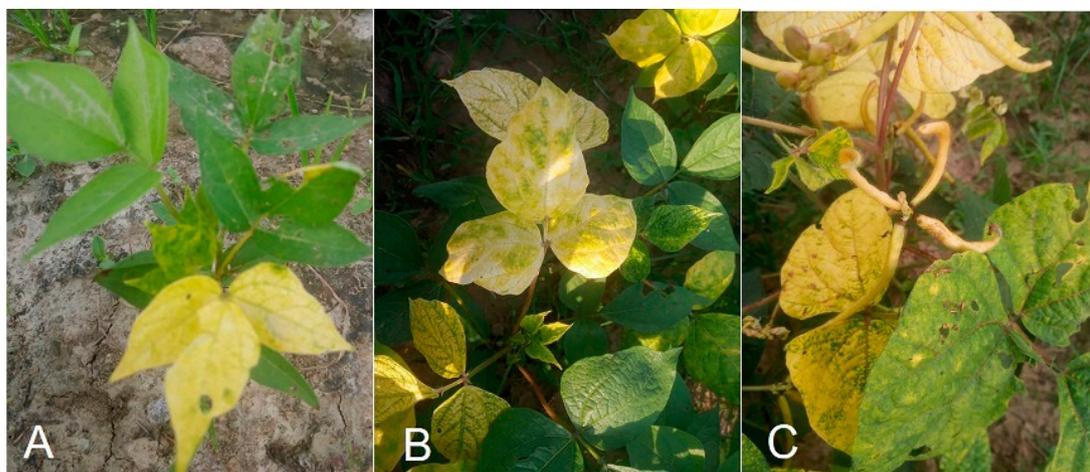


Figure 2. Various stages of mungbean plants; (A) seedling, (B) vegetative, (C) pod filling stage infected by *Mungbean Yellow Mosaic India Virus*.

5. Host-Pathogen Resistance

Due to the involvement of whitefly in spreading YMD from plant to plant, the population load of whitefly and its feeding behavior are important. Studies on the different aspects of YMD-causing viruses, host plants, and vector behavior may aid in developing disease mitigation strategies.

5.1. Physical Basis of Resistance

Identification of plant morphology may be made to manage the whitefly population and to aid the development of non-preferable cultivars. These traits may help in managing diseases that are transmitted by insect vectors. The morphological features of these crops may inhibit the feeding and oviposition of the vector(s). Hairiness is one of these inhibitory features that may be utilized in developing insect-resistant varieties of crops [65,66]. Long and dense hairs may help to reduce the whitefly infestation, which results in the low infection of YMD. Thus, it will be very interesting to transfer the hairiness phenotype into YMD-susceptible cultivars in an attempt to develop insect-resistant varieties. Fatokun and Singh [67] reported that wild accessions of *Vigna* spp., including *V. pubescens*, confer some degree of insect resistance in cowpea. Likewise, many researchers also worked on the hairiness trait and reported the possibility of developing insect-resistant varieties in various crops, viz., urdbean [68], cowpea [66], alfalfa (*Medicago sativa*) [69], and soybean [70]. Singh et al. [71] studied the hair density in mungbean plant parts, but they did not test the effect of hair density toward conferring insect-resistance or on the disease. Comprehensive research on hairiness is required in order to obtain a deeper understanding of the genetics involved, and its subsequent incorporation into related wild *Vigna* species. Moreover, the effects of hairiness on behavior, oviposition, and the whitefly life cycle will give more comprehensive ideas on preventing YMD transmission.

Inbar and Gerling [72] studied leaf characteristics, along with certain constitutive and induced chemical profiles (i.e., defensive and nutritional elements), that are involved in the regulatory mechanism of whitefly. One of the important resistance factors is the presence of trichomes, which potentially affects adult insect oviposition and immature insect attachment. Feeding of whiteflies may vary depending on the angle of trichome to the leaf surface, as well as varying based on leaf color, length, and plant variety [73]. Leite et al. [74] reported a non-occurrence of oviposition by *B. tabaci* on

the apical leaves of eggplant with high trichome density. A significant negative correlation between the number of leaf hairs per unit area and whitefly count has also been reported in pumpkin [75]. On the contrary, several authors have found that in cotton, the trichome density was positively correlated with the whitefly population [76,77]. Whitefly-resistant genotypes of mungbean possess thinner leaf lamina in comparison to susceptible genotypes [78]. A positive correlation between leaf trichome density and resistance to *B. tabaci* was also recorded in black gram [79]. *Cotton Leaf Curl Virus (CLCuV)*-resistant cotton cultivars were reported to have high epicuticular wax content on their leaves as compared to susceptible cultivars [80]. Young leaves of cotton have stem terminals with a high number of trichomes compared to those of older leaves; moreover, it was also reported that young leaves were infested with few silver leaf whitefly eggs, nymphs, and adults [81]. Similarly, a negative correlation between leaf trichomes and the whitefly population was reported in brinjal [82].

5.2. Biochemical Basis of Resistance

Wynd [83], Selman et al. [84], Bozarth and Diener [85], and Chhabra et al. [86,87] performed studies on peach, tomato, tobacco, mungbean, and urdbean. They recorded significant differences in reducing and non-reducing sugars, total phenols, and free amino acids between virus-infected and virus-free plants. Biochemical analysis of the contents of the resistant genotypes revealed that the percentages of reducing and non-reducing sugars and amino acids in the resistant genotypes were significantly higher compared to those of the susceptible genotypes [88].

A higher amount of total phenols was observed in black gram cultivars resistant to *MYMV* than in susceptible cultivars [87]. Thind et al. [89] reported an increased amount of total phenols in *MYMV*-infected mungbean compared to that in healthy plants. An increase of total phenols was observed in *YMV*-infected resistant and susceptible soybean cultivars compared to healthy cultivars [90]. Likewise, high phenolic content was also observed in yard long beans (*Vigna unguiculata* ssp. *sesquipedalis*) infected with *MYMV* [91]. Mali et al. [92] reported a significant increase in total phenols with an increased level of *YMV* infection in moth bean. Infection with *YMD*, in general, tends to decrease total protein content in susceptible mungbean. Total protein content was observed to be higher in the healthy leaves of *YMV*-resistant urdbean varieties than in the highly-susceptible and susceptible varieties [93]. Total soluble proteins increased with increasing levels of *YMV* infection in *YMV*-susceptible moth bean [92] and mungbean genotypes [94].

A significant reduction in chlorophyll and carotenoids has been reported in black gram and moth bean plants infected with *MYMV*, but the reduction in total chlorophyll was higher in susceptible genotypes [92,95]. Thind et al. [89] recorded a decrease in total chlorophyll content in *MYMV*-infected mungbean plants compared to healthy plants. A reduced amount of chlorophyll was observed in the *MYMV* infected mungbean cultivar ML-267 [96]. Sinha and Srivastava [94] reported low levels of total chlorophyll, chlorophyll a and b, in *MYMV*-infected mungbean plants than in the healthy plants. Total chlorophyll, as well as chlorophyll a and b, were higher in healthy yard long bean compared to *MYMV*-infected plants [91]. Hemida [97] reported a reduction of photosynthetic pigments in broad bean (*Vicia faba*) and common bean (*P. vulgaris*) infected by the *Bean Yellow Mosaic Virus*. Increased peroxidase (PO) and PPO activities were recorded in *MYMV*-infected cultivars of mungbean, H45 (susceptible), and LM 170 (resistant).

YMD-resistance in plants may be altogether attributed to obstruction of viral invasion in the plant cell wall, restriction in viral DNA replication, and early onset of antioxidant defense responses. Chakraborty and Basak [59] observed that the accumulation levels of the early as well as late expressed genes/transcripts of *MYMIV* were low and high in the resistant and susceptible plants, respectively; whereas an opposite response was exhibited by membrane stability index (MSI). A decrease in the malondialdehyde levels along with an increase in the activities/levels of different antioxidant enzymes, total phenol, and H_2O_2 was also noted during the early stages of infection in the resistant plants, which acts in various signaling pathways by activating the defense-related genes, which in turn leads to local or systemic resistance. Accumulation of ROS prevents the invading of the pathogen by cross-linking

with lignin, proteins, and phenolic compounds [98], which leads to an oxidative burst of the invading pathogen [99]. It also leads to distortion of the plasma membrane and causes nucleic acid degradation. Likewise, phenols are converted to defensive substances, which restrict the invasion of pathogens [100] and lead towards enhancing host resistance. The inhibition of *ac1* and *ac2* genes have been observed in resistant urdbean plants indicating the defensive mechanism against virus pathogen [59].

5.3. Genetic Basis of Resistance

Before the genomic era, several conventional studies suggested the monogenic inheritance of YMD resistance [101–106], with modifiers [68,107,108]. The involvement of complementary recessive genes, as well as the effects of digenic inhibitory gene interaction in governing resistance, have also been reported [104,108–112]. However, the successful development of YMD-resistant varieties through conventional approaches is still difficult due to the complex mechanisms behind YMD resistance [113]. The inheritance of different *LYMVs* is listed in Table 2.

Table 2. The inheritance pattern of different *LYMVs*.

Disease	Cross Combinations	Population Type	Population Size	Inheritance Pattern	References
Yellow Mosaic Disease (YMD)	NM 1-32-1 × NM 6-68-2	F ₂	150	Two major genes with additive effects	[114]
<i>MYMIV</i> Disease	KPS2 × NM10-12-1	RILs	122	QTL	[17]
	BM1 × BM6	F ₂	–	QTL	[112]
<i>MYMV</i> Disease	NM92 × VC2272	F ₂	300	Single recessive gene	[115]
	6601 × VC2272	F ₂	240	Single recessive gene	[115]
	6601 × Pusa Baisakhi	F ₂	240	Single recessive gene	[115]
	VC3902A × NM92	F ₂	340	Single recessive gene	[115]
	VC3902A × ML-5	F ₂	360	Single recessive gene	[115]
	NM92 × Pusa Baisaki	F ₂	220	Single recessive gene	[115]
	VC 1560D × 6601	F ₂	400	Single recessive gene	[115]
	VC 1560D × NM92	F ₂	400	Single recessive gene	[115]
	NM 92 × NM 98	F ₂	–	Single recessive gene	[116]
VBN(Gg)2 × KMG189	F ₂ /RILs	–	–	Single recessive gene	[51]

As YMD is a vector-transmitted disease, field screening of the breeding materials may sometimes be misleading due to the high variability in the causal viruses. In this situation, YMD-linked markers can be proven as being more useful for varietal screening and early generation selection against virus-specific YMD. Before the availability of the whole-genome sequences of the YMD-causing viruses, the disease was simply known as “yellow mosaic disease.” Several researchers used common terms, such as YMD or *MYMV*, to describe the disease without the proper molecular detection of virus species [117]. The genome sequences clearly distinguished between these disease-causing viruses. Therefore, the diagnosis of a specific virus that leads to YMD development, and the cross-talk that occurs among these viruses requires special attention. Several attempts have been made to tag markers needed to accelerate the marker-assisted breeding (MAB) program in *Vigna*. The molecular evidence suggests the involvement of quantitative trait loci (QTLs) in governing *MYMIV*-resistance in mungbean. To date, seven QTLs associated with four linkage groups, namely, LG2, LG4, LG6, and LG9, have been identified as being involved in *MYMIV*-resistance in mungbean [17,112]. The involvement of a single dominant gene in governing *MYMIV*-resistance in black gram [118] and soybean [119] have been reported. Likewise, the single monogenic inheritance of *MYMV*-resistance has also been reported by Singh and Patel [101], Sandhu et al. [120], Malik et al. [102,121], Malik [122], Saleem et al. [103], and Khattak et al. [16]. The involvement of two recessive genes in *MYMV*-resistance in mungbean has also been reported [123,124]. A recent report by Sai et al. [51] indicated the monogenic inheritance pattern of *MYMV*-resistance in mungbean. Finally, several groups have attempted to tag the resistance gene and develop linked primers to accelerate MAB in mungbean and its related *Vigna* species.

6. Breeding Strategies for Enhancing LYMV Resistance

6.1. Pathogen Identification

After the availability of the whole-genome sequences of LYMVs, several PCR-based diagnostic markers have been developed to diagnose the specific virus leading to YMD, which is helpful in detecting and determining the virus species that lead to YMD development (Table 3). ICAR-Indian Institute of Pulses Research, Kanpur, India has developed a specific virus detection kit, which is able to simultaneously detect the presence of all four legumoviruses, i.e., MYMIV, MYMV, HgYMV, and DoYMV [35,36].

Furthermore, these four YMD-causing LYMVs are well characterized, and their sequence information is available in the public domain. Based on sequence information, we can try to get restriction maps and use different combinations of enzymes to see the differential pattern obtained from these YMD-causing viruses to confirm the causal species. For example, *XbaI* is the non-cutter of the *DoYMV* genome, and complete digestion of DNA with *XbaI* will give an idea about the presence or absence of *DoYMV*, while subsequently confirming the presence of the other three viruses. Likewise, *PmeI*, a cutter of *MYMIV*, but non-cutter of *MYMV*, can confirm the absence of the *MYMV* genome. Similarly, another enzyme *HindIII* can also be used, which is a single cutter of *MYMIV* and a multiple cutter of *HgYMV*. In this case, the presence of a single linear fragment indicates a *MYMIV* infection, whereas the appearance of multiple fragments indicates *HgYMV* as the cause of YMD development. PCR-based detection and restriction digestion analysis will help in detecting the virus species that lead to YMD and, thus, help in developing virus-specific YMD-resistant cultivars (Figure 3).

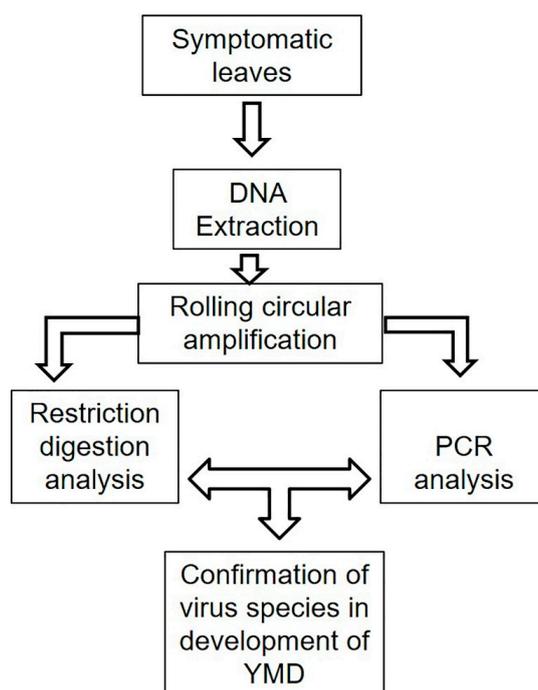


Figure 3. Detection of virus species in the development of yellow mosaic disease.

In addition, many more techniques have been made available to detect the virus. Suruthi et al. [12] suggested using of DAS-ELISA for detection of the virus. Based on the results of DAS-ELISA, they reported the seed-borne nature of *DoYMV*. Similarly, DAS-ELISA and immunosorbent electron microscopy have been performed to detect the presence of virus particles by Kothandaraman et al. [125] in black gram.

Table 3. PCR based molecular diagnostics of viruses causing *LYMV*s.

Primer Name	Accession Number	Forward-Sequence	Reverse-Sequence	Product Size (bps)	Detected DNA	References
NM 1 (AV1P) F/NM 2 (AV1P) R	FJ821189	GTA TTT GCA KCA WGT TCA AGA	AGG DGT CAT TAG CTT AGC	1000	MYMIV/DNA A	[35,37]
AC1P	FJ663015	AGT TGA TAT GGA TGT AATAGC3	ACA AAA ACG ACT TCA AATATG CCA A	1100	MYMIV/DNA A	[35,37]
MYMIV-MP	FJ663015	ATG GAA AAT TAT TCA GGT GCA	CTA CAA CGC TTT GTT CAC ATT	900	MYMIV/DNA B	[35,37]
AC2P	FJ663015	AGC TAA TGA CCC CTA AAT TAT	GAG TAC TTG GAT GAA GAG AAC	480	MYMIV/DNA A	[35,37]
AC3P	FJ663015	TTA TGA TTC GAT ATT GAA TTA ATA	CTG AAG TGTGGG TGT AGC TAT	450	MYMIV/DNA A	[35,37]
AC4P	FJ663015	CAA ATT ACAATT TAA GTT ATG	ACT TCT AGCCTT GTC AAC ACC AG	390	MYMIV/DNA A	[35,37]
MYMV-Coat protein (CP)	AY271896	ATG GG (T/G) TCC GTT GTA TGC TTG	GGC GTC ATT AGC ATA GGC AAT	1000	MYMV/DNA A	[35,37]
MYMV-MP	AY271896	ATG GAG AAT TAT TCA GGC GCA	TTA CAA CGC TTT GTT CAC ATT	900	MYMV/DNA B	[35,37]
HYMV-CP	NC_005635	ATG CTT GCA ATT AAG TAC TTG CA	TAG GCG TCA TTA GCA TAG GCA	1050	HgYMV/DNA A	[35,37]
HYMV-MP	NC_005635	ATG GAG CAT TAT TCC GGT GCA	TTA CA(G/A) GGT TTT GTT TAC AGT	900	HgYMV/DNA B	[35,37]
DoYMV-CP	AY309241	CTG TGA AAT TTG TGC AGG	TAC GCG GTT GCG AAT ATG TAT	900	DoYMV/DNA A	[35,37]
Rep VI	AF361431.1	AATGTAAGGCGACTCATA	GAGAATTCACCGGTCGCGGGCA	566	MYMIV	[38]
CP	AY271896	ACACGAGCTCCTCTACCCCG ATATCGAATG	ACACGGATCCGTTGCATACAC AGGATTG	750	MYMV/DNA-A	[39]
Deng-F/R	–	TAATATTACC(GT)G(AT)(GT)G (AGC)CC(GC)C	TAATATTACC(GT)G(AT)(GT)G (AGC)CC(GC)CCCTCACA	530	MYMIV/partial DNA A	[40]
PAL1v1978/PAR 1c496	–	GCATCTGCAGGCCACATY GTCTTYCCNGT	AATACTGCAGGGCTTYC TRTACATRGG	1.5 kbp	MYMIV/partial DNA A	[40]
AV 494/AC 1048	FJ821189	GCC(C/T)AT(G/A)TA(T/C)AG (A/G)AAGCC(A/C)AG	GG(A/G)TT(A/G/T)GA(G/A)GCATG (T/A/C)GTACATG	500–600 bp	MYMIV/partial DNA A	[40]
AC-abut/AV-abut	MF818048.1	GTAAAGCTTTACGCATAATG	AAAGCTTACATCCTCCAC	2.7 kbp	MYMIV Full length DNA A	[40]
BV-abut/BC-abut	MF818046.1	CCAGGATCCAATGATGCCT	ATTGGATCCTGGAGATTCA	2.7 kbp	MYMIV Full length DNA B	[40]
RHA-F/AC-abut	–	TCAAGCTCCCGGTGCATGTTGCA	GTAAAGCTTTACGCATAATG	920 bp	MYMIV Right half of DNA-A	[40]
PAR1v772/1c1960	–	GGNAARATHHTGGATGGA	ACNGGNAARACNATGTGGGC	1.1 kb	MYMIV/DNA A	[40]

6.2. Effective Phenotyping Procedure

There are various methods adopted by several researchers for screening germplasm for disease resistance. The infector-row technique is routinely used for screening the germplasm against diseases under natural conditions. Singh et al. [117], Bhanu et al. [126], Suman et al. [127], Deepa et al. [50], Nair et al. [41], and Khaliq et al. [128] screened mungbean genotypes for *YMV*-resistance under natural conditions. The infector-row technique, grafting, and sap inoculation technique has also been demonstrated in *Dolichos* against the *DoYMV* [129]. Some of the researchers focused on the force-feeding methods for *YMV* screening [130]. Malathi and John [22] stated that viruliferous adults are more suitable for virus transmission due to their high transmission efficiency. Moreover, female adults are more efficient than males in spreading *YMD* [46].

The environmental conditions such as temperature, humidity, and rainfall also affect the life cycle of the whitefly as well as its disease inoculum load [21,131–133], therefore, the multiplication of whitefly in controlled conditions is more effective. This method also provides the natural environment and ideal condition for host–virus interaction. Nevertheless, the major limitation of the natural condition is that the appearance of the disease is highly affected by the environment as well as the population load of the insect vector. As for the transmission of *LYMV*s only through whitefly, the presence of primary inoculum on the alternate and collateral hosts is required for the dispersion of disease through the insect vector. It is also challenging to maintain the purity of the virus strain under the natural condition, as we know that the viruses have very high mutability, which leads to the evolution of new plant viruses in legumes [21]. Therefore, developing an efficient artificial inoculation technique using specific virus(es) at an early stage will prove to be more useful in phenotyping plant materials against the specific virus and breeding for enhanced resistance. Ariyo et al. [134] used wedge grafting, biolistic inoculation of total DNA extracted, and biolistic inoculation of the cloned virus DNA A+B genomic components to test virus transmission in cassava. Jamsari et al. [135] described a new, promising method, i.e., an injection technique for transmission of Geminivirus in chili, but they reported a maximum of 53% disease expression. They also documented other artificial techniques such as grafting [136,137], mechanical infection via wounding [138], agro-inoculation [139,140], and particle bombardment [141] for the transmission of DNA viruses. Due to the herbaceous nature of mungbean, the inculcation of viruses through grafting is very tedious. Injection techniques may require standardization to achieve easy inoculation. For the screening of breeding lines against viruses, the agro-inoculation methods are now gaining popularity because of their ability to easily incorporate a specific virus into the plant system [142]. This method provides a means to avoid the presence of multiple viruses and assures the presence of a specific pathogen of *LYMV*. Several researchers developed the agro-infectious clones of specific virus(es) and successfully used these to screen various pulses (Table 4). To supplement further, the studies of Kartikeyan [143,144] and Bashir [145] suggested molecular markers that can be used for the indirect selection of resistant mungbean genotypes.

Table 4. Agro-inoculation technique of artificial disease phenotyping against *LYMV*s.

Viruses	Legume Crop	References
<i>MYMV</i>	Mungbean	[15,51,131,146–148]
	Urdbean	[142]
<i>MYMIV</i>	Mungbean	[9,149–151]
	Urdbean	[9,58,149]
	Cowpea	[9,149,150,152]
	Pigeon pea	[54]
<i>HgYMV</i>	Moth bean	[21]

6.3. Durable Sources of *LYMV* Resistance

Tagging the source for resistance to any of the biotic and abiotic stresses is essential in order to get breed resistant cultivars. Several methods for identifying and characterizing resistance sources are now available. Phenotyping is the most important step in identifying donors for resistance. However, due to

the vector transmission of *LYMVs*, it is very difficult to screen the breeding materials or genotypes under natural climatic conditions. Conditions like temperature, humidity, wind, and velocity affect whitefly multiplication and movement, which may cause uneven population load and distribution of whitefly over the crop population. The whitefly population also depends upon the alternate and collateral hosts present in the nearby the experimental field. Sometimes, the whitefly may also spread more viruses at the same time, which can affect the target virus multiplication into the plant system and affect YMD expression. Therefore, artificial screening techniques are more useful in generating the appropriate data to tag the true resistance source(s) for the specific virus(es) leading to YMD development. Previously, most of the researchers, such as Singh et al. [117], used the spreader row technique to create sufficient inoculum loads that are used to screen the mungbean genotypes for YMD resistance. Some of the researchers [153,154] have emphasized multi-environment screening of the germplasm to discover the YMD-resistant genotypes. Because of constraints in natural screening and the chances of mixed infection, implementing an artificial screening technique is strongly needed to mitigate this issue. Several groups, including Jacob et al. [147], Gupta et al. [118], Sudha et al. [15], and Sai et al. [51], have used artificial screening techniques combined with agro-inoculation for the strict screening of breeding material against the specific YMD-causing viruses. In our opinion, agro-inoculation coupled with the spreader row and force-feeding techniques will give more precise information about the true resistant genotypes by confirming all the involved parameters, like the susceptible host, natural environment, and the presence of virus and vector. The development of resistant cultivars is the most efficient and economical way to manage YMD. Some efforts have been made towards the identification of resistant donors in mungbean [43,155], black gram [156,157], soybean [95], and cowpea [158]. Some of the wild relatives of mungbean, such as some accessions of *V. radiata* var. *sublobata* [159], *V. trilobata* [160], *V. mungo*, and *V. umbellata* [160], have been reported as resistant to YMD.

6.4. Exploiting *LYMV*-Linked Markers and QTLs

Before the release of the mungbean genome, the researchers frequently identified the transferable markers from other *Vigna* backgrounds and used these to detect genetic variation and for the mapping of several important mungbean traits, including YMD [161–163]. To date, seven QTLs have been reported for *MYMIV* resistance in mungbean, whereas none of the QTL involvement was detected for *MYMV*-resistance, which indicates a possibly monogenic inheritance pattern of the *MYMV* resistant gene. After the genome sequence of mungbean, it has been easier to design primer pairs based on the whole genome in order to detect the novel QTLs governing *MYMIV* resistance. This gives a clear idea about the location of QTLs involved in YMD resistance. All the QTLs for mungbean were detected by linkage analysis using cultivated mungbean genotypes. Nevertheless, we also need to adopt other approaches, such as association mapping, to detect more QTLs for multi-virus YMD resistance over the natural mungbean population. For broadening the genetic base of mungbean and for the development of highly resistant genotypes, we also need to study the inheritance of YMD resistance using some of its interspecific and wild relatives as a donor. This will aid in detecting the novel QTLs and enhancing the level of resistance for mungbean breeding. Tagging of those genes will accelerate the marker-assisted breeding with special attention to YMD resistance.

The development and identification of tightly linked markers will help in the indirect selection of material for breeding and enable the precise introgression of resistant genes required to develop the *LYMV*-resistant genotypes. Several research groups have attempted to tag the resistance gene and develop linked primers to accelerate MAB in mungbean and urdbean. The *CYR1* marker is reported as completely linked to *MYMIV*-resistance, whereas *YR4* is partially linked to *MYMIV*-resistance in mungbean and urdbean [14]. Likewise, *RGA-1* [164], *CEDG180* [118], *ISSR811* [165], and *YMV1* [166] were reported as tightly linked to *MYMIV* resistance in urdbean, whereas the linkage of these markers with *MYMIV*-resistance gene in mungbean is not observed. Identification of more tightly linked markers for *MYMIV*, *MYMV*, and *HgYMV* resistance is, therefore, required. After the validation of these linked markers, they may be used in marker-assisted breeding programs for developing

LYMV-resistant genotypes. The scarcity of studies on the genetics and genomics of *HgYMV* has led to the development of YMD research in mungbean, which opens the door for researchers to explore untapped areas. Attention should also be placed on the development of linked markers to *HgYMV*-resistance.

6.5. Transcriptomics Approaches

Due to the large data size obtained from genomic tools, researchers are now shifting towards a transcriptomic approach, which provides a better understanding of the mechanisms behind the biotic and abiotic stress resistance. Expression profiling has now become a well-established and high-throughput method for acquiring quantitative information on a sample's transcriptome and for studying differential gene expression. Reports on some of the differentially expressed transcripts in *Vigna* spp. against *YMV* are available. Chakraborty and Basak [59] studied the transcriptional modifications in *V. mungo* during *MYMIV* resistance, as well as in susceptibility. They identified 145 and 109 differentially expressed transcripts in resistant and susceptible plants, respectively, through suppression subtractive hybridization. About 43% of the unique expressed sequence tags (ESTs) shared homology with *G. max* sequences, while 14%, 13%, and 9% of the ESTs showed homology with *V. radiata*, *V. angularis*, and *P. vulgaris*, respectively. They also observed major changes in the gene expression in resistant genotypes upon *MYMIV* infection in the jasmonic acid biosynthesis and signal transduction pathways, cell wall modifications, and metabolic pathways (Table 5). Likewise, Kundu et al. [167] found 345 candidate genes that illustrate differential expression during compatible or incompatible interactions. They categorized ESTs into nine different functional categories based on their putative functions. These ESTs involved in various metabolic pathways, such as glycolysis, showed upregulation in their expression. Since ESTs do not provide any quantitative estimation of gene expression, gene expression of selected ESTs through quantitative PCR will give more accurate information. Kundu et al. [167] studied 19 genes related to R-protein such as, the suppressor of the G2 allele of *skp1* (SGT1) and heat shock protein 90 (HSP 90), systemic acquired resistance (SAR) indicator gene, pathogenesis-related (PR) genes, genes related to reactive oxygen species (ROS) homeostasis (superoxide dismutase (SOD), ascorbate peroxidase (APOX), thioredoxin (TRX), and metallothionein (MET)), signal transduction gene (such as calmodulin (CAM), and other genes like WRKY, glutathione S transferase (GST), phenylalanine ammonia lyase (PAL), ubiquitin ligase, tryptophan synthase (TS), cysteine protease (CSP), rubisco activase (RuAc), MADS box protein (MADS), auxin response factor (ARF), and oxygen-evolving complex (OEC)), at different time point intervals in both the resistant and susceptible cultivar of *V. mungo* against *MYMIV*. They also observed the coordinate action of salicylic acid-responsive pathways, Ca²⁺ signaling, redox imbalance, and PR genes. Chakraborty and Basak [59] functionally validated the differential expression of 12 different genes like phytohormone jasmonic acid, detoxification proteins, PR protein, ROS regulatory transcripts, and metabolic pathways for the systemic resistant response in *MYMIV*. In contrast, susceptible plants demonstrated a weak implementation of these pathways, differing in the induction kinetics and transcript dynamics of stress-responsive genes (Table 5). Kundu et al., [167] demonstrated about a 9-fold change in the expression of *Arabidopsis thaliana* mitogen-activated protein kinase (*AtMAPK6*) at 12 h post-inoculation in the resistant background and observed a notable difference in its abundance in the *MYMIV*-infected resistant VMR84 strain, which suggests its participation in SA signaling. Maiti et al. [168] characterized the *Non-TIR-NBS-LRR* encoding candidate gene *CYR1*, which is tightly associated with *MYMIV*-resistance in urdbean. Yadav et al. [169] also identified the *LRR LIKE-PROTEIN KINASE* gene as *MYMIV*-resistance in soybean. Likewise, Naresh et al. [170] characterized the NBS-LRR class disease resistance gene analogs leading to multi-virus resistance in chilli, indicating the need for deep analysis of NBS-LRR candidates for YMD-resistance in mungbean. These candidates will also help in developing gene-specific markers for resistance breeding.

Table 5. Candidate genes associated with *LYMV*s.

Sl. No.	Candidate Genes Associated with <i>LYMV</i> s	Function of Gene	Expression in <i>MYMIV</i> Resistant Genotypes at Different Time Intervals	Expression in <i>MYMIV</i> Susceptible Genotypes at Different Time Intervals	References
1	Allene oxide cyclase (AOC: XM_014661012.1), Allene oxide synthase (AOS: XM_017565246.1), Acyl activating enzyme 1 (AAE1: XM_014663780.1)	Jasmonic acid biosynthesis pathway	Upregulated	Downregulated	[59,167]
2	Glycine-rich protein (GRP: XM_017558252.1), Proline-rich protein encoding gene (PRP: PRP-U72769.1), Hydroxy-proline-rich glycoprotein encoding gene (HRGP: XM_017559232.1)	Cell wall synthesis	Upregulated	Downregulated	[59,167]
3	Lipid transfer protein (LTPs: XM_014636461.1), PR proteins such as PR1 (JZ168313), PR2, PR3, PR4, PR5 (JZ168398)	Pathogenesis-related proteins	Upregulated	Downregulated	[59,167]
4	Pyridoxin (PDX1: XM_014661423.1), Glutathione-S-transferases (GST: XM_014648972.2), Peroxiredoxins (JK086508.1), Cu/Zn superoxide dismutase (JZ168376.1), and Ferredoxin-like protein such as PRX (JZ168355.1), SOD, and TRX	Reactive oxygen scavengers	PDX1 up and early expression of ROS gene	PDX1 Down but in the late expression of ROS genes	[59,167]
5	RUBISCO (XM_014661061.1), PEPC, ribose-5-phosphate isomerase and aldehyde dehydrogenase (JZ168331.1)	Metabolic pathways genes	Upregulated	Downregulated	[59,167]
6	WRKY transcription factor	Transcription factor	Upregulated	Downregulated	[59,167]
7	MAP kinase (JZ168283.1)	Protein kinases	Upregulated	Downregulated	[59,167,171]
8	Drought responsive ESTs (CPRD2 and CPRD14: JZ168282.1)	Drought responsive	Upregulated	Downregulated	[59,167]
9	Ubiquitin ligase	Ubiquitin proteasome system	Upregulated	Downregulated	[59,167]
10	Ca ²⁺ responsive calreticulins (CRTs) and calmodulins (CAM) (JZ168377)	Ca ²⁺ response	Upregulated	Downregulated	[59,167]
11	Phenylpropanoid pathway (PAL: JZ168300.1)	Biosynthesis of lignin	Upregulated	Downregulated	[59,167]
12	R gene NBS-LRR (JZ168271.1), HSP90 (JZ168383)	Plant disease resistance against pathogens	Upregulated	Downregulated	[59,167]

6.6. Genome Editing Approaches

The Clustered Regularly Interspaced Short Palindromic Repeat (CRISPR) system, an adaptive immune system found in bacteria, has been successfully utilized by several researchers for the development of resistance to viruses. Prior to CRISPR, zinc finger nucleases (ZFNs) or transcription-activator-like effectors nucleases (TALENs) were traditionally used for genome editing, but these methods required a new nuclease pair for every genomic target region. CRISPR has rapidly become the most popular genome editing approach. This system contains two components: a guide RNA (gRNA) and a CRISPR-associated endonuclease (Cas protein). Thus, one can change the genomic target of the Cas protein by simply changing the target sequence present in the gRNA. CRISPR was employed to knock out target genes in various cell types, but modifications to various Cas enzymes have extended CRISPR to selectively activate/repress target genes, purify specific regions of DNA, and precisely edit DNA and RNA. Several works have successfully used genome-editing techniques against the Begomoviruses. There is a strong need to deploy this technique against the YMD-causing viruses as well.

7. Concluding Remarks and Future Prospects

It is evident that more than one virus species are involved in causing the yellow mosaic disease in *Vigna* crops, although, to our best knowledge, instances of mixed infections have not been reported. It may be due to the occurrence of different virus species causing YMD. However, the presence of multiple viruses causing different viral diseases in mungbean and urdbean have been reported [172], indicating that the presence of multiple viruses causing YMD may cause more damage to mungbean. Keeping in mind the prevalence of different species of viruses in *Vigna*-cultivation areas and the complexity of host–vector–virus interactions, mapping these viruses is required as a top priority, aside from the continuous monitoring of YMD outbreaks in newer areas. Establishing the accurate identity of viruses at the molecular level should be a preferred approach in order to create an effective YMD management strategy. Aside from the molecular marker-based detection of the specific viruses causing YMD, the development of a set of differential hosts for the identification of different viruses will further help in the establishment of the viral identities at the field level and, consequently, aid in deploying a proper management strategy. Simultaneously, investigation on the virus evolution and co-evolution at definite time intervals are also required if any changes in symptoms and expression are observed. Once it is done, tagging and deployment of appropriate resistance genes will be the best strategy for combating YMD. For developing cultivars with a good level of resistance, we need to identify the genes/QTLs governing resistance towards specific viruses by using artificial screening procedures, and once these resistance genes/QTLs have been tagged, their pyramiding will be the best mitigation strategy (Figure 4).

Difficulties in reproducing the results of QTLs for specific resistance may pose additional challenges while breeding for YMD resistance. It may be due to the differential genetics of highly resistant donors used in the study, as well as the phenotyping procedures involved. Some researchers have been using a 0 to 5 numerical scale, whereas the others have been using a 0 to 9 scale to rate the disease. While most of the researcher's report <5% of disease severity as highly resistant, some of the reports exhibit up to 10% disease severity as highly resistant. The use of different rating scales and disease severity percentages considered for selecting highly resistant genotypes can mislead researchers into utilizing the previously reported donors, as the currently available information seems to be unclear. The use of different plant stages (such as seedlings, flowering, and pod filling/maturity stage) for disease analysis can also be a source of confusion. Therefore, we need to develop a stage-specific universal scale for YMD screening to effectively utilize the identified HR-donors for YMD-resistance. The major role of the vector interaction causing YMD in the field also serves as a challenge to YMD-phenotyping under natural conditions. Thus, the development of an artificial screening procedure under laboratory conditions will save time and can better generate effective and authentic data for YMD. Pratap et al. [173] reviewed different platforms of high throughput phenotyping for harnessing the gains of genomics.

Likewise, we need to deploy such platforms for strict phenotyping of YMD at different plant growth stages. The development of agro-infectious clones of LYMV is a very tedious procedure due to the low concentration recovery of pure viral DNA after its cloning. Therefore, PCR-based cloning by using overlapping primers to develop dimeric agro-infectious clones for the respective virus will be an easier approach. Ferro et al. [174] have also developed the agro-infectious clones of a circular plant DNA virus by overlapping PCR-based amplification, and they have successfully validated this method on the mungbean.

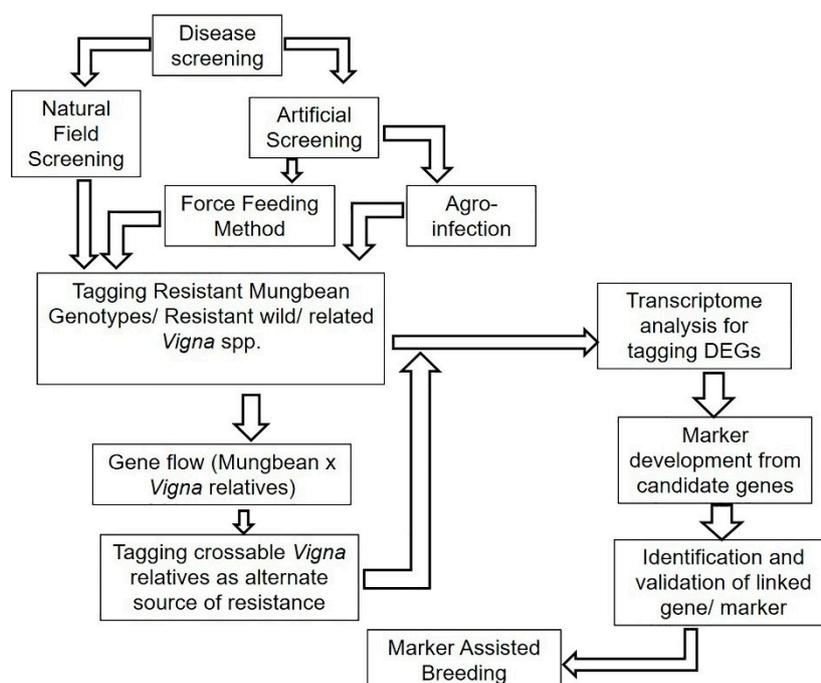


Figure 4. Marker-assisted breeding/gene introgression through transcriptomic and genomics approaches.

Several wild *Vigna* accessions belonging to different species have been identified as showing a high level of resistance to the different YMD-causing virus species. Some of the researchers have successfully studied the gene flow from wild to cultivated *Vigna*, and they have noticed that there is enough opportunity to facilitate gene transfer. The resistant gene of YMD has been successfully incorporated into urdbean [175] and mungbean [176,177]. Likewise, Mathivatahana et al. [178] used rice bean as a donor for MYMV-resistance in mungbean. Sudha et al. [179] used the rice bean as MYMV-resistant donor for developing the cross-specific markers for resistance breeding. Sudha et al. [180] identified 24 accessions of rice bean as highly resistant donors for MYMV, which can be used in developing MYMV-resistance mungbean genotypes. Chen et al. [181] identified the QTLs for governing resistance to MYMIV in mungbean using *V. radiata* var. *sublobata*, a wild progenitor of mungbean. Pandiyan et al. [182] also studied the gene flow from several wild relatives of *Vigna*, i.e., *V. radiata* var. *sublobata*, *V. silvestris*, *V. haniana*, *V. umbellata*, *V. glabrescense*, *V. pilosa*, *V. acconitifolia*, *V. stipulacia*, *V. trilobata*, *V. bournea*, *V. khandalensis*, *V. dalzelliana*, into mungbean and noticed fertile hybrid generation, indicating the possibility of incorporating the important genes from wild species to elite cultivars. Furthermore, we need to study the gene flow and inheritance patterns and compare the data obtained between resistant and wild *Vigna*, including mungbean progenitor, non-progenitors, and susceptible mungbean cultivars. If any novel inheritance pattern (i.e., Oligogenic inheritance pattern) is observed between HR-wild and HS-mungbean, it will be easier to incorporate the candidate genes rather than pyramiding a large number of QTLs.

Lately, there have been impressive advancements in the development of genomic and transcriptomic resources. These resources can be effectively employed for the marker-trait association

and QTL identification, as well as for contributing to the initiation of marker-assisted breeding and selection. Due to the large data size of the genome, it is very difficult to tag the specific region for resistance. Combining both genomic and transcriptomic approaches will be sounder in deploying the host plant with specific resistance, which appears to be the best strategy to manage this disease. In 2016, Liu et al. [183] successfully tagged the resistance locus and developed the tightly-linked marker for bruchid resistance in mungbean by harnessing the potential of wild relatives and combining genomic and transcriptomic approaches. Furthermore, there is also a need to explore plant innate immunity against the virus-vector to generate YMD-resistance in mungbean as an alternate way to induce resistance in mungbean plants. Characterizing NBS-LRR candidates in wild *Vigna* relatives will give a clue for understanding the mechanism of YMD-resistance. Harnessing the potential of wild/weedy and interspecific *Vigna* gene pools through genomics, transcriptomics, proteomics, and plant innate immunity approaches will be highly effective for identifying and tagging candidate genes for YMD resistance, as well as for determining their precise introgression in mungbean to develop high yielding multi-virus YMD-resistant mungbean cultivars.

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