Despite concerted efforts to generate transgenic resistance to BBTV, no successful glasshouse or field results have yet been reported. However, some promising strategies are being developed at Queensland University of Technology involving the following steps.

1. Transdominant negative strategies to interfere with replication, by constitutive overexpression of mutated Rep proteins, are employed. Single mutations in either of two motifs involved with rolling-circle replication render the Rep inactive, and in transient assays with constitutive overexpression, virus replication is significantly reduced, but not abolished.

2. Rep-activated cell death is carried out using a so-called suicide gene. DNA-R intergenic sequence is cloned within an intron and flanked by a split barnase gene construct. The suicide gene is only activated in the presence of the Rep protein, resulting in cell death and containment of the virus.

See also: Nanoviruses.

Further Reading


Barley Yellow Dwarf Viruses

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Introduction

Barley yellow dwarf (BYD) is the most economically important virus disease of cereals, and is found in almost every grain growing region in the world. Widespread BYD outbreaks in cereals were noted in the United States in 1907 and 1949. However, it was not until 1951 that a virus was proposed as the cause of the disease. The causal agents of BYD are obligately transmitted by aphids, which probably delayed the initial classification of BYD as a
virus disease. Subsequently, BYD was shown to be caused by multiple viruses belonging to the species barley yellow dwarf virus (BYDV) and cereal yellow dwarf virus (CYDV). Depending on the virulence of the virus strain, infection may contribute to winter kill in regions with harsh winters, induce plant stunting, inhibit root growth, reduce or prevent heading, or increase plant susceptibility to opportunistic pathogens and other stresses. Yield losses to wheat in the United States alone are estimated at 1–3% annually, exceeding 30% in certain regions in epidemic years. The effects of BYD in barley and oats typically are more severe than in wheat; sometimes resulting in complete crop losses. The existence of multiple strains of viruses that are transmitted in strain-specific manner has made BYDV and CYDV model systems to study interactions between viruses and aphid vectors in the circulative transmission of plant viruses. In addition, the compact genomes of the viruses have provided useful insights into the manipulation of host translation machinery by RNA viruses.

**Taxonomy and Classification**

The viruses that cause BYD are members of the family *Luteoviridae*, and were first grouped because of their common biological properties. These properties included persistent transmission by aphid vectors, the induction of yellowing symptoms in grasses, and serological relatedness. Different viruses are transmitted more efficiently by different species of aphids, a fact that was originally used to distinguish the viruses. Around 1960, the viruses were separated into five ‘strains’ (now recognized as distinct species) based on their primary aphid vector(s). BYDVs transmitted most efficiently by *Sitobion* (formerly *Macrosiphum*) *avenae* were assigned the acronym MAV, for *Macrosiphum avenae* virus. Similarly, viruses transmitted most efficiently by *Rhopalosiphum maidis* and *Rhopalosiphum padi* were assigned the acronyms RMV and RPY, respectively. Viruses transmitted most efficiently by *Schizaphis graminum* were assigned the acronym SGV. Finally, vector-nonspecific viruses, that is, viruses transmitted efficiently by both *R. padi* and *S. avenae* were assigned the acronym PAV.

Based on genome organization and predicted amino acid sequence similarities, BYDV-MAV, -PAS, and -PAV have been assigned to the genus *Luteovirus*, and CYDV-RPS and -RPV to the genus *Potyvirus*. The RNA-dependent RNA polymerases (RdRps) encoded by open reading frames (ORFs) 1 and 2 of BYDVs resemble those of viruses in the genus *Potyvirus* (Figure 1). In contrast, the predicted amino acid sequence of the RdRps encoded by ORFs 1 and 2 of CYDVs resemble those of viruses in the genus *Sobemovirus*. The two polymerase types are distantly related in evolutionary terms.

For this reason, viruses for which RdRp sequences have not been determined (BYDVs GPV, RMV, and SGV) have not been assigned to a genus. These observations suggest that the genomic RNAs of BYDV and CYDV resulted from recombination between RNAs expressing a common set of structural and movement proteins and RNAs expressing two different sets of replication proteins. Because of these differences, it has been suggested that BYDVs should be placed in the family *Tombusviridae* and CYDVs in the genus *Sobemovirus*.

**Viron Properties and Composition**

All BYDVs and CYDVs have nonenveloped icosahedral particles with diameters of 25–28 nm (Figure 2). Capsids are composed of major (22 kDa) and minor (65–72 kDa) coat proteins (CPs), which is formed by a carboxy-terminal extension to the major CP called the read-through domain (RTD). According to X-ray diffraction and molecular mass analysis, virions consist of 180 protein subunits, arranged in *T* = 3 icosahedra. Virus particles do not contain lipids or carbohydrates, and have sedimentation coefficients *s*₂₀,₀ in (Svedberg units) that range from 115–118 S. Buoyant densities in CsCl are approximately 1.4 g cm⁻³. Virions are moderately stable, insensitive to freezing, and are insensitive to treatment with chloroform.
or nonionic detergents, but are disrupted by prolonged treatment with high concentrations of salts. The single encapsidated genomic RNA molecule is single-stranded, positive-sense, and lacks a 3'-terminal poly(A) tract. A small protein (VPg) is covalently linked to the 5'-terminus of CYDV RNAs. CYDV-RPV also encapsidates a 322-nucleotide satellite RNA that accumulates to high levels in the presence of the helper virus. Complete genome sequences have been determined for BYDV-MAV, -PAS, and -PAV and CYDV-RPS and -RPV (Table 1). For several viruses, notably BYDV-PAV, genome sequences have been determined from multiple isolates.

**Genome Organization and Expression**

Genomic RNAs of BYDVs and CYDVs for which complete nucleotide sequences are available contain five to six ORFs (Figure 3). ORFs 1, 2, 3, and 5 are shared among all BYDVs and CYDVs. BYDVs lack ORF0. Genomic sequences of some BYDVs contain one or two small ORFs, ORFs 6 and 7, downstream of ORF5. In CYDVs, ORFs 0 and 1 and ORFs 1 and 2 overlap by more than 600 nucleotides. In BYDVs, ORF1 overlaps ORF2 by less than 50 nucleotides. In BYDV and CYDV genome sequences, ORF4 is contained within ORF3. An amber (UAG) termination codon separates ORFs 3 and 5.

BYDVs and CYDVs have relatively short 5' and intergenic noncoding regions. ORFs 2 and 3 are separated by about 200 nucleotides in BYDVs and CYDVs. The lengths of noncoding sequences downstream of ORF5 are very different between BYDVs and CYDVs. BYDV-PAV contains over 860 nucleotides downstream of ORF5 compared to just 170 nucleotides for CYDV-RPV.

The expression of BYDV-PAV RNA has been studied in detail and has revealed a complex set of RNA–RNA and RNA–protein interactions that are employed to express and replicate the virus genome. Less experimental data are available for CYDVs. However, expression and replication strategies and gene functions can be inferred from those of closely related luteoviruses, particularly beet western yellow virus (BWYV) and potato leaf roll virus (PLRV). ORFs 0, 1, and 2 are expressed directly from genomic RNAs. Downstream ORFs are expressed from subgenomic RNAs (sgRNAs) that are transcribed from internal initiation sites by virus-encoded RdRps from negative strand RNAs and are 3'-coterminal with the genomic RNA. Since the initiation codon for ORF0 of CYDVs is upstream of that of ORF1, translation of ORF1 is initiated by "leaky scanning" in which ribosomes bypass the AUG initiation codon of ORF0 and continue to scan the genomic RNA until they reach the initiation codon of ORF1. The protein products of ORF2 are expressed only as a translational fusion with the product of ORF1. At a low frequency during the expression of ORF1, translation continues into ORF2 through a −1 frameshift that produces a large protein containing sequences encoded by both ORFs 1 and 2 in a single polypeptide. In BYDV-PAV, frameshifting between ORFs 1 and 2 is dependent upon the interaction of RNA sequences close to the site of frameshifting and a

![Figure 2](https://example.com/image.png) Scanning electron micrograph of barley yellow dwarf virus-PAV particles, magnified 200000×. Virions are c. 25 nm in diameter, hexagonal in appearance, and have no envelope.

<table>
<thead>
<tr>
<th>Genus</th>
<th>Virus (alternative name)</th>
<th>Abbreviation</th>
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<tbody>
<tr>
<td>Luteovirus</td>
<td>Barley yellow dwarf virus-MAV</td>
<td>BYDV-MAV</td>
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<td></td>
<td>Barley yellow dwarf virus-PAS</td>
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<td>Barley yellow dwarf virus-PAV</td>
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<td>(Barley yellow dwarf virus-RGV) (rice giallume)</td>
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<tr>
<td>Polerovirus</td>
<td>Cereal yellow dwarf virus-RPS</td>
<td>CYDV-RPS</td>
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<td></td>
<td>Cereal yellow dwarf virus-RPV</td>
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<tr>
<td>Unassigned</td>
<td>Barley yellow dwarf virus-GAV</td>
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<td>Barley yellow dwarf virus-SGV</td>
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Figure 3  Genome organizations of barley yellow dwarf virus-PAV (BYDV-PAV) and cereal yellow dwarf virus-RPV (CYDV-RPV). Individual open reading frames (ORFs) are shown as staggered open boxes. The predicted sizes of the protein products are indicated. The genome-linked protein (VPg) attached to the 5' terminus of CYDV RNA is indicated by a solid circle. Based on homology to other viruses ORF0 encodes a silencing suppressor and ORFs 1 and 2 encode replication-related proteins. ORFs 3 and 5 encode the major coat protein and readthrough domain, respectively. ORF4 encodes a protein required for virus cell-to-cell movement. The BYDV translation enhancer (BTE) facilitates translation initiation of BYDV-PAV genomic RNA and subgenomic RNA1 (sgRNA1). In both BYDV-PAV and CYDV-RPV, ORF2 is expressed as a translational fusion with the product of ORF1 via a −1 frameshift. In BYDV-PAV, frameshifting requires interaction between the 5' frameshift signals and the long-distance frameshift element (LDFE). Dashed lines indicate long-distance RNA–RNA interactions.

long-distance frameshift element (LDFE) located 4000 nucleotides downstream in the 3' noncoding region of genomic RNAs. Mutations that disrupt the interactions between these two distal regions suppress frameshifting and abolish RNA replication.

ORFs 3, 4, and 5 are expressed from sgRNA1, the 5' terminus of which is located about 200 nucleotides upstream of ORF3, and extends to the 3' terminus of the genome. BYDV's produce a second sgRNA that contains ORF6. BYDV-PAV also produces a third sgRNA, which does not appear to encode a protein. ORF3 is translated from the 5' terminus of sgRNA1. ORF4 of BYDVs and CYDVs, which encodes a 17 kDa protein, is contained within ORF3 and is expressed from the same sgRNA as ORF3 through a leaky scanning mechanism much like that used to express ORF1 of CYDVs. In BYDV's and CYDV's, ORF5 is expressed only as a translational fusion with the products of ORF3 by readthrough of the UAG termination codon at the end of ORF3. This produces a protein with the product of ORF3 at its amino terminus and the product of ORF5 at its carboxyl terminus.

While genomic RNAs of CYDVs contain 5' VPgs that interact with translation initiation factors, BYDV-PAV RNA contains only a 5' phosphate. Unmodified 5' termini usually are recognized poorly for translation initiation. To circumvent this problem, a short sequence located in the noncoding region just downstream of ORF5 in the BYDV-PAV genome, called the BYDV translation enhancer (BTE), interacts with sequences near the 5' termini of the genomic and subgenomic RNAs to promote efficient cap-independent translation initiation.

Functions BYDV and CYDV proteins have been ascribed based on homology to virus proteins with known functions and mutational characterization of protein coding regions. Similarity to proteins encoded by BWYV and PLRV suggests that the 28–29 kDa proteins encoded by ORF0 of CYDVs are inhibitors of post-transcriptional gene silencing (PTGS). PTGS is an innate and highly
adaptive antiviral defense found in all eukaryotes that is activated by double-stranded RNAs (dsRNAs), which are produced during virus replication. The ORF1-encoded proteins of CYDVs contain the VPg and a chymotrypsin-like serine protease that is responsible for the proteolytic processing of ORF1-encoded polyproteins. The protease cleaves the ORF1 protein in trans to liberate the VPg, which is covalently attached to genomic RNAs. ORF2s of BYDVs and CYDVs, which are expressed as translational fusions with the product of ORF1, have coding capacities of 59–72 kDa and predicted amino acid sequences that are very similar to known RdRps and hence likely represent the catalytic portion of the viral replicase.

ORF3 encodes the major 22 kDa CP for both BYDVs and CYDVs. ORF5 has a coding capacity of 43–50 kDa, which is expressed only as a translational fusion with the product of ORF3 when translation reads through the termination codon at the end of ORF3 and continues through to the end of ORF5. The ORF5 portion of this readthrough protein has been implicated in aphid transmission and virus stability. Recombinant viruses that do not express ORF5 produce virions assembled from the major CP alone, which are not transmitted by aphid vectors and are less efficient in systemic infection of host plants than wild-type viruses. The amino-terminal portions of ORF5 proteins are highly conserved among BYDVs and CYDVs while the carboxyl termini are much more variable.

ORF4 of both BYDVs and CYDVs is contained within ORF3 and encodes a 17 kDa protein. Viruses that contain mutations in ORF4 are able to replicate in isolated plant protoplasts, but are deficient or delayed in systemic movement in whole plants. Hence, proteins encoded by ORF4 are thought to facilitate intra- and intercellular virus movement.

Some BYDV genomic sequences contain small ORFs (ORF6) downstream of ORF5. The predicted sizes of the proteins expressed by ORF6 range from 4 to 7 kDa. The predicted amino acid sequences of the proteins encoded by ORF6 are poorly conserved among BYDV-PAV isolates. Repeated attempts to detect protein products of ORF6 have been unsuccessful. In addition, BYDV-PAV genomes into which mutations have been introduced that disrupt ORF6 translation are still able to replicate in protoplasts. Based on these observations, it has been concluded that ORF6 is not translated in vivo.

Host Range and Transmission

BYD-causing viruses infect over 150 species of annual and perennial grasses in five of the six subfamilies of the Poaceae. The feeding habits of vector aphids have a major impact on the host ranges of virus species. Hence the number of species naturally infected by the viruses is much lower than the experimental host range.

As techniques for infecting plants with recombinant viruses have improved, the experimental host ranges of BYDVs and CYDVs have been expanded to include plants on which aphid vectors would not normally feed. For example, BYDV-PAV and CYDV-RPV have been shown to infect Nicotiana species when inoculated using Agrobacterium tumefaciens harboring binary plasmids containing infectious copies of the viruses, which had not been described previously as experimental hosts for the viruses.

Viruses that cause BYD are transmitted in a circulative strain-specific manner by at least 25 aphid species. Circulative transmission of the viruses is initiated when the piercing–sucking mouthparts of aphids acquire viruses from sieve tubes of infected plants during feeding. Aphids that do not probe into and feed from the vascular tissues of infected plants do not transmit the viruses. The viroids of BYDVs and CYDVs travel up the stylet, through the food canal, and into the foregut (Figure 4). After 12–16 h, virions then are actively transported across the cells of the hindgut into the hemocoel in a process that involves receptor-mediated endocytosis of the viruses and the formation of tubular vesicles that transport viruses.

![Figure 4](image-url)

Circulative transmission of BYDVs and CYDVs by vector aphids. While feeding from sieve tubes of an infected plant, an aphid (shown in cross section) acquires virions, which travel up the stylet, through the food canal, and into the foregut. Virions are actively transported across cells of the hindgut into the hemocoel. Virions then passively diffuse through the hemolymph to the accessory salivary gland where they are again actively transported into the lumen of the gland. Once in the salivary gland lumen, the virions are expelled with the saliva into the vascular tissue of host plants. Viruses that are not transmitted by a particular species of aphid often accumulate in the hemocoel, but do not traverse the membranes of the accessory salivary gland.
through epithelial cells and into the hemocoel. Virions then passively diffuse through the hemolymph to the accessory salivary gland where virions must pass through the membranes of accessory salivary gland cells in a similar type of receptor-mediated transport process to reach the lumen of the gland. The accessory salivary gland produces a watery saliva, containing few or no enzymes, that is thought to prevent phloem proteins from clogging the food canal. Once in the salivary gland lumen, virions are expelled with the watery saliva into vascular tissues of host plants. Typically hindgut membranes are much less selective than those of the accessory salivary glands. Consequently, viruses that are not transmitted by a particular species of aphid often are transported across gut membranes and accumulate in the hemocoel, but do not traverse the membranes of the accessory salivary gland. The specificity of aphid transmission and gut tropism has been linked to the RTD of the minor capsid protein. Even though large amounts of virions can accumulate in the hemocoel, there is no evidence for virus replication in their aphid vectors. Aphids may retain the ability to transmit virus for several weeks.

Genetic and biochemical studies have been conducted to identify aphid determinants of strain-specific transmission of BYDV-MAV and BYDV-PAV. Protein–protein and protein–virus interaction experiments were used to isolate two proteins from heads of vector aphids that bind BYDV-MAV that were not detected in nonvector aphids. These two proteins are good candidates for the cell-surface receptors that are thought to be involved in strain-specific transport of viruses into accessory salivary gland lumens. In addition, endosymbiotic bacteria that reproduce in specialized cells called mycetocytes in abdomens of aphids express chaperonin-like proteins that bind BYDV particles and the amino-terminal region of recombinant BYDV-PAV RTD proteins. However, the role of these proteins in aphid transmission is unclear since they are found in both vector and nonvector aphid species. Interactions of virus particles with these proteins seem to be essential for persistence of the viruses in aphids. The proteins may protect virus particles from degradation by aphid immune systems.

**Replication**

Like other viruses of the family *Luteoviridae*, BYDVs and CYDVs infect and replicate in sieve elements and companion cells of the phloem and occasionally are found in phloem parenchyma cells (Figure 5). BYDVs and CYDVs induce characteristic ultrastructural changes in infected cells. BYDV-MAV, -PAV, and -SGV induce single-membrane-bound vesicles in the cytoplasm near plasmodesmata early in infection. Subsequently, filaments are observed in nuclei, and virus particles are first observed in the cytoplasm. In contrast, BYDV-RMV and CYDV-RPV induce double-membrane-bound vesicles in the cytoplasm that are continuous with the endoplasmic reticulum. Later, filaments and tubules form in the cytoplasm, and BYDV-RMV and CYDV-RPV particles are first observed in nuclei.

The subcellular location of viral RNA replication has not been determined unequivocally. However, early in infection, negative-strand RNAs of BYDV-PAV are first detected in nuclei and later in the cytoplasm, which suggests that at least a portion of the BYDV-PAV replication occurs in the nucleus. A nuclear location for replication is supported by the observation that the movement protein encoded by ORF4, which also binds single-stranded RNA, localizes to the nuclear envelope and is associated with virus RNA in nuclei of infected cells. Synthesis of negative-strand RNA, which requires tetra-loop structures at the 3' end of BYDV-PAV genomic RNAs, is detected in infected cells before the formation of virus particles. Because tetra-loops have been implicated in RNA–protein interactions, these structures could be binding and/or recognition sites for BYDV replication proteins. BYDV-PAV sgRNAs are synthesized by internal initiation of RNA synthesis on negative-strand RNAs from three dissimilar subgenomic promoters. Late in infection, the BTE near the 5' terminus of BYDV-PAV sgRNA2 inhibits translation from genomic RNA, which may promote a switch from translation to replication and packaging of genomic RNAs. In addition to genomic RNAs, CYDV-RPV replicates a satellite RNA by a rolling-circle mechanism that generates multimeric satellite RNAs that self-cleave to unit length.

**Virus–Host Relationships**

Visible symptoms induced by BYDVs and CYDVs vary greatly depending on the host and strain of the virus. The most common symptoms are stunting and chlorosis. While some infected plants display no obvious symptoms, most BYDVs and CYDVs induce characteristic symptoms that include stunting, leaves that become thickened, curled or serrated, and yellow, orange or red leaf discoloration, particularly of older leaves of infected plants. These symptoms result from phloem necrosis that spreads from inoculated sieve elements and causes symptoms by inhibiting translocation, slowing plant growth, and inducing the loss of chlorophyll. Symptoms may persist, vary seasonally, or may disappear soon after infection. Temperature and light intensity often affect symptom severity and development. In addition, symptoms can vary greatly with different virus isolates or strains and with different host cultivars. Yield losses caused by BYD are difficult to estimate because the viruses are so pervasive and symptoms often are overlooked or attributed to
Figure 5  Barley yellow dwarf virus-Pav life cycle. Virus particles are deposited in sieve elements by aphid vectors. By a yet unknown process, single-stranded messenger-sense genomic RNA is released from virus particles and translated by host translation machinery, which is facilitated by long-distance RNA–RNA interactions. Open reading frames (ORFs) 1 and 2, which encode the viral replicase, are expressed first because of their proximity to the 5’ termini of genomic RNAs. Virus encoded replicase then synthesizes negative-sense RNAs that are used as templates for the production of new full-length positive-sense genomic RNAs and subgenomic RNAs. Production of subgenomic RNAs results in synthesis of structural and cell-to-cell movement proteins. Subgenomic RNA2 suppresses translation from genomic RNAs, furthering the switch from early to late gene expression. Full-length positive-sense genomic RNAs and structural proteins then assemble into virions in cells of phloem tissues where they can be ingested by aphid vectors to start the process again.

other agents. In Australia alone, losses in barley production have been valued at over 100 million US dollars annually. Plants infected with BYD at early developmental stages suffer the most significant yield losses, which often are linearly correlated with the incidence of virus infection.

Epidemiology

BYD infections have been reported from temperate, subtropical, and tropical regions of the world. Even though the incidence of infections of individual viruses varies from year to year and can differ among annual and perennial hosts, BYDV-PAV usually is the most prevalent of the viruses causing BYD in small grains worldwide, followed by CYDV-RPV or BYDV-MAV. The remaining BYD-causing viruses are typically much less prevalent. BYDVs and CYDVs must be reintroduced into annual crops each year by their aphid vectors. Alate, that is, winged aphids may transmit viruses from local cultivated, volunteer, or weed hosts. Alternatively, alate aphids may be transported into crops from distant locations by wind currents. These vectors may bring the virus with them, or they may first have to acquire virus from locally infected hosts. In temperate regions of Europe and North America moderate and long-distance migration of viruliferous aphids is important to development of BYD epidemics. In Australasia, and other regions with Mediterranean climates, alate aphids usually transmit viruses from relatively close infected plants. Secondary spread of the viruses is often primarily by apterous, that is, wingless aphids. The relative importance of primary introduction of viruses by alate aphids and of secondary spread of viruses by apterous aphids in disease severity varies with the virus, aphid species, crop, and environmental conditions.

Diagnosis

Accurate diagnosis of infections has been important in understanding the transmission and epidemiology of BYDVs and CYDVs and developing control strategies for BYD. Because BYD symptoms resemble those caused
by other biotic and abiotic factors, visual diagnosis is unreliable and other methods have been developed. Initially, infectivity, or biological, assays were used to diagnose infections. In bioassays, aphids are allowed to feed on infected plants and then are transferred to indicator plants. These techniques have also been used to determine vector specificities of viruses causing BYD and to identify viruliferous vector aphids in epidemiological studies. These techniques are very sensitive, but they can require several weeks for symptoms to develop on indicator plants. The viruses causing BYD are strongly immunogenic, which has facilitated development of genus- and even strain-specific antibodies that have been used extensively in BYD diagnosis. Because the viruses causing BYD are present in infected tissues at very low levels, mice have been used to produce monoclonal antibodies against the viruses. Mice typically require much less viral antigen per immunization than rabbits, and hybridoma cell lines that produce monoclonal antibodies can be stored for extended periods and used for many years, which further reduces the amount of antigen needed to produce diagnostic antibodies. Techniques have also been developed to detect viral RNAs from infected plant tissues by reverse transcription polymerase chain reaction, which can be more sensitive and discriminatory than serological diagnostic techniques. Even so, serological tests are the most commonly used techniques for the detection of infections because of their simplicity, speed, and relatively low cost.

Control

Planting of insecticide-treated seeds that protect emerging seedlings from aphid infestation has been shown to reduce losses caused by BYD in North America, Australasia and Africa. Foliar applications of insecticides on older plants typically have been less effective. Alternatively, planting of tolerant or resistant cereals has proved to be a much more cost-effective and sustainable management strategy for BYD. Breeding programs have successfully integrated genes conferring high levels of tolerance into barley and oat and to a lesser extent in wheat. Even though a limited number of single genes for BYD resistance/tolerance genes have been identified in cultivated barley and rice, in most instances, tolerance to BYD is conditioned by multiple genes in a quantitative fashion, which has made moving BYD tolerance into new plant lines challenging. Particularly in barley, molecular markers have begun to facilitate the process of breeding for BYD tolerance. Because of a lack of effective single-gene resistance in cultivated wheat, some researchers have moved BYD resistance genes from wheat grasses (Thinopyrum intermedium and Thinopyrum ponticum) into wheat, which have provided high levels of resistance. The lack of naturally occurring resistance in cereals to BYD has made transgene-mediated resistance very attractive. Even though the expression of CP sequences in transgenic plants has conferred resistance in several other plant–virus systems, it has not provided significant resistance to BYD in barley, oat, or wheat. In contrast, transgenic barley and oat plants have been produced that express either intact or inverted copies of BYDV-PAV replicase genes, which conferred high levels of resistance to BYDV-PAV and closely related viruses.

In many small grain growing regions, viruliferous aphids arrive at similar times each spring and fall even though sizes of the aphid populations can vary significantly from year to year. In these areas, it is sometimes possible to plant crops so that young, highly susceptible plants are not in the field when the seasonal aphid migrations occur. However, crops planted later typically do not yield as well as those planted early in the growing season. Consequently, growers must weigh the probability of obtaining higher yields against possible yield losses caused by BYD. In some instances, biological control agents such as predatory insects and parasites have reduced aphid populations significantly.

See also: Cereal Viruses: Wheat and barley; Luteoviruses.

Further Reading


