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Unraveling yellow vein mosaic disease of Okra: a *Review*

Abdul Majid Ansari[1], Nishar Akhtar[2] and Shahina Perween*[3]

[1]*Zonal Research Station (Birsa Agricultural University, Ranchi), Chianki, Palamau-822102, Jharkhand, India*
[2]*Agriculture College Garhwa, (Birsa Agricultural University, Ranchi), Garhwa-822114, Jharkhand, India*
[3]*Department of Genetics and Plant Breeding, Birsa Agricultural University, Ranchi-834006, Jharkhand, India*

ABSTRACT

Okra (Abelmoschus esculentus L. Moench) is an important vegetable crop valued for its nutritional contribution, market demand and adaptability to diverse agro-climatic conditions. However, its productivity is seriously constrained by yellow vein mosaic disease (YVMD), one of the most important viral diseases affecting okra production in tropical and subtropical regions. The disease is associated mainly with begomoviruses and is disseminated by the whitefly Bemisia tabaci, which facilitates rapid movement of the pathogen between infected and healthy plants. The disease is characterized by vein clearing followed by yellowing of veins and veinlets, progressive chlorosis, reduction in leaf development, stunting and poor fruit formation, with infection at an early crop stage generally causing greater yield reduction. This review critically synthesizes the available knowledge on the occurrence and distribution of okra yellow vein mosaic disease, associated viruses, genome organization, disease development, symptom expression and virus transmission, with particular emphasis on the biological and epidemiological significance of the whitefly vector. Different management approaches are discussed, including field sanitation, removal of infected plants, host resistance, vector surveillance, chemical and botanical interventions, biological control and integrated disease management. The review also highlights advances in pathogen-derived resistance and molecular approaches, including coat protein-mediated resistance, replicase gene-mediated resistance and RNA interference, which provide opportunities to interfere with viral multiplication and disease development. Despite substantial progress, effective and durable management remains difficult because of the genetic variability of begomoviruses, changes in vector populations, mixed infections and the potential emergence of resistance-breaking virus variants. Future research should therefore focus on broad-spectrum and durable resistance through integration of conventional breeding with genomics, transcriptomics, gene editing and multi-target RNAi. Emerging areas such as CRISPR/Cas-mediated antiviral strategies, host susceptibility-gene editing, host-induced gene silencing against the vector, vector microbiome manipulation, high-throughput pathogen surveillance and early disease detection offer promising new avenues for research. Integration of these emerging technologies with ecological vector management, climate-based disease forecasting and precision agriculture could shift YVMD management from conventional reactive control towards a predictive, sustainable and knowledge-driven system. Such multidisciplinary approaches may provide durable solutions for okra production and contribute to the broader management of whitefly-transmitted begomoviruses.

Keywords: *Abelmoschus esculentus; yellow vein mosaic disease; begomovirus; Bemisia tabaci; host resistance; RNA interference; CRISPR/Cas; pathogen-derived resistance.*

1. INTRODUCTION

Okra (*Abelmoschus esculentus* (L.) Moench), popularly referred to as bhindi or lady's finger, is an economically important vegetable crop belonging to the family Malvaceae. Although Africa is generally considered the probable center of origin, the exact origin of okra remains uncertain, as wild and related forms have been reported from both Africa and the Indian subcontinent [1]. The crop is extensively grown in tropical and subtropical areas due to its good adaptation to warm environments and comparatively short growing period.

Corresponding Author:* ***Shahina Perween*
Email Address: ***shahirose112@gmail.com***

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The tender, immature fruits are widely used as vegetables and provide valuable nutrients, including carbohydrates, proteins, dietary fiber, minerals and vitamins. In addition to their nutritional value, okra seeds are of considerable interest because they contain appreciable quantities of oil and protein, with reported levels of approximately 30–40% oil and 15–20% protein [1], [2]. Okra is important from nutritional, economic and agricultural perspectives, particularly in farming systems involving small and marginal growers. Its ability to perform under a range of agro-ecological conditions, coupled with the possibility of harvesting tender fruits repeatedly, makes it a useful source of regular farm income. The crop also has applications beyond direct consumption. Okra fruit mucilage has potential uses in food processing, pharmaceutical preparations and other industrial products, whereas the seeds are being explored for their oil and protein content [2].
Nevertheless, okra production is affected by numerous biotic and abiotic stresses.

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Major insect pests include aphids, jassids, thrips, whiteflies and fruit and shoot borers, while fungal, bacterial, nematode and viral pathogens also contribute to yield losses. Viral diseases are of particular concern because once systemic infection occurs, conventional chemical treatments generally cannot eliminate the virus. Furthermore, many plant viruses are transmitted efficiently by insect vectors, making disease suppression dependent on limiting both infection and vector-mediated spread [1].

Environmental stresses, including elevated temperature, drought, excessive precipitation, waterlogging, salinity and nutrient deficiencies or imbalances, can adversely influence vegetative growth, flowering, fruit formation and final yield.

Such environmental conditions may also alter the development and spread of viral diseases by affecting host susceptibility, viral multiplication and the abundance, survival and feeding activity of insect vectors. Consequently, the epidemiology of viral diseases in okra is influenced by the combined effects of the host plant, virus, vector and prevailing environmental conditions [2].

Among the viral diseases affecting okra, yellow vein mosaic disease (YVMD) is regarded as one of the most serious constraints to production. The disease was first documented in India by Kulkarni in 1924 and has subsequently become an important disease of okra across the Indian subcontinent and several other tropical and subtropical production regions [1], [3].

The major economic impact of YVMD results from reductions in plant vigour, fruit yield and marketable fruit quality. The extent of damage is closely associated with the stage of crop growth at which infection occurs. Plants infected at an early stage may exhibit pronounced stunting, reduced flowering and substantial decreases in the number and size of fruits. Yield losses reported in different studies range widely, approximately from 30 to 100%, depending on the cultivar, virus isolate, geographical region, environmental conditions and timing of infection [1]. Under favourable conditions for disease development, susceptible okra fields in India may experience nearly complete disease incidence, with severe infections resulting in yield reductions of approximately 50–94% [4].

A major reason for the widespread occurrence of YVMD is its efficient transmission by the whitefly *Bemisia tabaci* (Gennadius). The disease is characteristically expressed as yellow discoloration of the leaf veins and veinlets. Symptoms may begin as small chlorotic patches and gradually expand to form a prominent yellow network contrasting with the surrounding green leaf tissue. In advanced infections, most or all of the vein system may become yellow, whereas the interveinal tissue can remain green or show varying levels of chlorosis. The yellow vein mosaic syndrome of okra has been associated with several begomoviruses, including Okra yellow vein mosaic virus (OYVMV) and other closely related viral species [1], [5].

Control of OYVMD remains difficult because no curative treatment is currently available to remove the virus once systemic infection has been established. Disease management therefore needs to emphasize prevention, early intervention and restriction of virus transmission. An integrated disease-management programme involving resistant or tolerant cultivars, effective whitefly suppression, crop sanitation, cultural practices, biological and botanical interventions, and carefully targeted insecticide applications is more appropriate and sustainable than dependence on any individual control measure [1].

The present review provides a comprehensive overview of the agricultural and economic significance of okra and examines the importance and impact of yellow vein mosaic disease. It further discusses the taxonomy and characteristics of the associated begomoviruses, genome organization, molecular variation and genetic diversity, disease symptoms and development, and the biology and epidemiological significance of *Bemisia tabaci* as the principal vector. The review also considers interactions among the virus, vector and host plant, together with environmental factors that influence disease establishment and progression.

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Particular attention is given to integrated disease management, host resistance, vector suppression, molecular diagnostic techniques and emerging biotechnological approaches, with the overall objective of identifying effective, sustainable and durable strategies for reducing the impact of OYVMD in okra production.

2. OKRA YELLOW VEIN MOSAIC VIRUS

2.1 Geographical Distribution: Okra yellow vein mosaic virus (OYVMV) is a whitefly-transmitted begomovirus associated with yellow vein mosaic disease of okra (*Abelmoschus esculentus* L.). The disease was first recognized in India in 1924, and yellow vein mosaic has subsequently become one of the most important viral diseases of okra in tropical and subtropical production systems. However, molecular studies have established that the yellow vein mosaic syndrome does not have a single viral etiology. Several begomovirus species, including OYVMV, Bhendi yellow vein mosaic virus (BYVMV) and Okra enation leaf curl virus (OELCuV), may produce comparable symptoms in okra. Therefore, the geographical distribution of OYVMV should be distinguished from the much wider distribution of okra yellow vein mosaic disease [1], [3]. The Indian subcontinent is particularly important in the epidemiology and evolution of begomoviruses associated with okra. India has been considered a major centre of begomovirus diversity, and numerous virus species associated with yellow vein mosaic and related symptoms have been characterized from different parts of the country. The presence of different begomoviruses in geographically separated okra-growing regions indicates that the disease complex is genetically diverse and that the predominant virus can vary according to location. Molecular surveys have also demonstrated recombination and genetic differentiation among okra-associated begomoviruses, suggesting continuous evolution of these viruses under field conditions [6], [7]. OYVMV has also been characterized outside India, particularly from the neighbouring Indian subcontinent. A complete genome of an OYVMV isolate from Faisalabad, Pakistan, was reported by [8], providing an important reference sequence for subsequent molecular studies. The occurrence of closely related begomoviruses in India and Pakistan is epidemiologically significant because both countries have extensive okra cultivation and favourable conditions for multiplication of the principal vector, *Bemisia tabaci*.

The disease has also been reported from Sri Lanka and other Asian countries, although the virus responsible for the symptoms is not necessarily OYVMV in every location. For example, OYVMV was reported in association with yellow vein mosaic symptoms in Sri Lanka, while subsequent molecular investigations demonstrated the involvement of other begomoviruses in the Sri Lankan disease complex [9]. This observation illustrates the importance of molecular identification when reporting the geographical distribution of OYVMV. More broadly, begomoviruses capable of producing yellow vein mosaic, yellow mosaic, leaf curl or related symptoms in okra have been reported from Asia, Africa and the Americas. However, several of these viruses are taxonomically distinct from OYVMV. Consequently, reports of "okra yellow vein mosaic virus" in older literature should be interpreted cautiously because disease symptoms were historically used more frequently than sequence-based criteria for identification. Modern classification relies primarily on complete or partial genome sequences, phylogenetic relationships and established species demarcation criteria.

The distribution of OYVMV is closely linked to the ecology of its insect vector, *B. tabaci*. The vector has a broad host range and can acquire begomoviruses from infected plants and subsequently transmit them to healthy plants during feeding. Consequently, the movement and population dynamics of whiteflies, availability of infected plant material and alternate hosts, cropping pattern and climatic conditions can all contribute to the establishment and spread of OYVMV. The occurrence of the virus is therefore best understood as the outcome of a dynamic interaction between virus, host, vector and environment.

2.2 Okra Yellow Vein Mosaic Virus (OYVMV): OYVMV belongs to the genus Begomovirus of the family Geminiviridae**.** Begomoviruses are characterized by circular single-stranded DNA genomes enclosed within twinned, or geminate, particles. They predominantly infect dicotyledonous plants and are transmitted naturally by whiteflies, particularly members of the *Bemisia tabaci* species complex. Transmission occurs in a persistent-circulative manner, allowing the virus to move through the insect vector before being introduced into a new plant during feeding [5]. Unlike some begomoviruses that possess two genomic components, OYVMV is considered a monopartite begomovirus**,** with its essential genetic information present on a single circular DNA molecule generally referred to as DNA-A. The genome is approximately 2.7 kb in length; an OYVMV genome reported from Pakistan was approximately 2,741 nucleotides long. The relatively small genome nevertheless encodes proteins required for viral replication, regulation of gene expression, movement within the host and interactions with host defence mechanisms [8].

2.2.1 Genome organization: The genome of OYVMV contains several open reading frames (ORFs) arranged on both the virion-sense and complementary-sense strands. The organization is characteristic of Old World monopartite begomoviruses. The major genes include V1, V2, C1, C2, C3 and C4**,** although nomenclature can vary among studies according to the annotation system used [10], [11]. The V1/AV1 gene encodes the coat protein (CP), which is responsible for packaging the viral DNA into virus particles. In addition to its structural function, the coat protein contributes to virus movement and plays an important role in interactions with the whitefly vector. Because of its relative conservation and biological significance, the coat protein gene is frequently targeted for molecular detection, sequence comparison and phylogenetic analysis of begomoviruses. The V2/AV2 gene**,** commonly referred to as the pre-coat protein gene in Old World begomoviruses, is involved in virus movement and pathogenicity.

Its precise functions may vary among begomovirus species, but the protein is generally associated with the movement of viral infection from initially infected cells to neighbouring and systemic tissues [1].

Among the complementary-sense genes, C1/AC1 encodes the replication-associated protein (Rep). Rep is indispensable for viral genome replication because it recognizes the origin of replication, initiates the replication process and interacts with host cellular machinery. Begomoviruses replicate through a rolling-circle mechanism after their incoming single-stranded DNA is converted into a double-stranded DNA replicative form within the plant nucleus.

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The C2/AC2 gene encodes the transcriptional activator protein (TrAP), which regulates the expression of viral genes and also participates in suppression of host defence responses. The C3/AC3 gene encodes the replication-enhancer protein (REn), which assists efficient accumulation of viral DNA. The C4/AC4 gene is associated with pathogenicity and symptom expression and can interfere with several host regulatory and defence pathways [12].

Table 1: Major genomic regions/proteins of OYVMV and their principal functions

Genomic region	Encoded protein	Principal function
V1/AV1	Coat protein (CP)	Encapsidation, movement and vector interaction
V2/AV2	Pre-coat protein	Virus movement and pathogenicity
C1/AC1	Replication-associated protein (Rep)	Initiation and regulation of viral DNA replication
C2/AC2	Transcriptional activator protein (TrAP)	Regulation of viral gene expression and suppression of host defence
C3/AC3	Replication enhancer protein (REn)	Enhancement of viral DNA replication
C4/AC4	C4 protein	Pathogenicity, symptom development and modulation of host responses

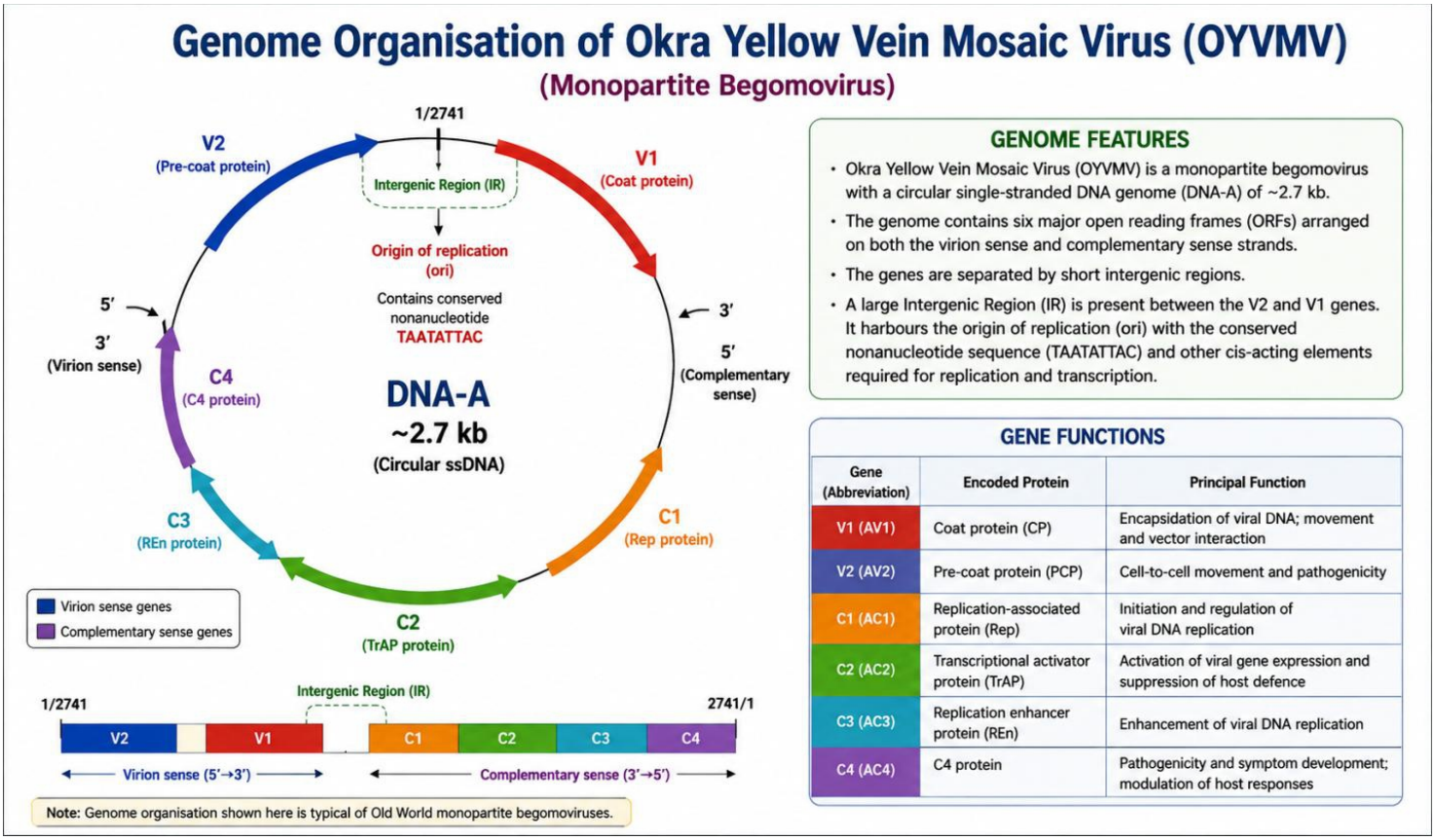

Fig. 1: Genome Organisation of Okra Yellow Vein Mosaic Virus

The genome also contains a non-coding intergenic region (IR) that includes the origin of replication and regulatory sequences controlling viral replication and transcription. A conserved stem-loop structure containing the characteristic nonanucleotide sequence TAATATTAC forms a critical part of the replication origin. Replication-associated proteins recognize sequence motifs within this region and initiate viral DNA replication [13].

2.2.2 Replication of OYVMV: Following inoculation by *B. tabaci*, the viral genome enters susceptible okra cells and reaches the nucleus. Because the incoming viral genome is single stranded, it is first converted into a double-stranded DNA form using host cellular enzymes. This double-stranded form serves as the template for transcription and production of viral proteins. The Rep protein then initiates replication of the viral genome through a rolling-circle replication mechanism**.** Viral DNA replication is closely associated with host cell-cycle machinery because begomoviruses rely heavily on host factors for synthesis of their DNA. Once sufficient viral DNA accumulates, newly synthesized genomes can be converted into single-stranded DNA molecules, which are subsequently encapsidated by the coat protein [12].

The virus subsequently moves from initially infected cells to adjacent cells and eventually establishes systemic infection throughout the plant. The movement process is particularly important for symptom development because systemic infection of young leaves and developing tissues leads to the characteristic yellowing of veins and veinlets and associated reductions in plant growth and fruit production.

3. VARIABILITY IN OKRA YELLOW VEIN MOSAIC VIRUS (OYVMV)

Genetic variation is an important feature of OYVMV and other begomoviruses. Comparative studies of OYVMV isolates from different geographical locations have revealed nucleotide substitutions and differences in individual genomic regions. The extent of variation is not uniform throughout the genome; some regions, particularly those involved in essential structural functions, tend to be relatively conserved, whereas pathogenicity-associated regions may exhibit greater sequence variability [14]. Recombination is another important evolutionary mechanism in begomoviruses. The coexistence of multiple begomovirus species in the same geographical region and host crop provides opportunities for exchange of genetic material.

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Evidence of recombination among begomoviruses associated with okra yellow vein mosaic disease has been reported from India, indicating that recombination may contribute to the emergence of genetically distinct virus populations [7]. Such genetic diversity has practical consequences. Variability among virus isolates may influence symptom severity, host range, vector transmission, molecular detection and the effectiveness of host resistance. It may also complicate the development of universal diagnostic primers and durable resistance against the disease complex.

3.1 OYVMV and the yellow vein mosaic disease complex: A critical consideration in studies of OYVMV is the distinction between the virus and the disease**.** Yellow vein mosaic disease of okra is not necessarily caused by OYVMV in every geographical region. Recent molecular investigations have established that OYVMD can result from infection by several distinct begomoviruses, including OYVMV, BYVMV, OELCuV and other okra-associated begomoviruses). Consequently, the presence of yellow vein symptoms alone cannot be considered sufficient evidence for identifying OYVMV. A reliable diagnosis should ideally combine field symptomatology with molecular detection and sequence analysis. PCR-based detection followed by sequencing of suitable genomic regions, particularly the coat protein region or complete DNA-A, can provide greater confidence in virus identification and facilitate differentiation among closely related begomoviruses.

This distinction is particularly relevant for epidemiological surveys and resistance-screening programmes. If different begomovirus species occur within the same region, a genotype that appears resistant to one isolate may not necessarily exhibit equivalent resistance against another virus. Therefore, future resistance-breeding programmes should evaluate germplasm against genetically diverse virus populations rather than relying on a single virus isolate.

4. SYMPTOMOLOGY

Okra yellow vein mosaic disease (OYVMD) is a major viral disease of okra (*Abelmoschus esculentus* L.) and is widely recognized as an important constraint to okra production. The disease is mainly characterized by progressive yellowing of the veins and veinlets, which produces a conspicuous yellow network on the otherwise green leaf surface. Although the disease is commonly associated with okra yellow vein mosaic virus, molecular studies have shown that several begomoviruses can be associated with yellow vein mosaic symptoms in okra. Consequently, the intensity and pattern of symptoms may differ according to the virus or virus complex involved, the cultivar, plant age at infection and prevailing environmental conditions [1.].

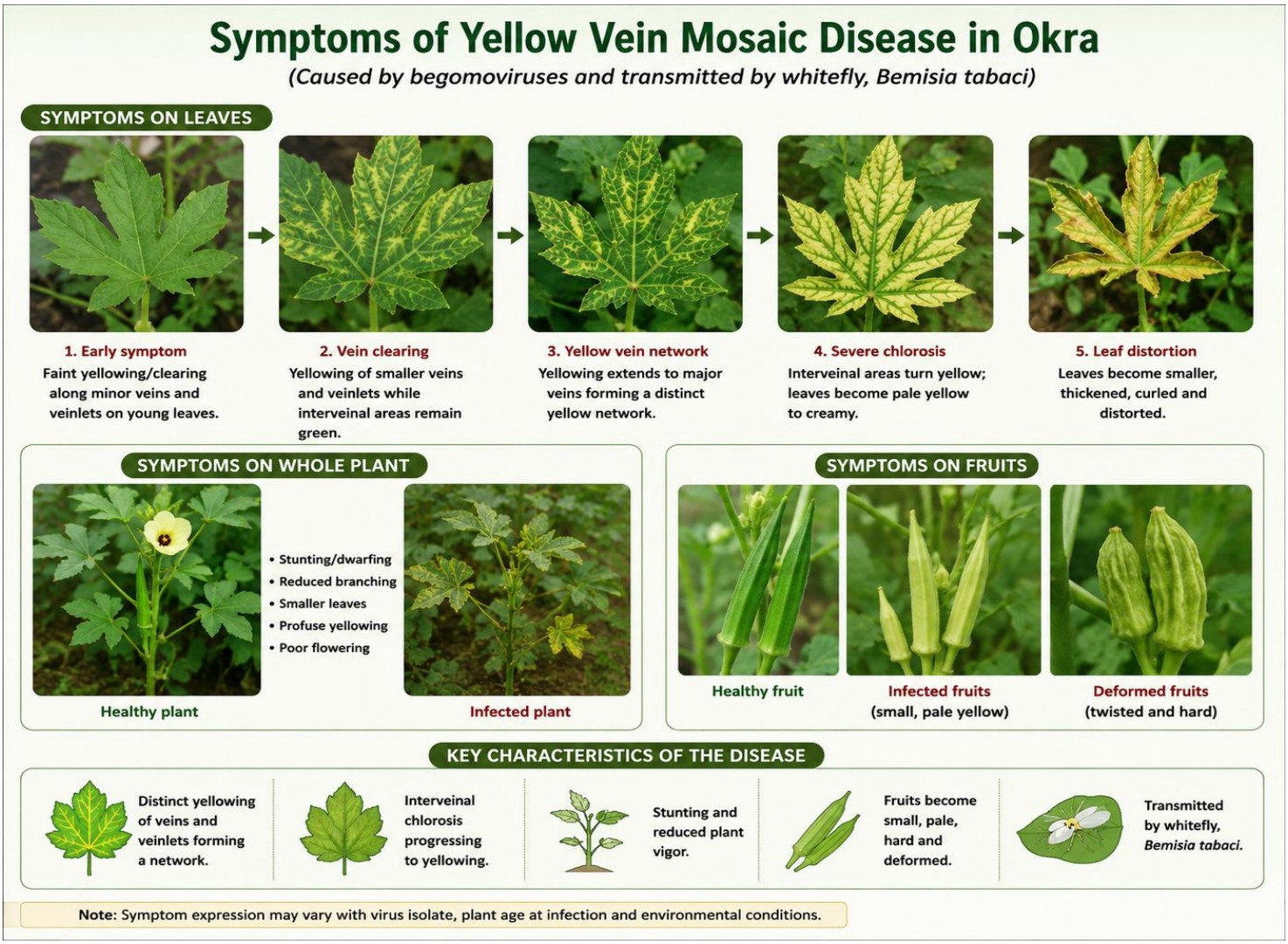

Fig. 2: Symptoms of Okra Yellow Vein Mosaic Virus

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4.1. Initial symptoms: The first visible symptoms generally develop on young leaves. Initially, the veins and smaller veinlets become lighter in colour, producing faint vein clearing or chlorotic patches. At this stage, the symptoms may be subtle and difficult to distinguish from other causes of leaf chlorosis. As the infection becomes established, yellowing becomes more pronounced and spreads along the secondary and tertiary veins.

4.2. Development of the yellow vein pattern: A characteristic feature of the disease is the formation of a distinct yellow network along the veins and veinlets**.** With disease progression, the yellow colour becomes increasingly prominent and extends from the minor veins towards the major veins. The contrast between the yellow vascular network and the surrounding green leaf tissue gives infected leaves their typical yellow vein mosaic appearance. In severely infected plants, chlorosis may spread beyond the veins and involve a large proportion of the leaf lamina, resulting in yellow to pale-yellow leaves [1], [3], [5].

4.3. Leaf symptoms: Infected leaves may show several morphological abnormalities in addition to vein yellowing. These include reduced leaf area, curling, distortion, vein thickening and abnormal leaf development**.** Newly emerging leaves are often more severely affected because systemic virus infection interferes with normal leaf development. In advanced stages, leaves may become markedly pale and distorted, with a substantial reduction in green, photosynthetically active tissue. The severity of leaf symptoms is strongly influenced by the age of the plant when infection occurs. Plants infected during the early vegetative phase generally develop more pronounced symptoms than those infected at a later stage. Early infection can result in continuous production of symptomatic leaves throughout subsequent plant growth [3], [4], [5].

4.4. Effects on plant growth:
The disease can adversely affect the overall growth and development of infected plants. Severe infections, particularly those occurring at an early growth stage, may result in plant stunting, reduced leaf production and restricted branching**.** The reduction in healthy leaf area decreases the plant's photosynthetic capacity and consequently affects biomass accumulation and reproductive development. Infected plants may also show delayed or reduced flowering. Because virus infection occurs systemically, its effects can continue throughout the growing period, particularly when plants are infected at a young age [1], [4].

4.5. Fruit symptoms: The disease also produces noticeable symptoms on okra fruits, which contributes directly to economic losses. Fruits produced by severely infected plants may be smaller than normal, pale green to yellowish, distorted and poorly developed**.** Some fruits may become twisted or malformed, resulting in reduced marketability and consumer acceptance. The reduction in fruit size and number is particularly severe when plants become infected before or during the reproductive phase. Consequently, the disease affects not only plant growth but also the quantity and quality of marketable produce.

4.6. Influence of plant age at infection: The stage at which infection occurs has a major influence on disease severity. Early infection generally results in more severe symptoms and greater yield reduction**,** whereas infection at alater stages tends to produce comparatively less severe effects.

Young plants infected systemically may exhibit pronounced vein yellowing, leaf deformation, stunting and substantial impairment of reproductive growth. Thus, the timing of infection is an important factor in determining the ultimate severity of OYVMD and its impact on crop productivity. Early infection allows the virus to establish throughout the plant before substantial vegetative and reproductive growth has occurred.

4.7. Diagnostic importance of symptoms: The characteristic yellowing of veins provides a useful field indication of OYVMD. However, visual symptoms alone cannot always establish the identity of the causal virus because different begomoviruses may induce similar symptoms in okra. Mixed infections can also modify the appearance and severity of disease symptoms. Therefore, molecular diagnostic techniques such as PCR and nucleotide sequencing are useful for confirming the identity of the virus associated with symptomatic plants. Such confirmation is particularly important in epidemiological investigations, virus characterization and studies aimed at identifying resistant or tolerant okra genotypes [1], [4].
Overall, the symptomatology of okra yellow vein mosaic disease is dominated by veinal chlorosis and the formation of a conspicuous yellow network**,** followed by leaf distortion, reduced leaf development, plant stunting and deterioration of fruit quality. The disease is most damaging when infection takes place during the early stages of plant growth. Early infection can interfere with both vegetative and reproductive development, ultimately resulting in fewer, smaller and poorly developed fruits. Although the characteristic yellow vein pattern is useful for field recognition, molecular confirmation is recommended because several begomoviruses can be associated with yellow vein mosaic symptoms in okra.

5. TRANSMISSION
Okra yellow vein mosaic disease is mainly transmitted by the whitefly *Bemisia tabaci* (Hemiptera: Aleyrodidae). The viruses associated with this disease, including okra yellow vein mosaic virus and bhendi yellow vein mosaic virus, belong to the genus *Begomovirus* and are transmitted by *B. tabaci* in a persistent-circulative manner. The transmission process begins when a non-viruliferous whitefly feeds on an infected okra plant and acquires virus particles along with phloem sap. During feeding, the insect inserts its stylets into the plant vascular tissues, allowing the virus to enter its digestive system. The virus subsequently passes through the insect's midgut and reaches the haemolymph before moving to the primary salivary glands [15], [16]. After acquisition, the virus undergoes a specific circulation pathway within *B. tabaci*. Begomoviruses are not simply carried externally by the insect; instead, they interact with internal tissues and cellular components of the vector. The viral coat protein plays an important role in the movement of virions across the midgut and into the haemolymph. From the haemolymph, virus particles are transported toward the salivary glands. Specific cells of the primary salivary glands are involved in the retention and subsequent release of begomoviruses during feeding [17].
When a viruliferous whitefly moves to a healthy okra plant and begins feeding, virus particles are introduced into the plant through the insect's salivary secretions. This process is known as inoculation. The virus then enters susceptible plant cells, multiplies and spreads systemically through the plant. As infection progresses, characteristic symptoms such as yellowing of the veins and veinlets become visible.

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Experimental studies have demonstrated that the duration of both virus acquisition and inoculation feeding can influence transmission efficiency. [18] reported differences in transmission efficiency between two genetic groups of *B. tabaci*, namely MEAM1 and Asia I, when transmitting bhendi yellow vein mosaic virus. MEAM1 showed greater transmission efficiency than Asia I under the experimental conditions used in the study. The transmission of begomoviruses by *B. tabaci* is generally considered persistent-circulative or circulative-nonpropagative. This means that the virus circulates through the insect's body and remains transmissible for an extended period, but it generally does not replicate within the vector. Consequently, a viruliferous whitefly can acquire the virus from an infected plant and subsequently transmit it to several healthy plants as it moves and feeds within the crop [15], [16].

The efficiency of transmission is influenced by several factors, including the virus isolate, genetic group of the whitefly, virus concentration in the source plant, acquisition access period, inoculation access period, age and susceptibility of the host plant, whitefly population density and environmental conditions. Differences between *B. tabaci* genetic groups are particularly important because this insect is a species complex consisting of genetically distinct groups with different abilities to acquire and transmit particular begomoviruses. The greater transmission efficiency observed for MEAM1 compared with Asia I in experimental studies demonstrates the importance of virus–vector compatibility in the development and spread of yellow vein mosaic disease [18].

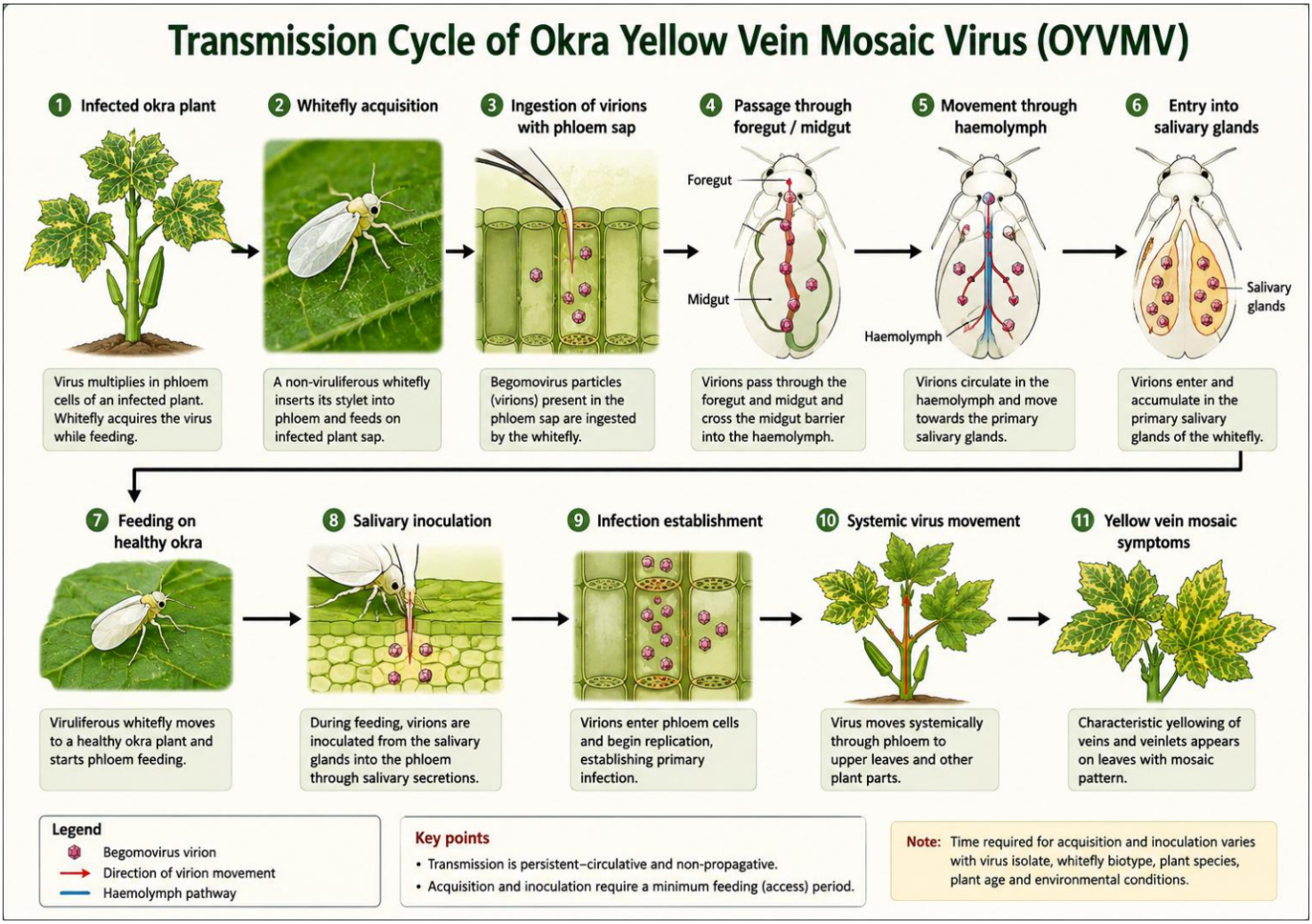

Fig. 3: Transmission Cycle of Okra Yellow Vein Mosaic Virus

The disease cycle can therefore be summarized as follows: an infected okra plant serves as a source of virus, a whitefly acquires the virus while feeding on the infected plant, the virus passes through the whitefly's internal tissues and reaches the salivary glands, and the viruliferous whitefly subsequently inoculates the virus into a healthy okra plant during feeding. The virus then establishes infection and spreads systemically, resulting in the characteristic yellow vein mosaic symptoms. Repeated movement of viruliferous whiteflies between infected and healthy plants can lead to rapid secondary spread of the disease within an okra crop.

Therefore, the interaction among the virus, *B. tabaci* and the host plant plays a central role in the epidemiology and development of okra yellow vein mosaic disease.

6. VECTOR

The major insect vector responsible for the transmission and spread of okra yellow vein mosaic disease is the whitefly, *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae). The disease is associated with begomoviruses such as Okra yellow vein mosaic virus and Bhendi yellow vein mosaic virus, which are transmitted by whiteflies from infected plants to healthy plants.

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Bemisia tabaci is a small, sap-sucking insect with a broad host range and is considered one of the most important vectors of begomoviruses affecting vegetable and other economically important crops. Its ability to feed on a wide range of plant species and move between infected and healthy plants contributes significantly to the rapid development and spread of yellow vein mosaic disease in okra fields [15]. The transmission process begins when a non-viruliferous whitefly feeds on an infected okra plant and acquires the virus along with the phloem sap. The period during which the insect feeds on the infected plant and obtains the virus is known as the acquisition access period. Following acquisition, the virus passes through the digestive system of the whitefly and moves internally through the haemolymph towards the salivary glands [19]. When the infected whitefly subsequently feeds on a healthy okra plant, virus particles are introduced into the plant through salivary secretions. The virus can then establish infection and spread systemically within the plant, eventually producing characteristic yellowing of the veins, chlorosis, reduction in leaf size and poor plant growth. Begomoviruses are generally transmitted by *B. tabaci* in a persistent-circulative manner, in which the virus passes through the insect's internal tissues before reaching the salivary glands. The virus generally does not replicate within the whitefly, but it can remain associated with the vector for a considerable period and be transmitted during subsequent feeding activities [15], [20].

The transmission efficiency of yellow vein mosaic viruses is influenced by the biological characteristics of both the virus and its whitefly vector. *Bemisia tabaci* is recognized as a species complex containing genetically distinct groups, commonly referred to as cryptic species. These groups can differ in their host preference, biological characteristics, insecticide susceptibility and capacity to transmit particular begomoviruses [18]. Demonstrated differences in the transmission of Bhendi yellow vein mosaic virus by two *B. tabaci* genetic groups, MEAM1 and Asia I. Their findings indicated that both groups were capable of transmitting the virus, although transmission efficiency varied between them. This variation demonstrates that the interaction between the virus and its vector is complex and that the genetic identity of the whitefly can influence the success of virus transmission. The number of whiteflies present on a plant is another important factor affecting disease development. A higher vector population increases the likelihood of contact between viruliferous insects and healthy plants and consequently increases the number of opportunities for virus inoculation. Female whiteflies may be particularly important in disease epidemiology because they contribute to the establishment and multiplication of vector populations on susceptible hosts. In addition, infected crop plants and alternative weed hosts can serve as sources of virus acquisition, allowing whitefly populations to acquire the pathogen and subsequently introduce it into healthy okra plants. Environmental conditions that favour whitefly multiplication can therefore indirectly enhance the development and spread of yellow vein mosaic disease.

The epidemiological importance of *B. tabaci* is consequently related not only to its ability to transmit the virus but also to its feeding behaviour, mobility, reproductive capacity and broad host range. The disease cycle can be broadly understood as a continuous interaction between infected plants, whitefly vectors and susceptible healthy plants. Whiteflies acquire the virus while feeding on infected plants and subsequently disseminate it to healthy plants during their feeding activities. Repeated movement of viruliferous whiteflies within a crop can result in the progressive increase of disease incidence and severity. For this reason, management of the vector is an important component of an integrated strategy for controlling okra yellow vein mosaic disease. Measures that reduce whitefly populations, minimize sources of infection and prevent the movement of viruliferous insects can help reduce secondary disease spread. However, vector management is most effective when combined with other approaches such as the use of tolerant or resistant cultivars, removal of infected plants, weed management and appropriate cultural and biological practices. Overall, *Bemisia tabaci* plays a central role in the epidemiology of okra yellow vein mosaic disease because it provides the principal biological pathway through which begomoviruses move from infected plants to healthy plants. Differences among whitefly genetic groups, virus isolates, vector density, feeding behaviour and environmental conditions can all affect transmission efficiency. A clear understanding of the biology and virus-transmission behaviour of *B. tabaci* is therefore essential for developing effective and sustainable management strategies against yellow vein mosaic disease in okra.

7. MANAGEMENT

Management of okra yellow vein mosaic disease (OYVMD) is challenging because the disease is primarily transmitted by the highly mobile whitefly, *Bemisia tabaci*, and may involve genetically diverse begomoviruses. Since the virus cannot be effectively eliminated once systemic infection is established, management is mainly directed towards preventing virus acquisition and transmission, reducing the vector population, minimizing sources of inoculum, and developing resistant host plants. Conventional vector management, cultural practices, host resistance, botanical and biological interventions, and molecular approaches can therefore be combined to reduce disease pressure. Recent research has also emphasized the need for integrated approaches rather than dependence on a single insecticide or disease-management component.

7.1. Vector Control: Since *B. tabaci* is the principal vector of begomoviruses associated with yellow vein mosaic disease, suppression of the vector is an important component of disease management. The objective is not merely to kill whiteflies after they have colonized the crop but to reduce the probability of virus acquisition from infected plants and subsequent inoculation into healthy plants. Vector management should begin during the early crop-growth period because plants infected at a young stage generally suffer greater growth reduction and yield loss. Regular monitoring of whitefly populations, removal of severely infected plants, weed management and prevention of overlapping susceptible crops can reduce the sources from which whiteflies acquire the virus. Reflective mulches and sticky traps can also contribute to reduction or monitoring of whitefly populations. Field validation of integrated pest-management programmes in okra has demonstrated the value of combining resistant cultivars, neem seed kernel extract, traps, removal of infected plants and need-based insecticide application.

7.1.1 Chemical management: Chemical control is principally directed against *B. tabaci* rather than the virus itself. Systemic and translaminar insecticides can reduce vector populations and consequently decrease the number of virus-transmission events. In field studies on okra, insecticides such as imidacloprid, thiamethoxam, malathion and other compounds

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targeting sucking pests have been evaluated for suppression of whitefly and reduction of yellow vein mosaic development. A field study by Borah RK and Nath PD 1995) [21], for example, reported that malathion 50 EC at 2 ml L^{-1} combined with sarpagandha leaf extract (10%) substantially reduced disease severity compared with the untreated control. Earlier work specifically examining OYVMV also demonstrated that certain compounds with potential virus-inhibitory or defence-inducing activity could reduce infection. Pun et al. reported significant reductions following treatment with acetyl salicylic acid, salicylic acid, benzoic acid and barium chloride**,** with barium chloride showing the greatest reduction among the tested chemicals under their experimental conditions. However, these results should be interpreted as experimental evidence and should not be considered a general recommendation for field application without crop-safety, regulatory and residue considerations.

For practical vector suppression, compounds registered for whitefly management in the respective jurisdiction may include insecticides from different modes of action, such as neonicotinoids, pyridine azomethines, sulfoximines, ketoenols or insect-growth regulators**,** depending on local registration and resistance status. Rotation of insecticides having different modes of action is important because repeated exposure to the same chemistry can select resistant *B. tabaci* populations. Therefore, chemical intervention should preferably be need-based and incorporated into an insecticide-resistance-management programme**,** rather than following repeated calendar sprays.

7.1.2 Botanical approaches: Botanical pesticides offer an important supplementary option because several plant-derived compounds possess repellent, antifeedant, oviposition-deterrent or insecticidal properties against *B. tabaci*. Neem **(***Azadirachta indica***)** is particularly important. Neem oil, neem seed kernel extract and azadirachtin-based formulations have been investigated against whitefly and OYVMD. Experimental work on OYVMV showed that 3% neem oil and 5% neem seed kernel extract significantly reduced virus infection when applied before inoculation with viruliferous whiteflies. Neem oil produced an 88.3% reduction in infection under the experimental conditions, while neem seed kernel extract produced an 86.7% reduction [22]. Other botanical materials investigated against the whitefly–OYVMD complex include Datura stramonium, ginger, tobacco, Vitex negundo, Ocimum sanctum, Adhatoda vasica, Bougainvillea spectabilis, Prosopis chilensis, Calotropis procera and Eucalyptus globulus**.** These materials have shown varying degrees of suppression of whitefly populations or disease development in experimental and field studies. Such variation is expected because efficacy depends on the plant species, plant part, extraction procedure, concentration, formulation and timing of application [23]. Botanicals should therefore be considered as components of an integrated programme rather than as direct curative treatments for virus-infected plants. Their greatest value lies in reducing vector activity and consequently lowering the probability of virus transmission**.**

7.2. Biological Control: Biological control of OYVMD is mainly indirect because biological agents are generally used to suppress *B. tabaci*, thereby reducing virus transmission. Natural enemies of whitefly include parasitoids, predators and entomopathogenic microorganisms.

More than 40 predator species, numerous parasitoids and several microbial pathogens have been reported in association with *B. tabaci*. Important parasitoid groups include *Encarsia* and *Eretmocerus*, while predators include coccinellids, lacewings and predatory bugs.

7.2.1 Parasitoids: *Encarsia formosa*, *Encarsia sophia*, *Eretmocerus eremicus* and *Eretmocerus mundus* are among the important parasitoids investigated for whitefly suppression. These parasitoids attack immature stages of *B. tabaci*, thereby reducing the number of adults that can acquire and transmit begomoviruses. *Eretmocerus* species are particularly important because they are capable of parasitizing *B. tabaci* and have been incorporated into biological control programmes in vegetable production systems [24]. Conservation of naturally occurring parasitoids is equally important. Broad-spectrum insecticides can eliminate beneficial parasitoids along with whiteflies and may consequently disrupt biological regulation of the vector. Thus, selective insecticides and compatible botanicals should be preferred where biological control is being employed.

7.2.2 Predators and Entomopathogenic fungi: Several predatory arthropods contribute to natural suppression of whitefly. Important examples include *Chrysoperla* spp., *Coccinella* spp., *Menochilus sexmaculatus***,** *Micraspis discolor***,** *Orius* spp. and predatory mites**.** Conservation of these organisms through reduced use of broad-spectrum insecticides can strengthen the natural biological-control component of OYVMD management [25]. Entomopathogenic fungi represent another promising biological approach against *B. tabaci*. Important candidates include: *Lecanicillium lecanii, Beauveria bassiana*, *Metarhizium anisopliae, Isaria fumosorosea*, etc. Among these, *L. lecanii* has shown particularly encouraging results against whitefly in okra. Mascarin, G. M et al, 2013 [26] found that *L. lecanii* reduced whitefly populations effectively under field conditions, and its combination with neem oil performed particularly well. The fungal treatments were also comparatively safe to naturally occurring ladybird beetles and spiders. The mechanism involves fungal spores attaching to the insect cuticle, germinating and subsequently penetrating the insect body. This makes entomopathogenic fungi fundamentally different from chemical insecticides and provides an additional mode of action that can be incorporated into resistance-management programmes.

7.3. Host Plant Resistance: Host plant resistance is considered one of the most desirable and sustainable approaches for managing okra yellow vein mosaic disease (OYVMD), as resistant cultivars can restrict disease development without requiring repeated chemical intervention. Extensive screening of cultivated and wild *Abelmoschus* germplasm has identified considerable variation in response to yellow vein mosaic disease, providing valuable sources for resistance breeding [27], [28]. The development of resistant varieties is particularly important because chemical management of a virus disease is largely indirect and depends mainly on suppression of its whitefly vector. Earlier studies have identified several resistant or moderately resistant genotypes and wild *Abelmoschus* species that can serve as sources of resistance in breeding programmes [27], [28]. Genetic studies have also indicated that resistance to yellow vein mosaic disease can involve both additive and non-additive gene effects, suggesting that appropriate breeding strategies are required for efficient transfer and accumulation of resistance factors [29].

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However, the durability of resistance remains a major concern. Different begomoviruses and virus strains occur in okra-growing regions, and resistance expressed against one virus population may not necessarily provide the same level of protection against another. The breakdown of resistance in previously resistant cultivars has been associated with the emergence and spread of new begomoviruses and changes in the virus–vector complex [11], [28]. Therefore, future breeding programmes should emphasize broad-spectrum and durable resistance**,** preferably by evaluating germplasm under both natural and artificial inoculation conditions. Molecular characterization of resistant and susceptible genotypes, identification of quantitative trait loci (QTLs), and integration of molecular breeding tools could accelerate the development of stable resistant cultivars [30].

7.4. Pathogen-Derived Resistance: Pathogen-derived resistance (PDR) is a molecular strategy in which selected genetic sequences originating from the pathogen are introduced into a susceptible plant to interfere with one or more stages of the viral infection cycle. The concept was originally developed from observations that expression of viral components in plants could provide resistance against subsequent virus infection [31]. For begomoviruses, several viral genes and genomic regions have been considered potential targets for PDR. These include the coat protein (CP), replication-associated protein (Rep), movement protein, antisense viral sequences, defective viral DNA and truncated or mutated replicase genes [30], [32]. The principal advantage of PDR is that viral components essential for replication, encapsidation or movement can be targeted directly. Nevertheless, the genetic diversity of begomoviruses associated with OYVMD represents a major obstacle. A construct designed against a highly variable region of one virus may provide limited protection against genetically distinct isolates. Consequently, conserved viral sequences and combinations of multiple targets should be considered when designing broad-spectrum resistance [30].

7.4.1 Coat Protein-Mediated Resistance: The coat protein (CP) is an important viral protein involved in encapsidation of viral DNA and plays an important role in the interaction between begomoviruses and their whitefly vector. Coat-protein-mediated resistance (CPMR) was among the earliest and most extensively investigated forms of pathogen-derived resistance. Expression of viral CP genes in transgenic plants has been shown to provide protection ranging from delayed infection and symptom attenuation to high levels of resistance, depending on the virus, host and transgene construct [31], [33], [34]. In begomoviruses, CP is an attractive target because it participates in viral encapsidation and vector interactions. Therefore, CP-mediated resistance has been proposed as a potential approach for managing begomoviruses infecting okra. Mishra et al. (2017) [30] specifically identified capsid protein-mediated resistance as one of the pathogen-derived strategies that could be exploited for developing resistance to YVMV and other begomoviruses in okra**.** However, CP-mediated resistance specifically developed and comprehensively validated in commercially cultivated okra remains an experimental approach. Future work should focus on CP sequences that are sufficiently conserved among the predominant begomoviruses occurring in different okra-growing regions. Combining CP-mediated resistance with RNAi or other viral targets may also provide a broader and more durable resistance response.

7.5. RNAi-Mediated Resistance: RNA interference (RNAi) is a sequence-specific antiviral defence mechanism in plants and has emerged as an important molecular approach for developing resistance against plant viruses. During viral infection, double-stranded RNA or RNA-derived structures can trigger the plant RNA-silencing machinery, resulting in the production of small interfering RNAs (siRNAs). These siRNAs guide silencing complexes towards complementary viral sequences and thereby suppress viral gene expression and multiplication [31], [35]. Begomovirus genomes contain several genes that can potentially be targeted through RNAi, including Rep, CP, AC2/TrAP, AC4 and other conserved sequences. Selection of an appropriate target is important because viral sequence variability can influence the effectiveness and durability of RNAi-mediated resistance. Targeting conserved regions or simultaneously targeting more than one viral gene may reduce the probability of resistance breakdown [30].

The potential of RNAi for begomovirus resistance in okra was already being investigated before 2018. Kurulekar et al. (2016) reported experimental work evaluating antisense and hairpin RNA constructs in okra against begomoviruses associated with yellow vein mosaic disease**,** providing an early proof-of-concept for exploiting RNA silencing as a resistance strategy in this crop [36]. The βC1 region of begomovirus-associated satellites is another particularly interesting target because βC1 is involved in symptom development and suppression of host defence. Earlier molecular studies demonstrated that viral proteins associated with begomoviruses can interfere with plant RNA-silencing pathways, highlighting the importance of targeting viral suppressor functions as part of a resistance strategy [37]. Thus, RNAi has considerable potential for developing broad-spectrum resistance against OYVMD**,** particularly if conserved sequences shared by predominant begomovirus populations are selected. Nevertheless, transformation efficiency, stability of transgene expression, viral sequence variation and biosafety considerations must be addressed before this approach can be translated into commercial cultivars.

7.6. Replicase Gene-Mediated Resistance: The replication-associated protein **(**Rep**)** is indispensable for begomovirus replication and therefore represents an important target for pathogen-derived resistance. Interference with Rep expression or activity can potentially restrict viral DNA replication at an early stage, thereby reducing subsequent systemic movement and symptom development. Rep-mediated resistance has been demonstrated in several plant-virus systems. Transgenic expression of modified, truncated or mutated viral replicase sequences has produced resistance by interfering with viral replication or through sequence-specific mechanisms [31]. For begomoviruses infecting okra, the Rep gene has been identified as one of the potential targets for pathogen-derived resistance. Mishra et al. (2017) discussed the use of truncated or mutated Rep sequences as a possible strategy for managing begomoviruses in okra [30]. The major challenge is that the Rep sequence may vary among different begomovirus species and isolates. Therefore, selection of conserved Rep regions is important if the objective is to obtain broad-spectrum protection. Combining Rep-mediated resistance with RNAi directed against an independent viral gene could potentially reduce the probability of resistance breakdown.

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7.7. Cross-Protection: Cross-protection is a biological phenomenon in which prior infection of a plant with a mild or attenuated strain protects it against subsequent infection or disease caused by a closely related severe strain. The phenomenon has been investigated for many plant viruses and has also contributed conceptually to the development of pathogen-derived resistance [38]. The protective effect may involve several mechanisms, including competition between virus strains, exclusion of the challenging virus from infection sites and RNA-silencing-mediated antiviral responses. However, the precise mechanism is dependent on the particular virus–host combination. For OYVMD, cross-protection remains largely a prospective research strategy rather than an established management practice**.** Its application would require identification of a genetically stable and biologically characterized mild strain that provides reliable protection against economically important severe strains without itself producing unacceptable symptoms or yield losses.

The considerable diversity of begomoviruses infecting okra, together with the possibility of recombination and mixed infections, makes this approach particularly challenging. Consequently, extensive biological, molecular and biosafety evaluation would be necessary before considering practical application.

7.8. Plant Defence Activators: Activation of the plant's endogenous defence mechanisms represents another complementary strategy for reducing the development of yellow vein mosaic disease. Salicylic acid (SA) is particularly important because it is associated with plant defence signalling and systemic acquired resistance. Treatment with defence-inducing compounds may strengthen host responses and restrict virus multiplication or reduce the severity of disease [39]. Specific studies on okra have investigated salicylic acid, acetyl salicylic acid, benzoic acid and barium chloride for their effects on OYVMV infection. Pun et al. (2002) reported that all tested chemicals significantly reduced OYVMV infection under glasshouse conditions. Barium chloride produced a 76.67% reduction in infection, while acetyl salicylic acid and salicylic acid produced reductions of 75.0% and 73.3%, respectively.

The same study also demonstrated considerable effects of botanical products, with 3% neem oil and 5% neem seed kernel extract reducing OYVMV infection by 88.3% and 86.7%, respectively, under the experimental conditions used [40]. Plant defence activators should not be interpreted as conventional antiviral chemicals that directly destroy virus particles. Rather, their potential value lies in priming or strengthening host defence mechanisms, thereby making the plant less favourable for virus establishment and multiplication. Other compounds, including benzoic acid, citric acid and monopotassium phosphate, have also been investigated as defence activators against OYVMD. Such treatments may influence defence-related biochemical pathways and improve the plant's ability to tolerate or restrict infection. These approaches are best considered as supplementary components of integrated disease management rather than replacements for genetic resistance or vector management.

7.9 Genome Engineering-Based Resistance: Genome engineering represents a promising future approach for managing okra yellow vein mosaic disease (OYVMD), particularly because its associated begomoviruses possess circular single-stranded DNA genomes that form double-stranded DNA intermediates during replication. These viral DNA forms provide suitable targets for programmable nucleases such as CRISPR/Cas systems. The first experimental demonstrations showed that CRISPR/Cas9 could target geminiviral DNA and substantially reduce viral accumulation and disease development. Baltes et al. (2015) demonstrated this approach against bean yellow dwarf virus [41], while Ji et al. (2015) obtained similar antiviral effects against beet severe curly top virus by targeting viral genomic sequences [42].

For OYVMD, CRISPR/Cas9-mediated targeting of conserved regions of the begomovirus genome**,** particularly the replication-associated protein (*Rep*) gene, coat protein (*CP*) gene and conserved intergenic/origin-of-replication region, could be investigated as potential resistance strategies. Targeting highly conserved sequences may be particularly useful because begomoviruses associated with yellow vein mosaic disease show considerable genetic variability. Studies with other begomoviruses have demonstrated that targeting the conserved intergenic region can provide activity against more than one virus, indicating the potential for developing broad-spectrum resistance.

Another possibility is host genome editing, in which CRISPR/Cas is used to modify host susceptibility genes required for viral infection rather than directly targeting viral DNA. Such an approach could potentially provide durable resistance while avoiding dependence on a particular virus isolate. Genome editing has already been proposed for targeting host susceptibility factors involved in plant-virus interactions [43].

TALENs and zinc-finger nucleases are alternative genome-engineering platforms that could theoretically be used for targeted modification of viral or host sequences. However, specific TALEN-, ZFN- or CRISPR/Cas-mediated resistance against OYVMD in okra has not been reported yet**.** Therefore, these technologies should presently be considered prospective research avenues rather than established management practices**.** Future work should focus on identifying conserved viral targets and host susceptibility genes in OYVMD and validating their effectiveness against diverse virus isolates and under field conditions.

7.10. Integrated Disease Management: Because OYVMD is a vector-borne disease, its successful management requires simultaneous consideration of the virus, vector, host and crop environment**.** An integrated disease-management programme should therefore begin before sowing by selecting a suitable sowing period, using healthy seed and adopting cultivars with the highest available level of resistance or tolerance. Fields should be kept free from volunteer okra and important weed hosts that can harbour whiteflies and virus inoculum. Early removal and destruction of symptomatic plants can reduce the number of virus sources available for acquisition by *B. tabaci*. During crop establishment, regular monitoring of whitefly populations should be undertaken using visual observations and yellow sticky traps. Reflective mulches and suitable border crops may further interfere with vector landing and movement. Where biological control is feasible, conservation of parasitoids such as *Encarsia* and *Eretmocerus*, predators such as coccinellids and lacewings, and entomopathogenic fungi such as *L. lecanii*, *B. bassiana* and *M. anisopliae* can contribute to vector suppression. In okra, *L. lecanii*, particularly when used in combination with neem oil, has shown promising suppression of whitefly while maintaining comparatively greater safety to natural enemies.

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Botanical products such as neem oil, neem seed kernel extract and azadirachtin can be incorporated where appropriate. Experimental studies have demonstrated considerable reduction in OYVMV infection following neem-based treatments, while field studies have also shown the usefulness of neem-based products against whitefly. Chemical insecticides should be reserved for situations where vector populations exceed economically or epidemiologically important levels. When required, insecticides should be selected according to current local registration and rotated among different modes of action. This is particularly important because *B. tabaci* has a well-documented capacity to develop insecticide resistance. Excessive and repeated spraying can also suppress parasitoids and predators, thereby weakening biological regulation of the vector. Field studies in okra have shown that some microbial and botanical treatments can be substantially safer to natural enemies than conventional insecticide treatments.

The longer-term component of IDM should focus on host resistance and molecular resistance technologies**.** Conventional breeding, marker-assisted selection and gene pyramiding can strengthen genetic resistance, whereas RNAi, CP-mediated resistance and Rep-mediated resistance offer opportunities for developing broad-spectrum resistance against diverse begomoviruses. Among these advanced approaches, βC1-targeted RNAi is particularly noteworthy because resistance-related transgenic research has already been demonstrated in okra. The emphasis should be on preventing infection rather than attempting to cure infected plants**.** Integration of these measures can reduce the initial virus inoculum, suppress the vector, restrict secondary transmission and increase the durability of host resistance. Such a multi-component strategy is likely to be more sustainable than reliance on insecticides alone.

8. CONCLUSION

Okra (*Abelmoschus esculentus* L. Moench) yellow vein mosaic disease (OYVMD) remains one of the most important constraints to profitable okra production, particularly in tropical and subtropical regions. The disease is primarily associated with begomoviruses and is efficiently disseminated by the whitefly *Bemisia tabaci*, making its epidemiology dependent on complex interactions among the virus, vector, host plant and environmental conditions. Early infection can severely restrict plant growth, reduce photosynthetic efficiency and cause substantial losses in marketable fruit yield. The characteristic yellowing of veins and veinlets, followed by extensive chlorosis and reduction in leaf area, makes the disease readily recognizable; however, symptom expression alone is insufficient for distinguishing the different begomoviruses and virus complexes involved. Molecular characterization has demonstrated considerable genetic diversity among begomoviruses associated with yellow vein mosaic disease, emphasizing the need for accurate diagnosis and region-specific disease-management strategies [11], [28], [30].

Management of OYVMD has traditionally focused on reducing populations of *B. tabaci*, since prevention of virus acquisition and transmission is more practical than attempting to eliminate the virus after systemic infection has occurred. Chemical insecticides can provide effective suppression of the vector, but their continuous and indiscriminate use may lead to insecticide resistance, destruction of beneficial organisms and environmental concerns.

Consequently, the future management programme should give greater emphasis to resistant cultivars, removal of infected plants, weed management, vector monitoring, botanical products, biological control agents and selective use of insecticides. Neem-based products, entomopathogenic fungi such as *Lecanicillium lecanii*, *Beauveria bassiana* and *Metarhizium anisopliae*, together with parasitoids and predators of *B. tabaci*, offer useful components for reducing vector pressure. However, these measures are unlikely to provide complete protection when used individually and should be incorporated into an integrated disease-management programme.

Host plant resistance remains the most attractive long-term approach because resistant cultivars can provide protection without repeated pesticide applications. Screening of cultivated and wild *Abelmoschus* germplasm has identified useful sources of resistance, but variation among begomoviruses and geographical populations of the pathogen makes durability of resistance an important concern [27], [28]. Future breeding programmes should therefore move beyond the identification of simply resistant or susceptible genotypes and focus on understanding the genetic and molecular basis of resistance. Integration of conventional breeding with molecular markers, QTL analysis, genomic selection and genome-wide association studies could accelerate the development of cultivars possessing broad-spectrum and durable resistance.

The advances in molecular biology have opened an entirely new dimension in OYVMD research. Pathogen-derived resistance, coat protein-mediated resistance, replicase-mediated resistance and RNA interference provide opportunities to interfere directly with viral replication, movement or gene expression. RNAi is particularly promising because conserved regions of essential viral genes can be targeted through sequence-specific silencing. Similarly, identification of host susceptibility factors may provide an opportunity to develop resistance by modifying plant genes required for successful viral infection. These technologies should not, however, be viewed as immediate replacements for conventional breeding and vector management; rather, they represent important research platforms for developing the next generation of resistant okra cultivars.

9 FUTURE PERSPECTIVES

The future of OYVMD research should move beyond the conventional approach of simply identifying the virus and controlling its whitefly vector. A major new research arena is the development of a comprehensive virus–vector–host interaction framework. High-throughput sequencing can be used to determine the complete diversity of begomoviruses, satellites and mixed infections occurring in different okra-growing regions. Establishment of a geographical and genomic database containing virus species, isolates, host range, vector populations and disease severity would greatly improve our understanding of disease emergence and resistance breakdown. Such information would also help identify conserved viral sequences that can be exploited for broad-spectrum molecular resistance. Another promising research direction is the discovery of new resistance genes and host susceptibility genes. Wild *Abelmoschus* species remain an underexploited reservoir of genetic diversity and could provide novel resistance alleles. Combining genome sequencing, transcriptomics, proteomics, metabolomics and GWAS could reveal the molecular pathways responsible for resistance.

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At the same time, identifying host proteins that are essential for begomovirus replication or movement could provide targets for CRISPR/Cas-based gene editing. Thus, future research may shift from merely transferring resistance genes into elite cultivars towards precisely modifying host factors that make plants susceptible to infection.

RNAi research can also be expanded from single-gene targeting to multi-target and conserved-sequence approaches. Simultaneous targeting of Rep, CP, AC2/TrAP, AC4 and, where relevant, βC1 could provide multiple barriers against viral infection. The identification of conserved sequences shared by the major begomoviruses infecting okra may permit development of broad-spectrum RNAi constructs capable of functioning against genetically diverse virus populations. Similarly, CRISPR/Cas technology offers an emerging possibility of directly targeting conserved regions of begomovirus DNA. Combining virus-targeted RNAi or CRISPR strategies with conventional genetic resistance could potentially generate multilayered resistance that is more difficult for the virus to overcome. The virus–whitefly interaction represents another relatively unexplored research arena. Future investigations should examine how different *B. tabaci* genetic groups acquire, retain and transmit different begomoviruses, and how temperature, plant genotype and vector microbiota influence these processes. The role of endosymbiotic bacteria in whitefly fitness and virus transmission is particularly promising and may eventually provide opportunities for manipulating vector competence rather than relying exclusively on insecticide-mediated mortality. Host-induced gene silencing (HIGS) could also be explored as a novel strategy in which plants produce silencing molecules targeting essential genes of *B. tabaci*, thereby simultaneously reducing vector fitness and virus transmission.

Climate change provides another important dimension for future OYVMD research. Changes in temperature, rainfall and humidity may alter whitefly multiplication, migration, virus replication and disease development. Consequently, long-term epidemiological studies should integrate climatic variables with vector abundance, virus incidence and crop growth stages. Combining these datasets with artificial intelligence and machine-learning techniques could facilitate the development of early-warning systems for OYVMD outbreaks. Such systems could predict periods of high disease risk and enable farmers to implement vector-management measures before widespread infection occurs.

The integration of remote sensing, hyperspectral imaging, thermal imaging, drones and artificial intelligence could further revolutionize disease diagnosis. Conventional visual assessment generally identifies disease after prominent symptoms have developed, whereas spectral changes associated with physiological stress may potentially be detected before visible symptoms appear. Development of smartphone- or drone-based diagnostic systems could therefore enable rapid identification and mapping of disease hotspots at field and landscape scales. This would facilitate targeted removal of infected plants and precision application of vector-management measures. Another important future requirement is the establishment of standardized OYVMD resistance-screening protocols. Differences in virus isolates, whitefly populations, inoculation techniques, vector density, environmental conditions and disease-rating scales can produce inconsistent assessments among studies.

Standardized screening using characterized virus isolates and vector populations, combined with molecular confirmation of infection and multi-location field validation, would improve the reliability of resistance identification and breeding programmes.

Ultimately, the most promising direction is the development of a multilayered disease-management system in which genetic resistance, vector resistance, molecular antiviral technologies, biological control, precision monitoring and predictive epidemiology operate together. Such an approach would transform OYVMD management from a predominantly reactive and pesticide-dependent system into a predictive, precision-based and environmentally sustainable programme. In conclusion, the greatest opportunity emerging from the present review is not the development of another individual control measure, but the integration of conventional pathology with genomics, vector biology, molecular biotechnology, artificial intelligence and precision agriculture. Research on OYVMD should increasingly focus on understanding the complete disease system raF3Fther than studying the pathogen, vector or host in isolation. The development of broad-spectrum, durable and multilayered resistance, combined with precise vector and disease forecasting, could provide a sustainable solution to yellow vein mosaic disease and may also establish a transferable framework for managing other economically important whitefly-transmitted begomovirus diseases.

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Authors' contributions:

Abdul Majid Ansari: Concept and designing of the research, contribution of the data collection and gathering technical information of the research, and overall supervision of the research as well as preparation of the manuscript and finalization.

Nishar Akhtar: Data collection, designing of the figures, and preparation of the manuscript.

Shahina Perween: Manuscript preparation, English editing, and contribution to the breeding aspects of the content.

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