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Review

Special Collection: Pulse Crop Insect Pests and Their Management Strategies

Vector-Borne Viruses of Pulse Crops, With a Particular Emphasis on North American Cropping System

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Abstract

Due to their nutritional value and function as soil nitrogen fixers, production of pulses has been increasing markedly in the United States, notably in the dryland areas of the Northern Plains and the Pacific Northwest United States (NP&PNW). There are several insect-transmitted viruses that are prevalent and periodically injurious to pulse crops in the NP&PNW and elsewhere in North America. Others are currently of minor concern, occurring over limited areas or sporadically. Others are serious constraints for pulses elsewhere in the world and are not currently known in North America, but have the potential to be introduced with significant economic consequences. Managing plant viruses and the diseases they cause requires effective diagnostics, knowledge of virus vectors, virus transmission biology and ecology. A comprehensive compendium to inform producers and researchers about viruses currently and potentially affecting pulses in North America is needed. Here we provide an overview of insect transmitted viruses and their biology, followed by descriptions of the structure, infection biology, host ranges, symptoms, interspecific interactions, and current management options including host plant resistance and vector control for 33 viruses affecting or potentially affecting pulses in the United States and Canada. These are organized based on their transmission biology into persistently transmitted (families *Geminiviridae*, *Luteoviridae* and *Nanoviridae*), semi-persistantly transmitted (*Secoviridae*), and nonpersistantly transmitted (*Betaflexiviridae*, *Bromoviridae* and *Potyviridae*) viruses. We conclude with an overview of the principles of managing insect-transmitted viruses and an outline of areas requiring further research to improve management of viruses in pulses currently and into the future.

Key words: legumes, virus transmission, integrated pest management, aphid, whitefly, leafhopper, beetle

Pulse crops are important sources of proteins and fiber in many arid and semi-arid regions around the world, including the United States, where the increase in pulse production is driven by the surge of demand in both domestic and international markets (Parr et al. 2017). In 2016 to 2017, U.S. exports for pulse crops reached 2.79 billion pounds, accounting for about 43% of total U.S. production (Wells and Bond 2016). In the United States, cool season legumes are grown primarily in the dryland areas of the Northern Plains (NP) (Montana, North Dakota, and South Dakota) and the Palouse area of the Pacific Northwest (PNW) (includes parts of Washington, Idaho, and Oregon). In Canada, pulse production and acreage has increased considerably over the past three decades, with Saskatchewan and Alberta being the leading provinces, placing the country among top producers in the world (Bekkering 2011). Similar to other crops, pulses can be infected by a wide range of viruses, many of which are transmitted by insect vectors. Currently, virus diseases are more prevalent in the PNW than in the NP and are rare in Canada. For the purpose of this article, insect-transmitted viruses will primarily be discussed for the following pulse crops: Common bean (*Phaseolus vulgaris* L. (Fabales: Fabaceae)), broad bean (*Vicia faba* L. (Fabales: Fabaceae)), dry pea (*Pisum sativum* L. (Fabales: Fabaceae)), lentil (*Lens culinaris* Medik. (Fabales: Fabaceae)), chickpea (or garbanzo bean) (*Cicer arietinum* L. (Fabales: Fabaceae)), and cowpea (*Vigna unguiculata* (L.) Walp. (Fabales: Fabaceae)).

Viruses are obligate acellular parasites consisting of a nucleic acid and, typically, a protein that reproduce inside living cells (Agrios 1997). They can be difficult to diagnose because their field symptoms appear similar to those of other disorders, such as environmental and nutritional stresses, herbicide injury, or phytotoxicity (Burrows 2012). Transmission of viruses to plants usually occurs by either an arthropod vector or infected seed. Hogenhout et al. (2008) reported that arthropod vectors transmit 75% of the 700 plant viruses recognized by the International Committee on Taxonomy of Viruses. In the field, arthropod vectors are the most common and economically important means of virus spread from plant to plant within crops.

The main insect vectors of viral diseases in pulse crops belong to the three orders Hemiptera, Thysanoptera, and Coleoptera. Hemiptera, which includes aphids, whiteflies, leafhoppers, planthoppers and true bugs, constitutes one of the most important groups of insect vectors and is known to transmit 55% of transmitted viruses (Hogenhout et al. 2008). These insects are efficient vectors due to their piercing-sucking mouthparts, which consist of two mandibular and two maxillary stylets (Cranston and Gullan 2003). Hemipteran herbivores feed on the phloem, xylem, or mesophyll tissue (Chapman 1998). Many plant viruses are phloem-limited and so are readily encountered by phloem-feeders. Other plant viruses are not phloem-limited and can be acquired or transmitted by the insects probing in other plant tissues while seeking a feeding site. The most economically important insect vectors within Hemiptera are aphids (family Aphididae) and leafhoppers (family Cicadellidae), which transmit approximately 325 plant virus species across all plant taxa (Hogenhout et al. 2008). One of the most common vectors of pulse crop viruses is the pea aphid (*Acyrthosiphon pisum* (Harris) (Hemiptera: Aphididae)) (Burrows 2012).

Thrips (Thysanoptera) use their piercing-rasping mouthparts, composed of two maxillary stylets and one mandibular stylet (Cranston and Gullan 2003), to puncture plant epidermis to feed on cell contents. They can transmit viruses that could infect pulses, e.g., *Ilarvirus* and Tospoviruses. Tospoviruses, however, are not currently considered as major pests of cool season legumes in North America.

Beetles (Coleoptera) can transmit viruses with their chewing mouthparts by injuring the leaf tissues and breaching cells during defoliation. The viruses transmitted by beetles are either circulative or carried on the mouthparts (Bradshaw et al. 2008, Smith et al. 2017).

The three main modes of virus transmission by insect vectors include persistent, semi-persistent, and nonpersistent (Nault 1997, Ng and Perry 2004). All three types occur among the viruses affecting pulse crops. In persistent transmission, a virus needs to be ingested and transferred through hemocoel to the salivary glands of the insect, thus typically exhibiting a relatively high degree of vector-specificity (Gray and Gildow 2003). Persistent viruses can be further divided into nonpropagative viruses that do not replicate within the vector (the majority infecting pulses) and propagative that do replicate within the vector (*Tospovirus* within *Bunyaviridae*). Semi-persistent viruses require acquisition and inoculation access periods ranging from several minutes to hours, thus do not necessarily require a latent period within their insect vectors as the virus is retained on the lining of the mouthparts, foregut, or both (Blanc et al. 2014). Finally, the nonpersistently transmitted viruses may be acquired immediately from the infected plants by brief probing and feeding by the insect vector (Blanc et al. 2014) and are retained at the very tip of the maxillary stylets in aphid vectors (Uzest et al. 2007).

Vector-borne viruses of pulse crops in the United States and Canada have received limited attention and have not been reviewed comprehensively. In this article, we introduced viruses that are currently present or have the potential to affect North American production. These include several persistently transmitted viruses from the families *Geminiviridae*, *Luteoviridae*, and *Nanoviridae*, semi-peristently transmitted viruses from *Secoviridae*, and nonpersistently transmitted viruses from the *Betaflexiviridae*, *Bromoviridae*, *Nanoviridae*, and *Potyviridae* families. Table 1 summarizes the host ranges, transmission mechanisms, prevalent management practices, and availability of host plant resistance to each of these viruses. Of the 33 viruses covered, only 13 are known to occur in North America, but the others are included because they could potentially infect pulses grown widely in North America and under the right conditions could be established as economic pathogens on the continent. Among the persistently transmitted viruses, only two species within the *Geminiviridae* occur in North American (*Bean golden mosaic virus* [BGMV] and *Spinach curly top Arizona virus*). The tropical and semi-tropical ranges of their vectors likely contribute to this pattern. The *Luteoviridae* are well represented (two of the four species), and all six of the nonpersistent viruses affecting pulses occur in North America. In addition to individual virus descriptions and vector-plant-pathogen interactions, current management options are also discussed for most of these viruses. Some selected areas of future research are also proposed in the conclusion of this article to fill existing gaps in our understanding of vector-borne pathosystems in pulse crops.

Persistently Transmitted Viruses

Geminiviridae

Geminiviridae is the second largest family of plant viruses (van Regenmortel et al. 2000). The Geminiviruses feature small single-stranded DNA (ssDNA) genomes and a geometry of two twinned segments which gives the family its name and which is a unique structure among viruses (Bennett et al. 2008, Jeske 2009). Within this twin structure, each particle contains either a single circular ssDNA or two ssDNA molecules with two components (Bennett et al. 2008). Single genomes range between 2.5 and 2.8 kb, while bipartite genomes are approximately 5.2 kb (Bennett et al. 2008). Bipartite Geminiviruses require both segments for complete infection (Jeske 2009). In either configuration, there is a single protein coat species (Goodman 1977, Harrison et al. 1977, Böttcher et al. 2004) and specificity between virus coat protein and the vector is thought to be determined exclusively by the coat protein (Briddon et al. 1990, Höhnle et al. 2001).

Geminiviridae contains between four and seven genera: *Becurtovirus, Begomovirus, Eragrovirus, Curtovirus, Mastrevirus, Topocuvirus*, and *Turncurtovirus*, which are classified based on a combination of the organization of their genomes, their insect vectors, and sequence similarity (Brown et al. 2012, Briddon 2015). All members of the genera *Topocuvirus*, *Mastrevirus*, and *Curtovirus*, in addition to some of the *Begomovirus*, have monopartite structures while the *Begomovirus* genus also contains bipartite genomes (Jeske 2009).

Geminivirus demonstrates pronounced biogeographic clustering in sequence comparisons allowing assignment to the new and old-world regions. These viruses exhibit a very high rate of evolution among DNA viruses, which is almost equivalent to the rate seen in RNA-based viruses (Duffy and Holmes 2008). It is possible that this, along with a high recombination rate (Lefeuvre et al. 2007), enables members of the family to adapt quickly to new host plants.

Geminiviridae are mostly phloem-limited viruses transmitted in a persistent manner by hemipteran vectors (Briddon 2015), specifically leafhoppers (in *Mastrevirus, Curtovirus*), treehoppers (in *Topocuvirus*) and by the whitefly species *Bemesia tabaci* (Gennadius) (Hemiptera: Aleyrodidae) (*Begomovirus*) (Byrne and Bellows 1991, Morales and Jones 2004). Recently, *B. tabaci* has been shown to be a complex of nearly three dozen cryptic species (Boykin and De Barro 2014). Over 80% of described Geminiviruses are in the genus *Begomovirus* and transmitted by whiteflies in the *B. tabaci* species

Table 1. A summary of the virus species, means and modes of transmission, host ranges, key management practices and availability of host plant resistance, covered in the present review

Mode of transmission	Family	Genus	Species	Vector	Other means of transmission	Main pulse hosts	Alternative host plant families	Present in North America	Current management	Host plant resistance identified
Persistent										
	Geminiviridae	*Mastervirus*	*Chickpea chlorotic dwarf virus*	Leafhopper	N/A	Bean (various), Chickpea,pigeon pea, lablab	9	N	Chemical	N
			Chickpea chlorotic dwarf Syria virus	Leafhopper	N/A	Bean (various), Chickpea, pigeonpea, lablab	Unknown	N	Chemical	N
			Chickpea chlorotic dwarf Pakistan virus	Leafhopper	N/A	Bean (various), Chickpea, pigeon pea, lablab	9	N	Chemical	N
			Chickpea chlorotic dwarf Sudan virus	Leafhopper	N/A	Bean (various), Chickpea, pigeon pea, lablab	9	N	Chemical	N
			Chickpea red leaf virus	Leafhopper	N/A	Bean (various), Chickpea	3	N	Chemical	N
			Chickpea yellows virus	Leafhopper	N/A	Bean (various), Chickpea	3	N	Chemical	N
			Chickpea chlorosis Australia virus	Leafhopper	N/A	Bean (various), Chickpea	3	N	Chemical	N
			Tobacco yellow dwarf virus	Leafhopper	Grafting	Bean (various), Chickpea	7+	N	Chemical	N
			Chickpea chlorosis virus	Leafhopper	N/A	Chickpea	Unknown	N	Chemical	N
			Bean yellow dwarf virus	Leafhopper	N/A	Bean (various), chickpea	Unknown	N	Chemical	N
		Begomovirus	*Bean golden mosaic virus*	Whitefly	Grafting, mechanical	Bean (various), pigeonpea	0	Y	Chemical	Y
			Bean dwarf mosaic virus	Whitefly	Grafting, mechanical	Common bean	1	Y	Chemical	Y
			Cowpea golden mosaic virus	Whitefly	N/A	Cowpea	0	N	Chemical	N
			Dolichos yellow mosaic virus[a]	Whitefly	Grafting	Dolichos (lablab)	Unknown	N	Chemical, weed control, various cultural	Y
			Horsegram yellow mosaic virus	Whitefly	N/A	Mungbean	Unknown		Chemical, weed control, various cultural	N
			Mungbean yellow mosaic India virus	Whitefly	Grafting	Bean (various), lentils, pigonpea	0	N	Chemical, weed control, various cultural	Y
			Mungbean yellow mosaic virus[b]	Whitefly	Mechanical	Common bean, mungbean, pigeonpea	< 3	N	Chemical, weed control, various cultural	Y
		Becurtovirus	*Beet curly top Iran virus*	Leafhopper	N/A	Common bean,cowpea, pigeonpea	1	N	Chemical	N
			Spinach curly top Arizona virus	Leafhopper	N/A	Common bean, cowpea	Unknown	Y	Chemical	N

Table 1. *Continued*

Mode of transmission	Family	Genus	Species	Vector	Other means of transmission	Main pulse hosts	Alternative host plant families	Present in North America	Current management	Host plant resistance identified
	Luteoviridae	*Luteovirus*	*Bean leaf roll virus*	Aphid	N/A	Broad bean, chickpea, lentil, pea	0	Y	Chemical, planting date	Y
		Polerovirus	*Chickpea chlorotic stunt virus*	Aphid	N/A	Broad bean, chickpea	Unknown	N	Chemical	N
			Beet western yellows virus	Aphid	N/A	Broad bean, chickpea, lentil, pea	20+	Y	Chemical, weed control	N
		Enamovirus	*Pea enation mosaic virus*	Aphid	Mechanical	Broad bean, chickpea, lentil, pea	2	Y	Chemical, weed control	Y
	Nanoviridae	*Nanovirus*	*Faba bean necrotic yellows virus*	Aphid	N/A	Chickpea, common bean, cowpea, lentil, pea	1	N	Chemical, various cultural	Y
Semi-persistent										
	Secoviridae	*Comovirus*	*Broad bean stain virus*	Beetle (weevils)	Seed	Broad bean, chickpea, lentil, pea	0	N	Certified seed, chemical, weed control	N
			Bean pod mottle virus	Beetle (weevils)	Mechanical	Common bean, cowpea	2	Y	Chemical, planting date, trap crop	N
Nonpersistent										
	Potyviridae	*Potyvirus*	*Bean yellow mosaic virus*	Aphid	Seed, mechanical	Broad bean, chickpea, pea	10	Y	Certified seed, chemical, weed control	Y
			Bean common mosaic virus	Aphid	Seed, mechanical	Bean (various)	1	Y	Certified seed, chemical	Y
			Pea seedborne mosaic virus	Aphid	Seed, mechanical	Broad bean, chickpea, lentil, pea	11	Y	Certified seed, chemical, weed control	Y
	Betaflexiviridae	*Carlavirus*	*Pea streak virus*	Aphid	N/A	Chickpea, pea, lentil	4	Y	Chemical	N
			Red clover vein mosaic virus	Aphid	Seed, mechanical	Broad bean, lentil, pea[c]	1	Y	Chemical	N
	Bromoviridae	*Alfamovirus*	*Alfalfa mosaic virus*	Aphid	Seed, mechanical	Broad bean, chickpea, lentil, pea	71	Y	Certified seed, chemical, various cultural	N
		Cucumovirus	*Cucumber mosaic virus*	Aphid	Seed, mechanical	Broad bean, chickpea, common bean, cowpea, lentil, pea	84	Y	Certified seed, chemical, weed control	N

[a]Semi-persistent transmission reported.
[b]Nonpersistent transmission reported.
[c]Not seed transmitted.

complex (Boykin and De Barro 2014). In general, leafhopper-transmitted mastreviruses infect monocotyledonous plant hosts while curtoviruses infect dicotyledonous hosts.

Geminivirus infection is responsible for disease in numerous host plants and is associated with major losses in pulses (Varma et al. 1992) and other crops (e.g., Moffat 1999, Briddon and Markham 2001). Symptoms generally can include stunting, chlorosis, vein swelling, leaf curling, and other tissue abnormalities (Schwinghamer et al. 2011).

Geminiviridae infection in pulse crops is almost exclusively restricted to *Mastrevirus* and *Begomovirus* infections of broad bean, chickpea, pigeon pea (*Cajanus cajan* (L.) Milsp. (Fabales: Fabaceae)), and lablab (*Lablab purporeus* L. Sweet (Fabales: Fabaceae)), with most reports from Australia, Africa or Asia. This may be a function of the range and distribution of the vectors, which are mostly tropical and subtropical.

Consequently, very little *Geminivirus* infection has been reported for pulse crops in North America. Further, those infections that have been detected are limited to the whitefly vectored species in the genus *Begomovirus*. The most comprehensive work is that of Brown and colleagues in Puerto Rico and Mexico (Brown et al. 1999, Idris et al. 1999). While there is currently limited concern from *Geminivirus* in pulse crops in North America, these are major pests of pulse crops in other regions, thus only a brief description of each of these viruses are presented below.

The Genus *Mastrevirus*

The genus *Mastrevirus* contains numerous species that are known to be infectious on pulse crops and other legumes. All of these species are transmitted by leafhopper (Hemiptera: Cicadellidae) species in the subfamily Deltocephalinae. These viruses are relatively common in chickpea and beans (but are rarer in lentil, Schwinghamer et al. 2011). Some species are also known to infect broad bean, pigeon pea, and lablab (Schwinghamer et al. 2011).

Chickpea chlorotic dwarf virus (CpCDV)

CpCDV is transmitted by the leafhopper *Orosius orientalis* (Matsumura) (Hemiptera: Cicadellidae). It has been associated with up to 90% field loss in Sudan (Hamed 2000). It has recently been proposed that CpCDV is actually one of multiple species that cause chickpea stunting disease (Nahid et al. 2008). CpCDV is associated with disease in both chickpea and broad bean (Makkouk et al. 1995a), it is also associated with lentil but at very low rates (Makkouk et al. 2002a,b). There are confirmed reports for North Africa and the Indian subcontinent (Thomas et al. 2010). It has been suggested that *Chickpea chlorotic dwarf Syria virus* (CpCDSV), *Chickpea chlorotic dwarf Pakistan virus* (CpCDPKV), and *Chickpea chlorotic dwarf Sudan virus* (CpCDSDV; Ali et al. 2004) are all strains of a single species of CpCDV (Thomas et al. 2010).

Chickpea red leaf virus (CpRLV), *Chickpea yellows virus* (CpYV), *Chickpea chlorosis Australia virus* (CpAV) represent three of the five recently identified species in Australia along with *Tobacco yellow dwarf virus* (TYDV), and *Chickpea chlorotic virus* (CpCV) (Hadfield et al. 2012). These viruses are known to infect chickpea and bean. TYDV infects at least seven plant families including bean and chickpea but is rare in lentils (Thomas and Bowyer 1984, Trebicki et al. 2010). Symptoms can include both stunting and chlorosis (Schwinghamer et al. 2010). Some bean cultivars are highly susceptible to infection, as are early infected chickpea (Horn et al. 1995).

CpCV is associated with infection of chickpea but is rare or unknown in lentils. Symptoms include stunting and yellowing. In addition to Australia, CpCV is also reported from India, Pakistan, and Africa (Hamed and Makkouk 2002, Hadfield et al. 2012).

Bean yellow dwarf virus (BeYDV)

This virus is generally a pest of common bean, but also infects chickpea. Infected plants die rapidly following infection, which is also associated with symptoms in young leaves including shortened internodes and downward curling (Rybicki and Pietersen 1999). BeYDV has been reported from Africa and Pakistan (Thomas et al. 2010).

The Genus *Begomovirus*

The genus *Begomovirus* contains numerous virus species associated with disease in a variety of dicotyledonous crop and noncultivated species. It is arguably the most destructive group of plant viruses in tropical and subtropical regions (Seal et al. 2006). All known species are transmitted by the whitefly *B. tabaci*. Disease outbreaks are associated with large populations of the vector (Fauquet and Fargette 1990, Cohen et al. 1992).

Bean dwarf mosaic virus (BDMV)

The BDMV infection is characterized by stunted plant growth and mottled leaves (Seo et al. 2004). Severely-affected plants lose flowers or may produce malformed pods (Levy and Tzfira 2010). The common bean is the most important host of BDMV. The Middle American genotypes of common bean, originating from Mexico and Central America (Levy and Tzfira 2010), are either resistant or partially resistant to BDMV (Seo et al. 2004).

Bean golden mosaic virus (also *Bean golden mosaic begeminif virus*)

BGMV is a substantial constraint on bean production in parts of South and Central America in addition to the Caribbean and southern United States. Infected plants are most commonly identified by golden mosaic on leaves. Additional symptoms include reduced pod numbers, prolonged vegetative growth, and stunting. Hosts of BGMV include species of *Vigna, Phaseolus,* and *Capopogonium* (Brown 1990).

Bean golden yellow mosaic virus (BGYMV)

BGYMV is associated with BGMV disease, along with BGMV. It differs from BGMV in nucleic acid sequence, but infection results in similar symptoms and is also transmitted by *B. tabaci.* BGYMV has also been reported in various countries within Tropical North and South America (Brown 1990).

Cowpea golden mosaic virus (CPGMV)

CPGMV is a pest of cowpea primarily in Africa (Singh and Allen 1979) and India (Sharma and Varma 1976).

Dolichos yellow mosaic virus (DYMV)

This virus affects the production of dolichos (*Lablab pupureus*) in which it is responsible for dolichos yellow mosaic disease (Capoor and Varma 1950). DYMV is currently restricted to the Old World. Unlike the other listed Geminiviruses, DYMV is transmitted in a nonpersistent manner (Brunt et al. 1996).

Yellow mosaic disease is associated with multiple *Begomovirus* species which typically share sequence identity with either *Mungbean yellow mosaic India virus* or *Mungbean yellow mosaic virus,* which is a semi-pesistently transmitted virus (Brunt et al. 1996). It is associated with damage and losses in numerous legume crops including: Lima bean (*Phaselous lunatus* L. (Fabales: Fabaceae)), common bean, cluster bean (*Cyamopsis tetragonoloba* (L.) Taub. (Fabales: Fabaceae)), lablab, and pigonpea (Capoor and Varma 1950, Varma and Malathi 2003). These viruses are primarily associated with Old World locations in Asia and Africa.

The Genus *Becurtovirus*

The genus *Becurtovirus* contains two species, *Beet curly top Iran virus* (BCTIV) and *Spinach curly top Arizona virus* (Varsani et al. 2014). BCTIV is associated with damage in common bean and cowpea (*Vigna unguiculata*). Both species are leafhopper transmitted particularly by *Circulifer haematoceps* Baker (Hempitera: Cicadellidae) (Heydarnejad et al. 2013).

There are limited approaches available to manage Geminiviruses. Insecticides have been mostly unsuccessful, often requiring multiple applications without fully suppressing disease (Briddon 2015). Similarly, there has been limited to no success in identifying sources of resistance or developing resistant cultivars. Further, there is evidence of recombination in species of *Begomovirus* that breaks natural plant resistance to infection (Briddon 2015). There is evidence that natural resistance to species of *Begomovirus* can be overcome (Briddon 2015). Some species of Geminiviruses are the subject of molecular studies aimed at developing management strategies or engineered resistance (Ramesh et al. 2017).

Luteoviridae

Viruses in the *Luteoviridae* family have simple nonenveloped (lacking the external lipid membrane) outlines, are 25–30 nm in diameter, and icosahedral in shape (Gray and Gildow 2003, Hogenhout et al. 2008). While genetic variations (i.e., genome organization, gene expression, and sequence) define three distinct genera: *Polerovirus*, *Luteovirus*, and *Enamovirus* (D'Arcy et al. 2000), they all possess a single-stranded genome with 5 to 6 open reading frames, named ORF 0 through ORF 6. The length of the overlap between ORF1 and ORF2, the size of the intergenic region between ORF 2 and ORF 3, and the absence of ORF 0 (encoding a protein with unknown function) would differentiate the *Luteovirus* genus from the *Polerovirus* and *Enamovirus* genera. The absence of the movement protein encoding reading frame, ORF 4, in the genus *Enamovirus* differentiates this group from both *Polerovirus* and *Luteovirus* genera (Domier et al. 2002). In spite of these subtle differences, the transmission biology is closely similar among the three genera, all being transmitted by their aphid vectors in a persistent manner (Gray and Gildow 2003). Luteoviruses can alter host attractiveness to the vectors (Eigenbrode et al. 2002, Jimenez-Martinez et al. 2004, Medina-Ortega et al. 2009), as well as vector preference for the infection status of host plant (Srinivasan et al. 2006, Werner et al. 2009, Ingwell et al. 2012). Infected plants also tend to become better reproductive hosts for the aphid vectors (Castle and Berger 1993, Wu et al. 2014). All these processes are expected to facilitate and enhance virus spread. The circulative nature of virus-aphid interactions in *Luteoviridae*-associated pathosystems, also enables aphids to transmit virus persistently for an extended period of time, or life time. It is important to note that while the two genera *Luteovirus* and *Polerovirus*, are phloem-limited and exclusively transmitted by the phloem-feeding aphids, *Enamovirus* can also penetrate plant through epidermis, infecting cells other than phloem cells (Hogenhout et al. 2008), facilitating acquisition and making transmission through mechanical means possible.

The Genus *Luteovirus*

Bean leafroll virus (BLRV)

Initially described by Boning (1927), BLRV was first isolated in 1954 by Quantz and Volk, in Germany (Ashby 1984). BLRV is now known to be present in Africa (Najar et al. 2000b, Bekele et al. 2005, Makkouk and Kumari 2009), America (Thottappilly et al. 1977, Trucco et al. 2016), Asia (Kaiser and Danesh 1971, Horn et al. 1996, Makkouk et al. 2003), Australia (Schwinghamer et al. 1999), and Europe (Ortiz et al. 2005). In the United States, the virus was first detected in alfalfa (*Medicago sativa* L. (Fabales: Fabaceae)) in Michigan (Thottappilly et al. 1977). Later on, between 1980 and 1983, BLRV caused significant damage to southern Idaho pea production (Hampton 1983).

BLRV has been reported from a variety of cultivated and noncultivated hosts (Ashby 1984, Guy 2010, Jones 2012). Among cultivated pulse crops, it primarily infects broad bean, lentil, pea, and chickpea (Makkouk et al. 2003). Although alfalfa is considered to be a host, and a significant reservoir along with clover (*Trifolium* spp.), neither are significantly affected by BLRV infection (Bos et al. 1988). As alfalfa may also serve as an overwintering host for the aphid vectors of BLRV, this crop may play a key role in disease epidemiology in the PNW, an aspect that needs further research.

Following a 2- to 4-wk incubation period, the initial BLRV symptoms are expressed as interveinal chlorosis, followed by upward rolling of the fully expanded leaves and reduced pod numbers, resulting in yield losses of up to 80% (Heathcote and Gibbs 1962). Stunting and overall yellowing in common bean, chickpea, cowpea, lentil, and pea have also been associated with BLRV infections (Ashby 1984, and references within). In broad bean, while infections that occur before blooming may result in complete losses, inoculations conducted at full-bloom and post-bloom developmental stages resulted in nearly 89 and 50% losses in seed yield, respectively (Kaiser 1973a). Generally, in spring planted legumes, BLRV infections that occur at the later stages of plant development are expected to be less damaging than early occurring infections (Bos et al. 1988). In lentil and pea in the PNW, inoculations occurring approximately 1 mo after plant emergence do not cause economical yield losses (Stokes 2012, Paudel 2014, and Paudel et al. in review). In fall-planted pulses, complete crop failure due to fall infections has been reported in Syria (Bos et al. 1988).

Although polyclonal and monoclonal ELISA antibodies are available for BLRV detection (Makkouk and Kumari 2009, Vemulapati et al. 2014), nucleic acid-based molecular approaches appear to be favored, capable of detecting minute amounts of *Luteovirus* RNA within plant tissues (Figueira et al. 1997, Ortiz et al. 2005, Trucco et al. 2016). Currently available reverse-transcription polymerase chain reaction (RT-PCR) primers (Prill et al. 1990, Makkouk and Kumari 2009) can detect BLRV presence in both aphid and plant tissues (Ortiz et al. 2005).

BLRV shows high levels of vector specificity and is known to be transmitted by the pea aphid *Acy. pisum*, the black bean aphid *Aphis fabae* Scopoli (Hemiptera: Aphididae), the cowpea aphid *Aphis craccivora* Koch (Hemiptera: Aphididae), and the green peach aphid *Myzus persicae* (Sulzer) (Hemiptera: Aphididae) (Kaiser 1973a), in a non-propagative and persistent manner. However, in a study by Ortiz and colleagues (2005) *A. fabae*, *A. craccivora*, and *Myz. persicae* failed to transmit the virus successfully to uninfected broad bean, despite the aphids testing positive for the pathogen, which indicates that these species may not be as efficient vectors as the pea aphid *Acy. pisum*. More recently, a study by Peck et al. (2012) showed that the bluegreen aphid *Acyrthosiphon kondoi* Shinji (Hemiptera: Aphididae) may also transmit BLRV to the clover host *Trifolium subterraneum*. Davis et al. (2017) showed that BLRV provides the pea aphid *Acy. pisum* with fitness advantages, an effect which is expected to promote virus spread within a field, and likely, spillover to nearby patches as aphid density continues to increase. Moreover, the uninfected *Acy. pisum* shows preference to feed on BLRV infected hosts as the virus alters the plant olfactory cues, rendering it more attractive to the aphid vectors (Wu et al. 2014). Preference toward an infected host is predicted to increase the rate of pathogen spread at the initial stages of an epidemic (McElhany 1995, Sisterson 2008, Zeilinger and Daugherty 2014).

Acy. pisum is a complex species that includes several biotypes, distinguished by ecological and genetic differences (Via et al. 2000, Tsuchida et al. 2004), each having specific associations with particular host plants (Peccoud et al. 2010). In addition to genetic variation, this level of host plant specialization may also be driven by the endosymbiont community associated with the aphid biotypes (Tsuchida et al. 2004). Most recently, it has been documented that BLRV infection may interfere with host discrimination and host preference of *Acy. pisum* biotypes, in a genotype-specific manner (Davis et al. 2017). Specifically, BLRV infection improved the performance of a pink morph of *Acy. Pisum* adapted to alfalfa on its relatively less optimal pea host. BLRV infection had no impact on a green morph of *Acy. Pisum* on alfalfa, as both alfalfa and pea remained equally suitable hosts for this aphid biotype. The BLRV presence also had no effect on host suitability of the *Acy. pisum* biotype associated with pea, as optimal performance was associated with pea host regardless of the plant's infection status (Davis et al. 2017). These findings have direct implications in BLRV epidemiology, as virus infection could potentially facilitate vector colonization of the plant species, which may otherwise be ineffective hosts in supporting large vector populations. Several biotypes of *Acy. pisum* occur naturally throughout the PNW and in particular where they migrate annually into pulse crops (Eigenbrode et al. 2016). Further studies are warranted.

Neonicotinoid seed treatments have been used to limit primary BLRV infections due to initial infestation by the aphid vectors (Makkouk and Kumari 2001) and demonstrably can reduce secondary spread prebloom in controlled plot studies (Wu and Eigenbrode, unpublished). Although alfalfa and several weedy species are known BLRV reservoirs, spatial isolation from these potential sources of infection virus sources per se may not be sufficient in preventing infections, because the aphid vectors transmit the virus in a persistent manner and may move long distances (Eigenbrode et al. 2016). Changing planting dates may also be used to reduce losses (Johnstone and Rapley 1979). Information on various management practices is provided below under 'Managing Vector-Borne Viruses of Pulse Crops'.

Several studies have been conducted to identify sources of BLRV resistance. In peas, BLRV resistance and tolerance are controlled by the recessive genes *lr* and *lrv*, respectively (Makkouk et al. 2014). Despite the lack of immunity, planting resistant Australian pea varieties and several advanced breeding lines proved effective against BLRV in a series of experiments conducted in Syria (van Leur et al. 2013). Makkouk et al. (2002a) screened 358 broad bean genotypes worldwide to detect sources of resistance to BLRV; 15 genotypes were identified (Makkouk et al. 2014). As for lentil, several pea genotypes have been registered for resistance to BLRV (Makkouk et al. 2001).

The Genus *Polerovirus*

Beet western yellows virus **(BWYV)**

Initially, referred to as *Radish yellows virus*, James E. Duffus (1960) first described BWYV in the Northwestern United States. To date, BWYV occurrence has been confirmed in Central, Western (Makkouk et al. 2003, Makkouk et al. 2014), and Eastern Asia (Shiying et al. 2007), Northern Africa (Najar et al. 2000a, Bekele et al. 2005), Australia (Latham and Jones 2001a), Europe (Duffus and Russell 1970), Mexico, New Zealand (Johnstone et al. 1989), and the United States (Duffus 1961). This virus is known to affect pea, lentil, broad bean, chickpea, and other legume and nonlegume host plants, belonging to more than 20 families (Duffus 1964, Duffus and Russell 1970, Makkouk et al. 2014). This wide host range would make possible overwintering of this virus (Duffus 1964) in most regions including the pulse growing regions of the United States.

Similar to other viruses in the *Luteoviridae* family, BYWV infections may be characterized by yellowing, rolling, and thickening of leaves and stunting of plants (Shiying et al. 2007, Makkouk et al. 2012). Initial symptoms of chlorosis and leaf curl would appear between 10 and 20 d after inoculation. Tissue-blot immunoassay (TBIA) (Latham and Jones 2001a, Shiying et al. 2007), ELISA (Carazo et al. 1993, Freeman and Aftab 2011), and PCR (Fortass et al. 1997, Freeman and Aftab 2011, Makkouk et al. 2012, Yuan et al. 2015) are laboratory approaches used to confirm BWYV presence. Studies have yet to estimate yield losses to BWYV, both alone and in mixed infections with other viruses, in pulse crops.

Brachycaudus helichrysi Kalt. (Hemiptera: Aphididae), *Myzus ornatus* Liang (Hemiptera: Aphididae), *Myz. persicae*, *A. craccivora*, *Aulacorthum solani* Kalt. (Hemptera: Aphididae), and *Acy. pisum* have been identified as BWYV vectors (Duffus 1960, Makkouk and Kumari 2009). However, the green peach aphid *Myz. persicae* is the most important vector of BWYV (Duffus and Russell 1970). *Myz. persicae* can acquire the pathogen within minutes of feeding and is capable of efficiently transmitting the virus following a 12- to 24-h incubation period (Duffus 1960, Tamaki et al. 1979). The average transmission success of an individual of this species has been estimated at 41.7%, and this rate reached more than 87% when there were several aphids feeding on test plants for a 48-h inoculation access period (Duffus 1960).

Chemical management of aphids has been recommended in conjunction with aphid monitoring in orchards (primary aphid hosts) and in cultivated and noncultivated hosts (e.g., weeds and sugar beet fields) (Tamaki et al. 1979), and as a result, weed management has been recommended as a management approach (Freeman and Aftab 2011). Since perennial alfalfa is also known to host BWYV, it may act as a source of infection for multiple years.

Chickpea chlorotic stunt virus **(CCSV)**

The virus was first described by Abraham et al. (2006) who reported yellowing and stunting of chickpea and broad bean plantings in Ethiopia. Molecular characterization confirmed it was an as-then unknown or uncommon member of *Luteoviridae*. It has subsequently been reported across West Asia (Bananej et al. 2010, Mustafayev et al. 2011) and North Africa (Kumari et al. 2008, Abraham et al. 2009). Genetic diversity within the virus has been documented across its range, including Egypt, Morocco, Sudan, and Syria (Abraham et al. 2009). In addition to its generic symptoms, the virus can be identified based on PCR primers (Abraham et al. 2009). Knowledge of this virus is otherwise very limited. Since it is within *Luteoviridae*, it presumably will be persistently transmitted by aphids, likely the species that affect the pulse crops within its current known range, including *Acy. pisum*, *A. craccivora*, *A. fabae*, and *Myz. persicae*. Thus, when infection risk is deemed to be high, aphid control through insecticides may be indicated. Although the virus has not been detected outside of West Asia and North Africa, it will merit monitoring in the future wherever pulse crops are grown.

The Genus *Enamovirus*

Pea enation mosaic virus **(PEMV)**

PEMV was first described by Taubenhouse (Taubenhaus 1914), and Osborn (Osborn 1935) first used the term 'enation' to describe the characteristic symptom in pea. Stubbs (1937) named the virus PEMV and described its symptoms, insect transmission, and temperature relationships. Although these first descriptions were based

on occurrences in North America, the virus occurs throughout the world in temperate and subtropical regions where legumes are grown (Hagedorn 1996, Makkouk et al. 1999). It was determined to be the main cause of a viral disease outbreak in Washington and Oregon in 1990 (Klein et al. 1991) and periodic severe virus disease episodes that occurred for decades previously were likely caused primarily by PEMV (Clement et al. 2010). Since then, PEMV along with BLRV have been frequently injurious to field pea, chickpea, and lentil in the PNW (Clement et al. 2010) and in broad bean, *Vicia faba*, in various locations in Europe (Hagedorn 1996 and reference within).

PEMV is a bipartite virus comprised of two single-stranded RNAs: RNA-1 and RNA-2 that form a virtually obligate symbiosis (Skaf and de Zoeten 2000). Based on genomic sequence and functions, RNA-1 falls within the *Luteoviridae*, but has been placed in its own genus, *Enamovirus*, and RNA-2 is an *Umbravirus*. Although each RNA is capable of infecting plant tissue independently in protoplasts, normal transmission, and replication requires coinfection. In coinfection, both RNAs are separately encapsidated in two distinct particles. RNA-1 codes for the common coat protein and a protein responsible for aphid transmission, while ORFs in RNA-2 code for a cell-to-cell movement protein (de Zoeten and Skaf 2001), which are evidence for the codependency of the two viruses. Genetic variability among strains of PEMV has been detected in North America (PNW) (Vemulapati et al. 2014) and elsewhere (Šafarova and Navratíl 2014), but more work is needed to assess the extent of this variation within and among regions.

The host range of PEMV is limited mainly to Leguminosae, including a number of economically important genera: *Lens, Cicer, Pisum, Medicago, Melilotus, Phaseolus, Trifolium,* and *Vicia* (Skaf and de Zoeten 2000). It can also infect nonleguminous plants in Chenopodaceae and Solanaceae (Skaf and de Zoeten 2000). Thus, nonlegumes potentially serve as reservoirs for the virus in working landscapes.

Infection by PEMV can be recognized in pea approximately 1 wk after inoculation by downward curling and chlorotic or translucent spots on leaves. As the infection proceeds, growth deformations of various kinds including stunting, rugosity, and loss of apical dominance are evident. Approximately 3 wk after inoculation, enations (hyperblastic growths on leaves) and warts on the pods appear and pods can be distorted. When these symptoms are severe, seed set is severely limited (de Zoeten and Skaf 2001). Although early symptoms can be confused with other diseases or nutritional problems, the later symptoms with enations are definitive for PEMV in pea. In lentil, symptoms are less distinctive and typically include growth reduction and leaf rolling, accompanied by tip wilting or necrosis (e.g., Makkouk et al. 1999). Similar symptoms to those observed in lentil also occur in chickpea (Wu and Eigenbrode, unpublished). Detection of PEMV can be achieved through ELISA (Vemulapati et al. 2014) and commercial kits for Direct Antigen Coating ELISA are available. Detection methods for RNA-1 and RNA-2 by polymerase chain reaction are also available (Timmerman-Vaughan et al. 2009, Doumayrou et al. 2017, Lorenzen et al. unpublished) and are used for detection as part of monitoring efforts in Idaho and Washington (http://www.ag.uidaho.edu/aphidtracker/index.asp).

PEMV is transmissible in a persistent manner by at least 10 aphid species: *Acy. pisum*, *Acy. solani*, *A. gossypii*, *Aul. solani*, *Macrosiphum avenae*, *Macrosiphum euphorbiae* Thomas (Hemiptera: Aphididae), *Myz. ornatus*, *Myz. persicae*, *Rhopalosiphum padi* L. (Hemiptera: Aphididae), and *Schizaphis graminum* Randoni (Hemiptera: Aphididae), among which *Acy. pisum* and *Myz. persicae* are the most important. Most strains of PEMV can also be transmitted mechanically, and if this is continued the strains can lose aphid transmissibility through mutation (Demler et al. 1997). RNA-1 alone can be transmitted mechanically, but it is movement defective and dependent upon RNA-2 for that function (Skaf et al. 1997). Vertical transmission to seed is negligible or nonexistent (Timmerman-Vaughan et al. 2009). Since these vectors and PEMV have multiple hosts, interspecific transmission contributes to PEMV epidemiology.

PEMV has been studied for its indirect effects on the primary vector *Acy. pisum* through infected plants (Hodge and Powell 2008, 2010; Wu et al. 2014). Under some conditions, PEMV-infected plants are superior hosts for the aphid, or elicit greater production of alates, which might facilitate virus spread (Hodge and Powell 2008, 2010), but this has not been shown consistently (Wu et al. 2014). PEMV-infected pea plants are also more attractive to *Acy. Pisum*, and this is at least partially due to aphid responses to differences in volatiles released from infected and noninfected plants (Wu et al. 2014).

Typically, *Acy. pisum* overwinters on perennial legumes which can serve as reservoirs for PEMV. It is therefore prudent to avoid planting annual pulse crops near perennial legumes (Skaf 2000). This practice might have limited value in the PNW region of the United States, where annual flights of immigrating viruliferous aphids evidently come from distances of up to 200 km or more, based on genotyping (Eigenbrode et al. 2016). The overwintering source of PEMV remains uncertain. In the PNW of the United States, alfalfa, which is by far the most abundantly grown perennial legume in the region, is a non-host of PEMV (Larsen et al. 1996b). However, common vetch is frequently infected with PEMV (Eigenbrode et al. unpublished), as it is in the United Kingdom, (Cockbain and Gibbs 1973), so it remains a possible candidate, but is not extensively grown in the PNW. Finally, as noted previously, PEMV can infect nonleguminous hosts and, although it seems unlikely, these hosts could at least contribute to PEMV inoculum entering cultivated pulses. In short, there seem not to be effective ways to reduce the sources of inoculum to manage PEMV in annual legumes in the PNW.

In commercial settings, the primary method for managing PEMV has been and continues to be through reducing aphid populations with insecticides (Davis et al. 1961, Weigand et al. 1994). To be effective, this method requires aggressive control, which poses a problem because the prevalence of PEMV is irregular among years. In the Palouse region, virus incidence monitoring based on samples of migrating aphids and plant tissue samples are provided along with decision support tools to help producers decide whether to treat the aphids for PEMV (and BLRV) (http://www.cals.uidaho.edu/aphidtracker/).

Longer-term, host plant resistance to PEMV remains the most promising management tool. Sources of resistance, traced back to Iranian and Indian origins, are available in pea, lentil, and chickpea (Larsen and Porter 2010, Jain et al. 2014), and resistant varieties have been released. Inheritance of PEMV resistance is simple in pea. For example, PEMV resistance in Geneva Selection 168 is controlled by the single dominant gene *En*, which is used in the U.S. pea breeding programs (Jain et al. 2013, Makkouk et al. 2014).

Nanoviridae

The *Nanoviridae* family consists of the two genera *Babuvirus* and *Nanovirus* (Vetten et al. 2005, Vetten 2008). Members of *Nanoviridae* possess multipartite genomes of single-stranded, circular, positive-sense DNA, and each of them is encapsidated in an isometric particle having a diameter of 18 nm. All DNAs have similar structures containing a conserved stem-loop and other conserved domains in the noncoding region (NCR) (Vetten et al. 2005). Twelve distinct DNA components have been identified in members of *Nanoviridae*. *Babuvirus* and *Nanovirus* comprise six and eight distinct ssDNAs, respectively (Karan et al. 1994, Burns et al. 1995, Karan et al. 1997,

Vetten et al. 2005, Timchenko et al. 2006, Sharman et al. 2008, Vetten 2008, Grigoras et al. 2009). DNA-R, -S, -C, -M, and -N are homologous DNA components shared by Babu- and Nanoviruses and encode for master Rep (M-Rep), structural (capsid), cell-cycle link, movement, and nuclear shuttle proteins, respectively (Vetten 2008). The functions of DNA-U1, -U2, and -U4 proteins identified from Nanoviruses and DNA-U3 identified from Babuviruses are unknown (Karan et al. 1997, Sharman et al. 2008, Vetten 2008, Grigoras et al. 2009). DNA components encoding other Rep proteins associated with several Babu- and Nanoviruses isolates have also been identified (Hu et al. 2007, Vetten 2008).

Viruses of the *Nanoviridae* have very narrow host range. Natural hosts of the *Nanovirus* species are restricted to legumes, whereas only few monocots like the Musaceae and Zingiberaceae have been reported as hosts for *Babuvirus* species. All of these viruses can be transmitted in a persistent manner by aphids. Viruses in the *Nanoviridae* family have been recorded from across Asia as well as Northern and Eastern Africa and some are more of economic importance than the others (Vetten 2008). The geographic distribution of the *Nanovirus*, *Faba bean necrotic yellow virus* (FBNYV), is wider than many other members of *Nanoviridae*, thus this species is introduced in more detail.

The Genus *Nanovirus*

***Faba bean necrotic yellow virus* (FBNYV)**

FBNYV was first isolated from broad bean near Lattakia, Syria (Katul et al. 1993). Currently, the virus has been reported from Central (Makkouk et al. 1998) and Western Asia (Katul et al. 1993, El-Muadhidi et al. 2001, Makkouk et al. 2002a,b), Northern Africa (Katul et al. 1993, Najar et al. 2000a, Makkouk et al. 2003, Kumari et al. 2008) and Europe (Ortiz et al. 2006). FBNYV has a narrow host range; while the main natural host is broad bean, it can also infect other pulse crops such as chickpea, lentil, common bean, pea, and cowpea (Makkouk et al. 1992, Katul et al. 1993, Franz et al. 1995, Horn et al. 1995). Several wild legume species, as well as perennial species from *Onobrychis* and *Medicago* genera are also listed as FBNYV hosts. The virus may also infect nonleguminous species including *Amaranthus blitoides* S.Watson (Caryophyllales: Amaranthaceae), *Amaranthus retroflexus* L. (Caryophyllales: Amaranthaceae), and *Amaranthus viridis* L. (Caryophyllales: Amaranthaceae) (Mouhanna et al. 1994, Franz et al. 1997).

Infected broad bean plants are stunted, with poorly developed new shoots, leaves, and flowers. Leaf symptoms show progression over time: from interveinal chlorosis, 2 wk after inoculation, to necrosis forming 3–4 wk after inoculation. Young leaves are small and rolled upward, whereas older leaves are rolled downward. Infected plants may die within 5–7 wk after infection. Similar symptoms may be observed in other chickpea, lentil, common bean, pea, and cowpea varieties (Katul et al. 1993). FBNYV, and another two *Nanoviruses*, *Milk vetch dwarf virus* (MDV) and *Subterranean clover stunt virus* (SCSV), are taxonomically close and cause symptoms that are quite similar among legumes (Franz et al. 1996, Sano et al. 1998, Timchenko et al. 2000, Vetten et al. 2005). Yield loss can be up to 100% when young plants are infected with FBNYV. In Egypt, the FBNYV epidemic on broad bean during the 1991–1992 growing season led to 80–90% yield losses (Makkouk et al. 1994).

ELISA or TBIA using either polyclonal (Katul et al. 1993, Kumari et al. 2001) or monoclonal antibodies (Franz et al. 1996) and by dot-blot hybridization (Katul et al. 1995, Franz et al. 1996) are some of the available FBNYV detection methods. In addition, virus-specific primers are developed and available for FBNYV detection by PCR (Shamloul et al. 1999, Kumari et al. 2010).

FBNYV is phloem-limited and is not known to be transmitted by seed or other mechanical means. FBNYV is primarily transmitted by the aphid species *Acy. pisum* and *A. craccivora* in a circulative (and nonpropogative) manner (Franz et al. 1998, Ortiz et al. 2006). *A. fabae* is known to be a poor vector of FBNYV (Katul et al. 1993; Franz et al. 1995, 1998).

Cultural practices, such as delayed planting, roguing, weed management, and chemical control of aphid vectors have been recommended to manage FBNYV (Makkouk and Kumari 2001). Detailed information on these approaches are presented below under 'Managing Vector-Borne Viruses of Pulse Crops'. Although FBNYV resistance has yet to be identified in broad bean, resistant lentil genotypes have been identified (Makkouk et al. 2014). Studies have been performed to develop pathogen-derived resistance against FBNYV, but transgenic broad bean lines with high level of FBNYV resistance are not yet available.

Semi-Persistently Transmitted Viruses

The Family *Secoviridae*

The family *Secoviridae* comprises eight genera: *Cheravirus*, *Sadwavirus*, *Torradovirus*, *Sequivirus, Waikavirus, Comovirus*, *Fabavirus*, and *Nepovirus* as well as some unassigned virus species. Based on phylogenetic analyses, *Comovirus*, *Fabavirus*, and *Nepovirus* are now assigned to the *Comovirinae* subfamily within *Secoviridae* by the International Committee on Taxonomy of Viruses (ICTV) (Thompson et al. 2017). Members of *Secoviridae* possess mono- or bipartite genomes of single-stranded, linear, positive-sense RNA. Virions are non-enveloped, and the genome is encapsidated in isometric particles having a diameter of 25–30 nm. Majority members of *Secoviridae* possess bipartite genomes (*Sequivirus* and *Waikavirus* are monopartite) divided between RNA1 and RNA2. RNA1 encodes a polyprotein with all the information required for replication, while structural proteins are contained in polyprotein encoded by RNA2.

Host ranges of viruses in *Secoviridae* range from narrow to wide. Symptoms on infected plant vary depending on virus and host species. Although transmission of some Sequiviruses requires a helper virus, natural vectors of Sadwaviruses have not been identified. However, many viruses in the family have a known biological vector such as beetles, aphids, nematodes, whiteflies and leafhoppers. Many viruses in family *Secoviridae* can be transmitted by seed and by mechanical inoculation (Thompson et al. 2017).

The Genus *Comovirus*

***Broad bean stain virus* (BBSV)**

BBSV was first isolated from broad bean displaying systemic mottling and leaf deformation in the United Kingdom (Lloyd et al. 1965). BBSV has been found in Africa, Asia, Europe, and the Middle East (Makkouk et al. 1988, Brunt et al. 1996). The natural host range of BBSV is restricted to Fabaceae. BBSV can infect a range of temperate pulses such as lentils, peas, and broad beans (Gibbs et al. 1968, Cockbain et al. 1975, Jones 1978, Makkouk et al. 1992, Kumari et al. 1993, Agarwal and Prasad 1997, Bayaa and Erskine 1998).

The symptoms caused by BBSV range from mild mottling, stunting, deformed pods, and severe necrosis, which may eventually lead to plant death (Kumari and Makkouk 1996, Hamdi and Rizkallah 1997, Al-Khalef et al. 2002). The mottle or mosaic symptoms developed on leaves of infected host plant can be confused with those

caused by other viruses, especially *Broad bean true mosaic virus* (BBTMV) (Gibbs et al. 1968, Cockbain et al. 1976). Chewing marks on the leaf margins caused by beetle vectors may help to distinguish BBSV from others. Serological methods are commonly used for BBSV detection. ELISA and TBIA have been developed for field surveys of BBSV (Kumari and Makkouk 1993, Musil and Gallo1993, Makkouk and Comeau 1994, Ouizbouben and Fortass 1997, Tadesse et al. 1999, Makkouk et al. 2003). Sequence data are very limited for BBSV, and RT-PCR using specific primers is not yet applicable.

Infection of BBSV through broad bean seed has been found to greatly reduce the number of pods formed on the plants reducing seed yield (Vorra-Urai and Cockbain 1977). Incidence of BBSV combined with BBTMV in broad bean in England ranged from 2 to 92% in the field and virus infection resulted in 70% yield loss (Cockbain 1972). Seed yield reductions in lentils have also been reported by several studies with yield losses reaching up to 77% (Kumari et al. 1993, Mabrouk and Mansour 1998). BBSV can also affect broad bean quality and marketability by causing a characteristic staining pattern or brown necrosis and crinkling of the testa (Russo et al. 1982, Omar et al. 1990, El-Dougdoug et al. 1999).

The weevils *Apion arrogans* Wenck., *A. vorax* Herbst, *Sitona crinita* Herbst, and *Sitona lineatus* L. (all Coleoptera: Curculionidae) are known to transmit BBSV. *Apion vorax* can transmit the virus with much higher efficiency than *S. lineatus* (Cockbain et al. 1975, Edwardson and Christie 1991, Makkouk and Kumari 1995b). BBSV can also be transmitted by seed with high efficiency: up to 20% in broad beans (Edwardson and Christie 1991, Mali et al. 2003), 50% in field peas (Musil and Kowalska 1993, Fiedorow and Szlachetka-Wawrzyniak 2002) and 27% in lentils (Kumari et al. 1993, Kumari and Makkouk 1996, Al-Khalaf et al. 2002). Infection of BBSV at pre-flowering stage in some lentils could result in 77% seed yield losses (Mabrouk and Mansour 1998).

The control measures for BBSV include the use of healthy seeds, cultural controls like weed management to reduce alternate hosts of the virus, and beetle control to reduce the virus spread. Dry heat treatment at 70°C for 28 d can help to eliminate virus from the infected seed but reduces germination by 57% (Kumari and Makkouk 1996).

Bean pod mottle virus (BPMV)

BPMV was originally described in common bean in the United States by Zaumeyer and Thomas (1948). However, it became an epidemic in the early 2000s, threatening soybean production (Giesler et al. 2002). In addition to North America, BPMV has also been reported from Asia (Shahraeen et al. 2005), Africa (Odedara et al. 2007), and South America (Zettler et al 1989). Through mechanical inoculation, plants from the three families Apocuanceae, Chenopodiaceae, and Fabaceae have been successfully infected with BPMV (Bradshow et al. 2007). However, the knowledge of natural host plants susceptible to both vectors and BPMY is limited (Bradshaw et al. 2007).

Foliar symptoms in soybean may range from mild mottling to sever mosaic of primarily young leaves, delayed maturity, terminal necrosis and plant death (see Giesler et al. 2002). While BPMW infection of common bean resulted in severe mosaic and malformation of leaves, cowpea (cv. Mashad) remained asymptomatic (Shahraeen et al. 2005). ELISA, RT-PCR, and reverse-transcription loop-mediated isothermal amplification (RT-LAMP) are methods which have been used in detecting BPMV (Wei et al. 2012, and references within).

Several beetles from the families Chrysomelidae, Coccinellidae and Meloidae may transmit BPMV, but the bean leaf beetle *Cerotoma trifurcata* (Förster) (Coleoptera: Chrysomelidae) appears to be the main, and a highly efficient, vector. The virus is noncirculative, remains in the insect digestive system, and is detectable in overwintering adult beetles (Giesler et al. 2002). Although the virus is mechanically transmittable, seed transmission of BPMV is either negligible or nonexistent (Giesler et al. 2002, Krell et al. 2003, Bradshow et al. 2007).

Managing beetle populations with insecticide may reduce BPMV spread. In soybean, delayed planting resulted in increased preclonization mortality of the bean leaf beetle. In addition, early planted trap crops can be used to attract colonizing beetle population allowing a more targeted management of the potential vectors (Giesler et al. 2002).

Nonpersistently Transmitted Viruses

Bromoviridae

The family *Bromoviridae* comprises six genera: *Alfamovirus*, *Anulavirus*, *Bromovirus*, *Cucumovirus*, *Ilarvirus*, and *Oleavirus*. Virions are non-enveloped, having an icosahedral symmetry and a 26–35 nm diameter (genera *Anulavirus*, *Bromovirus*, *Cucumovirus*, and *Ilarvirus*) or pleomorphic, i.e., icosahedral/bacilliform (genera *Alfamovirus*, *Ilarvirus*, and *Oleavirus*) with a diameter of 18–26 nm and lengths of 30–85 nm (Bujarski et al. 2012). The genomes of viruses in *Bromoviridae* consist of three single-stranded, positive-sense RNAs, 5′ end of the RNA particles possesses a cap and 3′ terminus forms either a tRNA-like structure that can be aminoacylated (genera *Bromovirus* and *Cucumovirus*) or forms other structures that cannot be aminoacylated (genera *Alfamovirus*, *Anulavirus*, *Ilarvirus* and *Oleavirus*) (Gallie 1991, Bujarski et al. 2012). RNA1- and RNA2-encoded proteins (1a and 2a) act with host factors as the viral replicase, and RNA3 encodes a movement protein and a coat protein expressed from a sub-genomic RNA which are involved in virus movement. Members of *Cucumovirus* and *Ilarvirus* (subgroups 1 and 2) express a smaller, 2b protein from an additional sgRNA (sgRNA4A), and 2b protein is involved in cell-to-cell movement and post-transcriptional gene silencing (Sztuba-Solinska and Bujarski 2008).

The natural host ranges of *Alfalfa mosaic virus* (AMV) and *Cucumber mosaic virus* (CMV) are extremely broad among the *Bromoviridae*. Most viruses in *Bromoviridae* are transmitted by insects; Alfamoviruses and Cucumoviruses are transmitted by many aphid species in a nonpersistent manner, while most Bromoviruses are transmitted by beetle vectors with low efficiency. *Ilarvirus* and *Anulavirus* are transmitted mechanically by thrips feeding on pollen containing the virus. *Oleavirus*-infected hosts are asymptomatic, and the virus can be transmitted mechanically, but no natural vector has been reported (Bujarski et al. 2012). Some viruses in *Bromoviridae* are seed-borne with varying efficiency depending on the host and the viruses.

The Genus *Alfamovirus*

Alfalfa mosaic virus (AMV)

AMV was first identified as a viral disease infecting alfalfa in the United States, and now is found distributed worldwide. Host range of AMV is very broad and includes at least 697 species in 167 genera of 71 families (Edwardson and Christie 1997). The main host of this virus in nature is alfalfa, but it can infect temperate pulses including chickpeas, broad beans, field peas, and lentils (Hull 1969).

Symptoms induced by AMV infection are affected by factors such as virus strains, host varieties, time of infection, and environmental conditions. In common bean, many AMV strains produce localized necrotic lesions on inoculated leaves (Makkouk et al. 2012). A survey

conducted in Australia showed that stunting, chlorosis, necrosis or streaking on older leaves could be observed on field peas and broad beans, reduced number and deformity of pods may also be observed at later stage of infection; shoot tip necrosis may develop in chickpea and lentils, twisting, leaf deformation, and stunting could also be found in lentils (Aftab and Freeman 2005a). Localized lesions could be induced by virus inoculation in cowpea, but no systemic infection was reported (Aftab and Freeman 2005a). Accurate serological or molecular diagnostic tools such as ELISA and TBIA using polyclonal and monoclonal antibodies, and RT-PCR using virus-specific primers can also be applied for virus detection and to confirm visual diagnoses (Makkouk et al. 1987, Bailiss and Offei 1990, Hajimorad et al. 1990, Bariana et al. 1994, Makkouk and Kumari 1996). Host range test and observation of virus infection in indicator species are widely used to differentiate AMV strains. Serological tests using monoclonal antibodies can also help to distinguish between strains (Hajimorad et al. 1990).

For many years, AMV was not considered of economic importance in cool-season legumes. However, substantial yield loss was reported in both lentils and chickpeas (Kaiser 1973b, Bosque-Pérez and Buddenhagen 1990). Studies performed in Australia showed that in broad bean (cv. Fiord), late virus infection reduced shoot dry weight and seed yield almost in half. In lentil (cv. Matilda), AMV infection decreased shoot dry weight by 74–76%, seed yield by 81–87% and individual seed weight by 10–21%. In chickpea (cv. Tyson), early infection of AMV killed plants while later infection decreased shoot dry weight by 50%, seed yield by 98% and individual seed weight by 90% (Latham et al. 2004). These data indicate AMV may induce an important viral disease of pulse crops.

AMV is transmitted in a nonpersistent manner by over 20 aphid species which include *Acy. pisum*, *A. craccivora*, *A. fabae*, and *Myz. persicae* (Edwardson and Christie 1997). The virus can be mechanically transmitted by sap and can also be seed transmitted in some species. Seed transmission rates of 0.1–5% in lentils, 0.1–1% in chickpeas, and 0.04% in broad bean seeds have been reported (Jones and Coutts 1996, Latham et al. 2004).

So far, no effective host-derived AMV resistance has been identified in peas, chickpeas, and lentils (Ford and Baggett 1965; Hagedorn 1968; Timmerman-Vaughan et al. 2001; Latham and Jones 2001a,b). As AMV is transmitted in a nonpersistent manner by its aphid vectors, chemical control of aphids may not be an effective method for virus management. Use of healthy seed, managing weeds and other overwintering hosts by spatial separation, sowing early to generate early canopy closure, and other cultural practices to minimize virus spread in the field are recommended. Detailed information management options are presented under 'Managing Vector-Borne Viruses of Pulse Crops'.

The Genus *Cucumovirus*

Cucumber mosaic virus (CMV)

CMV is distributed worldwide and its host range is extremely broad, including 85 plant families and up to 1,000 species (Bujarski et al. 2012). Yield loss caused by CMV infection in common bean has been reported throughout the world, especially in the tropics and southern Europe (Palukaitis et al. 1992, Gallitelli 2000). Other major pulses such as chickpea, lentil, lupin, pea, and broad bean are also hosts of CMV.

Symptoms induced by strains of CMV in common bean range from mild mosaic to severe plant malformation (Bird et al. 1974). Chickpeas develop leaf chlorosis, stunting, and reddening or yellowing of whole plants. Chlorosis, leaf distortion, and stunting of the plant can also be observed on lentils and peas. Broad beans may remain asymptomatic or exhibit severe systemic necrosis resulting in plant death. Symptoms caused by CMV in pulses can be very mild and difficult to observe (Aftab and Freeman 2005b). In Western Australia, yield losses of up to 60% caused by CMV infection in lupin crops have been reported (Jones 2000). In Western Australian field experiments, great losses of shoot dry weight (72–81%), seed yield (80–90%) and individual seed weight (17–25%) caused by CMV infection in lentils (cv. Matilda) have been recorded (Latham et al. 2004).

Several hosts like *Chenopodium amaranticolor* Coste and Reyn (Caryophyllales: Amaranthaceae), *Chenopodium quinoa* Willd. (Caryophyllales: Amaranthaceae), *Cucumis sativus* L. (Cucurbitales: Cucurbitaceae), and chickpea may be used for diagnostic purposes, where CMV infection may be visualized as chlorotic and/or necrotic lesions to systemic mosaic. CMV strains and isolates, however, cannot be differentiated through host range evaluations. A rapid CMV detection assay in leaf extracts can be performed through a commercially available immunostrip test (Ohki and Kameya-Iwaki 1996). For routine detection of CMV, ELISA, and RT-PCR methods exist (Wahyuni et al. 1992, Bariana et al. 1994, Elliott et al. 1996, Uga 2005).

CMV is predominantly transmitted in a nonpersistent manner by over 80 species of aphids (Palukaitis et al. 1992, Gallitelli 2000). *Myz. persicae* and *A. gossypii* can transmit the virus efficiently. CMV can also be transmitted mechanically by sap and seed. Seed transmission rates of 10% in common bean, 1% in lentil, 2% in chickpea and a very low rate in pea and broad bean have been reported in previous studies (Hampton and Francki 1992, Latham and Jones 2001a).

Several weed species are hosts of CMV and can serve as virus reservoirs adjacent to cultivated fields. Thus, cultural practices such as barrier crops and weed management are recommended to reduce the risk of crop infection (Makkouk et al. 2014). Since CMV can be transmitted by over 80 aphid species in a nonpersistent manner, vector control is likely ineffective for managing the virus. Although no immunity has yet been detected in chickpea and lentil, variations in symptom expression and susceptibility have been observed (Makkouk et al. 2014).

Potyviridae

Potyviridae is comprised of positive-sense RNA viruses, with non-enveloped flexible, filamentous virus particles measuring 680 to 900 nm in length and 11 to 13 nm in width (Sorel et al. 2014, Valli et al. 2015). The family currently contains the eight genera *Brambyvirus*, *Bymovirus*, *Ipomovirus*, *Macluravirus*, *Poacevirus*, *Potyvirus*, *Rymovirus*, and *Tritimovirus* (Wylie et al. 2017). The genera all have monopartite genomes except for *Bymovirus* which is bipartite (Sorel et al. 2014). The distinctions between virus families and genera are primarily based on nucleotide sequences of the 3′ NCR and the nucleotide sequencing and the amino acid profile of the coat protein (Rybicki and Shukla 1992, Ward et al. 1995, Berger et al. 1997). The family *Potyviridae* is characterized as having terminal untranslated regions with an ORF that is translated into a large polyprotein by a single overlapping ORF and cleaved by enzymes into 10 individual proteins (Adams et al. 2005, Gibbs and Ohshima 2010). A defining microscopic feature in the family is the development of pinwheel-shaped cylindrical inclusions formed in infected plant tissue directly related to a cylindrical inclusion helicase protein associated with virus replication (Danci et al. 2009, Sorel et al. 2014).

Most member of the *Potyviridae* family can be transmitted by mechanical methods but the primary vectors are arthropods or plasmodiophorids (Valli et al. 2015). The principle viruses in the *Potyviridae* family infecting pulses are all from the genus *Potyvirus*. This genus contains the largest number of species of any of the virus

genera (150) all of which are aphid-transmitted (Ward and Shukla 1991, Valli et al. 2015). Three species from the *Potyviridae* family that are known to impact pulses in the Americas are *Bean yellow mosaic virus* (BYMV), *Bean common mosaic virus* (BCMV), and *Pea seedborne mosaic virus* (PSbMV).

The Genus *Potyvirus*

Bean yellow mosaic virus (BYMA)

BYMV was initially described by Doolittle and Jones (1925) when isolated from common bean in the United States and the Netherlands. Virus particles of BYMV are characterized as flexuous, rod-shaped, and measure approximately 750 nm in length (Moghal and Francki 1981) with some variants infecting pea measuring between 788 to 846 nm (Bos et al. 1974, Moghal and Francki 1981). The virion genome is monopartite consisting of positive-sense single-stranded RNA (Makkouk et al. 2012). Analysis of coat protein sequences from isolates collected across four continents revealed seven distinct groups (Wylie et al. 2008). BYMV has been isolated from legumes from Africa (Habib et al. 1981, Yahia et al. 1997), Asia (Kaiser 1973b, Azza and Makkouk 1985, Sharma et al. 2015), Australia (Abu-Samah and Randles 1983), Europe (Doolittle and Jones 1925, Boning 1927, Saiz et al. 1995, Lisa 2000), North America (Doolittle and Jones 1925), and South America (Campos et al. 2013). Thirty-five plant genera, from 11 families, have been reported as BYMV hosts, including Fabaceae (e.g., Bos 1970. Blaszczak 1965, Kaiser and Danesh 1971, Abu-Samah and Randles 1983, Yahia et al. 1997, Gibbs et al. 2000, Uga et al. 2004, Skelton et al. 2007, Kumar et al. 2009). BYMV is not considered to be a major pathogen on peas, chickpeas and lentils in the United States, but has been shown to cause severe yield losses in the past on broad bean in Canada (Frowd and Bernier 1977).

Primary symptoms in pea are expressed as vein clearing, random dark green patches on leaves and more leaf mottling than a mosaic (Larsen 2001a). Additional symptoms on pea associated with early infection include mild stunting, and malformation of leaves and pods. Symptoms expressed on lentil consist of stunting, yellowing, mild mosaic, curled leaves with marginal necrosis, reduced flower and pod formation, and mottling (Larsen and Schwinghamer 2011a). Infected chickpeas exhibit wilting, yellowing, shoot tip necrosis, reddish leaf margins, stunting, leaf deformation, prolific formation of secondary shoots, phloem discoloration, and early senescence (Kaiser and Danesh 1971). Early chickpea infections may result in disfigured leaflets that are narrower than normal. Symptoms in broad bean are greatly impacted by virus strain and broad bean genotype but include yellowing, mosaic, mottling, and green vein banding (Makkouk et al. 2012). Severe infections in broad bean can also result in necrosis of stem and tip tissues and premature death (Frowd and Bernier 1977, Makkouk et al. 2012). Infected broad bean pods may develop necrotic ring spotting with discolored seed (Kaiser 1973a). Laboratory diagnosis is needed to confirm BYMV presence. Polyclonal and monoclonal antibodies are available for detecting BYMV in plant tissue (Werkmeister and Shukla 1991, Ali 2017) and primers have been developed for BYMV (Sharma et al. 2015). DAS-ELISA, PCR, one-step RT-PCR, real-time (rt)-RT-PCR or Immuno Capture (IC)-rt-RT-PCR have been successful in detecting BYMV in plant tissue with rt-RT-PCR and IC-rt-RT-PCR being the most sensitive (Duraisamy et al. 2011, Sharma et al. 2015).

The virus is seed transmitted in pea (Bos et al. 1988), lentil (Makkouk et al. 1992, Kumari et al. 1993) and broad bean (Evans 1973, Makkouk et al. 1992) and chickpea seed (Yahia et al. 1997). Moreover, 21 aphid species have been identified as being vectors of BYMV (Kennedy et al. 1962). The aphid species transmitting BYMV to pea in the Mediterranean region include *Acy. pisum*, *A. fabae*, *A. gossypii*, *Aul. solani*, *Brevicoryne brassicae*, *Myz. persicae*, and *Rhopalosiphum maidis* (Makkouk et al. 2012). In the PNW, aphids transmitting BYMV ranked in order of ability to transmit the virus to pea were *M. euphorbiae*, *Acy. pisum* (two 'biotypes'), *Myz. persicae*, *A. fabae*, *Neomyzus circumflexus*, *M. rosae*, *Thrioaphis riehmi*, *B. helichrysi*, and *Cavariella aegopodii* (Sohi and Swenson 1964). Sohi and Swenson (1964) found that genotypes ('biotypes') of *Acy. pisum* differed substantially in their ability to transmit the BYMV. In the greenhouse, *A. fabae, A. craccivora, Myz. persicae*, *Acy. pisum*, and *Acy. sesbaniae* were able to transmit BYMV to broad bean at transmission efficiencies of 70, 65, 60, 20, and 20%, respectively (Kaiser 1973a).

Aphids transmit BYMV in a nonpersistent manner making it difficult to control this virus using insecticides. In addition, distance from alfalfa, clover, vetch, and gladiolus production fields may play a role in BYMV spread since these crops can harbor both the virus and its aphid vectors. Most pea varieties are resistant to BYMV since they have been bred to possess the single recessive gene *mo* which confers resistance to this virus (Yen and Fry 1956). An additional single recessive resistant gene in pea, *Pmv*, also has been shown to confer resistance to BYMV (Provvidenti 1990). Chickpeas with resistance to BYMV have not been identified and certain lentil lines have been determined to be tolerant, but not resistant (Larsen and Schwinghamer 2011a). Resistance in broad bean to BYMV has been identified with accession 2N138 showing immunity to two different BYMV strains (Gadh and Bernier 1984) and eight genotypes immune to a Syrian strain of BYMV (Makkouk and Kumari 1995a). In addition, two recessive resistant genes *bym-1* and *bym-2* have been identified in broad bean conferring resistance to BYMV (Rohloff and Stulpnagel 1984, Schmidt et al. 1985). The use of clean seed is highly recommended to avoid introduction of BYMV into fields and new growing regions.

Bean common mosaic virus (BCMV)

First described by Stewart and Reddrick in 1917 and Pierce in 1930 (Morales and Bos 1988), BCMW has now been reported from Asia (Shahraeen et al. 2005, Makkouk et al. 2012), Europe (Bos et al. 1988) and North America (Silbernagel et al. 1986). Various beans including broad bean and other plants from the Fabaceae family may host BCMV. The two solanaceous species *Nicotiana clevelandii* and *N. benthamiana* have been reported as nonlegume hosts (Morales and Bos 1988).

Virus particles are filamentous, 750 nm in length and 12–15 nm in width (Hema et al. 2014). Along with *Bean common mosaic necrotic virus*, BCMV has been reported to cause considerable losses that could reach as high as 80% (Hema et al. 2014). Symptoms may vary depending on the virus strains and host genotype. In beans, symptoms include deformed and curled leaves and green vein banding (common mosaic; dark green veins and yellowish or light green interveins) or systemic necrosis, followed by plant death (black root) (see Makkouk et al. 2012 and Hema et al. 2014 for reviews). ELISA and PCR approaches can be used to confirm BCMV infections.

BCMV can be transmitted mechanically and is also known to be seed-borne with the transmission success rates of up to 83% in broad bean and up to 22% in tapari bean (Hema et al. 2014). Several aphid species including *A. fabae*, *Acy. pisum*, and *Myz. persicae* can transmit BCMV in a nonpersistent manner.

As the virus is seed-transmitted, planting virus-free seed can prevent primary infections. Chemical control of vectors and oil applications may also limit secondary pathogen spread. Resistant genotypes are available in common bean (Makkouk et al. 2012). Genotypes

with the *I* gene were protected against BCMV strains including common mosaic symptoms (Hema et al. 2014).

***Pea seedborne mosaic virus* (PSMV)**

PSbMV was initially described by Musil (1966) when isolated from pea in Czechoslovakia. Virus particles of PSbMV are characterized as flexuous, rod-shaped, and measure approximately 750 nm in length and 12 nm in width (Inouye 1967, Hampton et al. 1981, Makkouk et al. 1993). The virion genome is monopartite consisting of positive-sense single-stranded RNA (Makkouk et al. 2012). The primary strains of PSbMV are designated as P-1, P-2, P-3, P-4 and L-1, with P-1 and P-4 being the most prominent strains throughout the United States (Alconero et al. 1986). PSbMV has been isolated from legumes from Northern Africa (Makkouk et al. 1993), throughout Asia (Inouye 1967, Thakur et al. 1984, Makkouk et al. 1993), Australia (Fry and Young 1980, Bos et al. 1988), Europe (Musil 1966, Thottappilly and Schmutter 1968, Bos 1970, Milicic and Grbelja 1977, Kowalska 1979, Pelet 1980), and North America (Hampton and Muehlbauer 1977, Hamilton 1997, Hampton et al. 1981). At least 21 plant genera belonging to 11 families have been reported to host PSbMV (Inouye 1967, Mink et al. 1969, Aapola et al. 1974, Makkouk et al. 1993). PSbMV is considered to be a major pathogen on pea and chickpea and to a lesser degree on lentil and broad bean in the United States.

Severity and type of symptom expression in pulse crops is influenced by cultivar, environment, and virus pathotype. Symptoms in pea are comprised of mosaic leaves, downward or upward leaf curling, tendrils slightly thickened and tightly curled, shortened internodes, malformation and stunting of plant canopy, chlorosis, terminal rosetting of flower structures, vein clearing, seed with striped markings, split or cracked seed coats of fresh or dry seed, and small deformed pods with aborted seed (Mink et al. 1969, Larsen 2001b).

Severity of symptoms on chickpeas are influenced by plant growth stage and virus pathotype but may involve abnormally narrow leaflets that are twisted and curl downward, mosaic, mottling, chlorosis, reddening or necrotic lesions, shoot tip necrosis, stunting, and pod abortion (Larsen and Schwinghamer 2011b). The seed of kabuli-type chickpeas can be reduced in size with abnormal necrotic rings or line markings on the seed coat. Lentils demonstrate the same symptoms as chickpeas except that necrotic rings or line markings on seed is not normally observed (Larsen and Schwinghamer 2011b). Symptoms in broad bean consist of downward curling of leaves, mild mosaic, stunting and reduction in size of tip leaves (Makkouk et al. 1993).

Enzyme-linked immunosorbent assay (ELISA) and immunosorbent electron microscopy (ISEM) have been used to successfully diagnose plants infected with PSbMV, with ISEM being more sensitive to detection than ELISA (Hamilton and Nichols 1978). PSbMV has also been detected using RT-PCR (Kohnen et al. 1992).

PSbMV can be transmitted mechanically through infected plants coming in contact with healthy plants (Congdon et al. 2016). In addition, over 21 aphid species have been documented as transmitting PSbMV to pea (Khetarpal and Maury 1987). Viruliferous *Myz. persicae* were able to transmit the virus to pea after a few minutes of acquisition access period (Stevenson and Hagedorn 1969). *Myzus persicae*, *Acy. pisum*, and *M. euphorbiae* were capable of transmitting the virus to pea in a nonpersistent manner (Gonzalez and Hagedorn 1970), and *M. ephorbiae* was shown to be the most efficient vector followed by *Myz. persicae* and lastly *Acy. pisum* when all three were compared directly in transmission efficiency to pea (Gonzalez and Hagedorn 1971). In addition, alatae were shown to be more efficient than aptere in transmitting the virus to pea (Gonzalez and Hagedorn 1971).

Five aphid species *Myz. persicae*, *A. fabae*, *Acy. pisum*, *A. craccivora*, and *R. padi* demonstrated the ability to transmit PSbMV to broad bean at 100, 94, 90, 88, and 48% transmission success rates, respectively, under greenhouse conditions (Makkouk et al. 1993).

A primary means of managing PSbMV is to plant virus-free seed. The virus is seed-borne in pea (Inouye 1967), lentil (Hampton and Muehlbauer 1977), chickpea (Makkouk et al. 1993) and broad bean (Makkouk et al. 1993), with transmission frequencies that range widely in pulses depending on the cultivar (Hampton and Muehlbauer 1977, Khetarpal and Maury 1987, Makkouk et al. 1993, Coutts et al. 2010). Aphids transmit PSbMV in a nonpresistent manner making it difficult to control this virus using insecticides. Several dry pea cultivars with resistance to PSbMV have been identified in the United States and several pea accessions from the *Pisum* Core Collection located at the USDA Western Regional Plant Introduction Station in Pullman, Washington, have been identified as resistant to all three pathotypes of PSbMV (Alconero et al. 1986). Resistance to PSbMV in pea is conferred by single recessive genes termed *sbm*-1, *sbm*-2, *sbm*-3, and *sbm*-4 (Hagedorn and Gritton 1973, Provvidenti and Alconero 1988, Khetarpal et al. 1990). Four lentil lines with resistance to PSbMV have also been identified with the single recessive gene *sbv* being associated with the resistance (Haddad et al. 1978); however, current cultivars lack this gene and are susceptible to the virus. Chickpea lines screened for resistance to PSbMV have been susceptible to the virus (Alconero et al. 1986) and currently there are no known resistant genes in chickpea or broad bean.

Betaflexiviridae

Betaflexiviridae is comprised of positive-sense, monopartite RNA viruses, with flexible, filamentous particles with helical symmetry measuring 600 to over 1,000 nm in length and 12 to 13 nm in width (Adams et al. 2012). The family currently contains six genera of viruses: *Capillovirus*, *Carlavirus*, *Citrivirus*, *Foveavirus*, *Trichovirus*, and *Vitivirus* (Adams et al. 2012). The distinction between genera is based on virion morphology, genome organization, modes of transmission, coat protein sequences and polymerase gene sequences (Martelli et al. 2007). A defining characteristic of this family of viruses is the alphavirus-like replicase proteins that are always coded for in the first open reading frame going from the 5′ to the 3′ end of the RNA (Martelli et al. 2007). Viruses within this family can be transmitted by mechanical inoculations (Martelli et al. 2007), but vectors include aphids (Hampton and Weber 1983), mites (Malagnini et al. 2016), pseudococcid mealybugs (Bertin et al. 2010, Buzkan et al. 2012), scale insects (Hommay et al. 2008), and white flies (Rosario et al. 2014). The principal viruses in the *Betaflexiviridae* infecting pulses are all from the genus *Carlavirus* and include *Pea streak virus* (PeSV) and *Red clover vein mosaic virus* (RCVMV), discussed below.

The Genus *Carlavirus*

***Pea streak virus* (PeSV)**

PeSV was initially described by Linford (1929) when observed on pea in Maryland and New Jersey and later characterized by Zaumeyer (1937, 1938). Virus particles of PeSV are characterized as flexuous and rod-shaped, measuring 600 to 700 nm in length (Kaiser et al. 1993, Sarkisova et al. 2016). The virion genome is monopartite consisting of positive-sense single-stranded RNA (Adams et al. 2012). Distinct strains of PeSV have not been identified. PeSV has been isolated from plants from Europe (Czech Republic, Germany)

(Wetter and Quantz 1958, Bos et al. 1988, Sarkisova et al. 2016), and North America (United States) (Zaumeyer 1938). Twenty-five susceptible plant genera from Plantaginaceae (Hampton et al. 1978), Solananceae (Kim and Hagedorn 1959), Amaranthaceae (Hampton and Weber 1983), Fabaceae (Zaumeyer 1938, Kim and Hagedorn 1959, Hampton et al. 1978, Kaiser et al. 1993, Sarkisova et al. 2016) and Asteraceae (Kim and Hagedorn 1959) have been reported. PeSV can cause major damage to pea crops and serious epidemics have occurred in Washington and Eastern Oregon in 1983 and 1990 (Larsen 2001c). PeSV has also caused major epidemics in chickpea and lentils in the Palouse region of eastern Washington in 1983, 1990, 1996, and 2005 (Larsen 2011).

Symptom expression in pea results in brown or purple steaks on above-ground plant parts, absence or reduced pod-fill, die-back at shoot tips, pods containing multiple sunken brown/purple spots, general yellowing of plant, and wilting. Early infection can result in premature death of plant prior to pod set. Seeds formed in pods are usually small discolored and malformed. Symptoms in chickpeas are associated with general yellowing of foliage, stunting, necrosis of leaflets, wilting at shoot tips, and browning of phloem tissues (Kaiser et al. 1993). Early chickpea infections can lead to premature death of seedlings (Kaiser et al. 1993). Symptoms in lentils include shoot tip necrosis, general stunting, yellowing of shoot tissue, wilting, and discoloration of vascular tissue. Seeds in pods are small, malformed and reduced in numbers. ELISA has been used successfully to identify isolates of PeSV infecting plants (Kaiser et al. 1993).

PeSV was successfully transmitted from alfalfa to pea at a frequency of 25 to 35% by *Acy. pisum* (Hampton and Weber 1983). After acquisition periods of 1.5 min, *Acy. pisum* was able to still transmit the virus 2 h post-acquisition at 22°C (Hampton and Weber 1983). *Acy. pisum* was also able to transmit PeSV from infected broad bean to both chickpea and broad bean in a nonpersistent manner with transmission frequencies of 5–10% and 50–70%, respectively (Kaiser et al. 1993).

Aphids transmit PeSV in a nonpersistent manner making it difficult to control this virus using insecticides. Locate pulse fields as far away as possible from alfalfa fields since this crop is a susceptible host to PeSV and can harbor virus and viruliferous aphids (Kaiser et al. 1993). PeSV is not known to be seed transmitted. Currently there are no pea or chickpea lines identified with complete resistance to PeSV (Kaiser et al. 1993).

Red clover vein mosaic virus (RCVMV)

RCVMV was initially described by Osborne (1937) when isolated from *Trifolium pretense* L. (Fabales: Fabaceae) in the United States. Virus particles of RCVMV are characterized as flexuous, rod-shaped, and measure 650 nm in length and 12 nm in width (Fletecher et al. 2016). The virion genome is monopartite consisting of positive-sense single-stranded RNA (Adams et al. 2012). RCVMV has been isolated from plants from Asia (Khan et al. 1997, Al-Shahwan et al. 2016), Europe (Bos et al. 1988), and North America (United States) (Osborne 1937). Susceptible plant genera to RCVMV include *Cicer* (Larsen and Myers 1998), *Chenopodium* (Fletcher et al. 2016), *Crotalaria* (Hagedorn et al. 1959), *Lathyrus* (Hagedorn et al. 1959), *Lens* (Larsen and Myers 1998), *Medicago* (Graves and Hagedorn 1956), *Melilotus* (Graves and Hagedorn 1956, *Nicotiana* (Fletcher et al. 2016), *Ornithopus* (Hagedorn et al. 1959), *Phaseolus* (Fletcher et al. 2016), *Pisum* (Fletcher et al. 2016), *Lens* (Larsen and Myers 1998), *Melilotus* (Hagedorn et al. 1959), *Trifolium* (Osborne 1937, Khan et al. 1978), and *Vicia* (Fletcher et al. 2016). RCVMV is currently not considered to be a major issue on peas, chickpeas, broad beans or lentils in the United States. In New Zealand, RCVMV was detected in 66, 36, and 41% of the processing crops of broad beans, pea and common beans, respectively, but the infected plants were mostly asymptomatic and the impact on yield of these crops was not considered to be serious (Fletcher et al. 2016).

Symptoms in pea include chlorosis, mosaic, and vein clearing. Early infections can stunt plant growth, reduce pod fill and kill plants prior to maturity. Rosetting and/or shoot growth from axillary buds can result from infection (Larsen 2001d). Symptoms on chickpea are characterized by stunting, mosaic, axillary bud proliferation, and deformation of leaves and branches (Larsen et al. 1996a). Symptoms on lentil include chlorosis of plant tissue, severe stunting, prolific growth from axillary branches and premature death (Larsen and Myers 1998). Symptoms on infected broad bean in New Zealand were considered to be minimal (Fletcher et al. 2016). Indirect plate-trapped antigen (PTA)-ELISA, DAS-ELISA and RT-PCR has been used to successfully detect RCVMV (Fletcher et al. 2016).

Vectors of RCVMV in pea that transmitted the virus in a non-persistent fashion with as little as a 10-min acquisition time included *Acy. pisum* and *Myz. persicae*, but *A. fabae* was not able to transmit the virus (Hagedorn et al. 1959). Transmission of RCVMV by the clover aphid, *Myzocallis ononidis* (Kalt.) (Hemiptera: Aphididae), has also been observed in clover and is believed to be involved in the high incidence of RCVMV in clover populations in the United States (Graves and Hagedorn 1956). There is very limited information in pulses regarding vector transmission of RCVMV.

Aphids transmit RCVMV in a nonpersistent manner, making it difficult to control this virus using insecticides. Resistant cultivars of pea, chickpea, lentil or broad bean have not been identified, but some pea cultivars with tolerance to RCVMV have been identified (Larsen 2001d).

Managing Vector-Borne Viruses of Pulse Crops

Managing vector-borne viruses in pulses can be challenging since biotic and abiotic variables may impact the outcomes of various management practices. Climate change is one example, where environmental variations can differentially affect different components of such complexes (Coakley et al. 1999, Elbakidze et al. 2011). Ecological and epidemiological knowledge (e.g., Jones 2004, Makkouk and Kumari 2009, Jones 2012) of any particular vector-borne virus complex is important in order to select, develop, and apply the most effective integrated pest management (IPM) practices to minimize both virus and vector reservoir(s), and to reduce both vector movement and population size. For example, the efficacy of some management option may vary based on the mode of transmission, in persistent and nonperistent viruses. Moreover, regional agricultural practices and cropping systems need to be taken into consideration; for instance, planted pasture legumes (annuals and perennials) and/or cover crops may serve as overwintering reservoirs for both viruses and their insect vectors, thus contributing to virus spread and potential epidemics.

Planting resistant genotypes can be considered one of the most important components of IPM, particularly in vector-borne pathogen complexes. These mostly virus-specific approaches, were presented under each of the discussed virus diseases reviewed above. However, this approach currently has limitations; resistance is only available for 11 of the 30 viruses reviewed here, and only seven of those currently affecting pulses in North America (Table 1). On the other hand, genomic resources, such as bacterial artificial chromosome libraries, are available for various pulse crops including peas, chickpea, and beans, enabling the development of molecular markers for marker-assisted selection towards improved yield and resistance

to a wide range of pathogens (see Yu 2012 and Meziadi et al. 2017 for reviews). The bacterial artificial chromosome cloning is a cost-effective method to maintain and manipulate large sequences of DNA (Yu 2012).

Despite having the first report of a transgenic pulse crop in the 80s, advances have primarily been limited to laboratory trials and have not been commercialized at the large scale (Eapen 2008), with the exception of the release of a RNAi-mediated resistant pinto bean to BGMV in Brazil (Bonfim et al. 2007, Tollefson 2011). Environmental risks, consumer concerns and current marketing restrictions are examples of limitations in implementing this available technology. Additionally, cultural and chemical management approaches are recommended to manage virus and vector spread, which should be considered in the development of location-specific IPM protocols.

Removing Weeds and Volunteers

This management approach seeks to minimize sources of infection within and around pulse fields by eliminating potential sources of vectors and viruses, weeks prior to planting. In addition to supporting local vector populations, volunteers and weedy hosts can harbor vectors immigrating from remote overwintering sites, prior to crop emergence. Weed management has been practiced in pulse growing regions of southeastern Australia (Freeman and Aftab 2011) and can be adapted in other pulse growing regions as a component of an IPM approach.

Planting Date and Early Maturing Varieties

Changing planting dates and using early maturing varieties are examples of cultural approaches recommended to manage viral diseases in legumes and other crops (see Makkouk and Kumari 2009 for a review). In general, the more mature the growth stage of the pulse crop prior to infection, the less likely the yield reduction associated with the infection. Planting early when cool environmental conditions favor plant growth but limit vector development, activity and reproduction, provides an advantage to the host, and early maturing varieties can accelerate plants reaching a less vulnerable growth stage prior to infection. Decisions about planting dates need to be made based on clear understandings of the time of vector arrival and their overwintering sites, crop developmental stages, season, climate, and pathosystem specifics.

Roguing

Mostly applicable to small-scale pulse farms and research plots, physical removal of affected plants from fields at the initial stages of infection and vector infestation, may help to reduce virus spread. This approach, however, is not a feasible practice in large-scale farming in the United States (Makkouk and Kumari 2009) and could potentially stimulate aphid dispersal where large populations are present.

Increased Seeding Rate and Ground Cover

This management recommendation is implemented to reduce visual contrasts favored by insects that promote the landing of winged vectors migrating to a crop field. This is because insect vectors, particularly aphids, are known to use color contrast against the background as a cue to select landing sites (Thresh 1982, Kendall et al. 1991). While such practices seem appealing, its efficiency in large-scale farming and/or where overwintering populations of vectors are present needs to be investigated.

Chemical Control

Although herbicides may be applied to eliminate weed and volunteer pulse plants, here we are primarily focused on insecticide applications aiming to limit vector numbers. Several factors, however, need to be considered prior to employing such aggressive measures in pulse and legume production, including accurate identification of the pathosystem and understanding of the ecology of its predominantly present vector. Environmental and human health risks, potential for the development of resistance in vector populations, and the risk of secondary pest outbreaks due to targeting natural enemies are a few reminders, to promote responsible, calculated, and targeted use of insecticides.

Chemical applications may not be as effective where the transmission mechanism is nonpersistent. For instance, for systemic insecticides to work, the vector needs to initiate feeding, which would be sufficient for the virus to be transmitted. Systemic chemistries, however, could reduce the overall vector numbers and limit secondary infections that could result from established vector populations. Moreover, contact insecticides may fail to protect plants against vectors that may move into the field after chemical applications, especially against those that may transmit the virus simply by probing plant tissue (see Makkouk and Kumari 2009 and references within). Neonicotinoid seed treatments proved to be effective where the virus transmission by its vector is persistent (e.g., Makkouki and Kumari 2001). It is, however, important to note that timing of planting, i.e., seed treatment application, plays an important role in determining the effectiveness of the approach as the efficacy of systemic seed treatments is diminished after a few weeks. Thus, later-arriving vectors may be able to feed for longer periods of time before chemicals take effect. Thus, applying chemicals, even seed treatments, must be based on effective monitoring of aphid populations. The potential link between bee mortality and these second-generation insecticides (Godfray et al. 2014), especially in legumes that are frequently visited by pollinators, is another reminder for a responsible and wise use of insecticides.

Future Prospects

Pulses are important rotation crops since they are capable of fixing nitrogen through symbiotic relationships with rhizobium bacteria and are excellent sources of plant protein in developed and developing nations. However, due to their limited acreage in the recent past in the United States, and increased acreage in new growing regions (Montana, North/South Dakota and Nebraska), with different environments and disease pressures, research on the major pulse diseases has been limited. While decades-long research in Mediterranean countries, Europe and Australia has led to major progress in virus classifications, diagnostics, and management in pulse crops and pastures, numerous research avenues have yet to be explored, especially in light of continuing advances in science and technology.

Obtaining location-specific ecological data in the context of our ever-changing climate is a research area that needs continuous attention. In addition, recent findings on host plant associations among aphid biotypes, mixed virus infections, and the role of endosymbionts, and/or pathogens (i.e., viruses) in vectors, in host plant specificity and preference are areas that need further investigation and implementation in epidemiological models. Moreover, identifying virus and vector reservoirs and environmental factors that promote aphid movement into field crops are elements that need to be investigated in further detail.

Many of the vectors of viral pathogens of pulse crops are not endemic or otherwise found in North America. This has so far

protected North American pulse growers from many of the most problematic viruses found in more tropical and Old-World locations (Table 1). However, with increasing global trade and climate change, it is increasingly important to take whatever measures are possible to prevent the accidental introduction of vector insect species or of virus species which can be transmitted by native aphid species.

Host plant resistance to virus and insect vectors, in combination with other cultural management practices, would offer an effective and accepted control approach since nonintegrated control measures have often failed to protect pulse crops (Makkouk et al. 2014). However, in nonpersistently transmitted viruses, resistance to aphid per se may not be sufficient. Efforts toward identifying sources of resistance were outlined in this review. Additional screening studies are needed to identify sources of resistance, specific to virus strains prevalent in specific geographical regions, to render resistance relatively more effective.

Several viruses of pulse crops may also infect perennial alfalfa and other pasture legumes, thus establishing sources of infections in the pulse growing regions of the United States. Although estimates of economic losses to different viruses in alfalfa may not be available, and is perhaps negligible, using less susceptible varieties to aphids and viruses may help to reduce sources of infection and subsequent virus spread into pulse crops; this possibility merits future investigation.

Considerable advances have been made in generating transgenic pulse crops with resistance to pathogens and improved yield (see Eapen 2008, for a review). However, the durability, heritability, and risk assessments (i.e., regarding possible horizontal genetic spill into closely related plants) of particular developed traits needs intensive field research. Hostility towards such efforts has slowed transgenic research in some parts of the world. Limitations and deficiencies exists; as a part of nature however, we are set to evolve and improve our ability to utilize available resources, while minimizing our negative impact on the landscape and the environment, by wise and responsible use of knowledge and technology.

Viral diseases are responsible for major losses in agricultural production and here we presented some of the management practices, which have been practiced for decades in other pulse growing regions of the world. Some of these approaches could be considered by pulse growers in the United States and Canada to develop IPM strategies, which would fit their production system and marketing preferences. Pulse crop production is rapidly expanding in North America, and in this review, we intended to present some of the previous and ongoing efforts to limit losses to vector-borne viruses, and highlight areas that need further investigation, with the aim to improve our regional, and subsequently, global production of pulse crops.

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