HEMIPTERAN-TRANSMITTED PLANT VIRUSES: RESEARCH PROGRESS AND CONTROL STRATEGIES

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HIGHLIGHTS
● Research findings on the insect-virus interaction
● Influences of immunity, feeding and microorganisms on virus transmission
● Latest applications for virus control strategies

ABSTRACT About 80% of plant viruses are transmitted by specific insect vectors, especially hemipterans with piercing-sucking mouthparts. Many virus-transmitting insects are also important crop pests that cause considerable losses in crop production. This review summarizes the latest research findings on the interactions between plant viruses and insect vectors and analyzes the key factors affecting insect transmission of plant viruses from the perspectives of insect immunity, insect feeding, and insect symbiotic microorganisms. Additionally, by referring to the latest applications for blocking the transmission of animal viruses, potential control strategies to prevent the transmission of insect-vectored plant viruses using RNAi technology, gene editing technology, and CRISPR/Cas9 + gene-driven technology are discussed.

KEYWORDS control strategies, feeding, immunity, insect vector, microorganism, plant virus
1 CHALLENGE OF INSECT-VECTORED PLANT VIRUSES

Plant viruses are an important factor that decreases the yield and quality of grain, vegetables, flowers, fruits, and other crop products. Due to the lack of effective control strategies, plant viruses cause an estimated economic impact of > 30 billion USD annually[1]. Insects are important vectors of plant viruses. Among the 1100 known plant viruses, about 80% can be transmitted by vector insects including aphids, planthoppers, whiteflies, leafhoppers and thrips[2]. In addition to growing virus-resistant plants, the integrated management of vector insects is also critical in the control of plant virus diseases[3]. At present, pest control relies mostly on pesticide application, which is often incompatible with the natural enemies and so can result in pest resurgence. Additionally, the excessive use of pesticides leads to environmental pollution and is inconsistent with the sustainable development of agriculture[4]. Therefore, it is important to achieve the comprehensive control of plant viruses based on knowledge of the interactions between insects and plant viruses, searching for the key factors that influence virus transmission, and circumvention of the transmission routes.

2 CHARACTERISTICS OF PLANT-VIRUS TRANSMISSION BY HEMIPTERANS

Based on transmission mode, plant viruses can be classified as nonpersistent, semipersistent and persistent[5]. In nonpersistent transmission, viruses are retained in the distal parts of stylets from a few minutes to several hours, and in semipersistent transmission, viruses primarily bind to the anterior of the digestive tract. Generally, nonpersistent and semipersistent viruses lose transmissibility when the vector molts and viruses cannot replicate within the vector insects. The period of acquiring and transmitting semipersistent viruses is longer than that of nonpersistent viruses. Persistent viruses can be further divided into proliferative and nonpropagative types. In persistent nonpropagative transmission, viruses do not lose transmissibility when the vector molts and this mode of transmission is found mainly in Luteoviridae[3] (Table 1). In persistent propagative transmission, most plant viruses multiply and circulate inside the vector insects following the route: midgut-hemolymph-salivary gland, which is associated with multiple interaction among viruses, insects and plants[2,6].

The binding sites of nonpersistent and semipersistent viruses are located at the tip of the stylet or in the anterior of digestive tracts, where viruses specifically interact with insect receptors through coat proteins and helper proteins. The cauliflower mosaic virus (CaMV) transmitted by Brevicoryne brassicae has been extensively investigated. In this system the transmission of CaMV requires the viral protein 2 (P2) and viral protein 3 (P3). P2 is a helper protein with its N terminus binding to the aphid stylet and C terminus binding to the P3 on the surface of the virus, thereby building a bridge between the virus particle and the insect receptor[7–9]. In CaMV-infected plants, P2, P3 and virus particles aggregate to form transmission bodies. When the insects probe infected plant tissues the action of feeding triggers a rapid and massive influx of tubulin into the transmission body, leading to transmission activation[7,10]. CaMV P2 binds to insect receptors at the tip of the stylet. However, the identification of this receptor is challenging as the stylet tip consists of highly cross-linked chitin fibers and cuticular proteins[11–13]. In 2018, Webster et al. were the first to identify two receptor proteins (Stylin-01 and −02) for CaMV and demonstrated the potential target for controlling noncirculative plant viruses[14].

Interactions between vector insects and persistent transmitted viruses are more complex. The viruses circulate within the insects from the gut lumen into the hemolymph or other tissues and finally invade the salivary glands from which the viruses are released into new hosts during insect feeding. Also, some persistent viruses can break through the ovarian barrier for vertical transmission[9]. In the case of rice viruses transmitted by planthoppers or leafhoppers (Table 1), there are four barriers in horizontal transmission comprising the membrane invasion barrier of the midgut lumen, the basal lamina release barrier of the midgut, and the invasion barrier and release barrier of salivary glands. Qin et al. (2018) found that the rice stripe virus (RSV) uses the Laodelphax striatellus sugar transporter 6 to infect the insect midgut, but the rice grassy stunt virus does not, which is partially associated with their capacity for binding to this receptor[15]. RSV infection also requires the involvement of the virus-encoded glycoprotein NSvc2. This protein acts by helping the virus to successfully enter the midgut epithelial cells, with its N terminus binding to intestinal surface receptors and its C terminus inducing host cell membrane fusion[16].

Additionally, the passage of RSV through midgut and salivary glands needs the assistance of L. striatellus α-tubulin, leading to the dissemination of the virus to other organs in the insect vector[17]. The southern
rice black-streaked dwarf virus (SRBSDV) and rice dwarf virus (RDV) form tubular structures when they pass through the basal lamina release barrier of the midgut\cite{18,19}. The tubular structure of SRBSDV is formed by the interaction between the viral nonstructural protein P7-1 and the insect actin. The inhibition of P7-1 synthesis significantly reduces SRBSDV infection\cite{19}. Rice gall dwarf virus (RGDV) also forms tubular structures when it gets through the midgut barrier. However, it forms virus-induced filaments to perform an exocytosis-like process that enables the virus passage through the salivary gland barrier\cite{20}. To achieve vertical transmission, RSV invades female ovaries by interacting with insect vitellogenin\cite{21,22}, whereas RGV can bind to heparan sulfate proteoglycan on the surface of sperm and be transmitted by sperm\cite{23}. Additionally, the rice ragged stunt virus (RRSV), RDV, RGDV and SRBSDV form viroplasms during infection\cite{6}. Huang et al. reported that RRSV Pns10, which is an important component of viroplasms, interacts with insect mitochondrial membranes to help the virus make better use of host energy in the process of proliferation\cite{24}.

### 3 KEY FACTORS IN INSECT TRANSMISSION OF PLANT VIRUSES

#### 3.1 Insect immunity

A persistently transmitted virus that circulates within a vector insect is also an invader from the perspective of insects. Therefore, the proliferation and spread of plant viruses will inevitably activate the immune system of the host (Fig. 1). Insects do not have acquired immunity. Immune responses to viruses depend mainly on pathways such as RNAi, Toll, Imd, JAK/STAT and autophagy\cite{25} (Fig. 2). For example, infection with the rice black-streaked dwarf virus (RBSDV), maize mosaic rhabdovirus, RSV, RGDV, and SRBSDV activates the siRNA antiviral pathway of the host\cite{26–28}. *L. striatellus* cannot transmit SRBSDV\cite{29}. However, inhibition of the siRNA pathway significantly increases virus accumulation and promotes SRBSDV transmission by *L. striatellus*\cite{29}. It is universal that the insect RNAi pathway inhibits the spread of plant viruses. When the key gene in the siRNA pathway (*dicer-2*) is silenced, the increased virus accumulation leads to abnormality of the intestine caused by excessive amounts of virus\cite{30}. Therefore, the siRNA pathway may be key to balancing the amount of virus in insects without causing pathological damage. In addition to the siRNA pathway, activation of the Toll pathway has also been reported in *L. striatellus* infected with RSV\cite{31}. Induction of the Toll pathway was initiated by interaction between a Toll receptor and RSV nucleocapsid protein, and knockdown of Toll increased the proliferation of RSV in vector insects\cite{31}. Additionally, the prophenoloxidase cascade is one of the major innate immune pathways that can restrict virus infection. Chen et al. (2020) reported that RSV attenuates the prophenoloxidase response of the host to ensure virus stability in the hemolymph of vector insects\cite{32}. Some animal viruses employ the mitogen-activated protein kinases of the host to establish successful infection. Wang et al. (2017) found that RSV activates Jun N-terminal kinase (JNK) during infection. Inhibition of the JNK signal significantly restricts virus proliferation, indicating that different types of virus use similar strategies to regulate host immunity\cite{33}.

**Fig. 1** Key factors affecting insect transmission of plant viruses. Influences of insect immunity, insect feeding behavior, insect salivary effectors and insect symbiotic microorganisms on viral transmission are discussed in this review.
Autophagy is a basic metabolic activity that maintains cell homeostasis. When insects cope with an invading virus, autophagy may either have an antiviral role or be exploited by the virus. In *Bemisia tabaci*, infection with the tomato yellow leaf curl virus (TYLCV) activated the autophagy pathway, with autophagosomes formed to degrade the virus coat proteins and genomic DNA[34]. Inhibition of autophagy increased virus accumulation and enhanced the virulence of *B. tabaci*, whereas activation of autophagy significantly reduced the transmission efficiency of TYLCV[38]. Chen et al. reported RGDV- and RDV-induced autophagy in leafhoppers. However, the autophagy was exploited by viruses to promote their spread in this system, implicating the complex roles of autophagy in virus infection[35]. Virus infection is often accompanied by cell apoptosis, which is generally regulated by the intracellular cysteine protease[36]. Available evidence has demonstrated that some plant viruses may use apoptosis to promote virus transmission. In *Nilaparvata lugens*, RRSV-induced apoptosis was observed in salivary glands[37]. Inhibition of cell apoptosis by silencing caspase genes did not affect the proliferation of RRSV but significantly reduced the virus transmission efficiency from insects to rice plants. It was hypothesized that RRSV-induced cell apoptosis might contribute to the release of the virus[37]. A similar phenomenon was observed in *Recilia dorsalis* infected with RGDV, with improved virus transmission being observed after apoptosis induction[38]. Additionally, recent studies have demonstrated that apoptotic neurodegeneration induced by TYLCV caused sensory defects in infected *B. tabaci*, which removed the steady preference of the insect for virus-infected plants, thereby enhancing the probability of virus transmission to uninfected plants[39]. Compared with animal viruses, our understanding of the role of autophagy and apoptosis in insect transmission of plant viruses is limited and the relationship between insect immunity and plant viruses needs further investigation.

![Fig. 2](image_url)  
**Fig. 2**  Schematic representation of antiviral response pathway in vector insects. Five pathways, comprising siRNA, autophagy, JAK-STAT, Toll and IMD, are illustrated. (1) siRNA pathway: viral dsRNA is recognized by Dicer-2, and processed into siRNAs. The siRNAs are loaded onto the RNA interference silencing complex (RISC) that contains argonaute 2 (Ago2), then recognize and cleave target viral RNA. (2) Autophagy pathway: the transmembrane receptor Toll-7 recognizes the viral components and induces autophagy. It might be negatively regulated by the phosphatidylinositol 3-kinase (PI3K)-Akt kinase pathway. (3) JAK-STAT pathway: activation of the JAK-STAT pathway upon virus infection is likely mediated by binding of a cytokine of the unpaired (Upd) to their receptor, dome. Then, the JAK-tyrosine kinase hopscotch mediates the recruitment of Stat92E. After Jak-mediated phosphorylation, Stat92E proteins dimerize and translocate to the nucleus and regulate corresponding genes. (4) Toll pathway: recognition of Gram-positive bacteria, fungi and viruses by pattern recognition receptors results in proteolytic maturation of Spätzle (Spz). The cleaved Spz binds to Toll, which further recruits three death domain-containing adapter proteins MyD88, Tube and Pelle. Then, Cactus is phosphorylated, and induces the translocation of the Rel transcriptional factors, Dif and Dorsal, to the nucleus. (5) IMD pathway: recognition of Gram-negative bacteria and viruses by transmembrane receptors PGRP-LCs resulted in signal transduction to the IMD, which is localized in the cytoplasm. IMD activation recruits dFADD that recruits a caspase, DREDD. Activation of DREDD resulted in polyubiquitination of IMD. Then, TAK1 binds to the polyubiquitin chain and is responsible for the assembly and activation of the IKK complex (IKKβ and IKKγ). Phosphorylation of Relish is mediated by IKK complex, which is further cleaved by DREDD. The N-terminal DNA binding domain of Relish translocates to the nucleus and regulates transcription of corresponding genes.
3.2 Insect feeding behaviors

Plant viruses closely interact with plant hosts and vector insects in long-term coevolution. Most vector insects are attracted to plants infected with viruses and this may be associated with changes in plant hormones, plant metabolism and plant physical structures\(^{[40,41]}\) (Fig. 1). As an example, in tomato yellow leaf curl China virus the \(\beta C1\) protein encoded by the virus interferes with the polymerization of MYC2, a key transcription factor in the JA pathway, thereby inhibiting the synthesis of two volatile terpene compounds, finally attracting whitefly feeding and increasing their fitness for infected plants\(^{[42]}\). Additionally, the \(\beta C1\) protein hijacks WRKY20, a key immune regulatory factor, to prevent nonvectors (e.g., *Helicoverpa armigera*) from feeding, finally achieving the goals of promoting vector performance and inhibiting nonvector herbivores\(^{[42-49]}\). Reciprocity between plant viruses and vector insects occurs in the turnip mosaic virus (TuMV), tomato spotted wilt virus, barley yellow dwarf virus (BYDV), and potato leafroll virus\(^{[46-50]}\). However, there are some exceptions. The cucumber mosaic virus is a nonpersistent virus transmitted by aphids. Although infected plants are attractive to vector insects they are not suitable for aphid reproduction, which leads the aphids to transfer the virus to healthy plants after brief probing, thereby accelerating the spread of the virus\(^{[51]}\).

Based on the influences of plant viruses on vector insects, Mauck et al. proposed a model for insect vector behavior in relation to virus transmission\(^{[52]}\). Both persistent and nonpersistent viruses tend to enhance the attractiveness of infected plants to vector insects but they have contrasting effects on vector settling and feeding behavior. Specifically, persistent viruses tend to improve the plant quality and extend the vector feeding time whereas nonpersistent viruses tend to decrease plant quality and promote rapid dispersal. This model, to a certain extent, explains insect-virus-plant interactions, but there are still some limitations. For example, virus-free *Sogatella furcifera* is attracted to feed on SRBSDV-infected rice plants, but they are then attracted to feed on healthy rice plants after acquiring SRBSDV\(^{[53]}\). TuMV is a nonpersistent virus but it promotes vector insects feeding on infected plants\(^{[54]}\). These exceptions indicate that viruses have other strategies by which to influence insect-plant interactions in addition to manipulating plants.

3.3 Insect salivary effectors

Saliva mediates the interactions between insects and plants. In vector insects, when the virus is secreted along with saliva the interaction between the saliva and plants not only regulates the plant defense but also affects the virus spread\(^{[55]}\). In *L. striatellus*, RSV and RBSDV infection stimulate the insect to secrete more salivary proteins\(^{[56]}\). As an example of salivary mucin, which is an important component of the salivary sheath that accounts for continual insect feeding\(^{[57-59]}\), RSV infection significantly induces the expression of mucin, which might promote insect performance and virus transmission\(^{[60]}\). The regulation of salivary secretion by viruses was also reported in TYLCV-infected *B. tabaci*\(^{[61]}\). In addition, salivary effectors such as Bt56, Te84 and HARP1 affect the accumulation of plant hormones\(^{[62-64]}\). These hormones are extensively involved in plant-virus interactions\(^{[41]}\). Whether plant viruses alter the saliva secretion of vector insects to modulate plant antiviral defense remains to be further investigated. Currently, the study of the influence of saliva on virus transmission focuses primarily on several blood-feeding insects such as mosquitoes, ticks and sand flies\(^{[65-67]}\). With the successful identification of the salivary components of aphids, planthoppers, leafhoppers, whiteflies and other vector insects, it will be possible to manipulate insect-virus-plant interactions using a saliva approach.

3.4 Insect symbiotic microorganisms

In addition to host plants and vector insects, the infection cycle of plant viruses also involves symbiotic microorganisms (Fig. 1), with symbiotic bacteria being most extensively studied. Van den Heuvel et al. (1994) were the first to find that the potato leafroll virus (PLRV) can bind to the GroEL protein produced by symbiotic bacteria in insect hemolymph. Antibiotic treatment reduced PLRV transmission by more than 70%, indicating that symbiotic bacteria are beneficial in PLRV transmission\(^{[68]}\). GroEL also contributes in the transmission of TYLCV by *B. tabaci* and the transmission of BYDV by *Acyrthosiphon pisum*\(^{[69,70]}\). Inhibiting GroEL with antibodies reduced the TYLCV transmission by more than 80% \(^{[70]}\). Gottlieb et al. demonstrated that GroEL, which had a protective effect on TYLCV, was secreted by symbiotic bacteria *Hamiltonella*, rather than by the symbiotic bacteria *Rickettsia* or *Portiera*. By binding to the coat protein of TYLCV, *Hamiltonella* GroEL protected TYLCV in the hemolymph and promoted viral entry into the salivary glands\(^{[71]}\). Although *Rickettsia* GroEL could not bind to TYLCV, the symbiotic bacteria prolonged
the virulence period of *B. tabaci* and nearly doubled the virus transmission efficiency, indicating that the symbiotic bacteria promote the virus transmission independently of GroEL[72]. In recent years, increased attention has been given to the role of symbiotic bacteria in the vertical transmission of viruses. It has been reported that RDV is carried by both *Sulcia* and *Nasusia* to ensure their simultaneous transovarial transmission, enabling the virus to exploit an ancient oocyte entry path of symbiotic bacteria in *Nephotettix cincticeps*[73,74]. RGDV is carried with the sperm of leafhoppers for paternal transmission, which scarcely affects the fitness of adult males or their offspring but facilitates long-term virus persistence[23]. In the case of mosquito-vectored animal viruses, different symbiotic bacteria exert different effects on virus transmission. For example, *Wolbachia* inhibits or blocks insect infection by a variety of arboviruses[75], whereas *Serratia* marcescens enhances the ability of the virus to infect intestinal cells[76]. Recently, Gong et al. introduced *Wolbachia* into an insect vector of plant viruses and found that *Wolbachia* inhibited infection with and transmission of rice viruses, facilitating the development of symbiotic bacteria-based strategies against agricultural pests and their transmitted pathogens[77].

Insects also harbor a large number of insect-specific viruses (ISFs), which replicate only in their insect hosts[78]. Although ISFs cannot replicate or proliferate in plants, some are closely related to plant viruses[79]. It remains unclear whether there is a superinfection exclusion effect among them. The functions of ISFs in mosquito-vectored arboviruses have been thoroughly investigated. ISFs such as the cell-fusing agent virus, *Nhumirim* virus and *palm creek* virus, have been reported to have inhibitory effects in vertebrate virus transmission[80–82]. In contrast, knowledge of ISFs affecting insect transmission of plant viruses is limited. Li et al. found that the symbiotic Himetobi P virus promotes RSV accumulation, indicating that ISFs also contribute to transmission of plant viruses[83]. Many insect symbiotic microorganisms reside within vector insects[79,84]. Understanding their relationship with insect-vectored parasites will help make better use of these natural resources.

### 4 Potential Strategies for Vector Control

There are many similarities between the animal viruses transmitted by dipteran insects (represented by mosquitoes) and plant viruses transmitted by hemipteran insects (represented by planthoppers, leafhoppers and whiteflies). For example, vector insects acquire a virus by feeding on infected animal/plant hosts and transmit the virus to healthy animal/plant hosts upon refeeding. In biological transmission of animal viruses or propagative transmission of plant viruses, the viruses initially entered the intestinal epithelium from where they spread into the hemolymph and salivary glands, then the viruses escaped the salivary glands and secreted into a new host along with saliva. The intestinal tract, salivary glands and other tissues of vector insects are important barriers to the circulation of animal/plant viruses. Also, the feeding behavior, immunity and symbiotic microorganisms of vector insects were reported to affect the transmission of animal/plant viruses[85]. Therefore, approaches to controlling animal viruses can provide a good reference and guidance for the integrative management of plant viruses, and vice versa.

Due to the advantages of environmental protection, high efficiency, and low cost, the biological control of mosquito-vectored viruses based on symbiotic bacteria has attracted increased attention. *Wolbachia* are intracellular bacteria that infect about 40% of arthropod species. Apart from regulating host reproduction through cytoplasmic compatibility, *Wolbachia* reduces the infection of mosquitoes with the dengue virus, Chikungunya virus, West Nile virus, yellow fever virus and zika virus[75,86,87]. An innovative control strategy involving the release of mosquitoes infected with the intracellular bacterium *Wolbachia* is currently being developed[88]. The latest field experiment shows that a mosquito population carrying *Wolbachia* strain wAlbB could be stably maintained in the urban environment, and the release of antiviral mosquitoes could reduce the incidence of dengue fever[89]. By referencing the control strategy of animal viruses, *Wolbachia* was recently employed to control agricultural pests[77]. Stable artificial *Wolbachia* infection helped modified *N. lugens* rapidly invade wild-type populations, and inhibited both the infection and transmission of RRSV[77]. In addition to *Wolbachia*, modification with other microorganisms can help in the prevention and control of mosquito-vectored diseases[90]. For example, *Serratia*, which is stably colonized in mosquito populations, was genetically engineered to secrete effectors against plasmodium parasites, thereby achieving the purpose of blocking malaria transmission[91]. *Asaia* was engineered to conditionally express the antiplasmodial protein only when a blood meal was present, allowing antiplasmodial bacterial strains to survive and be transmitted through mosquito populations[90].

The development of gene editing technology has provided the possibility of efficiently controlling vector insects. Application of RNAi technology to insects, including planthoppers, leafhoppers, whiteflies, and
aphids, has been extensively reported\cite{33,35,39}. It is workable to control vector insects by directly targeting genes that are essential for survival\cite{57}. Also, silencing virus proteins in infected insects or knocking down insect proteins that interact with virus proteins are promising fields of pest control research in future\cite{18,24,33}. In recent years, a gene-driven system based on CRISPR/Cas9 has made it possible to directly modify vector insects. In this system, with the help of DNA homology-directed repair, a small number of modified insects can be introduced to spread the mutation to the whole population, and finally the pest population can be continuously suppressed or even replaced\cite{93}. Gantz et al. successfully integrated antimalarial effector genes and autonomous gene-drive components into the mosquito genome and realized population replacement\cite{94}. Currently, CRISPR/Cas9 technology has been used for gene editing in planthoppers, whiteflies and aphids\cite{95-97}. In the future, gene editing technology, RNAi technology and CRISPR/Cas9 + gene-drive technology are expected to be used to prevent the transmission of important arboviruses to realize the prevention and treatment of plant virus diseases.

5 PROSPECT

Plant virus transmission is a complex process involving a series of complex interactions between host plants, viruses, vector insects and even symbiotic microorganisms. These interactions give rise to a distinct specificity between the virus and the transmitting insect, that is, a virus can only be transmitted by specific insects. Knowing how to use plant-insect-virus-symbiotic microorganism interactions to interfere in the vector transmission of virus will be key to virus control. Nonpersistent viruses and semipersistent viruses bind to the insect stylet or foregut. The elucidation of the binding and releasing mechanisms between viruses and receptors in these two tissues may lead to the development of methods to prevent virus transmission. Persistent viruses multiply and circulate inside the vector insects, acting as an invader of the insect. During infection, persistent viruses need to cope with the insect immune system to maintain homeostasis as well as overcome the barriers in the intestine, salivary glands, and ovaries. Therefore, in-depth investigation of the interaction between viruses and vector insects will help in developing methods to block or inhibit virus infection. Research on the mechanisms of insect immunity, insect feeding and insect symbiotic microorganisms in virus transmission, as well as the application of new gene editing technology, RNAi technology and CRISPR/Cas9 + gene-driven technology, will provide new strategies for controlling important plant viruses.

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Compliance with ethics guidelines

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| Virus                              | Family/Genus                  | Plant host                        | Hemipteran vector | Transmission mode          |
|-----------------------------------|------------------------------|-----------------------------------|-------------------|---------------------------|
| Barley yellow dwarf virus (BYDV)  | Luteoviridae, Luteovirus, (+)ssRNA | Barley, oats, wheat, etc.         | > 25 aphid species | Persistent-nonpropagative |
| Cauliflower mosaic virus (CaMV)   | Caulimoviridae, Caulimovirus, dsDNA | Radish, cauliflower, cabbage, etc. | Brevicoryne brassicae | Nonpersistent             |
| Cucumber mosaic virus (CMV)       | Bromoviridae, Cucumovirus, (+)ssRNA | Cucumber, spinach, pepper, etc.   | > 60 aphid species | Nonpersistent             |
| Maize mosaic rhabdovirus (MMV)   | Rhabdoviridae, Nucleorhabdovirus, (−)ssRNA | Maize, teosinte, itchgrass, etc. | Peregrinus maidis | Persistent-propagative    |
| Potato leafroll virus (PLRV)      | Luteoviridae, Polerovirus, (+)ssRNA | Potato                            | Aphis fabae, Aphis gossypii, Aulacorthum solani, Macrosiphum euphorbiae, Myzus persicae | Persistent-nonpropagative |
| Rice black-streaked dwarf virus (RBSDV) | Reoviridae, Fijivirus, dsRNA | Rice, wheat, maize, etc.          | Laodelphax striatellus, Unkanodes sapporona, Unkanodes albifascia, | Persistent-propagative |
| Rice dwarf virus (RDV)            | Reoviridae, Phytoreovirus, dsRNA | Rice                             | Nephotettix cincticeps, Recilia dorsalis, Nephotettix virescens, Nephotettix nigropictus, Nephotettix nigropictus, Nephotettix cincticeps, Recilia dorsalis | Persistent-propagative |
| Rice gall dwarf virus (RGDV)      | Reoviridae, Phytoreovirus, dsRNA | Rice                             | Nilaparvata lugens, | Persistent-propagative    |
| Rice ragged stunt virus (RRSV)    | Reoviridae, Oryzavirus, dsRNA  | Rice                             | Laodelphax striatellus, Unkanodes sapporona, Unkanodes albifascia, Terthron albovittatum | Persistent-propagative, |
| Rice stripe virus (RSV)           | Phenuiviridae, Tenuivirus, (−)ssRNA | Rice, wheat, maize               | Sogatella furcifera | Persistent-propagative,   |
| Southern rice black-streaked dwarf virus (SRBSDV) | Reoviridae, Fijivirus, dsRNA | Rice, maize, Chinese sorghum, etc. | Bemisia tabaci | Persistent-propagative,   |
| Tomato yellow leaf curl China virus (TYLCCNV) | Geminiviridae, Begomovirus, ssDNA | Tomato, tobacco, petunias, etc.   |                   | Persistent-propagative,   |
| Virus                                   | Family                  | Hosts                                      | Vector           | Mode of Propagation     |
|-----------------------------------------|-------------------------|--------------------------------------------|------------------|-------------------------|
| Tomato yellow leaf curl virus (TYLCV)   | *Geminiviridae,* *Begomovirus,* ssDNA | Tomato, eggplants, potatoes, etc.          | *Bemisia tabaci* | Persistent-propagative  |
| Turnip mosaic virus (TuMV)              | *Potyviridae,* *Potyvirus,* (+)ssRNA | Turnip, lettuce, watercress, etc.          | > 89 aphid species | Nonpersistent            |
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