

Review on Barely Yellow Dwarf Viruses

Alemu Nega

Department of Horticulture and Plant Science, Jimma University, P. O. Box 307, Jimma, Ethiopia

*Corresponding Author: Alemu Nega. Email Address: alemunega531@gmail.com

ABSTRACT

Barley yellow dwarf virus (BYDV) is distributed worldwide, and infects most cereals and grasses. It is a phloem-restricted pathogen, causing yellowing, reddening, and brittleness of leaves, dwarfing, and reduction in size and number of ears and grains. BYDV is a luteovirus with small isometric particles containing an ssRNA genome, and is transmitted persistently by more than 20 aphid species. Five virus isolates have been distinguished and divided into two subgroups on the basis of cytopathology and serology. Recent serological evidence also indicates that BYDV isolates are related to other luteoviruses, suggesting that a continuous, overlapping range of viruses may be implicated in the barley yellow dwarf syndrome. Until future research clarifies this point, the term BYDV continues to be used to indicate the agent(s) involved. Perennial wild or cultivated grasses constitute a large and permanent virus pool. Primary and secondary virus spread depends on the aphid vector reproduction and flight which, in turn, are influenced by climatic conditions. Recent research on monitoring and control of aphid vectors and on development of resistant cereal cultivars has improved the prospect of minimizing losses from BYDV infections. Because of the economic importance of the BYDVs, more research is needed. The specific locations and timing of virus outbreaks, and the particular causal isolates, need to be monitored. This will allow breeders to decide which BYDV isolate to target with transgenic resistance in a given locality. It will help growers decide whether to pay the extra premium for BYDV-resistant crops. Another area of applied research may be to engineer aphid-resistant crops. With the growing number of sequenced or partially sequenced isolates of BYDV and CYDV around the world, it's important 1) to develop rapid means of nucleic acid-based detection (e.g., PCR), 2) to understand the epidemiology of BYDV/CYDV, and 3) to develop transgenic and other means of disease control. The better understanding of BYDV molecular mechanisms that ultimately lead to new means of controlling or mitigating the effects of the disease, and it sheds light on processes relevant to medically important viruses. In addition, further review is needed to identify all recovered BYDV and evaluation of promising treatments for use in integrated disease management strategy to manage not only BYDV but also other related viral diseases of plant.

Keywords: Barley, Barley yellow dwarf virus (BYDV), and luteovirus.

1. INTRODUCTION

Plant viruses may affect the fitness of their hosts by reducing host survivorship, fecundity or competitive ability relative to uninfected individuals in the population. Virus strains may differ markedly in the severity of symptoms they induce (Anderson *et al.*, 1991, Bencharhi *et al.*, 1999) and their ability to infect a given host (Moury *et al.*, 2001, Sacristan *et al.*, 2005). Variation among strains in virulence, infectivity and transmission may affect patterns of disease spread and, thereby, host population dynamics in natural systems (Raybould *et al.*, 1999) or crop yield in agricultural systems. The behavior and constraints of a virus upon host infection surely have a genetic underpinning. Thus, it is necessary to acquire knowledge of genetic diversity in pathogen populations to better understand the role they play in ecological processes and as impediments to agricultural production. Barley yellow dwarf virus (BYDV) is the most economically important virus disease of cereals, and is found in almost every grain growing region in the world. Widespread BYDV 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 ears. The effects of BYDV 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. Every year barley yellow dwarf viruses (BYDVs) cause substantial losses throughout the world wherever their hosts, mainly wheat, barley, and oats, occasionally rice and maize, are grown (Lister RM, Ranieri R., 1995). In addition to their economic importance, the gene expression mechanisms, evolution and taxonomy,

satellite RNA, and intimate interactions with their aphid vectors are quite fascinating and unlike those of any other viruses. Moreover, barley yellow dwarf disease is caused by *Luteoviridae* species in the genera *Luteovirus* and *Polerovirus*. Each virus species has a distinct aphid transmission phenotype and the acronym for the species is derived from this specificity. *Luteoviridae* species commonly isolated from grain crops include: GAV (*Luteovirus*) transmitted most efficiently by *Schizaphis graminum* and *Sitobion avenae* (Wang *et al.*, 2001), MAV (*Luteovirus*) transmitted most efficiently by *S. avenae* (formerly *Macrosiphum avenae*), PAV (*Luteovirus*) transmitted most efficiently by *Rhopalosiphum padi* and *S. avenae*, SGV (unassigned to a genus within the family) transmitted most efficiently by *S. graminum* and RPV (*Polerovirus*) transmitted most efficiently by *R. padi* (Rochow, 1969, Rochow & Muller, 1971). BYD disease has significant impacts in agricultural and natural plant communities. It is the most economically damaging viral disease of grain crops worldwide (Lister & Ranieri, 1995) and in grasslands it may contribute to shifts in community composition due to asymmetrical fitness effects on exotic and native grass species (Malmstrom *et al.*, 2005a, Malmstrom *et al.*, 2005b). Among the species listed above PAV is the most widely distributed and economically important. Significant impacts in agricultural and natural plant communities. It is the most economically damaging viral disease of grain crops worldwide (Lister & Ranieri, 1995) and in grasslands it may contribute to shifts in community composition due to asymmetrical fitness effects on exotic and native grass species (Malmstrom *et al.*, 2005a, Malmstrom *et al.*, 2005b). Among the species listed above PAV is the most widely distributed and economically important. To reflect significant variation in coat protein (CP) sequence among isolates PAV has recently been divided into two species, PAV and PAS (Mayo, 2002). Sympatric populations of both species have been identified in Morocco (Bencharaki *et al.*, 1999), New York State (Chay *et al.*, 1996a) and France (Mastari *et al.*, 1998). BYDV is composed of a single-stranded, positive sense RNA with six open reading frames (ORF) in total (Miller *et al.*, 2002). Three viral genes were analyzed in this study, ORFs 2, 3 and 4. ORF2 encodes the viral RNA-dependent RNA-polymerase (RdRp) and is responsible for the replication of all viral RNAs (Koev *et al.*, 2002). ORF3 encodes the major component of the CP which is required for virion assembly (Mohan *et al.*, 1995) and is thereby a prerequisite for aphid transmission (Gildow, 1987, Gildow, 1993) and systemic plant infection (Filichkin *et al.*, 1994). ORF 4 encodes the movement protein (MP) which is required for the virus to spread systemically in the host (Chay *et al.*, 1996b). ORF 4 is completely embedded within ORF 3 but is translated +1 base pair out of the CP reading frame (Dinesh-Kumar & Miller, 1993). The objective of this review is to give an overview on Barley Yellow Dwarf Viruses (BYDV).

2. History of Barley Yellow Dwarf Virus

Barley yellow dwarf symptoms were first observed in 1951 on barley (*Horedum vulgare* L.) in California (Oswald and Houston, 1951). The disease was later characterized in oat (*Avena sativa* L.) and wheat. Since then it has been identified worldwide. Barley yellow dwarf symptoms are often mis-diagnosed because they resemble plant nutrient deficiencies. The most obvious symptoms are stunting and leaf discoloration. The stunted plants often appear in circular patches or as randomly scattered plants within a field. Leaf discoloration varies from shades of yellow, to red or purple. Visual leaf symptoms begin at the leaf tip and progress toward the middle and base of the leaves. High light intensity and cool temperatures (15 to 18° C) have been found to favor expression of BYD symptoms (D'Arcy, 1995). Symptom expression is generally dependent on the time of infection. Seedling infection may be lethal or cause a distinct yellowing of older leaves (Wiese, 1977). Plots with post seedling infections have a yellowed or reddened flag leaf (Wiese, 1977). Disease symptoms usually appear in late spring at jointing. Symptoms at jointing are predominately from fall infections. Spring infections have delayed symptoms that are usually less severe. Carrigan *et al.* (1981) and Herbert *et al.* (1999) noted that fall infection reduced yield to a greater extent than spring infection. Cisar *et al.* (1982) indicated that fall infection reduced yield 63%. Yield loss from spring infection is generally lower due to the shortened period of virus replication in the plant. However, significant yield loss may still occur. Cisar *et al.* (1982) reported spring infection reduced yield by 41%. Though, fall infections tend to be more severe, Perry *et al.* (2000) reported that there may be little yield difference between fall and early spring infections. Barley yellow dwarf virus also weakens plants, making them more susceptible to winter injury. Stand loss may be attributed to winterkill, but other factors may have weakened the plants, putting them at greater risk for winterkill. Cook and Veseth (1991) reported that stand loss may be a warning that the entire crop is in trouble, the survivors may only appear healthy and the culprit may be BYDV. Additional BYD symptomology may include stiff leaves, underdeveloped root systems, decreased tillering, and inhibited head development and grain fill (Wiese, 1977).

2.1. Current Classification

BYDV is the sole member of genus *Luteovirus* and the type member of the *Luteoviridae* family (formerly luteovirus group) (D'Arcy *et al.*, 2000). BYDV serotypes were divided into two subgroups, which were subsequently reclassified as separate species. Currently, only BYDV-MAV (transmitted primarily by *Sitobion avenae*) and BYDV-PAV (transmitted efficiently by *S. avenae* and *Rhopalosiphum padi*) are barley yellow

dwarf viruses. Former BYDV serotype RPV (transmitted primarily by *R. padi*) was given a new name, *Cereal yellow dwarf virus-RPV* (CYDV-RPV) and placed in genus *Polerovirus* along with four non-BYDV viruses in the *Luteoviridae*. A third genus, *Enamovirus*, consists only of RNA-1 of the bipartite *Pea enation mosaic virus* (PEMV). Its organization resembles poleroviruses, but lacks open reading frame (ORF) 4. After publication of the BYDV sequence, the sequences of several other luteoviruses were determined in rapid succession. These revealed a taxonomic dilemma that has continued to this day. Essentially the replication machinery of the *Luteoviridae* has two different evolutionary histories, whereas the proteins that form the virus particles and interact with the aphid vectors clearly have a common origin. Functional and comparative genomic analyses of BYDV and related viruses indicate that, from a molecular virological point of view, BYDV belongs in the *Tombusviridae* family (Miller *et al.*, 2002). The replication proteins and the RNA sequences that control replication and translation most closely resemble those of viruses in the *Tombusviridae* family (Figure 1). Yet the coat protein, movement and aphid transmission proteins clearly resemble those of the other *Luteoviridae*, including genus *Polerovirus*. Like the *Tombusviridae*, BYDV RNA lacks a 5' cap or any other modification (Allen *et al.*, 1999), and terminates at the 3' end with the sequence CCC, preceded by a conserved stem-loop (Koev *et al.*, 2002). In contrast, the poleroviruses, including CYDVRPV have a protein (VPg) linked to the 5' end and terminate in GU. In poleroviruses ORF 0 codes for a suppressor of the post-transcriptional gene silencing defense response (Pfeffer *et al.*, 2002). This ORF is absent in BYDV. ORF 1 of poleroviruses encodes a proteinase and the VPg which also are lacking in BYDV. All of the luteovirus-like genes of BYDV can be deleted and the remaining RNA can still replicate in protoplasts. Thus, the core of the virus, i.e. the gene expression and replication framework is more closely related to the *Tombusviridae* family than to other members of the *Luteoviridae* (Miller *et al.*, 2002).

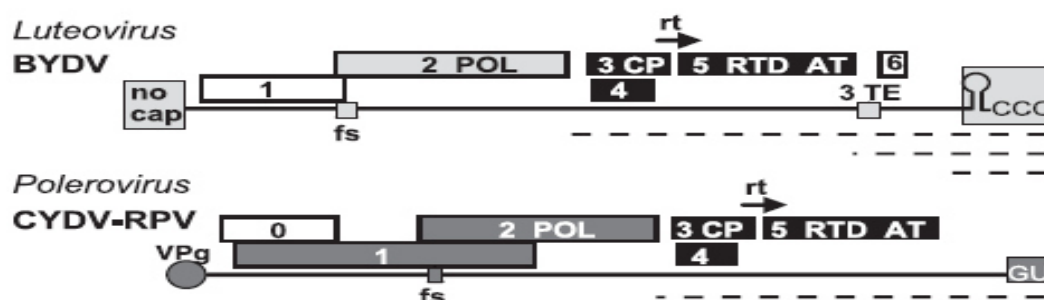


Figure 1. Genome organization of BYDV and CYDV-RPV.

Notes: Bold black line indicates genomic RNA; bold dashed line, subgenomic RNA. Open reading frames are numbered and functions indicated where known. POL, RNA-dependent RNA polymerase; CP, major coat protein; RTD, readthrough domain required for aphid transmission (AT); 3'TE, 3' cap-independent translation element; fs, frameshift signal; rt, readthrough site; VPg, viral genome-linked protein. Stemloops and terminal bases are shown at 3' ends of genomes. Light gray shading, features in common with at least one genus of the *Tombusviridae* and not with genus *Polerovirus*. Dark gray shading, shared among *Polerovirus* and *Enamovirus* genera and not genus *Luteovirus*. Black boxes, ORFs common to all *Luteoviridae* and absent in other families. White boxes, ORFs unique to the genus.

Yet with regard to symptomatology and practical plant pathology, BYDV is clearly a member of the *Luteoviridae*, defined as those viruses that: 1) are transmitted only by aphids in a persistent manner and not mechanically; 2) circulate but do not replicate in the aphid; 3) are confined to the phloem in the plant; and 4) have 25 nm icosahedral particles consisting of a major ~22 kDa coat protein and a minor component. None of these properties apply to the *Tombusviridae*.

2.2. Economic importance

BYDVs can have a serious impact on, and be an important limiting factor for, grain production wherever cereals are grown. However, global yield losses due to the BYDVs are difficult to estimate because of insufficient information. Average yield losses attributable to natural BYDV infection can range between 11 and 33% (Lister RM, Ranieri R. 1995); in some areas the losses were reported to reach up to 86%. The relationship between the disease incidence and yield loss was found to be linear in wheat and oats. A 1% increase in BYD disease incidence caused yield reduction to increase from 20 to 50 kg/ha in wheat and from 30 to 60 kg/ha in oats (F Nutter, personal communication). Hewings & Eastman (1995) calculated that hypothetical 5% losses caused by BYDVs in the United States in 1989 would result in crop losses valued at \$847.0 million for corn, \$387.1 million for wheat, \$48.5 million for barley, and \$28.0 million for oats. A PAV-like virus may also cause sugarcane yellow leaf disease in Brazil, Hawaii, and Australia Vega *et al.* (1997a). Thus the range of

economically important crops affected by BYDVs may be greater than previously thought.

2.3. Main Diseases

Causes stunting and chlorosis of a wide range of monocotyledonous species, including oats, barley, wheat, and many grasses (Bruehl, 1961; Rochow, 1961; Slykhuis, 1967). Losses may be great if infection occurs early in the growing season. BYD is diagnosed in the field by the presence of yellowish to reddish stunted plants grouped singly or in small patches among normal plants. Early infection of any of the cereals may result in severe stunting, excessive or reduced tillering, bright-yellowing or reddening of older leaves, delayed heading or ripening, increased sterility, and fewer and lighter kernels. In some oat cultivars, leaves become bronzed. This disease is not the only cause of red coloration in oats. Post-seedling infections are progressively less severe to the point where only the upper leaves, or the flag leaves, show discoloration. The leaves of plants infected with BYDV are shorter than normal and the flag leaf may be severely shortened. Leaves are often stiffer and more erect. Root systems are reduced and diseased plants are more easily pulled up than healthy ones (Wallwork, 1992; Watkins and Lane, 2004). On oats the first symptoms are yellowish-green spots or blotches near the tips of older leaves. Eventually these blotches enlarge and coalesce. Symptoms vary according to the variety, the virus strain, the growth stage of the plant at the time of infection, the general health of the plant, the temperature and other environmental factors. The main colour change is to shades of yellow, reddish-orange, reddish-brown, or purple (Martens, Seaman and Atkinson, 1985). In barley, the most characteristic symptoms are dwarfing and the brilliant yellow coloring of the leaves which extends from the tip towards the base. With late infections only the flag leaf may show symptoms. In wheat, severe dwarfing and general yellowing are less common, and the disease is more severe when infection takes place in the fall than in the spring. Although fall symptoms may be absent, infection predisposes the plants to winterkilling, and disease is severe when new growth starts the following spring. Yellowed or reddened leaves on otherwise normal plants are often typical of post seedling infections. Some cultivars show stunting but no leaf discoloration. Generally, the earlier the infection, the more severe the disease with the root system damaged as severely as the tops. Stunted plants result from the failure of stem internodes to elongate. This leads to a "telescoped" plant where the leaves may unfurl before they have fully emerged from the sheath of the previous leaf. Infected plants are "dwarfs" and have lost their normal conformation. Even the panicle fails to emerge fully or properly. Patterns of BYD in a field may be seen either as random within the crop or as circular or angular patches, which reflect the pattern of movement of the aphid vectors or carriers. Many infected plants ripen prematurely, after which they may be invaded by sooty moulds, which give them a dirty appearance and may lower germination of harvested seed (Watkins and Lane, 2004).

2.4. Geographical distribution

It is not surprising that BYDVs are of global importance (Lister and Ranieri, 1995) because they have a very wide host range in the Poaceae and can be spread efficiently by several aphid vectors that are prevalent worldwide. They are present in most of the countries where their vectors (*Rhopalosiphum padi*, *Sitobion avenae*, *Metopolophium dirhodum*, *Rhopalosiphum maidis*, *Schizaphis graminum* among others) have been reported.

2.5. Host Range

BYDVs are restricted in host range to the Poaceae. Cultivated hosts include all the major cereal crops: barley, maize, oat, rice, rye and wheat (*Triticum aestivum*, *T. durum*) (Oswald and Houston, 1951, 1953; Watson and Mulligan, 1960). Many annual and perennial lawn and weed pasture species are also hosts (D'Arcy, 1995). The pasture crops that are mostly affected include ryegrass (*Lolium perenne*, *Lolium multiflorum*) (Catherall and Parry, 1987; Eagling *et al.*, 1989), Fescue spp., Bromus spp. (Henry and Dedryver, 1991), cocksfoot (*Dactylis glomerata*), *Phalaris* (*Phalaris aquatica*) and Timothy grass (*Phleum pratense*) (Guy *et al.*, 1986; Guy, 1988). The known host range of the BYDVs includes more than 150 species (D'Arcy, 1995). D'Arcy (1995) lists wild Poaceae that have been reported to be naturally infected with BYDVs. These wild hosts may act as reservoirs for the virus. In addition, BYDV has been transmitted under experimental conditions to a wider range of Poaceae. In general, plants are more sensitive to BYDVs when they are infected at early growth stages. Smith and Sward (1982) showed nearly no damage occurred when the wheat was inoculated when the first node of the stem was visible, compared with up to more than 40% loss when inoculated before tillering. Comeau (1987) suggests that wheat has a temporary rise of resistance at the end of tillering, a period of higher sensitivity during stem elongation and second decrease in sensitivity at flowering time.

2.6. Symptom

Symptoms caused by BYDVs differ with the host species and cultivar, the age and the physiological state of the host plant at the time of infection, the strain and the environmental conditions and can be easily confused with nutritional and abiotic disorders. Symptoms include leaf discoloration from tip to base and from margin to centre. The discoloration takes on different colours depending on the plant. In barley, the leaf turns bright

yellow; in oat, an orange, red or purple discoloration is seen and in wheat, rye and triticale, the infected leaves are generally yellow and sometimes red. In maize, a conspicuous reddening occurs on the lower leaves, while in rice, infected leaves turn yellow to orange (D'Arcy, 1995) Some species of grasses show reddening or yellowing, but many of them are symptomless. Other leaf symptoms include serrations along leaf borders and corkscrew symptoms as observed on some wheat cultivars infected with RPV-Mexico. Plants are usually stunted, with a decrease in tiller number and biomass and a weak root system. Suppressed heading, sterility and failure of grains to fill occur in the most severe cases. In the field, symptoms appear usually as yellow or red patches of stunted plants. In hydroponic culture, the root system of BYDV-infected seedlings was initially more severely affected than the shoot, stunting was observed 4 days after infection in roots and only after 18 days in shoots (Hoffman and Kolb, 1997). In general, PAV causes severe symptoms, MAV moderately severe and RPV, RMV and SGV produce mild symptoms. However, there is a high variability amongst the severity of isolates from the same BYDV strain. Chay *et al.* (1996) reported PAV isolates ranging from mild to very severe, an RPV isolate producing corkscrew symptoms was isolated in Mexico, and in Ecuador. BYDVs are included in the *Luteovirus* genus. Their virus particles are isometric (measuring about 24-28 nm diameter), sedimenting as a single component at ca 104-118 S, and with a buoyant density in CsCl of ca 1.40g/cm³. The protein shell is composed of one polypeptide species of MW 23.5-24.4 x 10³ and the genome consists of one single molecule of positive sense single-stranded RNA (MWs: PAV, 1,850,000; MAV, 2,000,000; RPV, 1,850,000 - 2,000,000) (Waterhouse *et al.*, 1988). All of the *luteovirus* nucleotide sequences contain six open reading frames. The MAV and PAV (Group I) nucleotide sequences contain very small ORFs near the 3'-termini of their RNAs that are not present in the nucleotide sequences of the group II viruses (BYDV-RPV, BWYV, PLRV). The nucleotide sequences of the group II luteoviruses contain much larger ORFs near their 5'-termini than are found in the PAV and MAV sequences (Domier, 1995; Miller and Rosochová, 1997). BYDVs are distinct from the other viruses infecting cereals in their symptomatology, the morphology and size of their particles and the type of transmission. The symptoms can easily be confused with those of other biotic stresses. Therefore, diagnosis cannot be solely based on symptomatology but must be supported by other techniques such as transmission pattern, serology or PCR.

2.7. Strain of BYDV

There are several strains of BYDV; most are associated with a particular species of aphid (Table 1.1). The virus strains were originally differentiated by aphid vector, but now they are often differentiated by serotype-specific antibodies, with most of the strains classified under BYDV or *Cereal yellow dwarf virus* (CYDV). Barley yellow dwarf virus and CYDV are in the Luteoviridae family; however BYDV is in the genus *Luteovirus* and CYDV is in the genus *Polerovirus* (Miller *et al.*, 2002). The five common strains of BYDV/CYDV are PAV, SGV, RPV, RMV, and MAV. The RPV strain is now classified under CYDV. PAV and MAV remain classified under BYDV. However, SGV and MRV are currently not assigned to a Luteoviridae genus but remain under BYDV. PAV, a strain vectored by *R. padi* and *S. avenae* (Rochow 1979), is the most common and devastating strain in the Midwest. Aphid species, length of feeding period, and the physiological age of plant tissue influence transmission efficiency of various BYD viral strains (Gray *et al.*, 1991). Barley yellow dwarf virus is phloem restricted and is vectored more efficiently by different aphid species (Slykhuis 1967). Gray *et al.* (1991) reported that *R. padi* required a 1- to 2-hr or 2- to 3-hr acquisition access period (AAP) for 50% of the aphids to transmit PAV or RPV, whereas 50% of *S. avenae* required a 4- to 6-hr or 10- to 12-hr AAP to transmit MAV or PAV. It may take up to 48 hours for BYDV to be acquired from phloem sap, move through the aphid gut, and then be transmitted back into the phloem of another host plant (Zitter 2001).

Table 1. BYDV strains transmitted regularly by aphid species and Luteoviridae genus.

Virus	Efficient vector	Luteoviridae genus
RPV	<i>Rhopalosiphum padi</i> (BCOA)	Polerovirus (CYDV)
RMV	<i>Rhopalosiphum maidis</i> (CLA)	Not assigned
MAV	<i>Sitobion avenae</i> (EGA)	Luteovirus (BYDV)
SGV	<i>Schizaphis graminum</i> (GB)	Not assigned
PAV	<i>R. padi</i> and <i>S. avenae</i>	Luteovirus (BYDV)

Adapted from Compendium of Wheat Diseases and Miller *et al.* (2002).

Moreover, many variants have been identified on the basis of virulence, host range, and vector specificity. Recent evidence suggests that some of the variants are so different that they should perhaps be considered as distinct viruses, for not only do they seem serologically unrelated but their interactions in mixed infections and in cross-protection tests resemble those of unrelated viruses (Aapola, 1968). RMV - Weakly virulent in Coast Black oats, transmitted regularly by *R. maidis*, but infrequently by *R. padi*, *M. avenae*, and *S. graminum*. RPV - Weakly virulent in Coast Black oats, transmitted regularly by *R. padi*, erratically by *S.*

graminum, but rarely by *R. maidis* and *M. avenae*. MAV - Moderately virulent in Coast Black oats, transmitted regularly by *M. avenae*, but rarely by *R. padi*, *R. maidis*, and *S. graminum*. PAV - Strongly virulent in Coast Black oats, transmitted regularly by *R. padi* and *M. avenae*, erratically by *S. graminum*, but rarely by *R. maidis*. Three of these virus isolates (MAV, RPV, and PAV) have also been differentiated by serological techniques. SGV - Weakly virulent in Clintland 64 oats, transmitted regularly by *S. graminum*, but rarely if at all by *M. avenae*, *R. padi*, or *R. maidis*.

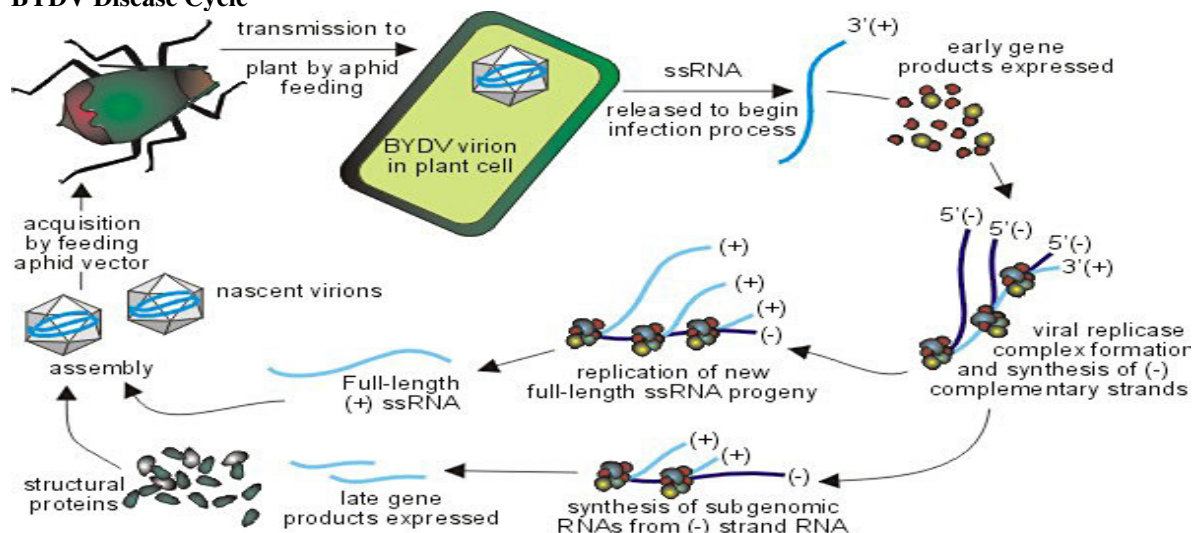
2.8. Morphology of BYDV

BYDVs are included in the Luteovirus genus. Their virus particles are isometric (measuring about 24-28 nm diameter), sedimenting as a single component at ca 104-118 S, and with a buoyant density in CsCl of ca 1.40g/cm³. The protein shell is composed of one polypeptide species of MW 23.5-24.4 x 10³ and the genome consists of one single molecule of positive sense single-stranded RNA (MWs: PAV, 1,850,000; MAV, 2,000,000; RPV, 1,850,000 - 2,000,000) (Waterhouse *et al.*, 1988). All of the luteovirus nucleotide sequences contain six open reading frames. The MAV and PAV (Group I) nucleotide sequences contain very small ORFs near the 3'-termini of their RNAs that are not present in the nucleotide sequences of the group II viruses (BYDV-RPV, BWYV, PLRV). The nucleotide sequences of the group II *luteoviruses* contain much larger ORFs near their 5'-termini than are found in the PAV and MAV sequences (Domier, 1995; Miller and Rosochova, 1997). BYDVs are distinct from the other viruses infecting cereals in their symptomatology, the morphology and size of their particles and the type of transmission. The symptoms can easily be confused with those of other biotic stresses. Therefore, diagnosis cannot be solely based on symptomatology but must be supported by other techniques such as transmission pattern, serology or PCR.

2.9. Disease Cycle of BYDV

Barley yellow dwarf virus (BYDV), a member of the *Luteoviruses*, is a group of five closely related virus strains. Strains of BYDV differ serologically and in virulence, host range and vector specificity. Virus particles are spherical. BYDV is transmitted by more than 20 aphid species. The most important are the oat bird-cherry aphid, the corn leaf aphid, the English grain aphid and the green bug. Aphids acquire BYDV by feeding on infected plants and transmit the virus in subsequent feedings (Watkins, J. 2003). The virus survives in perennial grasses and is spread by aphids to and within crops. Autumn and spring flights of aphids from infected grasses or crop plants establish colonies in crops. The disease is not transmitted by any other insects and aphids need to feed on an infected plant for at least 5 minutes followed by a latent period of 12 hours, before the virus will transmit to a healthy plant. Aphids remain infected for the rest of their life. However, infective aphids do not pass on the virus to their progeny. Losses depend strongly on time of infection. If plants are infected in the fall, losses can exceed 35%. If plants are infected after heading, losses are minimal (Bowden, R.L. 2000).

BYDV Disease Cycle



Source: (Zishan G, Aftab A.K. and Jamil K., 2011)

Figure 2. 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 (ORF5) 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.

2.10. Transmission of BYDV

BYDVs are not mechanically or seed transmissible, but are transmitted by aphids in a persistent, circulative but non-propagative manner. They are not transmitted to the progeny. Aphids acquire and transmit BYDVs while feeding on the phloem sieve tube elements of host plants. Minimum feeding access times reported for aphids to acquire or inoculate *luteoviruses* range from 0.1-4.0 h and 0.2-1.0 h, respectively. These reported times include the time required for the aphid's stylets to penetrate to the phloem tissue. Efficient transmission of most *luteoviruses* requires acquisition (AAP) and inoculation access period (IAP), each of 24 h. The minimum latent period (time from the start of acquisition feeding period until the insect becomes able to infect a plant) is normally between 12 and 24h (Waterhouse *et al.*, 1988). After acquisition through the phloem, virions are transported to the aphid hindgut. They cross the hindgut epithelium and are transported in the hemocoel in coated vesicles. The virions must then penetrate the accessory salivary gland basal lamina and plasmalemma to be released into the salivary canals. The virions will then be excreted while the aphid is feeding (Gildow and Gray, 1993). It is suggested that the accessory salivary gland basal lamina possess a selective function that regulates vector specificity through receptors on the plasmalemma and domains of the BYDV virions that interact with these receptors. Van den Heuvel *et al.* (1994) showed that the aphid protein symbionin was associated with luteovirus transmission and suggested that the interaction between virions and symbionin is involved in maintaining virus integrity and thus specificity. The specificity of transmission is high. Vector transmission pattern corresponds to serotypes in most cases. However, because more BYDV isolates are being characterized, inconsistencies between transmission pattern and serotypes are becoming more and more common (Power and Gray, 1995). Examples are given by Eweida and Oxelfelt (1985), Lister and Sward (1988), Creamer and Falk (1989) and Halbert *et al.* (1992).

2.11. Transmission by Aphid

BYDV virions pass through at least three barriers in the aphid (Figure 3) by specific uptake. They do not replicate in the aphid. Each BYDV serotype is transmitted efficiently by only a limited number of aphid species (PowerAG, Gray SM., 1995). The vector specificity of BYDVs does not always correlate with serotype (Halbert *et al.*, 1992, Lei *et al.*, 1995, Lister RM, Sward RJ., 1988). For example, Creamer & Falk (Creamer R, Falk BW., 1989) described an RPV isolate from California (RPV-CA) that is transmitted efficiently by *Sitobion avenae*, which gives it the transmission phenotype of PAV. The genomes of RPV-CA and the type RPVNY isolate exhibit sequence homology in the 30 halves but are unrelated in their 50 halves, based on the northern blot hybridization. The transmission phenotypes of BYDVs also may be altered transiently by heterologous encapsidation during mixed infections (Creamer R, Falk BW., 1990, RochowWF., 1970, Wen F, Lister RM., 1991).

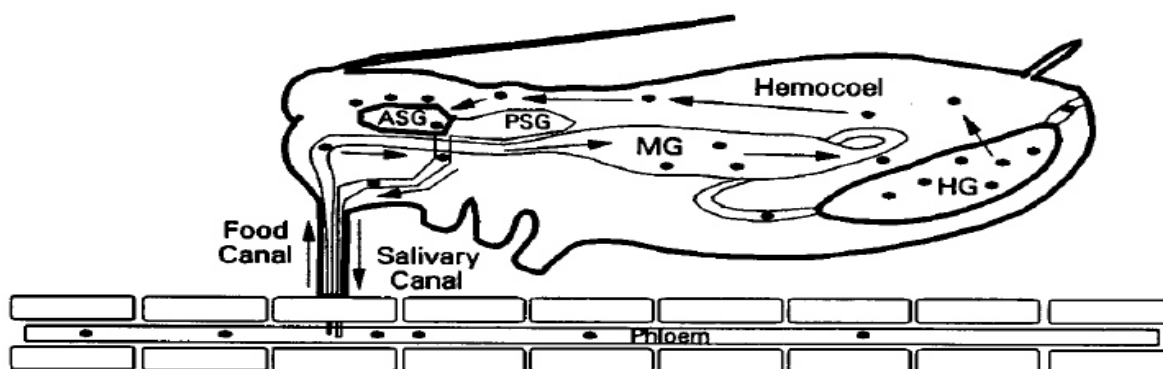


Figure 3. Schematic diagram of aphid feeding and luteovirus transmission (Chay *et al.*, 1996). Arrows indicate circulative pathway of transmission. ASG, the accessory salivary gland; HG, hindgut; MG, midgut; PSG, principle salivary gland.

2.12. Transmission by Dodder

Virus has been recovered by aphids feeding on dodder established on infected barley (Orlob & Medler, 1961),

and virus has been transmitted from barley to barley by *Cuscuta campestris* (Timian, 1964).

2.13. Serology

Serologically, BYDV viruses can be divided into subgroups: subgroup I includes PAV, MAV and SGV and subgroup II includes RPV and RMV. ELISA has been proven to be a fast, sensitive and versatile method to detect plant viruses (Clark and Adams, 1977) including BYDVs (Lister and Rochow, 1979; Rochow and Carmichael, 1979). There is a high serological specificity among BYDVs, so that individual strains can be easily identified. RMV reacts weakly with polyclonal antiserum to RPV, while MAV and SGV cross-react with polyclonal antiserum to PAV. ELISA can be used to detect BYDVs in air-dried samples (Lister *et al.*, 1985) allowing samples to be sent by mail to a testing laboratory. Monoclonal antibodies have been prepared against several of the BYDVs (Hsu *et al.*, 1984; Diaco *et al.*, 1986; Torrance *et al.*, 1986; Pead and Torrance, 1988; D'Arcy *et al.*, 1990). These can be used to better separate BYDV strains as no cross reaction exists. However, when used in a survey, they might be too restrictive, resulting in some particular isolates with different epitopes not being detected. For example, unusual epitope profiles of BYDVs have been identified in Asia using a range of polyclonal and monoclonal antibodies from various origins (McGrath *et al.*, 1996). Several private companies sell polyclonal and monoclonal antibodies and ELISA kits for the detection of the five BYDV strains. Immunosorbent electron microscopy (ISEM) can also be used to detect BYDVs and to separate strains (Forde, 1989), while the tissue-blot immunoassay (TBIA) has been used to evaluate resistance of wheat and barley germplasm to BYDV (Makkouk *et al.*, 1994; Makkouk and Comeau, 1994b). Detection at the level of a single aphid vector has been achieved by using an amplified ELISA (Torrance, 1987).

2.14. Polymerase Chain Reaction (PCR)

Luteovirus-specific PCR primers in RT-PCR (reverse-transcriptase PCR) allow PCR amplification of a 530bp cDNA fragment from all five strains of BYDV (Robertson *et al.*, 1991). Strains of BYDV can then be distinguished by cutting the PCR products with restriction endonucleases. With the sequence data of more BYDVs being made available, strain specific primers BYDV primers are being developed (French, 1997). BYDV has been detected in single aphids using the RT-PCR technique (Canning *et al.*, 1996).

2.15. Relationships between BYDV isolates

In the classification of luteoviruses agreed upon by the international committee on taxonomy of viruses (Randles and Rathjen, 1995), the five isolate of BYDV defined by Rochow (1969) and Jhonson and Rochow (1972) are placed in to two sub group as shown Table 2. This grouping is based on a range of criteria , including serology (Aapola and Rochow, 1971); Rochow and Carmichael, 1979), ultra structural symptomology (Gill and Chong, 1979), synergy and cross protection between isolates within the host (Halstead and Gill, 1971; Rochow, 1975; Wen *et al.*, 1991), and the size and number of double stranded RNAs found in infected plant tissue (Gildow *et al.*, 1983). Recently, the sequencing of the RNA genome of number of BYDV isolates in part or in full has provided additional information concerning the relationship between BYDV isolates (Miller *et al.*, 1988; Vincent *et al.*, 1990; Vincent *et al.*, 1991; Ueng *et al.*, 1992; Domier *et al.*, 1994). Most significantly the genome organization of the subgroup I isolate PAV and MAV was found to differ from the genome organization of the subgroup II isolate PAV (Miller *et al.*, 1995). In addition the degree of similarity between the nucleotide sequences of different BYDV isolate , shown by coat protein gene sequence comparison (Domier *et al.*, 1994) and nucleic acid hybridization studies (Fattouh *et al.*, 1990) have agreed with the classification of BYDV isolates in to two subgroups

Table 2. The classification of BYDV isolates in to two subgroups.

BYDV sub group	BYDV isolate
I	MAV PAV SGV
II	RPV RMV

The difference between sub group I and II BYDV isolates are so great that isolates from the two BYDV sub groups are considered by some to be two separate viruses (Miller *et al.*, 1995). Interestingly, the genome organization, nucleotide sequence and the serology of subgroup II BYDV isolates are more similar to the luteoviruse beet western yellow virus (BWYV) and potato leaf roll virus (PLRV), which infect dicotyledons, than to the BYDV isolates from subgroup I (Rochow and Duffus, 1978; Domier *et al.*, 1994; Miller *et al.*, 1995)

2.16. Stability in Sap

MAV has been recovered from crude sap (diluted as much as 1/1000) by *M. avenae* feeding through membranes on sap containing added sucrose. The thermal inactivation point (10 min) for RPV and MAV both in crude and

in partially purified preparations is between 65 and 70°C (Heagy & Rochow, 1965).

2.17. Purification of BYDV

MAV has been purified by chloroform clarification, differential centrifugation, and density gradient centrifugation (Rochow and Brakke, 1964). Yields of MAV are usually less than 100 µg/l of juice. Purified preparations of RPV, PAV and RMV have also been obtained by the same method, but yields of virus are usually less, especially with PAV and RMV. Precipitation by polyethylene glycol (M. Wt = 6000) (Hebert, 1963) has shown promise in preliminary tests. Growth of source plants at temperatures below 20°C and thorough extraction of virus from tissue are two critical steps in purification. Moreover, a vector-specific strain of barley yellow dwarf virus (transmitted selectively by *Macrosiphum avenae*) was partially purified from frozen oats by differential centrifugation following clarification by chloroform. Since the virus is not mechanically transmissible to plants, infectivity assays were based on feeding aphid vectors through membranes on test inocula. Concentrated virus preparations stored at 3°C remained infectious for up to 16 weeks; similar preparations were infectious following treatment (18 hours at 15°C) with any of several proteases and nucleases. A major limiting factor in purification appeared to be the low concentration of virus in source plants; usually the yield of purified virus from 1 liter of plant juice was only about 25-50 µg. Analytical sucrose density-gradient centrifugation of preparations made from infected oats revealed a component (sedimentation coefficient 115–118S) that was not present in comparable preparations made from healthy oats in each of three experiments. A dense polyhedral particle about 30µm in diameter was the major constituent of the extra component from infected plants. This polyhedral particle is believed to be the virus because it sedimented to the same depth as did the infectious entity in rate and equilibrium-zonal centrifugations, and it migrated with the infectious entity in Sephadex gel filtration. The particle also was obtained from viruliferous aphids. <http://www.sciencedirect.com/science/article/pii/0042682264901692>

2.18. Physicochemical properties of BYDV

2.18.1. Physical properties

Usually one sedimenting component in partially purified preparation; sedimentation coefficient is 115-118S (for BYDV-MAV). 1.4-1.405GCM IN CsCL (for BYDV-PAV). Density is 1.533g/cm³ in CsSO₄ for BYDV-PAV. In addition, sedimentation coefficient ($s_{20, w}$) is c. 115-118S for MAV. Other isolates studied appear to have a similar coefficient. A_{260}/A_{280} : c. 1.92 (MAV), 1.72 (RPV) (Brakke & Rochow, 1964).

2.18.2. Particle Structure

Particles are isometric; diameter c. 30 nm in shadowed preparations (Rochow and Brakke, 1964), c. 24nm in thin sections of host tissue (Jensen, 1969), and c. 20 nm in negatively stained virus preparations. Particles of four isolates appear similar, although RPV is less stable than MAV in phosphotungstate at pH 6.85. The particle may be an octahedron (Israel & Rochow, unpublished). Moreover, it has no information on particle compositions.

2.18.3. Particle Morphology

All BYDV have many common features. The virus particles is 25nm icosahedral (T = 3) virion. One major (22kda) and one minor (50-55kda) coat protein. 5.6-5.8kb positive sense RAN genome with no 5' cap and no poly (A) tail (Miller *et al.*, 2002)

2.18.4. Biochemical properties

Virion contains 28% of nucleic acid (in BYDV-MAV like isolates), 72% protein and no lipid (Paliwal, 1982). Genome consists of single stranded, linear RNA with molecular weight 2×10^6 d. Total genome size is 5.673kb (Gerlach *et al.*, 1987). Genome is unipartite, largest (or only) genome part 5.6kb (BYDV-RPV; 5.677 for BYDV-PAV). Genome nucleic acid was isolated by Brake and Rochow (1974). Base composition is 24.6%G, 29.6%A, 23.8%C AND 22%U. Poly A region is absent. Information on nucleotide sequence on various isolate of BYDV has been reviewed by Miller *et al.*, 1987.

2.19. Genome organization

The recent determination of the complete nucleotide sequence of three members of the luteovirus group has greatly enhanced our understanding of genome organization and gene-expression strategies of luteoviruses. The complete nucleotide sequence of P A V serotype of barley yellow dwarf virus (BYDV-PAV), PLRV (Keese *et al.*, 1990, Mayo *et al.*, 1989, Van der *et al.*, 1989), and BWYV (Veidt *et al.*, 1988), as well as the partial sequences of RNAs of SDV and CRL V, allows delineation of the features of genome organization that are most likely common to all luteoviruses, and other features unique to particular subgroups of the luteoviruses. Comparison of the BYDV-PAV, PLRV, and BWYV nucleotide sequences suggests that the luteovirus group contains at least two major genome organization maps. The two maps are represented, respectively, by PLRV/BWYV and BYDV-PAV. The organization and general features of the three fully sequenced luteovirus

genomes are shown in Figure 4 and Table 3. For this review we have reassigned the numbering of the open reading frames (ORFs) from those designated by the original investigators, including ourselves. The renumbered ORFs shown in Figure 4 reflect the sequence similarities found between the three luteoviruses. ORFs 1-5 of BYDV -PAY (Figure 4A) correspond in sequence similarity and likely function to ORFs 1-5 of PLRV and BWYV (Figure 5B). It remains to be determined if the two subgroups of genome organization represented by PLRV/BWYV and BYDV-PAV are the only ones among the luteoviruses. Partial sequences available for SDV, CRLV, and PEMV suggests that they too will have genome organizations corresponding to either BYDV-PAV or PLRV/BWYV genome organization. The genomes of all members of the luteovirus group share common structural and organizational features. They all have a positive-sense ssRNA genome of about 1.8-2 x 10⁶ Mr (5.5-6.0 kb) with a genome-linked protein (VPg) at the 5' end (Mayo *et al.*, 1982, Murphy *et al.*, 1989). Luteovirus genomes are most likely not 3' polyadenylated (Mayo *et al.*, 1982), nor do the 3' ends appear to possess tRNA-like structures such as that associated with other plant viral groups (Miller *et al.*, 1988). There is little noncoding sequence and all genomes possess at least four overlapping ORFs (Figure 4). All three viruses encode their ORFs in one or another of the three reading frames on the positive sense RNA (encapsidated RNA). The sizes of the predicted ORFs are indicated in Table 3. The BYOV-PAV genome is comprised of five large ORFs (ORFs 1-5) and a small 6.7K ORF 6, whereas both PLRV and BWYV encode six major ORFs (ORFs 0-5). There is no evidence that the small ORFs encoded by the complementary RNA are expressed. As is discussed below, each class of genome organization shares common properties that clearly identify each virus as a member of the luteovirus group. Common features of genome organization include; (a) overlapping ORFs 1 and 2; (b) ORF 3, which entirely encompasses ORF 4; and (e) an ORF 5 that is separated from the preceding ORF 3 by an amber termination codon. In general, the 3' halves of both genome organizations are similar. But the 5' halves are quite divergent. The genome organization of PLRV and BWYV is distinguished from BYDV-PAV genome organization by an additional ORF, designated ORF 0 at the 5' end. ORF 0 of PLRV and BWYV encode 28K and 29K products respectively. There is no corresponding ORF in the BYDV-PAV genome.

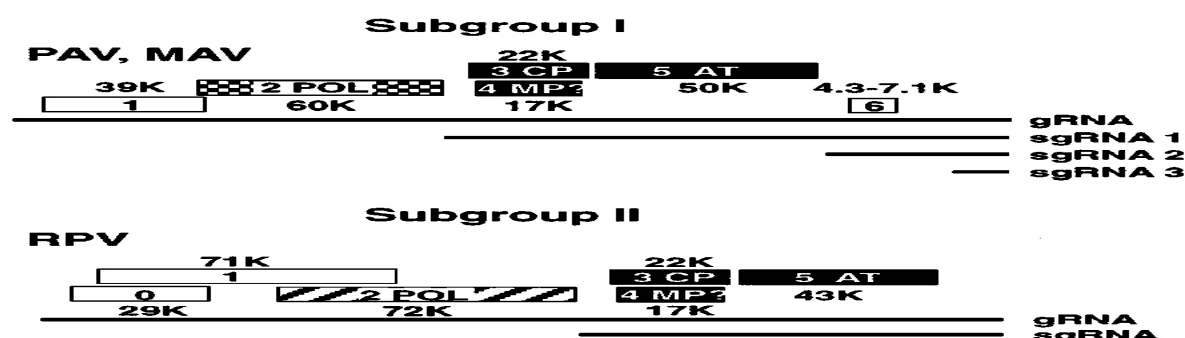


Figure 4. Genome organization of the BYDVs. Boxes indicate open reading frames, numbered as in Martin *et al* (1990), with molecular weight of protein products in kilodaltons (K). Black-shaded ORFs are conserved between subgroups. Abbreviations: CP, major coat protein gene; POL, putative polymerase gene; AT, readthrough domain probably required for aphid transmission; MP, putative cell-to-cell movement protein. Checkered POL ORF has homology to *Tombusviridae*, especially *dianthoviruses*. Striped POL ORF is homologous to *sobemoviruses*. Unshaded ORFs have no significant similarity to ORFs of any virus, with the exception of a possible protease motif in ORF 1 of subgroup II. Known positions of subgenomic RNAs are shown below the genomes.

Table 3. Size of proteins encoded by predicted ORFs of three luteoviruses and lengths of noncoding regions.

ORF or NCR'	Virus		
	BYDV-PAV	BWYV	PLRV
0	-	29k	28k
1	39	66	70
2	60	67	69
3	22	22.5	23
4	17	19.5	17
5	50	51.5	56
6	6.7	-	-
5'NeR	141ntb	31nt	71nt
INTERNAUNCR	116nt	202nt	196-197nl
3'NCR	568nt	146nt	141nt

• NCR = noncoding region, bnt = nucleotides

ORFs 1 and 2 in both types of genome organizations overlap, but the amount of overlap varies. ORFs of PLRV and BWYV overlap by 298nt or 474nt, respectively, but ORFs of BYDV-PAV overlap by only 13nt. ORFs 1 of BYDV -PA V, PLRV, and BWYV code for products of 39K, 70K, or 66K, respectively. As detailed below, ORF 2 does not encode a separate gene product but, rather, is expressed as a fusion protein with the product of ORF 1. ORF 3, which ranges in predicted size from 22K-23K, follows a tract of noncoding sequence of varying lengths beyond ORF 2 (Table 3). Contained entirely within ORF 3 is yet another ORF, designated ORF 4, that ranges in predicted size from 17K-20K (Figure 4). The last ORF shared by all three viruses is ORF 5, which is found in the same translational reading frame as the upstream ORF 3, but is separated from ORF 3 by an amber termination codon. The predicted molecular weights for these ORFs range from 50K-56K (Table 3). Evidence given below suggests that ORF 5 is expressed as a readthrough product of ORF 3. In addition to the large ORFs discussed above, BYDV-PA V has an additional ORF (with a 6.7K product) downstream of its ORF 5 (Figure 4). Finally, the 3' noncoding regions of PLRV and BWYV are relatively short (141nt and 146nt, respectively), compared to that of BYDVPAY (568nt) (Table 3).

2.19. 1. Replication Proteins

Gene functions are not well characterized for BYDVs and are currently under investigation in many laboratories. Sequence comparisons revealed that open reading frame (ORF) 2 encodes the catalytic domain of the RNA-dependent RNA polymerase (Figure 4) (Miller *et al.*, 1988, Ueng *et al.*, 1992, Vincent *et al.*, 1991). There is no evidence that the product of this ORF, P2, is translated by itself in luteoviruses of either subgroup. Rather, it appears to be expressed only fused to P1 (product of ORF 1) via translational frameshifting, in a P1-2 fusion (Figure 5). Consistent with a role in RNA replication, deletion mutations in ORFs 1 or 2 of PAV (Mohan *et al.*, 1995) or BWYV (Reutenauer *et al.*, 1993) destroyed the ability to replicate in plant cells. Because P1 is expressed by itself (the most abundant form) and fused to P2, it has two functions. In its rarer form, fused to P2, it is part of the RNA-dependent RNA polymerase. Its function when expressed by itself is unknown for PAV. Habili and Symons (1989) proposed that it is a helicase. It makes sense that a replicase-associated protein would have such a function, keeping (+) and (-) strands apart during RNA synthesis. However, Koonin & Dolja (1993) and Gibbs (1995) assert that this ORF has no homology to known helicases and that RNA viruses with genomes under 6 kb lack helicases. ORF 1 of subgroup II-like RNA1 of PEMV (Demler SA, de Zoeten GA., 1991) and all subgroup II luteoviruses including RPV (Miller *et al.*, 1985) have homology to the catalytic triad of chymotrypsin- like proteases. This implies that the P1-2 fusion protein has a protease in its N-terminal half, and the polymerase in its C terminus. Such an arrangement resembles poty-, como-, and picornaviruses that have a VPg-protease-polymerase polyprotein, which subsequently self-cleaves as replication initiates. The VPg is a genome-linked protein covalently attached to the 50 end of some viral RNAs including those of subgroup II luteoviruses (Mayo *et al.*, 1982, Murphy JF, D'Arcy CJ, Clark JMJ., 1989). Thus we speculated that the VPg of RPV might be encoded in the 50 end of ORF 1, upstream of the protease domain. Luteoviruses also appear to differ from others in that the Vpg would be produced in large molar excess to the polymerase which is expressed only as a result of a rare frameshift event (BraultV, MillerWA., 1992, Di *et al.*, 1993). In contrast to our speculation several years ago (Miller *et al.*, 1988), ORF 4 does *not* encode a genome-linked protein (VPg). Despite much phylogenetic (Miller *et al.*, 1995), biochemical (Tacke *et al.*, 1996), and genetic (Mohan *et al.*, 1995, Rathjen *et al.*, 1994) evidence that ORF 4 does not encode the VPg, some researchers still refer to ORF 4 as encoding the VPg.

2.19. 2. Structural Proteins

Much progress has been made recently in elucidating the roles of the proteins most conserved among all luteoviruses, those encoded by ORFs 3, 4, and 5. Besides its obvious function in forming virions, the coat protein (encoded by ORF 3) may have roles in virus movement in plants (Ziegler-Graff *et al.*, 1996) and in replication. Mutations that render ORF 3 untranslatable reduced accumulation of genomic RNA of both BWYV (Reutenauer *et al.*, 1993) and PAV (Mohan *et al.*, 1995). This may be due to simple increased sensitivity of the genomic RNA to nucleases during extraction because it cannot be encapsidated, or the CP may be involved more directly in RNA replication. The coat protein is obviously required for aphid transmission and it may confer aphid vector-specificity.

ORF 5 is expressed as a carboxy-terminal extension to the CP, produced in low abundance by in-frame readthrough of the CP ORF stop codon. The CP and the extended form containing the readthrough domain (RTD) make up the virion (Cheng *et al.*, 1994, Filichkin *et al.*, 1994, Wang *et al.*, 1995), with the RTD probably located on the surface (Filichkin *et al.*, 1997). A significant portion of the C terminus of the RTD is cleaved proteolytically to give the truncated form of the CP-RTD fusion (MW 51-58 kDa) that is found in purified virions. Substantial evidence indicates that this truncated CP-RTD is required for aphid transmission. The C-terminal portion that is cleaved off may be involved in systemic movement in the plant. None of the RTD is

required for virion formation. Deletions in this ORF in PAV and in BWYV actually increased RNA replication in protoplasts and did not affect the ability of the RNA to be encapsidated (Mohan *et al.*, 1995, Reutenauer *et al.*, 1993).

2.19. 3. Movement Protein

ORF 4 probably codes for a cell-to-cell movement protein. This protein may facilitate viral genome movement only through the specialized plasmodesmata of phloem cells and thus explain confinement of virus to these cell types. P4 of PLRV has many of the biochemical properties expected of a movement protein. It binds single-stranded nucleic acid nonspecifically (Tacke *et al.*, 1991); it has a protein-protein binding domain (Tacke *et al.*, 1993); and it localizes to the membrane fraction (Tacke *et al.*, 1996). Knocking out ORF 4 (but not the overlapping CP gene) did not affect accumulation of PAV (Mohan *et al.*, 1995) or BWYV (Ziegler-Graff *et al.*, 1996) in protoplasts. PAV mutants containing this mutation could not be transmitted to plants (Chay *et al.*, 1996). Virus from protoplasts mixedly infected with two mutant PAVs, one containing this ORF 4 knock-out mutation and the other containing a deletion in ORF 5, was able to infect plants. The only viral genome that accumulated in plants from this mixed inoculum was that containing the deletion in ORF 5 (Chay *et al.*, 1996). Thus P4 is required for systemic infection of plants but not for infection of protoplasts. This is consistent with a cell-to-cell movement function. In contrast, Ziegler-Graff *et al.* (1996) constructed a mutant BWYV genome with three stop codons interrupting ORF 4. Progeny virus replicated well, maintained the mutations, and was aphid transmissible to other hosts. In both PAV (Chay *et al.*, 1996) and BWYV (Brault *et al.*, 1995), mutations in the RTD reduced virus titer in plants, leading Ziegler-Graff *et al.* (1996) to propose that a domain in the C terminus of the RTD was required for movement in the plant, perhaps redundant to, or stimulated by, the P4 function.

2.19. 4. Gene expression

BYDVs use a combination of RNA-templated transcription and noncanonical translation mechanisms to express their six genes from a single genomic RNA. One of the most remarkable features of BYDVs, PAV in particular, is the plethora of unusual mechanisms by which the genes are translated. These include cap-independent translation, ribosomal frameshifting, in-frame stop codon readthrough, and leaky scanning (MayoMA, ZieglerGrafV., 1996, Miller *et al.*, 1995, and Miller *et al.*, 1997).

2.19. 5. Subgenomic RNA Synthesis

Viral RNAs with 5' truncations but the same 3' ends as genomic RNA are generated in infected cells. These subgenomic RNAs serve as messages for the 5'-distal open reading frames. The 5' end of subgenomic RNA 1 (sgRNA1) of PAV has been mapped to base 2769 by Dinesh-Kumar *et al.* (1992) and to base 2670 by Kelly *et al.* (1994). This difference may be due to strain variation, but we now have data that support the base 2670 start site. The apparent 5' end of 2769 is probably incorrect owing to an unlucky combination of misleading results. The 5' end determined by Kelly is appealing because it shares sequence with the 5' end of the genome: (A) GUGAAG (A in parentheses is absent in sgRNA1), and is similar to the 5' end of sgRNA2 at base 4809: AGUGAAGA (Kelly *et al.*, 1994). SgRNA1 is the mRNA for ORFs 3, 4, and 5 (Dinesh-Kumar *et al.*, 1992). SgRNA2 can act as mRNA for ORF 6 in vitro (Kelly *et al.*, 1994). The first ten codons of ORF 6 vary only in the wobble position giving silent mutations (no change in amino acid sequence), suggesting that its product, P6, is functional. However, the remaining codons are not conserved, as the rest of the ORF is the most variable region in the BYDV genome (Chaloub *et al.*, 1994). P6 has not been detected in vivo, despite considerable efforts (M Young, personal communication). Mutation of the ORF6 start codon reduces but does not eliminate genomic RNA replication in protoplasts (Mohan *et al.*, 1995). The role of sgRNA3, which seems to have no message function and which differs at its 5' terminus (GACGACC) (Kelly *et al.*, 1994) from the other viral RNAs, is unknown. Subgroup II BYDV sgRNAs have not been studied, but the 5' ends of genomic and sgRNAs of other subgroup II luteoviruses begin in ACAA (Miller *et al.*, 1991), as does genomic RNA. This sequence is also present within 20 bases of the 5' ends of PAV genomic and sgRNAs (Miller *et al.*, 1995). One candidate start site for sgRNA1 of RPV (Miller *et al.*, 1995) at base 3576 begins in ACAAACGUA, which is a perfect match with the start site of RCNMV sgRNA1 (Zavreiv *et al.*, 1996). If this is the start site for RPV sgRNA, we can expect that subgenomic promoter analysis of RCNMV may apply to RPV. Alteration of the ACAA to ACUAA in an infectious transcript of RCNMV had little effect on sgRNA synthesis (Zavreiv *et al.*, 1996). Changes that may weaken a proposed minus-strand stem-loop structure, which flanks the complement of the RCNMV sgRNA1 5' end, eliminated sgRNA synthesis (Zavreiv *et al.*, 1996). However, whether it is secondary structure or actual RNA sequence that provides promoter function was not determined. Alteration of the ORF6 start codon to AUC abolished accumulation of sgRNA2 (Mohan *et al.*, 1995). Either this mutation disrupted the promoter of sgRNA2, which begins 114 bases upstream, or by making sgRNA2 untranslatable, the stability is decreased. It has been assumed that sgRNAs are synthesized by internal initiation of the polymerase on full-

length minus stranded RNA (Zavreiv *et al.*, 1996), based on studies of brome mosaic (Miller *et al.*, 1985) and other viruses. This may be the case for BYDVs, but also plausible is the possibility that the replicase terminates prematurely at a defined site during minus strand synthesis (Miller *et al.*, 1997). Plus strand synthesis would then initiate at the 3' end of this 3' terminally truncated minus stranded RNA to make plus stranded sgRNA. The extensive homology between 5' termini of genomic and sgRNAs, and the abundant, subgenomic-sized double-stranded RNAs in BYDV-infected tissue (Gildow *et al.*, 1983), support this possibility. Functional dissection of subgenomic promoters will determine which mechanism applies.

2.19. 6. Translation

Leaky scanning: - In all luteoviruses, ORFs 3 and 4, and in subgroup II luteoviruses, ORFs 0 and 1 (Mayo *et al.*, 1989, Veidt *et al.*, 1992, Ziegler-Graff *et al.*, 1996), are translated by leaky scanning. According to this mechanism, if the first (5'-proximal) AUG on an mRNA is in a poor context, some scanning ribosomes can ignore this AUG and start protein synthesis at the second AUG. The AUGs of ORFs 4 and 1 are the second AUGs on their mRNAs and are indeed in better contexts than the first AUGs, which initiate ORFs 3 and 0, respectively (Miller *et al.*, 1995). Both products of ORFs 3 and 4 (CP and P4) can be translated from sgRNA1 of PAV (Dinesh-Kumar *et al.*, 1992), RPV (Vincent *et al.*, 1990), BWYV (Veidt *et al.*, 1988), and PLRV (Tacke *et al.*, 1990). In addition to the primary sequence context controlling AUG choice as expected, further observations led us to propose a new mechanism by which pausing of ribosomes during initiation at the second (ORF 4) AUG transiently "melts" secondary structure, which enhances initiation at the upstream (ORF 3) AUG by the following ribosome (Dinesh-Kumar *et al.*, 1993). The arrangement of ORF 4 completely nested within, and out of frame of, ORF 3 led to the hypothesis that ORF 4 evolved relatively recently as a kind of accident during out-of-frame translation of ORF 3 (Keese PK, Gibbs A., 1992).

Ribosomal frame shifting: - In all luteoviruses, the polymerase is translated by minus 1 (-1) ribosomal frameshifting. During the elongation process in translation of ORF 1, a small fraction of translating ribosomes slip back one base at a specific sequence, called the shifty heptanucleotide, and then resume translation in a new reading frame. This shift allows the ribosomes to bypass the stop codon of ORF 1. This has been demonstrated for several luteoviruses including PAV (Brault *et al.*, 1992, Di *et al.*, 1993, Garcia *et al.*, 1993, Miller *et al.*, 1995). The consensus signals known to facilitate -1 frameshifting for polymerase expression in corona-, retro-, and yeast viruses are the shifty site with the consensus XXXYYYZ, followed by a region of substantial secondary structure, usually a pseudoknot (Farabaugh PJ., 1996). These sequences and structures are present or predicted in all luteoviruses (Miller *et al.*, 1995). They are also present in other -1 frameshifting plant viruses, all of which are members of the groups most closely related to luteoviruses, including cocksfoot mottle sobemovirus (Makinen *et al.*, 1995), both PEMVRNAs (Demler SA, de Zoeten GA., 1991, Demler *et al.*, 1993), and the dianthoviruses (Kim KH, Lommel SA., 1994). The actual frameshift signals differ between the subgroups. PAV and MAV have GGGUUUU as the shifty site, followed by a region that can be folded into two stem-loops in which the loops base-pair to each other, or into a large stem-loop (Brault *et al.*, 1992). They favor the latter structure, based on phylogenetic comparisons (Miller *et al.*, 1995). A shifty site of GGGAAAC followed by a small, conserved pseudoknot has been found for BWYV (Garcia *et al.*, 1993) and predicted for RPV (Miller *et al.*, 1995). More recently, they found an additional sequence required for frameshifting by PAV. Remarkably, it is located four kilo bases downstream of the frameshift site (Wang S, Miller WA., 1995). This is higher than the very low level observed in reporter gene studies in oat cells using constructs that lacked the downstream region (Brault *et al.*, 1992). They have no idea of the mechanism, but this is only the first example of downstream elements controlling translation of PAV RNA.

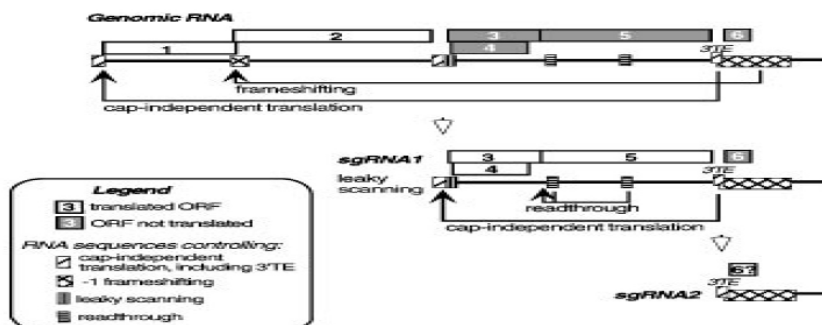


Figure 5. Map of *cis*-acting signals regulating PAV RNA translation (Miller *et al.*, 1997). **Bold line** indicates RNA on which boxes with different fill patterns demarcate the locations of sequences required for the indicated translational event. **Solid-headed arrows** indicated long-distance interactions. **Open-headed arrows** indicate

subgenomic RNA synthesis. sgRNA3 is not shown because it contains no ORFs and does not appear to be translated (Kelly *et al.*, 1994).

Readthrough:- As mentioned above, ORF 5 is expressed by readthrough of the coat protein gene stop codon during translation of sgRNA1 (Dinesh-Kumar *et al.*, 1992), i.e. when the ribosomes reach the stop codon, a small portion of them do not stop, but continue translating in the same frame 3' of the stop codon. The actual rate of readthrough is difficult to estimate because the ratio of CP-RTD fusion protein to CP in purified virions varies greatly (between 1:100 and 1:4) among BYDV serotypes (Filichkin *et al.*, 1994, Wang *et al.*, 1995) and even among individual virus preparations. In the most reliable cell-free wheat germ translation system, the rate was about one percent. The different researchers studied that *cis*-acting signals required for readthrough of the PAV CP ORF stop codon by translating an *in vitro* transcript of sgRNA1 containing various mutations (Brown *et al.*, 1996). In addition, these mutations were placed in a PAV cDNA clone such that resulting infectious transcripts contained a modified reporter gene (GUS) inserted in ORF 5 so that readthrough of the CP gene stop codon was required for GUS activity in oat protoplasts. Using these two assays, two regions 3' to the stop codon were identified as necessary for readthrough (Brown *et al.*, 1996). One is a repeated sequence motif: CCN NNN, located about 20 bases 3' to the stop codon. A second sequence, located 697 to 758 bases 30 of the stop codon, was also required (Figure 5). It occurs naturally within ORF 5, but functions well in the GUS-expressing virus, in which it is located two kilobases downstream of the CP stop codon and in the untranslated region following the GUS ORF. Highly conserved bases at and flanking the CP stop codon were not necessary. Deletions in and around the homologous regions in infectious clones of BWYV also destroyed or greatly reduced accumulation of RTD in infected plants (Bruyere *et al.*, 1997). In PEMV, a single, naturally occurring base change in the region homologous to the downstream readthrough element of PAV prevented readthrough and aphid transmissibility of the virus (Demler *et al.*, 1997). As in the case of frameshifting, they do not know the mechanism of readthrough. Long-distance base-pairing between the two required sequence elements can be imagined (Brown *et al.*, 1996) but this is not conserved among luteoviruses. Gibbs & Cooper provided evolutionary evidence that these two regions may interact during RNA replication (Gibbs MJ, Cooper JL., 1995). They proposed that strand-switching by the replicase facilitated recombination at these sites. Furthermore, Demler *et al.* (1997) found a natural PEMV deletion mutant in which a large region of the readthrough ORF, including portions of the proximal and distal elements and all the sequence between them, was deleted. This deletion could be explained by an intramolecular strand-switching event that would be favored if the two sequence elements were located in close proximity. If they are in proximity during replication, they could also interact during translation, and facilitate readthrough. The only type of readthrough control that remotely resembles this is that of selenocysteine-encoding genes. A sequence called the SECIS element in the 3'UTR, located kilobases downstream of a UGA (stop) codon, facilitates recognition of the UGA codon by a special tRNA charged with the amino acid selenocysteine (Walczak *et al.*, 1996). The luteovirus readthrough resembles this only in that the distal element functions at large, variable distances downstream. There is no structural similarity, nor is readthrough dependent on a UGA or any other particular stop codon (Brown *et al.*, 1996).

Cap-independent translation signal in the 3' untranslated region of PAV-RNA:- An unexpected finding was that a sequence we call the 3' translation enhancer (3'TE), located between ORFs 5 and 6, confers very efficient translation on mRNAs lacking the 5' cap structure that is normally required for translation of eukaryotic mRNAs (Figure 5) (Wang S, Miller WA., 1995). In order for the 3'TE to function, the mRNA must also contain the 5'UTR of either PAV genomic RNA or sgRNA1. This cap-independent translation has been observed in wheat germ translation extracts (Wang S, Miller WA., 1995) and in oat protoplasts (Wang *et al.*, 1997). Deletion or mutation of the 3'TE reduces translation of uncapped mRNA by more than an order of magnitude and renders the virus unable to replicate (Wang *et al.*, 1997). Addition of a cap restores translatability (Wang S, Miller WA., 1995). The only other known eukaryotic mRNA that has a cap-independent translation signal in its 3'UTR is satellite tobacco necrosis virus (STNV) RNA (Danthinne *et al.*, 1993, Timmer *et al.*, 1993). There is little or no sequence homology between STNV and PAV RNA. However, a portion of the 3'TE sequence is conserved in all subgroup I luteoviruses and in the 3'UTR of tobacco necrosis virus (TNV) RNA, the helper for STNV. Because TNV RNA is naturally uncapped, the sequence homologous to the 3'TE may facilitate translation initiation. As we would expect, PAV RNA also appears to lack a 5' cap (Wang *et al.*, 1997). This 3'TE phenomenon may be confined to subgroup I luteovirus and TNV RNAs. No sequence with homology to the 3'TE was detected in subgroup II luteoviruses, even though it is expected that subgroup II luteoviral genomes would also translate cap-independently because other VPg-containing genomes can do so (Carrington JC, Freed DD., 1990, Sarnow P., 1995).

2.19. 7. Satellite RNA

During the process of genome sequencing, a 322nt, single-stranded, noncoding satellite RNA (satRPV RNA)

was discovered serendipitously in an Australian isolate of RPV (Miller *et al.*, 1991). This mysterious RNA is difficult or impossible to detect in the field and has been found only in Australian RPV-like isolates only after greenhouse propagation. It has no significant sequence similarity to BYDV genomic RNA (Miller *et al.*, 1991) and it depends on RPV genomic RNA for replication (Silver *et al.*, 1994). SatRPV RNA is the only known satellite of a luteovirus, although a different class of satellite RNAs is associated with some luteo-like viruses (Demler *et al.*, 1996). SatRPV RNA replicates by a symmetrical rolling circle mechanism (Silver *et al.*, 1994). This resembles that of satRNA of tobacco ringspot virus (satTRSV RNA) (Bruening *et al.*, 1991) and several other viroid-like satellite RNAs (Bruening G., 1989). Linear and circular monomers and linear multimeric replication intermediates of both strands, which are formed during replication by this mechanism, were detected in satRPV RNA infected cells (Silver *et al.*, 1994). Newly formed multimers self-cleave into monomers in vitro at sequences that fold into hammerhead ribozyme structures, one in each strand (Miller *et al.*, 1991). The (+) strand hammerhead is an unusual derivation from the consensus structure. It has additional base-pairing that results in a pseudoknot that inhibits self-cleavage (Miller WA, Silver SL., 1991). This alternative conformation may serve as a molecular switch. The known range of helper viruses that support satRPV RNA is limited to subgroup II luteoviruses. RPV and BWYV support satRPV RNA replication (Rasochova *et al.*, 1997). PAV and a BWYV-associated RNA (ST9a RNA), which encodes a subgroup I-like polymerase, do not replicate satRPV RNA (Rasochova *et al.*, 1997, Silver *et al.*, 1994). SatRPV RNA can be transmitted to plants by aphids that acquired virus from infected protoplasts. It reduces accumulation of RPV helper virus RNA in oat plants and protoplasts, and ameliorates symptoms caused by RPV in oats (Rasochova *et al.*, 1996). SatRPV RNA had no effect on PAV RNA accumulation and did not affect symptoms caused by the severe mixed infection of RPV and PAV BYDVs in oats (Rasochova *et al.*, 1996) or by BWYV and ST9a RNA in shepherd's purse plants (Rasochova *et al.*, 1997). SatRPV RNA symptom modulation seems to be determined by the competition between the satRPV RNA and its helper virus for both replication and encapsidation. Because satRPV RNA can replicate and move systemically in monocotyledonous and dicotyledonous hosts, the helper virus (probably the replicase gene) and not the type of plant host range is the limiting factor for satRPV RNA replication (Rasochova *et al.*, 1997).

2.20. Relations with cells and tissues

Particles appear to be confined to the phloem (Jensen, 1969). Electron microscopy of leaves and roots of barley (*Hordeum vulgare* L.) infected with barley yellow dwarf virus (BYDV) has shown densely staining spherical particles, about 24 m μ in diameter, in certain phloem cells. *Luteoviruses* are tissue-specific and replicate in phloem cells of plant hosts. Virus particles are detected most readily in sieve elements, companion cells, and occasionally in phloem parenchyma. Particles were found only in phloem transfer cells of SCRLV-infected subterranean clover (Jayasena *et al.*, 1981). With one exception (Gill & Chong, 1981), *luteoviruses* have not been observed in epidermal, mesophyll or xylem tissues of infected plants. Evidence suggests that luteoviruses spread rapidly from cell to cell longitudinally through the vascular bundle; however, lateral spread is slow and not all phloem cells of a bundle may be infected (Jensen, 1969; Murrant & Roberts, 1979). The primary symptom of infection is necrosis of the phloem (Esau, 1957), which spreads from the initially inoculated sieve element to adjacent cells. Phloem necrosis is associated with resistance to translocation of plant metabolites, loss of chlorophyll, increased respiration, and decreased growth (Goodman *et al.*, 1965; Jensen, 1972; Orlob & Army, 1961). Ultrastructural examinations of infected plants have been reported for several *luteoviruses* (D'Arcy & De Zoeten, 1979; Esau & Hoefert, 1972; Faoro *et al.*, 1978; Jayasena *et al.*, 1981; Jensen, 1969; Kojima *et al.*, 1969; Kubo, 1981; Murrant & Roberts, 1979; Shepardson *et al.*, 1980). The most detailed study was that of Gill & Chong (1979) on BYDV isolates infecting oats, and showed that at least two distinct sequences of events may be involved in replication of viruses assigned to the *luteovirus* group. Based on cytopathological ultrastructure involving alterations of the nucleus, site of virus accumulation and types of vesicles produced, these authors suggested dividing BYDV isolates into subgroup 1 (MAV, PAV, SGV) and subgroup 2 (RPV, RMV). In oats infected with isolates of subgroup 1, virus particles seem to move from sites of initial inoculation in sieve elements through plasmodesmata into adjacent companion cells. Densely staining filaments (2-4 nm diameter) and single membrane-bound vesicles (50-230 nm diameter) form in the cytoplasm near the plasmodesmata. Filaments are later observed within the nucleus, which becomes distorted and begins to deteriorate soon after infection. Virus particles are first observed to occur in the cytoplasm, suggesting a cytoplasmic site for assembly. At about this time the mitochondria, plastids and ribosomes begin to disintegrate. Isolates in subgroup 2 initiate infection of companion cells in a similar manner; however, the vesicles produced are bounded by a second membrane which is continuous with the endoplasmic reticulum. A second type of membrane system (composed of tubules) proliferates, and the dense filaments are rare in the cytoplasm and do not occur in the nucleus. The nucleus does not deteriorate and virus particles are first observed surrounding the nucleolus within the nucleus. In addition, extensive wall thickenings develop in the infected parenchyma cells. Ultrastructural changes induced by BWYV and PLRV seem to be similar to those caused by BYDV isolates of subgroup 2 (Esau & Hoefert,

1972; Shepardson *et al.*, 1980). However, cytopathological effects vary with host species and virus isolates (D'Arcy & De Zoeten, 1979; Gill & Chong, 1981; Kojima & Yanase, 1984).

2.21. Management

BYD management is imperfect; complete control is rarely, if ever, achieved. Still, significant levels of BYD control can be achieved using multiple management tactics as part of an overall BYD management strategy. BYDV is controlled mainly by the use of plant lines that are tolerant or resistant, cultural practice, chemical control, biological control, and transgenic resistance to certain BYDV isolates to varying degrees. Spread of the disease can be controlled by aphicides (McKirby SJ, Jones RAC., 1996) or by carefully timed planting when aphid populations are monitored (Plumb RT., 1995). However, this is economically feasible only in the most intensive agricultural systems. Usually, especially in the developing world where disease pressure is high, growers simply live with losses to BYDV. Breeding for resistance, either by conventional or transgenic methods remains the most feasible means of disease control.

2.21.1. Cultural Practice

Cultural practices that could help reduce BYDVs incidence include changing sowing dates in order to avoid primary infection through viruliferous aphids, removal of cereal re- growths and stubble that can act as reservoirs of virus and vectors and the adoption of adequate cultivation methods (Plumb and Johnstone, 1995). In Australia, delaying sowing might be advisable in winter crops to minimize BYDV-induced grain yield losses. However, the yield benefits need to be balanced against possible yield reductions due to the late sowing (McKirby and Jones, 1997).

2.21.2. Chemical Control

As there is no chemical treatment effective against the virus, chemical control of BYDVs can only be achieved through control of its vectors. The critical time for control is at an early growth stage (Plumb and Johnstone, 1995). The need for aphid control can either be prophylactic or based on a forecasting system such as those described in Europe by Plumb *et al.* (1986) and Gillet *et al.* (1990). Mann *et al.* (1997) indicated that in England, UK, the spray regimes during spring are of little benefit and that sprays should be applied according to the time of aphid migration relative to crop development and the infectivity of the aphids migrating into the crop. The most commonly used aphicides are organophosphates or synthetic pyrethroids. Imidacloprid, an insecticidal seed treatment, reduced BYD infection under certain conditions (Gray *et al.*, 1996; McKirby and Jones, 1996). New generation synthetic pyrethroids (alpha-cypermethrin or beta-cyflurin) have been reported to be effective against BYDVs (McKirby and Jones, 1996).

2.21.3. Biological Control

Success with biological control has been reported from South America, where *Sitobion avenae* and *Metopolophium dirhodum*, were controlled through the introduction of *Coccinellid* predators and *Aphelinid* and *Aphidiid* parasites (Zuniga, 1990) and in New Zealand, with the introduction of *Aphidius rhopalosiph* (Farrell and Stufkens, 1990). In most areas, natural enemies limit aphid populations and it is important to integrate chemical and natural control methods.

2.21.4. Genetic Resistance

Incorporating resistance or tolerance (Cooper and Jones, 1983) to BYDVs or their vectors is one of the most promising approaches to control. Most of the screening for field 'resistance' to BYD has been directed to the identification of tolerance. In wheat, sources of tolerance have been reported by several researchers (Burnett *et al.*, 1995). Good tolerance has been reported in germplasm from South America (Ramirez, 1990). Tolerance in the variety Anza (Qualset *et al.*, 1984) has been associated with the presence of the gene Bdv1. It is a partially dominant, partially effective gene that induces slow yellowing (Singh *et al.*, 1993). It is probable that other genes are involved in tolerance to barley yellow dwarf. The winter wheat germplasm lines, Elmo and Caldwell were released as tolerant to BYDV (Ohm *et al.*, 1981; Patterson *et al.*, 1982). A decrease in virus multiplication has been reported from several wheat relatives, such as *Aegilops*, *Elymus*, *Elytrigia*, *Hordeum*, *Leymus* and *Thinopyrum* (Agropyron) (Sharma *et al.*, 1984, Larkin *et al.*, 1990; Makkouk *et al.*, 1994a; Xu *et al.*, 1994). Recently much effort has been directed toward incorporating these alien-derived resistances into wheat. Resistance is maintained in wheat x *Leymus* (Plourde *et al.*, 1992), wheat x wheatgrass (Sharma *et al.*, 1989; Goulart *et al.*, 1993), and wheat x *Agropyron* crosses (Comeau *et al.*, 1994). *Thinopyrum intermedium* has been widely used to produce resistant introgressed material such as the TC lines (Banks *et al.*, 1995b), Zhong 4 (Xin *et al.*, 1988) and Zhong 5-derived lines (Larkin *et al.*, 1995a). The 42 chromosome winter wheat line P29 and spring wheats TC5, TC6 and TC9 as well as the genetic stock Z1, Z2, Z6 with alien-derived resistance have been registered recently (Banks and Larkin, 1995; Larkin *et al.*, 1995b; Sharma *et al.*, 1997). In TC14, the alien

segment is located on 7DL (Hohmann *et al.*, 1996). Tolerance to BYDVs in barley was reported as early as 1961 (Bruehl, 1961; Rochow, 1961). Since then, several lines presenting tolerance to BYDVs have been reported. The resistance gene Yd2 (Rasmusson and Schaller, 1959) has been used extensively in barley breeding programmes and has been proven to be effective and stable over the years. Cultivars carrying this gene include Atlas 68, CM67 (Schaller and Chim, 1969), Shannon (Vertigan, 1979), Shyri (Vivar *et al.*, 1991), Vixen (Parry and Habgood, 1986), Nomini (Starling *et al.*, 1994) amongst others (Burnett *et al.*, 1995). Delogu *et al.* (1995) have incorporated the Yd2 gene to high yielding winter wheat. Another resistance gene, yd1, was identified in the cultivar Rojo (Suneson, 1955) but was rarely used in barley breeding programmes because of the low level of resistance it confers. The gene Yd2 operates by retarding virus multiplication (Jones and Catherall, 1970) and may sometimes lose its effect when placed in a slow growing background. Virus movement from the inoculated leaf towards the roots and subsequently to the growing point was significantly slower in the resistant than susceptible barley genotypes tested by Makkouk *et al.* (1994b) using ELISA and tissue-printing. Resistance and susceptible lines could be differentiated as early as 3-4 days after inoculation. Yd2 is very effective against the group I BYDVs (PAV, MAV) but only moderately effective against group II (RPV, RMV) (Skaria *et al.*, 1985; Herrera, 1989). In an ICARDA-CIMMYT programme, Yd2 was used extensively with other sources of resistance. Chalhouh *et al.* (1995) identified a Yd2 allele variant that does not originate from Ethiopia and that is overcome by one PAV isolate of BYDV. Yd2 is located close to the centromere of the long arm of chromosome 3 of barley (Collins *et al.*, 1996). A polypeptide marker of BYDVs resistance identified by Holloway and Heath (1992) can be used as a marker for Yd2. Effort is underway to clone the Yd2 gene. Sources of tolerance to BYDVs exist in oat but no true resistance has been reported. However, some tolerant lines significantly reduce virus multiplication (Gray *et al.*, 1993) and could qualify as resistant. Many researchers have shown that the tolerance is heritable (Cooper and Sorrels, 1983; Gellner and Sechler, 1986) and that two to four genes contribute to the tolerance (Landry *et al.*, 1984; McKenzie *et al.*, 1985). It appears that released varieties have shown both a good level of protection over a wide range of field conditions and stability over broad geographic areas (Burnett *et al.*, 1995). Selected oat cultivars tolerant to BYD include Otee (Brown and Jedlinsky, 1973); Ogle (Brown and Jedlinsky, 1983) and Hazel (Brown and Kolb, 1989) as the most notable (Burnett *et al.*, 1995). Three major quantitative loci for tolerance to BYDV-PAV have been identified by Jin *et al.* (1998) to contribute 25, 20 or 17% of the variability. In addition, good sources of tolerance to BYDVs have been found in other oat species, including *Avena sterilis*, *A. fatua* and *A. strigosa* (Rines *et al.*, 1980; Comeau, 1982, 1984; Jedlinski, 1984). Good sources of tolerance have been found in triticale (Collin *et al.*, 1990; Burnett and Mezzalama, 1992; Comeau and St-Pierre, 1992). The tolerance has been incorporated into wheat x triticale hybrids (Nkongolo, 1996).

2.21.5. Transgenic Resistance

McGrath *et al.* (1997) transformed oat with the coat protein (CP) genes of BYDV-PAV, BYDV-MAV and BYDV-RPV together with a construct containing the bar gene for herbicide resistance and the uidA reporter gene. Plants with reduced virus titers were found in the T2 (MAV), T3 (RPV) and T4 (PAV) generations. Using the same construct, a few barley plants transformed with CP-PAV showed moderate to high levels of resistance against BYDV-PAV. The most cost-effective and environmentally desirable form of disease control is genetic resistance. However, natural resistance genes to BYDV are few (Burnett *et al.*, 1995). Yet, because of the threat of BYDV, breeders are limited to lines that have significant natural BYDV resistance or tolerance. To allow breeders to expand beyond these limitations, they developed transgenic oats tolerant to BYDV (Koev *et al.*, 1998). Oats were transformed with a gene designed to express the 5' half of the BYDV genome driven by a CaMV 35S promoter. The most resistant line of transgenic oats initially showed mild symptoms but then recovered and grew to maturity. In laboratory growth conditions, yield was slightly reduced compared to uninoculated controls and virus was sometimes detectable, but the yield was infinitely greater than in the inoculated non-transgenic controls which were actually killed by virus infection long before flowering. The transgene was stably inherited in a Mendelian fashion. Field trials were less promising, mainly because the only line of oats that could be transformed (genetically engineered) at the time was not agronomically useful (Somers *et al.*, 1992). It was derived by artificial hybridization of *Avena fatua* with the Park cultivar of oat (*A. sativa*), followed by back crosses to oat. These plants (with or without a transgene) were smaller and less robust than agronomic cultivars, in the presence or absence of virus infection. Fortunately, recent improvements in technology now permit transformation of such agronomic lines as Bell. Peter Waterhouse and colleagues greatly improved design of transgenes to engineer resistant plants with high efficiency. Barley plants transformed with inverted sequences of BYDV genes, causing the transcripts to form long, double-stranded hairpin RNAs were immune to BYDV infection (Wang *et al.* 2000). Presumably the double-stranded RNA induces the host's post-transcriptional gene silencing system (Waterhouse *et al.*, 2001). To the best of our knowledge, no plants engineered for BYDV resistance have been released for use by growers. Unfortunately, due to the low value of oats as a profit-making enterprise, and perhaps due to concern about consumer acceptance of food derived from

GMO crops, corporate interest in funding transgenic oat research has waned. Perceived risks imposed by transgenic BYDV-resistant oats drew attention in a *Science* magazine article about an unpublished poster presentation at a scientific conference (Kaiser, 2001). The experiments alleging that pollen escape from transgenic BYDV-resistant oats could lead to “super weeds” were confined to the greenhouse and used no transgenic plants. Yet the benefits of new resistance genes, such as reduced pesticide inputs and increased yields, are clear (Miller *et al.*, 1997). Application of insecticides on wheat to control the aphid vectors of BYDV often results in substantial yield increases (Plumb and Johnstone, 1995) that are attributable to the absence of BYDV infection. One legitimate concern with regard to the applicability (but not safety) of virus-derived transgenes for resistance to BYDV is the wide range of sequence variation among isolates. BYDV isolates that lack high homology to the transgene would not be hindered by transgene-induced post-transcriptional gene silencing (Miller *et al.*, 1997; Wang *et al.*, 2000; Waterhouse *et al.*, 2001). Thus, virus-derived transgenes may confer resistance to only a subset of BYDV isolates in the field.

3. CONCLUDING REMARK

Barley yellow dwarf virus (BYDV) is distributed worldwide, and infects most cereals and grasses. It is a phloem-restricted pathogen, causing yellowing, reddening, and brittleness of leaves, dwarfing, and reduction in size and number of ears and grains. BYDV is a luteovirus with small isometric particles containing an ssRNA genome, and is transmitted persistently by more than 20 aphid species. Five virus isolates have been distinguished and divided into two subgroups on the basis of cytopathology and serology. Recent serological evidence also indicates that BYDV isolates are related to other luteoviruses, suggesting that a continuous, overlapping range of viruses may be implicated in the barley yellow dwarf syndrome. Until future research clarifies this point, the term BYDV continues to be used to indicate the agent(s) involved. Perennial wild or cultivated grasses constitute a large and permanent virus pool. Primary and secondary virus spread depends on the aphid vector reproduction and flight which, in turn, are influenced by climatic conditions. Recent research on monitoring and control of aphid vectors and on development of resistant cereal cultivars has improved the prospect of minimizing losses from BYDV infections. Because of the economic importance of the BYDVs, more research is needed. The specific locations and timing of virus outbreaks, and the particular causal isolates, need to be monitored. This will allow breeders to decide which BYDV isolate to target with transgenic resistance in a given locality. It will help growers decide whether to pay the extra premium for BYDV-resistant crops. Another area of applied research may be to engineer aphid-resistant crops. With the growing number of sequenced or partially sequenced isolates of BYDV and CYDV around the world, it's important 1) to develop rapid means of nucleic acid-based detection (e.g., PCR), 2) to understand the epidemiology of BYDV/CYDV, and 3) to develop transgenic and other means of disease control. The better understanding of BYDV molecular mechanisms that ultimately lead to new means of controlling or mitigating the effects of the disease, and it sheds light on processes relevant to medically important viruses.

In general, the developing world needs one of the most pressing reasons for comprehensive surveys of viruses affecting plants in the crop-growing areas to be aware of factors limiting crop yields. Although in many instances virus infections are suspected, these infections are never satisfactorily identified, and in many other cases it is not known whether crops are infected, let alone what effect the infection has on yield. Without a thorough understanding of the incidence and variety of virus infections in food crops in the developing world, it is hard to plan for improved crop yields. However, developing countries are often unable to do such surveys on their own due to lack of expertise and resources. Given the lack of respect of borders by plant (or any other) viruses, it is hoped that the more developed neighboring countries will assist those less well endowed in these endeavors. Another more future-oriented reason for surveys of viruses is the need to understand virus diversity in order to be able to combat virus diseases by genetic engineering of crop plants. Although there are many examples of crop plants transformed with viral genes or sequences exhibiting wide-spectrum resistance or tolerance to virus infection there are also many examples of an unexpected diversity of a given virus type being discovered within the crop in the same area or even within the same plants. Thus, it is quite possible that the use of transgenic material in areas where the virus diversity is not known could still result in crop failures. Moreover, the different viruses and transmission mechanisms that cause severe plant disease in plants in developing countries are that the main problem is often one of insect/vector control. If the vector populations or the interaction of vector populations with crop plants could be controlled or managed better, the incidence of severe disease could be drastically reduced. As a final note, it is well to realize that there are more direct threats to human health inherent in changing farming practices than simply a reduction in the amount of food being produced due to plant virus infections. In addition, further review is needed to identify all recovered BYDV and evaluation of promising treatments for use in integrated disease management strategy to manage not only BYDV but also other related viral diseases of plant.

4. Acknowledgements

The author is thankful to the department of horticulture and plant science of Jimma University, for allowing the

conveniences to review the BYDV.

5. REFERENCES

- Aapola, 1968. Serological relationships and in vivo interactions among isolates of barley yellow dwarf virus. Ph.D. Thesis, Cornell Univ.
- Anderson, E. J., Qui, S. G. & Schoelz, J. E., 1991. Genetic analysis of determinants of disease severity and virus concentration in cauliflower mosaic virus. *Virology* 181, 647-655.
- Baltenberger DE, Ohm HW, Foster JE., 1987. Reactions of oat, barley, and wheat to infection with barley yellow dwarf isolates. *Crop Sci.* 27:195-98.
- Banks PM, Larkin PJ, Bariana HS, Lagudah ES, Appels R, Waterhouse PM, Brettell RIS, Chen X, Xu HJ, Xin ZY, Qian YT, Zhou XM, Cheng ZM, Zhou GH., 1995. The use of cell culture for subchromosomal introgressions of barley yellow dwarf virus resistance from *Thinopyrum intermedium* to wheat. *Genome*, 38(2):395-405.
- Banks PM, Larkin PJ, Bariana HS, Lagudah ES, Appels R, *et al.*, 1995. The use of cell culture for subchromosomal introgressions of barley yellow dwarf virus resistance from *Thinopyrum intermedium* to wheat. *Genome* 38:395-405.
- Banks PM, Larkin PJ, 1995b. Registration of three BYDV - resistant wheat germplasms - TC5. *Crop Science*, 35:600-601.
- Bencharhi, B., Mutterer, J., El Yamani, M., Ziegler-Graff, V., Zaoui, D. & Jonard, G., 1999. Severity of infection of Moroccan barley yellow dwarf virus PAV isolates correlates with variability in their coat protein sequences. *Ann Appl Biol* 134, 89-99.
- Bowden, R.L., 2000. Barley yellow dwarf. Agricultural experimental station and cooperative extension service, Kansas State University, Manhattan.
- BrautV, MillerWA., 1992. Translational frameshifting mediated by a viral sequence in plant cells. *Proc. Natl. Acad. Sci. USA* 89:2262-66.
- BrautV, Van den Heuvel JFJM, Verbeek M, Ziegler-Graff V, Reutenauer A, *et al.*, 1995. Aphid transmission of beet western yellows luteovirus requires the minor capsid read-through protein P74. *EMBO J.* 14:650-59.
- Brown CM, Dinesh-Kumar SP, Miller WA., 1996. Local and distant sequences are required for efficient read-through of the barley yellow dwarf virus-PAV coat protein gene stop codon. *J. Virol.* 70:5884-92.
- Brown CM, Jedlinski H, 1973. Control of Barley Yellow Dwarf Virus (BYDV) in Oat and Wheat. Registration of Otee oats. *Crop Science*, 13:397.
- Brown CM, Kolb FL, 1989. Control of Barley Yellow Dwarf Virus (BYDV) in Oat and Wheat. Registration of 'Hazel' oat. *Crop Science*, 29:1573.
- Bruehl, 1961. Barely yellow dwarf, A virus disease of cereals and grasses. *Monogr. Am. phytopath. Soc.* 1.
- Bruening G., 1989. Compilation of selfcleaving sequences from plant virus satellite RNAs and other sources. *Methods Enzymol.* 180:546-58.
- Bruening G, Passmore BK, van Tol H, Buzayan JM, Feldstein PA., 1991. Replication of plant virus satellite RNA: Evidence favors transcription of circular templates of both polarities. *Mol. Plant- Microbe Interact.* 4:219-25.
- Bruyere A, BrautV, Ziegler-GraffV, Simonis M-T, van den Heuvel JFJM, *et al.*, 1997. Effects of mutations in the beet western yellows virus readthrough protein on its expression and packaging, and on virus accumulation, symptoms and aphid transmission. *Virology* 230:323- 34
- Burnett PA, Comeau A, Qualset CO, 1995. Host plant tolerance or resistance for control of barley yellow dwarf. In: D'Arcy CJ, Burnett PA, eds. *Barley Yellow Dwarf: 40 Years of Progress*. St. Paul, USA: APS Press, 321-343.
- Burnett PA, Mezzalama M, 1992. The Centro Internacional de Mejoramiento de Maíz y Trigo (CIMMYT) barley yellow dwarf program. Barley yellow dwarf in West Asia and North Africa. Proceedings of a workshop held at Rabat, Morocco, 19-21 November 1989., 33-45; 7 ref.
- Canning ESG, Penrose MJ, Barker I, Coates D, 1996. Improved detection of barley yellow dwarf virus in single aphids using RT-PCR. *Journal of Virological Methods*, 56:191-197.
- Carrigan, L. L., H. W. Ohm, J. E. Foster, and F. L. Patterson., 1981. Response of winter wheat cultivars to barley yellow dwarf virus infection. *Crop Sci.* 21: 377-380.
- Carrington JC, Freed DD., 1990. Capindependent enhancement of translation by a plant potyvirus 50 nontranslated region. *J. Virol.* 64:1590-97.
- Catherall PL, Parry AL, 1987. Effects of barley yellow dwarf virus on some varieties of Italian, hybrid and perennial ryegrasses and their implication for grass breeders. *Plant Pathology*, 36(2):148-153.
- Chay CA, Gunasinge UB, Dinesh- Kumar SP, Miller WA, Gray SM., 1996. Aphid transmission and systemic plant infection determinants of barley yellow dwarf luteovirus-PAV are contained in the coat protein readthrough domain and 17-kDa protein, respectively. *Virology* 219:57-65

- Chay CA, Smith DM, Vaughan R, Gray SM, (1996a). Diversity among isolates within the PAV serotype of barley yellow dwarf virus. *Phytopathology*, 86:370-77.
- Chay, C., D.M. Smith, R. Vaughan, and S.M. Gray., (1996b). Diversity among isolates within the PAV serotype of barley yellow dwarf virus. *Phytopathology* 86:370- 377.
- Chaloub BA, Kelly L, Robaglia C, Lapierre HD., 1994. Sequence variability in the genome-30-terminal region for 10 geographically distinct PAV-like isolates of barley yellow dwarf virus: analysis of the ORF6 variation. *Arch. Virol.* 139:403–16
- Chalhoub BA, Sarrafi A, Lapierre HD, 1995. Partial resistance in the barley (*Hordeum vulgare* L.) cultivar 'Chikurin Ibaraki 1' to two PAV-like isolates of barley yellow-dwarf virus: allelic variability at the Yd2 gene locus. *Plant Breeding*, 114(4):303-307; 16 ref.
- Cheng SL, Domier LL, D'Arcy CJ., 1994. Detection of the readthrough protein of barley yellow dwarf virus. *Virology* 202:1003–6
- Cisar, G., C. M. Brown, and H. Jedlinski., 1982. Effect of fall or spring infection and sources of tolerance of barley yellow dwarf of winter wheat. *Crop Sci.* 22: 474-478.
- Clark MF, Adams AN, 1977. Characteristics of the microplate method of enzyme-linked immunosorbent assay for the detection of plant viruses. *Journal of General Virology*, 34(3):475-483.
- Collin J, St-Pierre CA, Comeau A, 1990. Analysis of genetic resistance to barley yellow dwarf virus in triticale and evaluation of various estimators of resistance. In: Burnett PA, ed. *World Perspectives on Barley Yellow Dwarf*. Mexico: CIMMYT, 404-414.
- Collins NC, Paltridge NG, Ford CM, Symons RH, 1996. The Yd2 gene for barley yellow dwarf virus resistance maps close to the centromere on the long arm of barley chromosome 3. *Theoretical and Applied Genetics*, 92:858-864.
- Comeau A, 1982. Geographic distribution of resistance to barley yellow dwarf virus in *Avena sterilis*. *Canadian Journal of Plant Pathology*, 4(2):147-151.
- Comeau A, 1984. Barley yellow dwarf virus resistance in the genus *Avena*. *Euphytica*, 33(1):49-55.
- Comeau A, 1987. Effects of BYDV inoculations at various dates in oats, barley, wheat, rye and triticale. *Phytoprotection*, 68(2):97-109.
- Comeau A, Makkouk KM, Ahmad F, StPierre CA, 1994. Bread wheat x agroticum crosses as a source of immunity and resistance to the PAV strain of barley yellow dwarf luteovirus. *Agronomie*, 14(3):153-160; 3 ref.
- Comeau A, Makkouk KM, Ahmad F, StPierre CA, 1994. Bread wheat x agroticum crosses as a source of immunity and resistance to the PAV strain of barley yellow dwarf luteovirus. *Agronomie*, 14(3):153-160.
- Cook, J. R., and R. J. Veseth., 1991. *Wheat Health Management*. pgs. The American Phytopathological Society, St. Paul, MN.
- Cooper JI, Jones AT, 1983. Responses of plants to viruses: proposals for the use of terms. *Phytopathology*, 73(2):127-128.
- Cooper DC, Sorrells ME, 1983. Greenhouse screening and field evaluation of two oat populations segregating for barley yellow dwarf virus. *Cereal Research Communication*, 11:099-105.
- Creamer R, Falk BW, 1989. Characterization of a nonspecifically aphid-transmitted CA-RPV isolate of barley yellow dwarf virus. *Phytopathology*, 79(9):942-946; 32 ref.
- Creamer R, Falk BW., 1990. Direct detection of transcapsidated barley yellow dwarf luteoviruses in doubly infected plants. *J. Gen. Virol.* 71:211–17
- Danthinne X, Seurinck J, Meulewaeter F, van Montagu M, Cornelissen M., 1993. The 30 untranslated region of satellite tobacco necrosis virus RNA stimulates translation *in vitro*. *Mol. Cell Biol.* 13:3340–49.
- D'Arcy and De Zoeten, 1979. Beet western yellow virus in phloem tissue of *Thalspi arvensis* *Phytopathology* 69: 1194.
- D'Arcy CJ, Murphy JF, Miklasz SD, 1990. Murine monoclonal antibodies produced against two Illinois strains of barley yellow dwarf virus: production and use for virus detection. *Phytopathology*, 80(4):377-381.
- D'Arcy, C. J., 1995. Symptomology and host range of barley yellow dwarf. Pp. 9-28. In C. J. D'Arcy and P. A. Burnett (eds.), *Barley Yellow Dwarf: 40 Years of Progress*. The American Phytopathological Society, St. Paul, MN.
- D'Arcy, C.J., and P.A. Burnett ., 1995. *Barley Yellow Dwarf: 40 Years of Progress*. APS Press, St. Paul.
- D'Arcy, C.J., L.L. Domier, and M.A. Mayo., 2000. Family Luteoviridae. In: M.H.V. van Regenmortel, C.M. Fauquest, D.H.L. Bishop, E.B. Carstens, M.K. Estes, S.M. Lemon, J. Maniloff, M.A. Mayo, D.J. McGeoch, C.R. Pringle, and R.B. Wickner (eds.). *Virus Taxonomy: Seventh Report of the International Committee on the Taxonomy of Viruses*. Academic Press, San Diego. pp. 775-784.
- Delogu G, Cattivelli L, Snidaro M, Stanca AM, 1995. The Yd2 gene and enhanced resistance to barley yellow dwarf virus (BYDV) in winter barley. *Plant Breeding*, 114:417-420.
- Demler SA, de Zoeten GA., 1991. The nucleotide sequence and luteovirus-like nature of RNA 1 of an aphid nontransmissible strain of pea enation mosaic virus. *J. Gen. Virol.* 72:1819–34.

- Demler SA, Rucker DG, de Zoeten GA., 1993. The chimeric nature of the genome of pea enation mosaic virus: the independent replication of RNA 2. *J. Gen. Virol.* 74:1–14
- Demler SA, de Zoeten GA, Adam G, Harris KF., 1996. Pea enation mosaic enamovirus: properties and aphid transmission. In *The Plant Viruses*, Vol. 5: *Polyhedral Virions and Bipartite RNA Genomes*, ed. BD Harrison, AF Murant, pp. 303–44. New York: Plenum.
- Demler SA, Rucker-Feeney DG, Skaf JS, de Zoeten GA., 1997. Expression and suppression of circulative aphid transBYDV 187 mission in pea enation mosaic virus. *J. Gen. Virol.* 78:511–23
- Diacio R, Lister RM, Hill JH, Durand DP, 1986. Demonstration of serological relationships among isolates of barley yellow dwarf virus by using polyclonal and monoclonal antibodies. *Journal of General Virology*, 67:353-362.
- Di R, Dinesh-Kumar SP, Miller WA., 1993. Translational frameshifting by barley yellow dwarf virus RNA (PAV serotype) in *Escherichia coli* and in eukaryotic cell-free extracts. *Mol. Plant- Microbe Interact.* 6:444–52
- Dinesh-Kumar SP, Brault V, MillerWA., 1992. Precise mapping and *in vitro* translation of a trifunctional subgenomic RNA of barley yellow dwarf virus. *Virology* 187:711–22.
- Dinesh-Kumar SP, Miller WA., 1993. Control of start codon choice on a plant viral RNA encoding overlapping genes. *Plant Cell* 5:679–92
- Domier LL, 1995. Genome structure and function of barley yellow dwarf viruses. In: D'Arcy CJ, Burnett PA, eds. *Barley Yellow Dwarf - 40 Years of Progress*. St. Paul, USA: APS Press, 181-201.
- Eagling DR, Cunningham PJ, Sward RJ, Halloran GM, 1989. The incidence of barley yellow dwarf virus isolates in perennial ryegrass (*Lolium perenne*) in south-western Victoria. *Plant Pathology*, 38(3):408-413.
- Esau & Hoefert, 1972. Development of infection with beet western yellows virus in the sugar beet. *Virology* 48:724.
- Eweida M, Oxelfelt P, 1985. Purification and properties of two Swedish isolates of barley yellow dwarf virus and their serological relationships with American isolates. *Annals of Applied Biology*, 106(3):475-482.
- Faoro, Amici and Tornaghi, J., 1978. Barely yellow dwarf virus. *Submicroscop. Cytol.* 10: 126.
- Farabaugh PJ., 1996. Programmed translational frameshifting. *Microbiol. Rev.* 60:103–34.
- Farrell JA, Stufkens MW, 1990. The impact of *Aphidius rhopalosiphi* (Hymenoptera: Aphidiidae) on populations of the rose grain aphid (*Metopolophium dirhodum*) (Hemiptera: Aphididae) on cereals in Canterbury, New Zealand. *Bulletin of Entomological Research*, 80(4):377-383.
- Fattouh, F. A., UENG. P. P, KAWATA, E. E., BARBARA, B. A., LARKINS, B. A. and LISTER, R. M., (1990). Luteovirus relationships assessed by cDNA clone from barely yellow dwarf viruses. *Phytopathology* 80,913-920.
- Filichkin SA, Lister RM, McGrath PF, Young MJ., 1994. *In vivo* expression and mutational analysis of the barley yellow dwarf virus readthrough gene. *Virology* 205:290-99.
- French R, 1997. Rapid identification of barley yellow dwarf virus strains by RT-PCR and solution hybridization. *Phytopathology*, 87:S31.
- Forde SMD, 1989. Strain differentiation of barley yellow dwarf virus isolates using specific monoclonal antibodies in immunosorbent electron microscopy. *Journal of Virological Methods*, 23(3):313-319.
- Ford C, Collins N, Rathjen J, Shams- Bakhsh M, Paltridge N, Symons RH., 1996. *Barley Yd2, a naturally occurring gene that provides resistance against barley yellow dwarf virus*. Presented at Int. Congr. Virol., Jerusalem, 10th, p. 185 (Abstr.).
- Garcia A, van Duin J, Pleij CW., 1993. Differential response to frameshift signals in eukaryotic and prokaryotic translational systems. *Nucleic Acids Res.* 21:401–6.
- Gerlach, Miller and Waterhouse, 1987. *Barley Yellow Dwarf Virus. News*, 1:17.
- Gibbs M., 1995. The luteovirus supergroup: rampant recombination and persistent partnerships. In *Molecular Basis of Virus Evolution*, ed. AJ Gibbs, CH Calisher, F Garcia-Arenal, pp. 351-68. Cambridge: Cambridge Univ. Press.
- Gibbs MJ, Cooper JL., 1995. A recombinational event in the history of luteoviruses probably induced by base-pairing between the genomes of two distinct viruses. *Virology* 206:1129-32.
- Gildow FE, Ballinger ME, RochowWF., 1983. Identification of double-stranded RNAs associated with barley yellow dwarf virus infection of oats. *Phytopathology* 73:1570–72
- Gildow, F. E., 1987. Virus-membrane interactions involved in circulative transmission of luteoviruses by aphids. *Adv Dis Vect Res* 4, 93-120.
- Gildow, F. E., 1993. Evidence for receptor-mediated endocytosis regulating luteovirus acquisition by aphids. *Phytopathology* 83, 270-277.
- Gillet H, Dedryver CA, Robert Y, Gamon A, Pierre JS, 1990. Assessing the risk of primary infection of cereals by barley yellow dwarf virus in autumn in the Rennes basin of France. *Annals of Applied Biology*, 117:237-251.
- Gill and Chong, 1981. Differences in cellular ultra structural alterations between variants of barley yellow dwarf virus. *Virology* 114: 405.

- Gill and Chong, 1979. Differences in cellular ultra structural alterations between variants of barley yellow dwarf virus. *Virology* 95: 59.
- Gellner JL, Sechler DT, 1986. Generation mean analysis of inheritance of resistance to barley yellow dwarf in crosses involving Bates spring oats. *Plant Disease*, 70(8):795-797.
- Goodman, Watson and Hill, 1965. Sugar and fructosan accumulation in virus-infected plants: rapid testing by circular-paper chromatography. *Ann. appl. Biol.* 56: 65.
- Goulart LR, Mackenzie SA, Ohm HW, Lister RM, 1993. Barley yellow dwarf virus resistance in a wheat cross wheatgrass population. *Crop Science*, 33(3):595-599.
- Goulart LR, Mackenzie SA, Ohm HW, Lister RM, 1993. Barley yellow dwarf virus resistance in wheat cross wheatgrass population. *Crop Science*, 33(3):595-599; 16.
- Gray, S. M., A. G. Power, D. M. Smith, A. J. Seaman, and N. S. Altman., 1991. Aphid transmission of barley yellow dwarf virus: Acquisition access periods and virus concentration requirements. *Phytopathology*. 81: 535-545.
- Gray S.M, Smith D, Altman N, 1993. Barley yellow dwarf virus isolate-specific resistance in spring oats reduced virus accumulation and aphid transmission. *Phytopathology*, 83(7):716-720.
- Gray S.M, Bergstrom GC, Vaughan R, Smith DM, Kalb DW, 1996. Insecticidal control of cereal aphids and its impact on the epidemiology of the barley yellow dwarf luteoviruses. *Crop Protection*, 15:687-697.
- Guy P.L, Johnstone G.R, Duffus J.E, 1986. Occurrence and identity of barley yellow dwarf viruses in Tasmanian pasture grasses. *Australian Journal of Agricultural Research*, 37(1):43-53.
- Guy P.L., 1988. Pasture ecology of barley yellow dwarf viruses at Sandford, Tasmania. *Plant Pathology*, 37(43):546-550.
- Habili N, Symons R.H., 1989. Evolutionary relationship between luteoviruses and other RNA plant viruses based on sequence motifs in their putative RNA polymerases and nucleic acid helicases. *Nucleic Acids Res.* 17:9543-56
- Halbert S.E, Connelly B.J, Lister R.M, Klein RE, Bishop G.W, 1992. Vector specificity of barley yellow dwarf virus serotypes and variants in southwestern Idaho. *Annals of Applied Biology*, 121(1):123-132.
- Hebert, 1963. Barely yellow dwarf virus. *Phytopathology* 53: 362.
- Heagy and Rochow, 1965. Barely yellow dwarf virus. *Phytopathology* 55: 809.
- Henry M, Dedryver C.A., 1991. Occurrence of barley yellow dwarf virus in pastures of western France. *Plant Pathology*, 40(1):93-99.
- Herbert Jr., D. A., E. L. Stromberg, G. F. Chappell, and S. M. Malone., 1999. Reduction of yield components by barley yellow dwarf infection in susceptible winter wheat and winter barley in Virginia. *J. Prod. Agric.* 12: 105-109.
- Herrera GF, 1989. Interactions between host plants and British isolates of barley yellow dwarf virus. PhD Thesis. London, UK: University of London.
- Heuvel JFJM van den, Verbeek M, Wilk F van der, 1994. Endosymbiotic bacteria associated with circulative transmission of potato leafroll virus by *Myzus persicae*. *Journal of General Virology*, 75(10):2259-2565.
- Hewings AD, Eastman CE., 1995. Epidemiology of barley yellow dwarf in North America. See Ref. 24, pp. 75-106.
- Hoffman TK, Kolb FL, 1997. Effects of barley yellow dwarf virus on root and shoot growth of winter wheat seedlings grown in aeroponic culture. *Plant Disease*, 81:497-500.
- Hohmann U, Badaeva K, Busch W, Friebe B, Gill BS, 1996. Molecular cytogenetic analysis of Agropyron chromatin specifying resistance to barley yellow dwarf virus in wheat. *Genome*, 39:336-347.
- Holloway PJ, Heath R, 1992. Identification of polypeptide markers of barley yellow dwarf virus resistance and susceptibility genes in non-infected barley (*Hordeum vulgare*) plants. *Theoretical and Applied Genetics*, 85(2-3):346-352.
- Hsu HT, Aebig J, Rochow WF, 1984. Differences among monoclonal antibodies to barley yellow dwarf viruses. *Phytopathology*, 74(5):600-605.
- Jayasena, Hatta, Francki and Randles, Gen J., 1981. Subterranean clover red leaf *luteovirus*. *Virol.* 57:205.
- Jedlinski H, 1984. The genetics of resistance to barley yellow dwarf virus in oats. In: *Barley Yellow Dwarf. Proceedings of the Workshop*, 101-105.
- Jensen, 1969. Occurrence of virus particles in the phloem tissue of BYDV infected barley. *Virology* 38: 83.
- Jensen, 1972. Occurrence of virus particles in the phloem tissue of BYDV infected barley. *Phytopathology* 62: 587.
- Jin H, Domier LL, Kolb FL, Brown CM, 1988. Identification of quantitative loci for tolerance to barley yellow dwarf virus in oat. *Phytopathology*, 88:410-415.
- Jones AT, Catherall PL, 1970. The relationship between growth rate and the expression of the tolerance to barley yellow dwarf virus in barley. *Annals of Applied Biology* 65:137-145.
- Kaiser, J., 2001. Breeding a hardier weed. *Science* 293:1425-1427.

- Kelly L, Gerlach WL, Waterhouse PM., 1994. Characterisation of the subgenomic RNAs of an Australian isolate of barley yellow dwarf luteovirus. *Virology* 202:565-73.
- Keese, P., Martin, R. R., Kawchuk, L. M., Waterhouse, P. M., Gerlach, W. L., 1990. Nucleotide sequences of an Australian and a Canadian isolate of potato leafroll luteovirus and their relationships with two European isolates. *J. Gen. Virol.* 71:719-24.
- Keese PK, Gibbs A., 1992. Origins of genes: "big bang" or continuous creation? *Proc. Natl. Acad. Sci. USA* 89: 9489-93.
- Kojima, Shikata, Sugawara and Murayama, 1969. Physical and chemical properties of Japanese isolate of barley yellow dwarf virus. *Virology* 39: 162.
- Kojima and Yanase, 1984. Physical and chemical properties of Japanese isolate of barley yellow dwarf virus. *Bull. Fac. Agric. Niigata Univ.* 39:11.
- Kim KH, Lommel SA., 1994. Identification and analysis of the site of ψ 1 ribosomal frameshifting in red clover necrotic mosaic virus. *Virology* 200:574-82
- Koonin EV, Dolja VV., 1993. Evolution and taxonomy of positive-strand RNA viruses: implications of comparative analysis of amino acid sequences. *Crit. Rev. Biochem. Mol. Biol.* 28:375-430.
- Koev, G., B.R. Mohan, S.P. Dinesh-Kumar, K.A. Torbert, D.A. Somers, and W.A. Miller., 1998. Extreme reduction of disease in oats transformed with the 5' half of the barley yellow dwarf virus-PAV genome. *Phytopathology* 88:1013-1019.
- Koev, G., S. Liu, R. Beckett, and W.A. Miller., 2002. The 3'-terminal structure required for replication of barley yellow dwarf virus RNA contains an embedded 3' end. *Virology* 292:114-126.
- Kubo, 1981. Barley yellow dwarf virus (BYDV) encompasses a group of variously several luteoviruses. *CMI/AAB Descr. Pl. Viruses* 234, 4 pp.
- Landry B, Comeau A, Minvielle F, St-Pierre C-A, 1984. Genetic analysis of resistance to barley yellow dwarf virus in hybrids between *Avena sativa* 'Lamar' and virus-resistant lines of *Avena sterilis*. *Crop Science*, 24(2):337-340.
- Larkin PJ, Brettell RIS, Banks P, Appels R, Waterhouse PM, Cheng ZM, Zhou GH, Xin ZY, Chen X, 1990. Identification, characterization and utilization of sources of resistance to barley yellow dwarf virus. In: Burnett PA, ed. *Barley Yellow Dwarf*. Mexico: CIMMYT, 415-420.
- Larkin PJ, Banks PM, Lagudah ES, Appels R, Xiao C, *et al.*, 1995. Disomic *Thinopyrum intermedium* addition lines in wheat with barley yellow dwarf virus resistance and with rust resistances. *Genome* 38:385-94.
- Larkin PJ, Banks PM, Lagudah ES, Appels R, Xiao Chen, Zhiyong Xin, Ohm HW, McIntosh RA, 1995a. Disomic *Thinopyrum intermedium* addition lines in wheat with barley yellow dwarf virus resistance and with rust resistances. *Genome*, 38:385-394.
- Larkin PJ, Banks PM, Xiao C. (1995b). Registration of six genetic stocks of wheat with rust and BYDV resistance - Z1, Z2, Z3, Z4, Z5 and Z6 disomic addition lines with *Thinopyrum intermedium* chromosomes. *Crop Science* 35:604.
- Lister RM, Rochow WF, 1979. Detection of barley yellow dwarf virus by enzyme-linked immunosorbent assay. *Phytopathology*, 69(6):649-654.
- Lister RM, Sward RJ., 1988. Anomalies in serological and vector relationships of MAV-like isolates of barley yellow dwarf virus from Australia and the U.S.A. *Phytopathology* 78:766-70
- Lister RM, Clement D, Skaria M, McFatrige JA, 1985. Stability of ELISA activity of barley yellow dwarf virus in leaf samples and extracts. *Plant Disease*, 69(10):854-857.
- Lister RM, Ranieri R, 1995. Distribution and economic importance of barley yellow dwarf. In: D'Arcy CJ, Burnett PA, eds. *Barley Yellow Dwarf - 40 Years of Progress*. St. Paul, USA: APS Press, 29-53.
- Makinen K, Naess V, Tamm T, Truve E, Aaspollu A, Saarma M., 1995. The putative replicase of cocksfoot mottle sobemovirus is translated as a part of the polyprotein by ψ 1 ribosomal frameshift. *Virology* 207:566-71.
- Makkouk KM, Comeau A, 1994. Evaluation of various methods for the detection of barley yellow dwarf virus by the tissue-blot immunoassay and its use for virus detection in cereals inoculated at different growth stages. *European Journal of Plant Pathology*, 100(1):71-80.
- Makkouk KM, Comeau A, Ghulam W, 1994a. Resistance to barley yellow dwarf luteovirus in *Aegilops* species. *Canadian Journal of Plant Sciences* 74:631-634.
- Makkouk KM, Comeau A, Ghulam W, 1994b. Resistance to barley yellow dwarf luteovirus in *Aegilops* species. *Canadian Journal of Plant Sciences* 74:631-634.
- Mann JA, Harrington R, Carter N, Plumb RT, 1997. Control of aphids and barley yellow dwarf virus in spring-sown cereals. *Crop Protection*, 16:81-87.
- Malmstrom, C. M., Hughes, C. C., Newton, L. A. & Stoner, C. J. (2005a). Virus infection in remnant native bunchgrasses from invaded California grasslands. *New Phytol* 168, 217-230.
- Malmstrom, C. M., McCullough, A. J., Johnson, H. A., Newton, L. A. & Borer, E. T. (2005b). Invasive annual grasses indirectly increase virus incidence in California native perennial bunchgrasses. *Oecologia* 145, 153-164.

- Martens J.W., Seaman, W.L. & Atkinson, T.G. (eds). 1985. *Diseases of field crops*. In *Canada: An illustrated compendium*. Canadian Phytopathological Society, Harrow, Ontario, Canada. 160 pp.
- Martin RR, Keese PK, Young MJ, Waterhouse PM, Gerlach WL., 1990. Evolution and molecular biology of luteoviruses. *Annu. Rev. Phytopathol.* 28:341–63.
- Mastari, J., H. Lapierre, and J.T. Dessens., 1998. Assymetrical distribution of barley yellow dwarf virus PAV variants between host plant species. *Phytopathology* 88:818-821.
- Mayo MA, Barker H, Robinson DJ, Tamada T, Harrison BD., 1982. Evidence that potato leafroll virus RNA is positive-stranded, is linked to a small protein and does not contain polyadenylate. *J. Gen. Virol.* 59:163–67
- Mayo MA, Robinson DJ, Jolly CA, Hyman L., 1989. Nucleotide sequence of potato leafroll luteovirus RNA. *J. Gen. Virol.* 70:1037–51.
- MayoMA,ZieglerGrafV., 1996. Molecular biology of luteoviruses. *Adv. Virus Res.* 46:413–60.
- McGrath PF, Lister RM, Bertschinger L, Saari EE, 1996. Unusual epitope profiles of isolates of barley yellow dwarf viruses from Asia. *Phytopathologia mediterranea*, 35:169-176.
- McGrath PF, Vincent JR, Lei CH, Pawlowski WP, Torbert KA, Gu W, Kaeppler HF, Wan Y, Lemaux PG, Rines HR, Somers DA, Larkins BA, Lister RM, 1997. Coat protein mediated resistance to isolates of barley yellow dwarf in oats and barley. *European Journal of Plant Pathology*, 103:695-710.
- McKenzie RIH, Burnett PA, Gill CC, Comeau A, Brown PD, 1985. Inheritance of tolerance to barley yellow dwarf virus in oats. *Euphytica*, 34(3):681-687.
- Mckirdy SJ, Jones RAC, 1996. Use of Imidacloprid and newer generation synthetic pyrethroids to control the spread of barley yellow dwarf luteovirus in cereals. *Plant Disease*, 80:895-901.
- Miller WA, Dreher TW, Hall TC., 1985. Synthesis of brome mosaic virus subgenomic RNA *in vitro* by internal initiation on (j) sense genomic RNA. *Nature* 313:68–70.
- Miller WA, Waterhouse PM, Gerlach WL., 1988. Sequence and organization of barley yellow dwarf virus genomic RNA. *Nucleic Acids Res.* 16:6097–111.
- Miller WA, Hercus T, Waterhouse PM, Gerlach WL., 1991. A satellite RNA of barley yellow dwarf virus contains a novel hammerhead structure in the selfcleavage domain. *Virology* 183:711–20.
- Miller JS, Mayo MA., 1991. The location of the 50 end of the potato leafroll luteovirus subgenomic coat protein mRNA. *J. Gen. Virol.* 72:2633–38.
- Miller WA, Dinesh-Kumar SP, Paul CP., 1995. Luteovirus gene expression. *Crit. Rev. Plant Sci.* 14:179–211.
- Miller WA, Brown CM, Wang S., 1997. New punctuation for the genetic code: luteovirus gene expression. *Semin. Virol.* 8:3–13.
- Miller WA, Rasochová L, 1997. Barley yellow dwarf viruses. *Annual Review of Phytopathology*, 35:167-190.
- Miller, W. A., S. Liu, and R. Beckett., 2002. Barley yellow dwarf virus: Luteoviridae or Tombusviridae? *Mol. Plant Pathol.* 3: 177-183.
- Mohan B. R, Dinesh-Kumar SP, Miller WA., 1995. Genes and *cis*-acting sequences involved in replication of barley yellow dwarf virus-PAV RNA. *Virology* 212:186–95
- Moury, B., Cardin, L., Onesto, J. P., Candresse, T. & Poupet, A., 2001. Survey of Prunus necrotic ringspot virus in rose and its variability in rose and Prunus spp. *Phytopathology* 91, 84-91
- Murant and Roberts, 1979. *Ann. appl. Biol.* 92: 343.
- Murphy JF, D'Arcy CJ, Clark JMJ., 1989. Barley yellow dwarf virus RNA has a 50- terminal genome-linked protein. *J. Gen. Virol.* 70:2253–56.
- Nkongolo KK, 1996. Expression of barley yellow dwarf virus and Russian wheat aphid resistance genes and fertility of spring wheat x triticale hybrids and backcross lines. *Euphytica*, 337-344.
- Ohm HW, Patterson FL, Carrigan LL, Shaner GE, Foster JE, Finney RE, Roberts JJ, 1981. Registration of Elmo common wheat germplasm. *Crop Science*, 21:803.
- Orlob, Arny and Medler, 1961. Aphid transmission of barley yellow dwarf virus. *Phytopathology* 51: 515.
- Oswald, J. W., and B. R. Houston., 1951. A new virus disease of cereals, transmissible by aphids. *Plant Dis. Rep.* 35: 471-475.
- Oswald JW, Houston BR, 1953. The yellow-dwarf virus disease of cereal crops. *Phytopathology*, 43:128-136.
- Paliwal. Y.C., 1982. Identification and annual variation of variants of barley yellow dwarf virus in Ontario and Quebec. *Can. J. Plant Pathol.* 4: 59-64.
- Parry AL, Habgood RM, 1986. Field assessment of the effectiveness of a barley yellow dwarf virus resistance gene following its transference from spring to winter barley. *Annals of Applied Biology*, 108(2):395-401.
- Patterson FL, Ohm HW, Shaner GE, Finney RE, Gallun RL, Roberts JJ, Foster JE, 1982. Registration of Caldwell wheat (Reg. No. 660). *Crop Science*, 22(3):691-692.
- Pead MT, Torrance L, 1988. Some characteristics of monoclonal antibodies to a British MAV-like isolate of barley yellow dwarf virus. *Annals of Applied Biology*, 113(3):639-644.
- Perry, K. L., F. L. Kolb, B. Sammons, C. Lawson, G. Cisar, and H. Ohm., 2000. Yield effects of barley yellow dwarf virus in soft red winter wheat. *Am. Phytopath. Soc.* 90: 1043-1048.

- Pfeffer, S., P. Dunoyer, F. Heim, K.E. Richards, G. Jonard, and V. Ziegler-Graff., 2002. P0 of Beet western yellows virus is a suppressor of posttranscriptional gene silencing. *J. Virol.* 76:6815-6824.
- Plourde A, Comeau A, StPierre CA, 1992. Barley yellow dwarf virus resistance in *Triticum aestivum* cross *Leymus angustus* hybrids. *Plant Breeding*, 108(2):97-103.
- Plumb RT, Lennon EA, Gutteridge RA, 1986. Forecasting barley yellow dwarf virus by monitoring vector populations and infectivity. In: McLean GD, Garrett RG, Ruesink WG, eds. *Plant Virus Epidemiology: Monitoring, Modelling and Predicting Outbreaks*. Sydney, Australia: Academic Press, 185-198.
- Plumb RT, Johnstone GR, 1995. Cultural, chemical and biological methods for the control of barley yellow dwarf. In: D'Arcy CJ, Burnett PA, eds. *Barley Yellow Dwarf - 40 Years of Progress*. St. Paul, USA: APS Press, 307-319.
- Plumb RT., 1995. Epidemiology of barley yellow dwarf in Europe. See Ref. 24, pp. 107-27
- Power AG, Gray SM, 1995. Aphid transmission of barley yellow dwarf viruses: Interactions between viruses, vectors, and host plants. In: D'Arcy CJ, Burnett PA, eds. *Barley Yellow Dwarf - 40 Years of Progress*. St. Paul, USA: APS Press, 259-289.
- Ramirez I, 1990. A review of barley yellow dwarf virus in the southern cone countries of South America. In: Burnett PA, ed. *World Perspectives on Barley Yellow Dwarf*. Mexico: CIMMYT, 29-33.
- Randles JW and Rathjen JP., 1995. Genus *Luteovirus*. In: Murphy FA, Fauquet CM, Bishop DHL, Ghabrial SA, Jarvis AW, Martelli JP, Mayo MA and Summers MD (eds) *Virus Taxonomy, Sixth Report of the International Committee on Taxonomy of Viruses* (pp 379-383) Springer-Verlag, Wien New York.
- Rasmusson DC, Schaller CW, 1959. The inheritance of resistance in barley to the yellow-dwarf virus. *Agronomy Journal* 51:661-664.
- Rasochova L, Miller WA., 1996. Satellite RNA of barley yellow dwarf-RPV virus reduces accumulation of RPV helper virus RNA and attenuates RPV symptoms in oats. *Mol. Plant-Microbe Interact.* 9:646-50.
- Rasochova L, Passmore BK, Falk BW, Miller WA., 1997. The satellite RNA of barley yellow dwarf virus-RPV is supported by beet western yellows virus in dicotyledonous protoplasts and plants. *Virology* 231:182-91.
- Rathjen JP, Karageorgos LE, Habili N, Waterhouse PM, Symons RH., 1994. Soybean dwarf luteovirus contains the third variant genome type in the luteovirus group. *Virology* 198:571-79
- Raybould, A. F., Maskell, L. C., Edwards, M.-L., Cooper, J. I. & Gray, A. J., 1999. The prevalence and spatial distribution of viruses in natural populations of *Brassica oleracea*. *New Phytol* 141, 265-275.
- Reutenauer A, Ziegler-Graff V, Lot H, Scheidecker D, Guilley H, *et al.*, 1993. Identification of beet western yellows luteovirus genes implicated in viral replication and particle morphogenesis. *Virology* 195:692-99
- Rines HW, Stuthman DD, Briggles LW, Young VL, Jedlinski H, Smith DH, Webster JA, Rothman PG, 1980. Collection and evaluation of *Avena fatua* for use in oat improvement. *Crop Science*, 20:63-68.
- Robertson NL, French R, Gray SM, 1991. Use of group-specific primers and the polymerase chain reaction for the detection and identification of luteoviruses. *Journal of General Virology*, 72(6):1473-1477.
- Rochow, 1961. Description of plant virus. *Adv. Agron.* 13:217.
- Rochow and Brakke, 1964. Description of plant virus. *Virology* 24: 310.
- Rochow, W. F., 1969. Biological Properties of 4 Isolates of Barley Yellow Dwarf Virus. *Phytopathology* 59, 1580-1589.
- Rochow WF., 1970. Barley yellow dwarf virus: phenotype mixing and vector specificity. *Science* 167:875-78
- Rochow, W. F. & Muller, I., 1971. A fifth variant of barley yellow dwarf virus in New York. *Plant Dis Rep* 55, 874-877.
- Rochow, W. F., 1979. Field variants of barley yellow dwarf virus: Detection and fluctuation during twenty years. *Phytopathology* 69: 655-660.
- Rochow WF, Carmichael LE, 1979. Specificity among barley yellow dwarf viruses in enzyme immunosorbent assays. *Virology*, 95(2):415-420.
- Sacristan, S., Fraile, A., Malipica, J. M. & Garcia-Arenal, F., 2005. Analysis of host adaptation and its relationship with virulence in Cucumber mosaic virus. *Phytopathology* 95, 827-833.
- Sarnow P., 1995. *Cap-Independent Translation*. Berlin: Springer.
- Schaller CW, Chim CI, 1969. Registration of Atlas 68 barley. *Crop Science*, 9:521.
- Singh RP, Burnett PA, Albarrán M, Rajaram S, 1993. Bdv1: a gene for tolerance to barley yellow dwarf virus in bread wheats. *Crop Science*, 33(2):231-234.
- Sharma HC, Gill BS, Uyemoto JK, 1984. High levels of resistance in *Agropyron* species to barley yellow dwarf and wheat streak mosaic viruses. *Phytopathologische Zeitschrift*, 110(2):143-147.
- Sharma HC, Ohm HW, Lister RM, Foster JE, Shukle RH, 1989. Response of wheatgrasses and wheat cross wheatgrass hybrids to barley yellow dwarf virus. *Theoretical and Applied Genetics*, 77(3):369-374.
- Sharma H, Ohm H, Goulart L, Lister R, Appels R, Benhabib O., 1995. Introgression and characterization of barley yellow dwarf virus resistance from *Thinopyrum intermedium* into wheat. *Genome* 38:406-13.
- Sharma HC, Ohm HW, Perry KL, 1997. Registration of barley yellow dwarf virus resistant wheat germplasm

- line P29. *Crop Science*, 37:1032-1033.
- Shepardson, Esau and McCrum, 1980. Ultrastructure of potato leaf phloem infected with potato leafroll virus. *Virology* 105: 379.
- Silver SL, Rasochova L, Dinesh-Kumar SP, Miller WA., 1994. Replication of barley yellow dwarf virus satellite RNA transcripts in oat protoplasts. *Virology* 198:331-35
- Skaria M, Lister RM, Foster JE, Shaner G, 1985. Virus content as an index of symptomatic resistance to barley yellow dwarf virus in cereals. *Phytopathology*, 75(2):212-216.
- Slykhuis, 1967. Factors determining the development of wheat spindle streak mosaic caused by a soil borne virus in Ontario. *Rev. appl. Mycol.* 46: 401.
- Smith PR, Sward RJ, 1982. Crop loss assessment studies on the effects of barley yellow dwarf virus in wheat in Victoria. *Australian Journal of Agricultural Research*, 33(2):179-185.
- Smith, H.G., and H. Barker., 1999. *The Luteoviridae*. CABI Publishing, Wallingford, Oxon
- Somers, D.A., H.W. Rines, W. Gu, H.F. Kaeppler, and W.R. Bushnell., 1992. Fertile, transgenic oat plants. *Biotechnology* 10:1589-1594.
- Starling TM, Griffey CA, Price AM, Roane CW, Sisson WL, Brann DE, 1994. Registration of 'Nomini' barley. *Crop Science*, 34(1):300.
- Suneson CA, 1955. Breeding for resistance to yellow dwarf virus in barley. *Agronomy Journal*, 47:283.
- Tacke E, Pr'ufer D, Salamini F, Rohde W., 1990. Characterization of a potato leafroll luteovirus subgenomic RNA: differential expression by internal translation initiation and UAG suppression. *J. Gen. Virol.* 71:2265-72.
- Tacke E, Pr'ufer D, Schmitz J, Rohde W., 1991. The potato leafroll luteovirus 17K protein is a single-stranded nucleic acid binding protein. *J. Gen. Virol.* 72:2035-38.
- Tacke E, Schmitz J, Pr'ufer D, Rohde W., 1993. Mutational analysis of the nucleic acid-binding 17 kDa phosphoprotein of potato leafroll luteovirus identifies an amphipathic alpha-helix as the domain for protein/protein interactions. *Virology* 197:274-82.
- Tacke E, Salamini F, Rohde W., 1996. Genetic engineering of potato for broad spectrum protection against virus infection. *Nat. Biotechnol.* 14:1597-601.
- Timian, 1964. Barley yellow dwarf luteovirus. *Phytopathology* 54: 910.
- Timmer RT, Benkowski LA, Schodin D, Lax SR, Metz AM, *et al.*, 1993. The 50 and 30 untranslated regions of satellite tobacco necrosis virus RNA affect translational efficiency and dependence on a 50 cap structure. *J. Biol. Chem.* 268:9504-10.
- Torrance L, Pead MT, Larkins AP, Butcher GW, 1986. Characterization of monoclonal antibodies to a UK isolate of barley yellow dwarf virus. *Journal of General Virology*, 67:549-556.
- Torrance L, 1987. Use of enzyme amplification in an ELISA to increase sensitivity of detection of barley yellow dwarf virus in oats and in individual vector aphids. *Journal of Virological Methods*, 15(2):131-138.
- Ueng PP, Vincent JR, Kawata EE, Lei C-H, Lister RM, Larkins BA., 1992. Nucleotide sequence analysis of the genomes of the MAV-PS1 and P-PAV isolates of barley yellow dwarf virus. *J. Gen. Virol.* 73:487-92.
- Van der Wilk, F., Huisman, M. J., Cornelissen, B. J. C., Huttinga, H. , Goldbach, R., 1989. Nucleotide sequence and organization of potato leafroll virus genomic RNA. *FEBS Lett.* 245:51-56.
- Vega J, Scagliusi SMM, Ulian EC. 1997. Sugarcane yellow leaf disease in Brazil: evidence of association with a luteovirus. *Plant Dis.* 81:21-26.
- Veidt I, Lot H, Leiser M, Scheidecker D, Guilley H, *et al.*, 1988. Nucleotide sequence of beet western yellows virus RNA. *Nucleic Acids Res.* 16:9917-32.
- Veidt I, Bouzoubaa SE, Leiser R-M, Ziegler-Graff V, Guilley H, *et al.*, 1992. Synthesis of full-length transcripts of beet western yellows virus RNA: messenger properties and biological activity in protoplasts. *Virology* 186:192-200.
- Vertigan WA, 1979. Shannon: the first Tasmanian bred barley. *Tasmanian Journal of Agriculture*, 51:33-35.
- Vincent JR, Ueng PP, Lister RM, Larkins BA., 1990. Nucleotide sequences of coat protein genes for three isolates of barley yellow dwarf virus and their relationships to other luteovirus coat protein sequences. *J. Gen. Virol.* 71:2791-99.
- Vincent JR, Lister RM, and Larkins BA., 1991. Nucleotide sequence analysis and genomic organization of the NY-RPV isolate of barley yellow dwarf virus. *J Gen Virol* 72: 2347-2355.
- Vivar HE, Burnett PA, Fox PN, 1991. Andean subsistence farmers benefit from ICARDA-CIMMYT barley breeding program. *Diversity*, 7(1-2):64-65.
- Walczak R, Westhof E, Carbon P, Krol A., 1996. A novel RNA structural motif in the selenocysteine insertion element of eukaryotic selenoprotein mRNAs. *RNA* 2:367-79
- Wallwork, H., 1992. *Cereal leaf and stem diseases*. Grains Research and Development Corporation. Barton, Australia. 102p.
- Wang JY, Chay C, Gildow FE, Gray SM., 1995. Readthrough protein associated with virions of barley yellow dwarf luteovirus and its potential role in regulating the efficiency of aphid transmission. *Virology* 206:954-62.

- Wang S, Miller WA., 1995. A sequence located 4.5 to 5 kilobases from the 50 end of the barley yellow dwarf virus (PAV) genome strongly stimulates translation of uncapped mRNA. *J. Biol. Chem.* 270:13446–52.
- Wang S, Browning KS, Miller WA., 1997. A viral sequence in the 30 untranslated region mimics a 50 cap in stimulating translation of uncapped mRNA. *EMBO J.* 16. In press.
- Wang, M.-B., D.C. Abbot, and P.M. Waterhouse., 2000. A single copy of a virus-derived transgene encoding hairpin RNA gives immunity to barley yellow dwarf virus. *Mol Plant Pathol* 1:347-356.
- Wang, X., Chang, S., Jin, Z., Li, L. & Zhou, G., 2001. Nucleotide sequences of the coat protein and readthrough protein genes of the Chinese GAV isolate of Barley yellow dwarf virus. *Acta Virol* 45, 249-252.
- Waterhouse PM, Gildow FE, Johnstone GR, 1988. AAB Descriptions of Plant Viruses: Luteovirus Group, No. 339. Wellesbourne, UK: Association of Applied Biology, 9 pp.
- Waterhouse, P.M., M.B. Wang, and T. Lough., 2001. Gene silencing as an adaptive defence against viruses. *Nature* 411:834-842.
- Watkins, J., 2003. Barley yellow dwarf. Plant disease central, UNL Extension Specialist. University of Nebraska, NU Institute of Agriculture and Natural Resources Lincoln.
- Watkins, J.E. & Lane, L.C., 2004. Barley yellow dwarf disease of barley, oats, and wheat. In *NebGuide, University of Nebraska*. USA. Accessed on 12 July 2004 at <http://ianrpubs.unl.edu/plantdisease/g906.htm>.
- Watson MA, Mulligan TE, 1960. Comparison of two barley yellow dwarf viruses in glasshouse and field experiments. *Annals of Applied Biology*, 48:559-574.
- Wen F, Lister RM., 1991. Heterologous encapsidation in mixed infections among four isolates of barley yellow dwarf virus. *J. Gen. Virol.* 72:2217–23.
- Wiese, M. V., 1977. Compendium of wheat diseases, vol. 1. The American Phytopathological Society. St. Paul, MN.
- Xin ZY, Brettell RIS, Cheng ZM, Waterhouse PM, Appels R, Banks PM, Zhou GH, Chen X, Larkin PJ, 1988. Characterization of a potential source of barley yellow dwarf virus resistance for wheat. *Genome*, 30(2):250-257.
- Xu SJ, Banks PM, Dong YS, Zhou RH, Larkin PJ, 1994. Evaluation of Chinese Triticeae for resistance to barley yellow dwarf virus (BYDV). *Genetic Resources and Crop Evolution*, 41(1):35-41.
- Zavreiv SK, Hickey CM, Lommel SA., 1996. Mapping of the red clover necrotic mosaic virus subgenomic RNA. *Virology* 216:407-10.
- Ziegler-Graff V, Brault V, Mutterer D, Simonis M-T, Herrbach E, *et al.*, 1996. The coat protein of beet western yellows luteovirus is essential for systemic infection but the viral gene products P29 and P19 are dispensable for systemic infection and aphid transmission. *Mol. Plant-Microbe Interact.* 9:501–10.
- Zishan G, Aftab A.K. and Jamil K., 2011. Importance of barley yellow dwarf virus (bydv) disease in wheat. V01 (38-42).
- Zitter, T. A., 2001. Virus problems of sweet corn. Cornell University Fact Sheet http://vegetableonline.ppath.cornell.edu/factsheets/Virus_SweetCorn2.html
- Zuniga E, 1990. Biological control of cereal aphids in the southern cone of South America. In: Burnett PA, ed. *Barley Yellow Dwarf*. Mexico: CIMMYT, 362-367.

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