Research Article

Mungbean Yellow Mosaic Begomovirus: A Comprehensive Review

Hafiz Nawaz[1]*, Kashaf Nawaz[2], Attiq ur Rehman[1], Muhammad Bashir[3], Mussera Hira[2] and Mariyam Nawaz[4]

[1]Barani Agricultural Research Institute, Chakwal, Pakistan; [2]Center of Excellence for Olive Research and Training, Barani Agricultural Research Institute, Chakwal 48800, Pakistan; [3]Department of Botany, University of Central Punjab Quaid Campus, Rawalpindi, Pakistan; [4]Department of Bio Sciences and Management Sciences, COMSATS University, Islamabad 22060, Pakistan.

Abstract | Pakistan grows 200 thousand hectares of barani (rain-fed) and irrigated mungbean (*Vigna radiata* (L.) Wilczek). 80–85% of mungbean area is in Mianwali, Bhakkar, and Layyah of Punjab. Some viral diseases cause little economic damage, while some do. Thus, mungbean viral traits, differentiating features, and dispersion must be studied critically. Mungbean Yellow Mosaic Disease (MYMD) is a whitefly-transmitted viral disease. Infected plants have uneven yellow-green spots. Diseased plants develop late and produce few flowers and pods. MYMD causes 85% of economic losses. Begomovirus is a Begomovirus. DNA-A and DNA-B make up its single-stranded genome. The virus's virulence and symptoms need both components. The sequencing of both components showed considerable variation between old-world and new-world viruses. Cultural practices, insecticides, and virus-resistant mungbean cultivars may help suppress the disease. MYMD-resistant NIAB mungbean lines include 1429, 3198, 3845, 3850, 3854, and 3881 (Schreinemachers *et al*., 2019). A recent study has revealed resistant varieties NM-5, NM-7, NM-68, NM-69, M 20-21, and M 22-24. This review guides research and MYMD-resistant cultivar development in the nation. Mungbean virus behavior, evolution, and dispersion must be studied to protect mungbean farming. This will help produce resistant cultivars and effective management measures. To combat MYMD and preserve mungbean production in Pakistan, research institutes, agricultural extension agencies, and farmers must work together.

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***Correspondence** | Hafiz Nawaz, Barani Agricultural Research Institute, Chakwal, Pakistan; **Email:** h_husnain_012@yahoo.com
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Introduction

Pulses are one of the oldest food crops and have deep roots in the Indo-Pak subcontinent, originating in the fertile crescent of the Near East. Vegetarian communities in poor nations like India and Pakistan depend on them for proteins and vitamins (Kannaiyan, 1999). Mungbean is Pakistan's second-largest pulse crop after chickpea. Pakistan crops 22.8 million hectares, 0.7% of which are pulses (Arif *et al*., 2020). Rainfed and irrigated locations grow mungbeans. Mungbean needs less water than other summer crops

since it grows quickly (Anjum *et al*., 2006). Pakistan grows mungbean (*Vigna radiata* L.) for grains and green manure. It supplements Pakistan's rice diet with readily digested, low-flatulence proteins. Various climates support the mungbean. It requires a warm, humid environment with 25-30 °C and 400-550 mm of well-distributed rainfall over the 60-90-day growth season. Irrigated crops may survive in somewhat salty and nutrient-deficient soils. Mungbeans grow best at 28-30°C and produce seeds around 33-35°C. However, each degree beyond the ideal range reduces seed output by 35-40% (Ramakrishnan et al., 2019).

Mungbean crop in Pakistan

Mungbean is grown in Pakistan during the Kharif and summer seasons. NM 92 and NM 98 are popular mungbean varieties. With the introduction of high-yielding mungbean cultivars, farmers have seen a rise in area, production, and yield (kg/ha) during the previous several years. The Nuclear Institute for Food and Agriculture (NIFA) in Peshawar utilizes mutation breeding and the hybridization with locally adapted material to enhance mungbean genetics. NIFA tested advanced mungbean lines developed from hybridization of indigenous and foreign germplasm for different characteristics including the resistance of MYMV during the Kharif seasons of 2002 and 2003 (Khattak *et al*., 2001). Mungbean output dropped 55% to 76,000 tons from 118,000 tons in 2009-10 due to high rainfall and floods (Habib *et al*., 2013). Pakistan's supply-demand mismatch was 125,000 tons of mungbean. Thus, Pakistan imports most of its pulses. Despite increases in mungbean acreage, productivity, and yield, experimental yields remain much below national averages. Due to its economic value, mungbean cultivation is almost fixed. Mungbean as an intercrop or fallow land following wheat harvest in April/May might extend the area (Khattak *et al*., 2002). Mungbean are hypoglycemic, hypolipidemic, antihypertensive, anticancer, hepatoprotective, immunomodulatory, and anti-inflammatory. Mungbean grains include 49.4% carbs, 25% proteins, and 2-4% lipids. They have 365 mg of phosphorus and 134 mg of calcium per 100 grams of grains (Faizan et al., 2020). Punjab grows 88% of Pakistan's mungbeans and produces 85%. Layyah, Bhakkar, Mianwali, and Rawalpindi are major mungbean producers. With the economy under pressure to feed a rising population, rainfed areas in Pakistan must be used to increase food security by planting more crops (Mahmood *et al*., 1991). Table 1 lists Pakistan's high-yielding mungbeans.

Mungbean diseases

Fungi, viruses, bacteria, and nematodes infect mungbean. Powdery mildew caused by *Podosphaera fusca* (Fr.), Cercospora leaf spot (CLS) is caused by *Cercospora canescens*, anthracnose caused by *Colletotrichum acutatum*, and dry root rot, which is an emerging disease in mungbean. Bacterial leaf spot, tan spot, and halo blight are among the bacterial diseases that harm Mungbean (Nair *et al*., 2019). Mungbean yellow mosaic virus damages Pakistani mungbean fields. MYMV reduces Indian yields by 85% (Karthikeyan *et al*., 2014). ULCV (Urdbean leaf crinkle) causes leaf crinkle disease in mungbeans. Early-stage ULCV infections may cause plant sterility and significant losses (Bashir *et al*., 1991). Dry root rot has reduced mungbean yields by 10% to 44% in India and Pakistan. Pakistan has 70% MYMV prevalence (Bashir *et al*., 2006). Mungbean farmers must control these diseases to minimize their impact and maximize production.

Table 1: *High yielding recommended varieties of mungbean in Pakistan having specific characteristics.*

Name of variety	Developed by institution	Yield (kg\hac)	Characteristics
NM-2006	NIAB	20	High yielding, bold seeds and yellow mosaic resistant
AZRI-Mung-06	AZRI, Bhakkar	18	Non shattering, short duration, bold seeded, disease tolerant
NM-92	NIAB	18	Bold seeded, tolerant to Cercospora leaf spot and shiny colour
NM-54	NIAB	16	Dull seed color
NM-98	NIAB	15	High yielding, medium bold seeded and virus resistant
Chakwal Mung-97	BARI	15	Shiny green color, suitable for cultivation in Pothwar region and small seeded
NM-51	NIAB	15	Tolerant to Cercospora leaf spot, dull seed color
Chakwal Mung-06	BARI, Chakwal	15	Drought tolerant, high yielding and yellow mosaic tolerant

Source: *Agriculture service for farmers, Bakhabar Kissan(bkk.ag).*

Mungbean yellow mosaic virus (MYMV)

Mungbean mosaic Begomovirus destroys mungbean yields. Field whiteflies (*Bemisia tabaci*) spread the virus. Mungbean, Black gram, Pigeon pea, Soybean, Cowpea, and Navy Beans are all affected by this virus (Karthikeyan *et al*., 2014). Early infection symptoms may match those of other biotic and abiotic causes that create yellow flecks. The foregoing symptoms usually indicate pathogenic infection (Nawaz et al., 2022). Mungbean Yellow Mosaic Virus (MYMV) is distributed over a wide region in Pakistan and causes major losses in leguminous crop output. This disease most severely affects mungbean, black gram, and soybean (Khattak *et al*., 2000). MYMV is the worst summer mungbean disease in Pakistan (Ahmad, 1975). Whiteflies spread MYMV (Figure 1). Seeds, soil, and mechanical injection cannot transmit the virus (Nair and Nene, 1973). Variable cultivars have variable disease susceptibility, depending on their genetics (Brigneti *et al*., 2004).

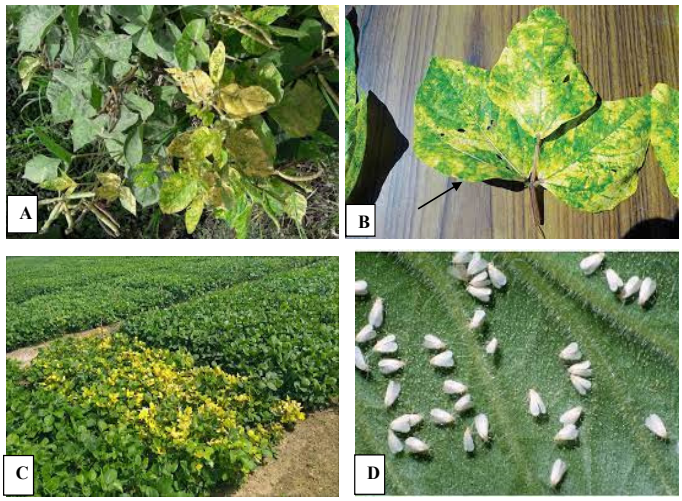

Figure 1: *Yellow mosaic virus-affected mungbean plants are depicted in the figure in actual field settings (A), Damaged leaves of mungbean (B), Symptoms of MYMD on susceptible cultivars (C), whitefly, the insect vector of MYMV (D).*

Losses caused by MYMV

The viral vector whitefly spreads swiftly, causing substantial production and quality losses in pulses. Mungbean yellow mosaic virus has devastated green gram in India for over 50 years (Mandhare and Suryawanshi, 2008). Geminiviridae family begomoviruses cause it. This virus is spreading and harming green gram crops economically to the tune of 85% (Karthikeyan *et al*., 2014). Chlorophyll, nitrogen, protein, phosphorus, and carbohydrate content are all affected by the mungbean yellow mosaic virus. Virus-infected mungbean plants had decreased glucose and chlorophyll levels. Virus-infected mungbean leaves had greater nitrogen, protein, and phosphorus content (Maravi *et al*., 2022).

Mungbean yellow mosaic virus (MYMV) is mechanically transmitted in Thailand by whiteflies. It can be mechanically transferred only to this isolate. Growth chamber plants display symptoms best at 25–30°C. MYMV only infects seven Leguminosae plants (Honda and Ikegami, 1986). A disease severity assessment scale has been created for field use (Bashir *et al*., 2005). Table 2 shows the scale.

Table 2: *Scale for evaluating the severity of the MYMV infection in field conditions.*

Disease severity scale	Percent infection by MYMV	Reaction type R
0	0%	HR (highly resistant)
1	1-10%	R (resistant)
2	11-20%	MR (moderately resistant)
3	21-30%	MS(moderately susceptible)
4	31-50%	S (susceptible)
5	More than 50 %	HS (highly susceptible)

Source: *Sources of genetic resistance in mungbean and black gram against Urdbean leaf crinkle virus (Bashir et al., 2005).*

Disease causing ability of yellow mosaic virus on different plants

Infected plants have brilliant yellow mosaic patterns on their leaves, causing considerable losses to mungbean harvests in Pakistan. Geminiviruses infect several agricultural plants, including dicots and monocots, causing severe damage. These viruses cause significant agroeconomic losses worldwide. Leguminous crops like pigeon peas (*Cajanus cajan*), soybeans (*Glycine max*), mat beans (*Phaseolus aconitifolius*), common beans (*Phaseolus aureus*), French beans *(Phaseolus vulgaris*), and black gram (*Vigna mungo*) are among the leguminous crops most commonly impacted by MYMV. Indirect ELISA (DAC-ELISA) testing of 540 mungbean samples against nine polyclonal antisera targeting legume viruses yielded 213 (39%) positive results. Interestingly, ELISA antisera did not react with 336 (63%) virus-like samples. 336 (63%) symptomatic samples were antiserum-negative. ELISA showed MYMV had the greatest disease incidence (15–36%), followed by ULCV (6–26%). CMV and BYMV were less common than MYMV and ULCV (Bashir *et al*., 2006). MYMV, ULCV, AMV, CMV, and BYMV were detected in

Pakistan using ELISA. Pakistan has received reports of the mungbean yellow mosaic virus on urdbean and Mungbean (Ahmad, 1975; Bashir *et al.*, 1991), while AMV, BYMV, and CMV were first found on mungbean.

Classification of MYMV

Baltimore (1970) classed the viruses as class II. In certain begomoviruses, their genomes are split into two components of 2600 to 2800 nucleotides each. These viruses feature elongated, geminate capsids linked at the missing vertex. Single-stranded or double-stranded DNA viruses are subgroups. These viruses reproduce their genomes using sigma or theta replication. A single-stranded DNA begomovirus called the mungbean yellow mosaic virus (MYMV) carries out rolling circle replication (RCR), also known as sigma replication (Gutierrez, 2000). This manner of replication defines the rolling circle mechanism and DNA molecule shape (sigma) during replication. The host cell's nucleus converts single-stranded DNA to double-stranded during replication. DNA replication begins in the protein-free intergenic region (IR). Viral gene transcription depends on this area.

Nature of viral genome (MYMV)

Phenol-sodium dodecyl sulphate extraction method used for nucleic acid particle extraction from the pure isolates of MYMV (Ikegami and Francki, 1975). The purified nucleic acid has ultraviolet spectra with 2.5 and 2.0 260/230 and 260/280 nm ratios. Diphenylamine reactions show deoxyribose in pure nucleic acid (Shatkin, 1969). Heat and formaldehyde affect single- and double-stranded DNA differently (Miura *et al.*, 1966; Robinson and Hetrick, 1969; Sinsheimer, 1959). Formaldehyde at normal temperature turns MYMV DNA into a single-stranded molecule. Formaldehyde adds 5 nm to the wavelength maximum and 18% hyperchromicity to isolated MYMV DNA in 10 minutes. Double-stranded calf thymus DNA does not change wavelength maxima or hyperchromicity. MYMV DNA absorbs UV light from 20 to 70°C, whereas calf thymus DNA has steep melting temperature (Tm) of 77°C. Polyacrylamide gel electrophoresis of 7 M urea-prepared MYMV nucleic acids shows two bands. DNase-treated MYMV nucleic acid is analyzed by polyacrylamide gel electrophoresis.

The cambium, seed coat hilum, and phloem parenchyma all contain begomoviruses (Rojas *et al.*, 2005; Kothandaraman *et al.*, 2016). Early indications, yellowing of trifoliate leaf of blackgram seedlings, suggest seed-borne YMV infection. Despite the fact that 32% of seedlings have MYMV DNA-A and DNA-B, seedling development assays do not reveal any signs of YMD (Kothandaraman *et al.*, 2016). The dynamic metabolic environment of seedlings may prevent viral accumulation and transmission, causing no symptoms. PCR-confirmed symptomless seedlings did not transmit the virus through whiteflies (Figure 2).

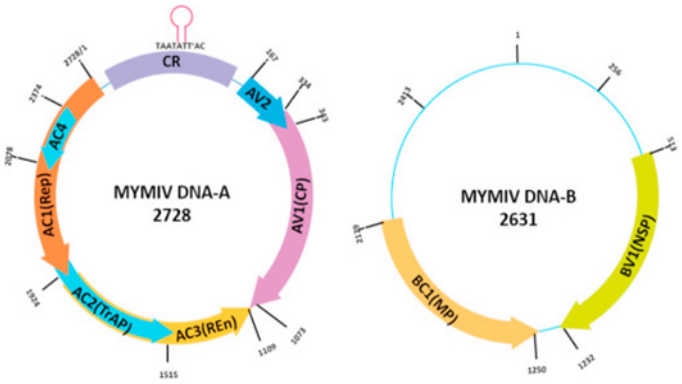

Figure 2: *DNA components A and B of MYMV.*

Life cycle of MYMV

Single or double-stranded DNA viruses exist. These viruses reproduce their genomes utilising sigma or theta replication. Sigma replication (RCR) is used by MYMV, a single-stranded DNA begomovirus (Gutierrez, 2000). The method is called "rolling circle replication" because DNA molecules resemble the Greek letter sigma. The host's nucleus initially converts single-stranded DNA into double-stranded DNA for replication. DNA replication begins in the protein-free intergenic region (IR). Viral gene transcription occurs here. The Rep protein attaches to the replication origin (ori), TAATATTAC in all geminiviruses (Stanely and Latham, 1992), starting replication. AATATT/AC, a nick at this conserved site following TT, protrudes the 5′ end. Recombination and component exchanges in begomoviruses may impact legume genotype resistance. However, legume-infecting begomoviruses develop separately from those infecting other plant groups since there is no extensive evidence of their interaction. The DNA-B component of a HgYMV isolate was 96% comparable to a soybean MYMV isolate, but only 70-73% similar to MYMV and MYMIV. This mismatch might be explained by component interchange (Qazi *et al.*, 2007). To confirm the pathogenicity of the cowpea strain, Kumar *et al.* (2017) agroinoculated

mungbean and cowpea with dimeric infectious clones carrying MYMIV DNA-A and DNA-B. Further genetic sequencing analysis unveiled the presence of a DNA-B component within the MYMV isolate responsible for the symptomatic manifestations. The insect vector may determine the natural host range. LYMV sequence data suggests that recombination with non legume viruses caused more virulent forms to attack legumes. According to Ilyas *et al.* (2010), overexpression of the IMYMV-Bg Rep protein in E. coli revealed that it binds to the CR sequences. Rep's structural changes and cleavage were brought on by ATP as well. When *Vigna mungo* was agroinoculated with a mixture of Agrobacterium cultures, the cloned DNA-A and five MYMV-Vig DNA-Bs co-infected the animal. This means that MYMV-Vig may infect *V. radiata* and *V. aconitifolia* thanks to a number of DNA-B components (Karthikeyan *et al.*, 2004). Thus, it is crucial to understand how different DNA components in different YMVs influence different Vigna species. Differential mungbean-YMV interactions may explain YMD resistance responses (Figure 3). Recessive inheritance means host gene loss, but dominant MYMV resistant gene activation means gain-of-function. Viral disease expression in the field is influenced by whitefly activity and environmental conditions (Sudha *et al.*, 2013b). Marker-assisted selection (MAS) is crucial for quick and accurate breeding programs since YMD resistance in mungbean is recessive (Chen *et al.*, 2013).

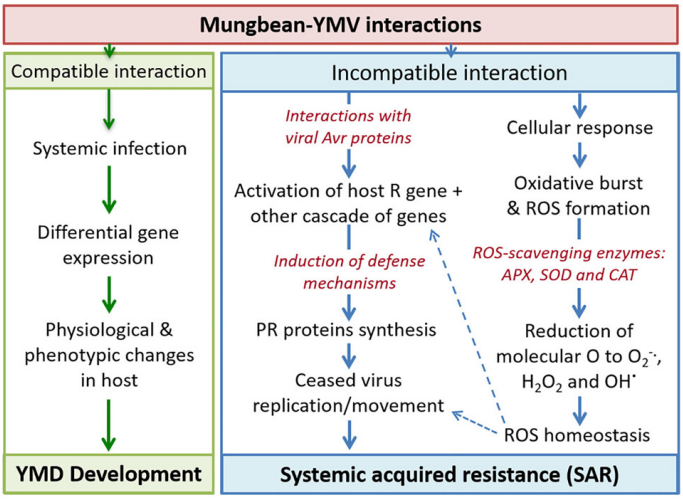

Figure 3: *Mungbean YMV interaction.*
Source: *Frontiers yellow mosaic disease of mungbean: Current status and management plant science.*

Sequence of MYMV genome

Complete or partial DNA-A and DNA-B sequencing has characterized MYMVs In Bangladesh and Pakistan, MYMVs infecting mungbean showed 97% and 94% sequence similarity in the CP and NSP genes of MYMIV, respectively (Hussain *et al.*, 2004). MYMV strain-A was found in Indonesian isolates, whereas strain-B was found in Vietnamese isolates (Tsai *et al.*, 2013). Following the sequencing of 44 components (23 DNA-A, 19 DNA-B, and 2 betasatellites), two kinds of MYMIV were discovered in Pakistani LYMVs (Ilyas *et al.*, 2010). According to a molecular investigation, MYMV-VSKN is present in *V. mungo* var. *Silvestris* (Naimuddin et al., 2011). The CP gene shown genetic diversity in MYMV-Tamil Nadu isolates from blackgram, cowpea, and mungbean samples (Maheshwari *et al.*, 2014).

Symptomology

Viruses cause many plant diseases, reducing agricultural yields. Plant viral infections defy chemical treatment, making them harmful. Viruses may infect cotton, tobacco, potato, papaya, rice, and pulses. Geminiviridae are the most significant plant disease-causing viruses. Twinned, quasi-isometric geminiviruses are single-stranded plant viruses (Bock *et al.*, 1974). The term geminiviruses comes from the twins' zodiac sign, "gemeni" (Harrison *et al.*, 1977). In 1995, the Geminiviridae family was formally named after the geminivirus group (Matthews, 1979). Whiteflies spread begomoviruses that infect only dicot hosts. Viruses are categorized into monopartite and bipartite types. DNA-A and DNA-B are the two DNA molecules that bipartite viruses have, whereas monopartite viruses only have one. Single-stranded DNA makes up the 2.8 kb bipartite begomovirus known as MYMV. Like other begomoviruses, it has isometric, geminate viral particles that are 18–30 nm in size. The single-stranded DNA molecules DNA-A and DNA-B have 2726 and 2775 nucleotides, respectively (Hull, 2004).

Viral strains

Initially monopartite, begomoviruses later developed DNA-B as a satellite that became a component of the genome. Bipartite begomoviruses with DNA-A and DNA-B varieties are the result of component exchange throughout evolution (Briddon *et al.*, 2010). On the viral sense strand of DNA-A, the AV1 and AV2 genes encode the coat protein (CP, 29.7 kDa), as well as the movement or pre-coat protein (12.8 kDa). According to Rouhibakhsh *et al.* (2011), the proportion of open circular and supercoiled DNA types influences the function of the Rep protein. On

the viral complementary sense strand, four proteins are encoded: Rep, REn, TrAP, and Rep. In DNA-B, BV1 (viral sense strand) and BC1 (complementary sense strand), respectively encode the nuclear shuttle protein and movement protein. The nuclear shuttle protein (NSP) transports viral DNA between the nucleus and cytoplasm of the host cell and through the host vascular system, while the movement protein (MP) regulates virus movement through plasmodesmata (Hanley-Bowdoin *et al.*, 1999). In monopartite begomoviruses without BV1 and BC1, CP (AV1) performs as NSP. For growth, multiplication, and cell-to-cell migration, the virus needs proteins from the mungbean plant, which has an impact on both the plant and the virus (Cayalvizhi *et al.*, 2015). Two begomoviruses that are bipartite share a 200-bp DNA-A/DNA-B region. According to Hanley-Bowdoin *et al.* (1999); Pant *et al.* (2001), an origin of replication (ori) is a highly conserved stem-loop or hairpin structure with the nonanucleotide pattern (TAATATTAC) and "iterons" or direct repetition motifs of 5-7 nucleotides in the intergenic region of begomoviruses.

Modes of viral transmission

Plants need viruses to survive and transmit disease. Insects, nematodes, and mechanical ways may spread viruses. Viruses are categorised by transmission. After eating infected plants, vectors may transfer certain viruses by attaching them to their mouthparts. Non-persistent viral transmission. Since they must reach insects' guts and saliva, some viruses take longer to spread. After constant feeding, these viruses may spread. Persistently spread viruses may be circulative or propagative. The polyphagous Indian whitefly transmits Mungbean yellow mosaic virus, which damages over 1,000 plant varieties. Whiteflies carry viral infections by feeding on plant sap (Fishpool and Burban, 1994). Nearly 300 virus species, including the Begomovirus (almost 90%), Carlavirus, Crinivirus, Closterovirus, and Ipomovirus (4%), may be spread by it. After feeding, the whitefly injects the virus into a healthy plant with its saliva. Before being transmitted to plants, the virus circulates without replicating in the whitefly's foregut, midgut, hindgut, hemolymph, and salivary glands (Fiallo-Olivé *et al.*, 2020). The vector needs 15–60 minutes and 15–30 minutes to acquire and inoculate the virus via phloem sap. Virus transmission requires a least 8-hour latent time between acquisition and injection (Ghanim *et al.*, 2001). The acquisition access period (AAP), gender, and age of the whitefly all affect how effectively the virus spreads (Czosnek *et al.*, 2002). Persistent transmission is determined by the minimum AAP and maximum viral retention (3 days for male whiteflies and 10 days for female whiteflies). Infected leaves may infect whitefly nymphs, but not their eggs. Both male and female whiteflies lose infectivity with time (Karthikeyan *et al.*, 2014). Begomovirus-whitefly interactions are determined by the highly conserved virus coat protein (CP) and whitefly gut and salivary gland receptors. Virus CP changes affect vector choices. Whitefly-encoded proteins such HSP70 help viruses spread (Brown and Czosnek, 2002). In blackgram, leaf trichomes and whitefly activity correlate with yellow mosaic virus (YMV) resistance, whereas there is no such link in mungbean. Begomoviruses increase transmission by decreasing whitefly lifespan and fertility. Whitefly feeding and behaviour may also affect viral genetics and evolution (Saxena and Tiwari, 2017). The common method for identifying the *B. tabaci* complex is by using the 3.5% and 4.0% thresholds for the divergence of the mitochondrial cytochrome oxidase subunit I (mtCOI) gene. Over 41 morphologically identical groupings or cryptic species were discovered by mtCOI sequencing analysis (Kanakala and Ghanim, 2019). The host range, pesticide tolerance, and dispersal of cryptic species differ (Nair *et al.*, 2017). While Q-biotype or Mediterranean (MED) species may be able to adapt to greenhouses, B-biotype or Middle East-Asia Minor 1 (MEAM1) species prosper in irrigated farming systems. Asia II-1 and MEAM1 whiteflies have different viral transmission and pesticide resistance genes, according to whole-genome sequencing (Hussain *et al.*, 2019). To control illnesses, additional sequencing information for whitefly biotypes globally is needed. Comprehensive investigations on the co-evolution between whiteflies and Begomoviruses, including transmission dynamics and viral mobility proteins within the whitefly, will help create new and more effective YMV control measures (Saxena and Tiwari, 2017).

Host range of MYMV

Yellow mosaic disease (YMD) caused by the Mungbean yellow mosaic virus can be controlled by removing perennial weeds (Karthikeyan *et al.*, 2014). The primary hosts of yellow mosaic virus are *V. radiata, V. mungo, V. aconitifolia, V. unguiculata*, *Cajanus cajan, Glycine max*, and *Phaseolus vulgaris* (Karthikeyan *et al.*, 2004; Qazi, 2007). *V. hainiana* and *V. trilobata* are

additional leguminous hosts (Naimuddin et al., 2011). The virus may also be hosted by "infected tolerant plants" and "symptomless carriers". Whiteflies in Northern and Southern India are dominated by the native cryptic species Asia II-1 and Asia II-8, respectively (Nair *et al*., 2017). Insecticides should be used rationally by knowing the quantity of whitefly species in a location since different species react differently to insecticides. Systemic pesticide combinations kill the vector and protect the plant against whiteflies during early development. Field cleanliness, plucking effected plants, water sprays and avoiding nitrogen fertiliser are further whitefly population control methods (Karthikeyan *et al*., 2014). Hydro-priming seeds for 8 hours also reduces yellow mosaic viraus infection in mungbean (Rashid *et al*., 2004).

Detection and identification techniques

Serological tests: 213 (39%) of 540 mungbean and mashbean samples evaluated utilizing indirect ELISA (DAC-ELISA) with nine polyclonal antisera against legume viruses were positive (Kaiser, 1979). Interestingly, 336 (63%) of the virus-like symptom samples did not respond with any of the ELISA antisera. Only four viruses were discovered in commercial plot samples: Mungbean yellow mosaic virus (MYMV), Urdbean leaf crinkle virus (ULCV), cucumber mosaic virus (CMV), and bean yellow mosaic virus (BYMV). AMV was absent from field samples taken from farmers. 336 (63%) symptomatic samples had no antisera reaction. ELISA findings showed MYMV (15–36%) had the greatest illness incidence, followed by ULCV (6–26%). CMV and BYMV were rarer than MYMV and ULCV. 43 experimental plots at various study locations yielded 225 mungbean and urdbean samples. Only 105 (46%) samples responded with legume virus antisera. Research station samples included AMV, BYMV, CMV, MYMV, and ULCV. Mungbeans were the only beans with AMV. MYMV (20–46%) and ULCV (20–33%) had the greatest ELISA incidence. AMV, BYMV, and CMV were less common (2–6%). No antisera reacted with 55% of samples. AMV was only found in mungbeans from research stations, not farmers' fields. ELISA showed 5–20% BYMV incidence. Farmer's field and research station samples showed mixed viral infections. MYMV-ULCV mixed infections were most prevalent. Experimental plots at the Plant Breeding and Genetics Department at the University of Agriculture, Faisalabad revealed mixed MYMV, ULCV, and BYMV infection. At NIBGE, Faisalabad, MYMV was detected using TAS-ELISA or PCR, and other viruses were detected using ELISA and mechanical transmission. DAC-ELISA examined several field samples without receiving any non-specific results. CSMV antiserum failed every test due to non-specific responses. Field surveys employ DAC-ELISA because plant extracts may coat plates without antisera or immunoglobulins. DAC-ELISA can test several samples during surveys (Hobbs *et al*., 1987). In ELISA, 63% of symptomatic samples from farmer's fields and 55% from research stations did not respond with any antisera. Nutrient shortage, physiological problems, or lack of antisera for other legume viruses may cause this (Makkouk *et al*., 2001).

Molecular tests: In Pakistan, mungbean leaves with severe yellow mosaic symptoms were gathered. The Mungbean Yellow Mosaic Virus (MYMV) coat protein gene was amplified and detected using PCR primers. The NCBI nucleotide database was used to align the MYMV coat protein gene sequences in order to identify the two regions that shared the most sequence homology. Forward and reverse primers were decided in these regions (Singh *et al*., 2020). DNA from virus-infected leaves was extracted using a modified CTAB method. On the collected DNA, PCR was conducted using CP-specific primers. Initial denaturation at 95˚C for 5 minutes, followed by 30 cycles of denaturation at 94˚C for 1 minute, annealing at 58˚C for 1 minute, extension at 72˚C for 30 seconds, and final extension at 72˚C for 5 minutes, were the PCR conditions. To confirm amplification, a PCR mix aliquot was resolved on a 0.8% agarose gel. Gel-purified PCR-amplified DNA samples were sequenced automatically. The NCBI BLAST programme was used to detect matches with other geminivirus coat protein genes globally, confirming their origin from MYMV. These sequences formed a phylogenetic tree. MYMV-infected plants have reduced development, fewer blooms, and smaller pods. The whole leaf and other plant components may become yellow in severe infestations. MYMV was evaluated on yellow mosaic virus-infected mungbean leaves. In this investigation, MYMV coat protein-specific primers were used to amplify genomic DNA from infected and uninfected leaf samples. PCR analysis amplified a ~750 bp CP gene fragment in the samples, as predicted. In tomato plants, ToLCV-specific primers identified molecular infection. Isolate

A and isolate B (Gazipur) were produced using one PCR-amplified sample from each site, gel purification, and automated sequencing.

Disease management and control strategies

Vector management: Controlling whiteflies the vector helps manage MYMV illness. Due to their high numbers and tolerance to pesticides, whiteflies are difficult to manage. Chemical control is popular. Systemic pesticides like acetamiprid, ethion, imidachlorpid, and triazophos kill whiteflies on contact and protect the plant for weeks. Insecticides operate best against first- and second-instar nymph whiteflies. Incomplete pesticide exposure of leaf undersides, where nymphs occur, may cause agricultural field whitefly control failure. Imidacloprid applied to leaf undersides reduces whitefly populations and MYMV dissemination, increasing seed output. Neem oil and NSKE may help control whiteflies. These botanicals inhibit nymphs from becoming adults, reducing whitefly populations on treated plants. Whitefly management might also include culture. Field cleanliness, including crop residue and weed clearance, may lower whitefly numbers. Manually removing leaves with non-mobile nymphal and pupal stages from badly affected plants reduces whitefly numbers, enabling natural enemies to manage them. Syringing or spraying may remove adult whiteflies. Avoid excessive nitrogen fertiliser, which promotes succulent growth and whitefly populations. MYMV disease management requires different approaches to control whiteflies without harming the environment.

Breeding for disease resistance: Since the 1970s, MYMV disease management has relied on mungbean breeding for resistance. Host resistance manages MYMV best. Plants may be sensitive or resistant to illness. Symptomless lines may be tolerant rather than resistant. Germplasm screening may find resistant or tolerant lineages for breeding.

Germplasm assessment should address viral strain diversity since germplasm sources have varying tolerance levels. Resistance crops need knowledge of gene inheritance and sources. MYMV disease resistance comes from single recessive, dominant, and complementing recessive genes. Intraspecific hybridization is utilised to improve MYMV resistance in mungbean, while wild mungbeans have resistance, permitting interspecific hybridization.

Intra- and interspecific hybridization has produced MYMV-tolerant, high-yielding mungbean lines for commercial agriculture. However, disease infestation, whitefly numbers, and the fast development of novel MYMV isolates have made traditional breeding for resistance difficult. For resistant lines, plant breeding and conventional techniques must be used together.

Marker-assisted selection (MAS): The fields of plant genetics and breeding have been irrevocably altered by the introduction of DNA markers such as RFLP, random amplified polymorphic DNA (RAPD) markers, simple sequence repeat (SSR), single-nucleotide polymorphism (SNP), and inter simple sequence repeat (ISSR). DNA markers can be used in breeding for a variety of purposes, but marker-assisted selection (MAS) offers the best prospects for cultivar growth. Research into the variety of germplasms, the discovery of the related marker for the resistant gene, and the construction of QTL maps using molecular markers have all increased the efficacy of breeding programs that result in MYV resistance (Sudha *et al*., 2013). By identifying the allele of a DNA marker, it is possible to identify plants with certain genes or quantitative trait loci (QTLs) based on their genotype rather than their phenotype. The MYMV resistance genes in mungbean have been identified using a variety of markers (Sudha *et al*., 2013), and the genetic diversity of the plant has been investigated (Chattopadhyay *et al*., 2005; Datta *et al*., 2012). In MYMV, MAS has achieved numerous notable successes in addition to considerably enhancing the efficacy of resistance breeding. One novel source of MYMV resistance has been identified as the use of donors from interspecific sources, and more modern molecular markers linked to resistance genes are now more readily available.

Pathogen-derived resistance (PDR): Pathogen-derived resistance (PDR) is one of the finest transgenic methods for crop virus resistance. Transgenic resistance utilising PDR is used in commercial mungbean cultivars without natural resistance. PDR disrupts pathogens by expressing viral genes in the host plant. PDR-mediated protection may delay symptom onset, minimise symptoms, and prevent virion buildup. These traits reflect numerous resistance mechanisms. In tobacco plants, full-length and shortened MYMV replication-associated protein (REP) genes suppressed viral replication, revealing REP sequences' stringent resistance. Gene silencing

and antisense RNA allow PDR without viral proteins. The DNA-based bidirectional promoter of MYMV is although transgenic plants were not resistant, a component stimulated post-transcriptional gene silencing (PTGS) against the yellow mosaic virus in blackgram. In other investigations, replication initiation protein (Rep) antisense RNA decreased the severity of mungbean plant symptoms and the proportion of infected plants. MYMV coat protein (CP) gene deletion affects systemic dissemination and pathogenicity in mungbean. Hairpin constructions targeting the CP gene employ MYMV sense and antisense sequences in a cloning vector. CP hairpin co-agroinoculation prevents viral pathogenesis. Mungbean germplasms may be screened for MYMV resistance utilising various MYMV isolates and agroinoculation methods. Transgenic techniques like PDR may help mungbean manage MYMV.

Conclusions and Recommendations

Understanding MYMV strain genomic variability requires categorization. However, MYMV strains are not classified globally. This lack of standardization has confused mungbean breeders and hampered disease-resistant breeding programmes. Due to non-homogeneous viral strain classifications, resistance sources, resistance information interchange, and germplasm use are uncertain. A uniform differential approach to identify strains consistently allows scientists to share knowledge and resistant germplasm. A more complete pathogen population structure is needed to create a viral isolation database. This will help create techniques for deploying resistance genes and introducing non-matching resistance genes into pathogen populations. Mungbean genome research lags behind soybean, cowpea, urdbean, and common bean. Despite advancements, mungbean contains fewer genetic resources and markers than azuki bean. Marker-assisted selection (MAS) has been accelerated by the development of RFLPs, RAPDs, AFLPs, SSRs, and ISSRs to uncover genetic diversity and associated markers for MYMV resistance genes in mungbean. Existing maps lack markers to cover all 11 connectivity groupings. This review summarises MYMV lifecycle, transmission, molecular biology, and integrated disease control. This review will inform MYMV control and mungbean production studies in Pakistan.

Novelty Statement

In the vast landscape of plant virology, 'Mungbean Yellow Mosaic Begomovirus: A Comprehensive Review' shines as a beacon of knowledge, illuminating the intricate dance between this viral antagonist and the resilient mungbean plant. This review not only elucidates the current state of our understanding but also charts new frontiers, revealing the hidden secrets of a tiny virus with a colossal impact on agriculture.

Author's Contribution

Hafiz Nawaz: Conceptualization, formal analysis, investigation, resources, supervision, visualization, writing original draft.
Kashaf Nawaz: Data curation, writing original draft, writing review and editing.
Attiq ur Rehman Nawaz: Investigation, supervision.
Muhammad Bashir: Conceptualization.
Mussera Hira: Formal analysis.
Mariyam Nawaz: Software.

Conflict of interest
The authors have declared no conflict of interest.

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