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Potato, Viruses, and Seed Certification in the USA to Provide Healthy Propagated Tubers

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ABSTRACT

Potatoes are vegetatively propagated and this can result in the dissemination of pathogens, and viruses in particular, in the tubers. Viruses infecting potato can be categorized by their mechanisms of transmission: aphid transmitted, mechanically transmitted, and soil-borne viruses. The most important viruses in North America include *Potato leafroll virus*, *Potato virus Y*, *X*, *A*, *S*, *M*, *Tobacco rattle virus*, and *Potato mop top virus*. The methods for chemical control of virus disease are greatly influenced by their mechanism of spread in the field. However, tubers play an important role in the spread of virus disease and this has led many regions to develop seed certification programs. The use of certified virus-free tubers by growers has been vital for control of disease worldwide. In addition, breeders have identified genetic resistance that can be introgressed into popular cultivated varieties and provides a method of control that is less costly than chemical application. In recent years there has been an emergence of viruses and recombinant virus strains that have posed new challenges to pathologists for seed certification and for breeders. Here we discuss the latest issues and challenges that viruses pose to potato production.

Keywords: emerging virus disease, genetic resistance, host plant resistance, potato virus, seed certification, tuber production

CONTENTS

INTRODUCTION

Cultivated potato (*Solanum tuberosum* ssp. *tuberosum*) is the world's third most important food crop and arguably is one of the most intensively managed, requiring irrigation, fertilization, and frequent pesticide applications to obtain competitive yields (Knutson *et al.* 1967). Since potatoes are vegetatively propagated, their production differs significantly from that of crops grown from true seed and this creates unique opportunities to propagate and spread many diseases, such as viruses (Khurana 2004). Commercial potato varieties are maintained in tissue culture, and the plantlets are transferred into hydroponic systems or greenhouses to obtain a first generation of potato tubers (**Fig. 1**) (Bohl *et al.* 2000). These "seed" tubers, which are destined only for planting, are multiplied for three or more years in the field before a final multiplication for consumption. Many vascular pathogens easily move into and survive in tubers (but not in true seeds) until the next growing season. Without proper management, seed potato stocks easily reach 100% disease incidence within a few years (Khurana 2004, Knutson *et al.* 1967). There are three major methods used in the US to control the spread of viruses. The first is chemical or cultural control of viruliferous vectors, which limits the spread of viruses from plant to plant. Second, the availability of certified seed that is free from virus prevents the introduction of viruses into production areas. Finally, incorporation of host resistance through breeding of new cultivars brings with it natural control mechanisms for several viruses and is particularly important in countries where seed potato certification programs are not effective.

There are several common aphid-transmitted potato viruses that seed potato certification programs have effectively controlled (not eradicated) (Khurana 2004), but new and emerging virus strains have undermined some of the efforts of these programs. The aphid-transmitted viruses have RNA genomes and belong to the genera *Polerovirus*, *Potyvirus*, and *Carlavirus* (Radcliffe and Ragsdale 2002). All potyviruses and carlaviruses are also mechanically transmissible. In the US, the *Potato leaf roll virus* (PLRV; a polerovirus) currently has a low incidence of occurrence. However, the incidence of *Potato virus A* (PVA; a potyvirus) may be increasing. The most common viruses in the US are *Potato virus Y* (PVY; a potyvirus) and *Potato virus S* (PVS; a carlavirus). The recent spread of necrotic PVY strains has created new challenges for seed potato certification programs. In particular, PLRV and some strains of PVY cause potato tuber necrosis and this impedes market-

Received: 24 August, 2010. Accepted: 10 April, 2011.

Invited Review

E

Tissue culture; 1-5 years; 0%
Planted into greenhouses

↓

Greenhouse; 1 year; 0%
Planted into field

↓

Foundation class seed potatoes;
Planted on seed potato farms
0.25% summer, 0.5% post-harvest

↓

Certified class seed potatoes;
Planted on commercial farms
3% summer, 5% post-harvest

↓

Ware potatoes; any virus level
acceptable, as long as tubers are not
necrotic

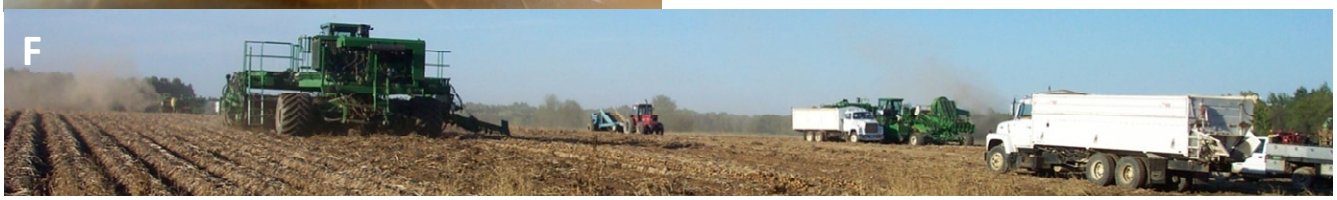

Fig. 1 Vegetative propagation and seed certification. The importance of virus incidence varies depending on the generation of seed potato. In certified seed potato production, there is a zero percent tolerance for virus in tissue culture and other protected environments, such as greenhouses. A tolerance is allowed for field production and this tolerance varies depending on whether the seed is destined for a seed potato farm or a commercial farm. Once potatoes have reached a commercial farm, no virus tolerances are enforced, although buyers may refuse potato tubers that are affected by necrosis. Certified seed potato prices are typically three to ten times higher than ware prices, thus a virus incidence above tolerance levels will cause a significant loss for a seed grower. Since virus levels tend to be low in certified seed, and potatoes can tolerate up to 10% virus incidence without significant losses, viruses such as PVY cause few problems for commercial potato growers. (**A**) potato plantlets in tissue culture; (**B**) potato hydroponic system; (**C**) potatoes growing in a hydroponic system; (**D**) post-harvest inspections of seed potatoes; (**E**) virus tolerances; percentages vary among certification programs; (**F**) potato harvest; potatoes are inspected at harvest and just prior to shipping for symptoms of necrotic viruses.

ability. Planting PVY infected seed tubers can limit marketable yield of some cultivars by ≤ 80% (Hane 1999). Once in the field, poty-, carla- and poleroviruses are spread either through feeding by insect vectors or through mechanical transmission. Aphid life cycles, control, and virus transmission in potato were extensively reviewed and will not be discussed here (Radcliffe and Ragsdale 2002). Only a few aphids colonize potato plants, including the green peach aphid (*Myzus persicae*), the potato aphid (*Macrosiphum euphorbiae*), and the glasshouse potato aphid (*Aulacorthum solani*) (Srinivasan *et al.* 2008). Systemic insecticides that are effective for controlling aphids and other pests include the organophosphate Monitor (methomidiphos) or the neonicotinoids, Admire (imidicloprid) and, more recently, Plati-

Table 1 Virus resistance genes in potato.

Gene	Virus	Source	Mechanism	Chromosome	Marker(s)[1]	Reference(s)
Rx1[3]	PVX	diploid P18	ER	12		Ritter *et al.* 1991
Rx2[3]	PVX	diploid P34	ER	5		Ritter *et al.* 1991
Rx_{adg}	PVX	*S. tuberosum* spp. *andigena* cv. Cara	ER	12		Bendahmane *et al.* 1997; van der Voort *et al.* 1999
Ry_{sto}	PVY	*S. stoloniferum*	ER	12	GP122 (RFLP/CAPS), STM003 (SSR)	Song *et al.* 2005; Valkonen *et al.* 2008
Ry_{adg}	PVY	*S. tuberosum* ssp. *andigena*	ER	11	ADG1, ADG2 (RGL)	Hämäläinen *et al.* 1998; Sorri *et al.* 1999
Ry_{chc}	PVY	*S. chacoense*	ER	9	38-530 (RAPD), CT220 (RFLP)	Hosaka *et al.* 2001; Sato *et al.* 2006
Rlr_{etb}	PLRV	*S. etuberosum*		4	C2_At1g42990 (COSII)	Kelley *et al.* 2009
Rtrv	TRV[2]	*S. tuberosum* clone PA95A33-1		9	AAC-CGT-0347, ACG-CTG-0588 (AFLP)	Khu *et al.* 2008
Na_{adg}	PVA/ PVY	*S. tuberosum* spp. *andigena*	HR	11	Linked to Ry_{adg}	Hämäläinen *et al.* 1998, 2000
Ns	PVS	S. tuberosum spp. andigena		8	$SC811_{454}$ (SCAR)	Szajko *et al.* 2008
Nx_{phu}	PVX	*S. phureja*	HR	9	TG424 (RFLP)	Tommiska *et al.* 1998
Nb	PVX	*S. tuberosum* cv. Pendland Ivory	HR	5	SPUD237, GP21 (SCAR)	De Jong *et al.* 1997a
Gm	PVM	*S. gourlayi*	Inoculation resistance	9	$SC878_{885}$ (SCAR)	Dziewonska and Ostrowska 1977; Marczewski *et al.* 2006
Rm	PVM	*S. megistracrolobum*	HR	11	$GP250_{510}$, $GP283_{320}$ (CAPS)	Ross 1986; Marczewski *et al.* 2006
ra	PVA	*S. tuberosum* spp. *andigena*	transport	unknown		Hämäläinen *et al.* 2000

[1] Abbreviations: RFLP=restriction fragment length polymorphism, CAPS=cleaved amplified polymorphic sequence, SSR=simple sequence repeat, RGL=resistance gene like fragments, RAPD=random amplified polymorphic DNA, COS=conserved orthologous sequence, AFLP=amplified fragment length polymorphism, SCAR=sequence characterized amplified region

[2] QTL analyses for corky ringspot disease, of which TRV is a major component

[3] Rx1 and Rx2 are the only genes known to encode CC-NB-LRR proteins.

num (thiamethoxam) (Unruh and Willett 2008; Cutler *et al.* 2009). Because of the widespread use of these insecticides in the US and Canada, the polerovirus PLRV, which is persistently transmitted by potato-colonizing aphids, such as the green peach aphid, is now rarely found (Mowry 2005). We discuss the challenges in controlling major potato viruses and their insect vectors in the following sections of this article.

There are also non-aphid transmitted viruses which are targeted by seed certification programs and breeders (Agindotan *et al.* 2007). Of these, the potexvirus *Potato virus X* (PVX) occurs worldwide, but is only mechanically transmissible. PVX can be effectively controlled (but not eradicated) by seed potato certification in the US and Canada, and is rarely found in other countries with similarly effective certification programs. The distribution of soil-borne viruses, *Potato mop top virus* (PMTV; a pomovirus) and *Tobacco rattle virus* (TRV; a tobravirus) in North America appears to be spreading (Kirk 2008, Tenorio *et al.* 2006, Xenophontos *et al.* 1998). Because PMTV and TRV cause tuber necrosis that limits marketability, farmers are becoming concerned about the potential impact of these viruses on production in the US and Canada. Soil-borne diseases, such as TRV and PMTV, are likely to be the biggest pathogen challenges for potato growers in the 21st century (Santala *et al.* 2010).

Recent reports of virus diseases causing substantial yield losses and diminishing tuber quality have led breeders to increase their focus on incorporation of virus resistant germplasm into their programs (Barker and Dale 2006, David *et al.* 2010, Gieck *et al.* 2007, Karasev *et al.* 2008). Potato breeders have difficulty visually assessing genetic virus resistance in field plots because some common potato viruses, such as PVY, TRV, PVS, and PVX may cause mild, transient, or no foliar symptoms, although the same viruses have significant effects on tuber yield and quality. Breeding for resistance traits in potato is further complicated because cultivated potato (*Solanum tuberosum* L.) is a tetraploid that acts as a self-compatible species due to heterozygosity at the self-incompatibility locus in the pollen. Because of its outcrossing nature, most loci are heterozygous, which impacts breeding efforts to introduce agronomic traits into advanced cultivars. Breeding for resistance is typically a multistage process that includes screening germplasm for resistance traits, determining the genetic inheritance of these traits, finding markers that can be used for marker assisted selection (Collard and Mackill 2008), and introgression of resistance into cultivars while preserving yield. This process can take up to 15 years once a specific trait is found and suitable crosses are made. Fortunately, wild species of potato provide ample diversity for sources of resistance traits and the development of markers that span the entire potato genome is rapidly progressing. In this article, we discuss recent progress in identification and cloning of useful virus resistance genes (**Table 1**).

There are three phenotypic host responses associated with potato virus resistance. First, extreme resistance (ER) is asymptomatic after virus inoculation. Typically, minimal or no virus can be detected using sensitive techniques (e.g. ELISA or PCR) in plants with ER. Genes that confer ER can be effective against multiple virus strains or multiple viruses (Solomon-Blackburn and Barker 2001, Song *et al.* 2005, Whitworth *et al.* 2009). Second, hypersensitive resistance (HR), is associated with programmed cell death and necrosis that occurs after inoculation and limits pathogen spread (Greenberg and Yao 2004, Heath 2000, Stuible and Kombrink 2004). In contrast to ER, virus can be detected in plants with HR and resistance is usually strain specific (Solomon-Blackburn and Barker 2001). Typically, viral resistance genes in potato are named based on the response they generate and the virus they recognize. For example, extreme resistance genes start with an "R" and genes for hypersensitive resistance begin with "N". Therefore, ER to PVY is conferred by the *Ry* gene and HR to PVY is conferred by *Ny*. If the genes come from a specific genetic source, the species abbreviation may be subscripted after the gene name (e.g. Ry_{sto}). The third host response is tolerance to virus infection (high virus titer in symptomless plants), which is not desirable for breeding programs (Thill and Mollov 2004). This phenomenon presents new challenges for potato breeders to select for resistant individuals based on symptom expression alone and for proper certification of disease-free seed. Propagating susceptible but symptomless plants can create an environment for viruses to thrive in locations that were once virus-free.

Biotechnology offers solutions to the losses incurred by potato viruses. Plants resistant to all of the major potato viruses have been constructed. This article will not discuss engineered resistance to potato viruses but there are a number of papers available on this topic (Kawchuk *et al.* 1991;

Tacke *et al.* 1996; (Ares *et al.* 1998, Barker *et al.* 1998, Bukovinszki *et al.* 2007, Ehrenfeld *et al.* 2004, Gargouri-Bouzid *et al.* 2006, Kawchuk *et al.* 1991, Melander *et al.* 2001, Tacke *et al.* 1996, Vazquez Rovere *et al.* 2001). In fact, potatoes resistant to PVY and PLRV have been commercially grown in North America (Coffin *et al.* 1997, Lawson *et al.* 2001). Unfortunately, they were not accepted by the processing industry or international trading partners and at this time, transgenic potatoes are not widely grown. Some potato viruses are important biotechnological tools; both PVX and TRV are commonly used as vectors for virus induced gene silencing and methods for using this technology are summarized nicely by (Lu *et al.* 2003). Technological advances have also improved our capacity to detect and diagnose potato viruses. ELISA is commonly used because of its simplicity and relatively low cost. However, multiplex PCR-based assays are now available for several potato viruses (Agindotan *et al.* 2007, Mortimer-Jones *et al.* 2009, Ryazantsev and Zavriev 2009). In addition, macroarrays detecting the most important potato viruses have been developed (Agindotan and Perry 2007, Agindotan and Perry 2008). Immunoassay strips, which allow quick and accurate diagnosis in the field, are increasing in popularity among inspectors and plant breeders. Even though there have been substantial advances in virus detection, indicator plants are still sometimes used when potato germplasm is moved across borders to inhibit the spread of uncharacterized viruses.

In this review, we have combined discussion of recent research related to the control of major potato viruses, all of which are positive-sense RNA viruses, including the biology of the viruses, their vectors, and the steps being taken to curtail their spread through certification and breeding of resistant varieties.

Aphid-transmitted potyviruses: *Potato virus Y* (PVY) and *Potato virus A* (PVA)

PVY and PVA are members of the genus *Potyvirus*, one of the two largest genera of plant-infecting viruses. Potyviruses are flexuous rods and have monopartite genomes that are just under 10-kilobases long. Potyviruses encode one polyprotein that is cleaved into 10 proteins (Urcuqui-Inchima *et al.* 2001) as well as a recently discovered protein embedded within the genome (Chung *et al.* 2008). PVY and PVA infect mainly *Solanaceae*, including potato, tomato, pepper, tobacco and eggplant. PVY is more widespread and causes greater losses than PVA, so this review will focus on PVY. Infection with PVY can reduce potato yields by over 60% (Nolte *et al.* 2003, Whitworth *et al.* 2006) and planting seed potatoes infected with PVY can reduce yields up to 80% (Bantarri *et al.* 1993, Hane 1999).

There are multiple strains of PVY that cause foliar symptoms ranging from mild mosaic to necrosis. The PVY^{O} strain is the common strain that causes mosaic symptoms in most hosts but can cause foliar necrosis on some potato varieties. For example, when the potato variety 'Goldrush' is infected with PVY^{O}, the leaves show a bright yellow mosaic disease and eventually turn necrotic and senesce early (**Fig. 2C**). PVY^{O} also causes foliar necrosis and on the potato variety 'Red Norland', and these plants die before tubers are set. PVY^{O} does not typically cause tuber necrosis, but causes significant yield reduction (**Figs. 2A, 2B**). PVY^{N} is a tuber necrotic strain of PVY that originated in South America (Inoue-Nagata *et al.* 2001, Weidemann 1988) and appeared later in Europe in the 1960s. It has since been reported in many countries around the world (Karasev *et al.* 2008, Volkov *et al.* 2009, Weidemann 1988). Until 1990, PVY^{O} was predominant in North America but several necrotic strains (PVY^{N}, PVY^{NTN}, and $PVY^{N:O}$) have begun to appear (Karasev *et al.* 2008). The tuber necrotic strain, PVY^{N} and a member of the PVY^{N} subgroup, PVY^{NTN}, sometimes cause a more mild mosaic disease on leaves than PVY^{O}, but cause tuber necrosis in certain potato varieties. This necrosis, referred to as potato tuber necrotic ringspot disease (PTNRD), diminishes tuber marketability.

The PVY genome has a high degree of genetic variability and there are frequent reports of recombination among PVY strains (Baldauf *et al.* 2006, Inoue-Nagata *et al.* 2001, Singh *et al.* 2003). Several PVY recombinants have been documented since the 1980s including PVY^{N}-Wi, $PVY^{N:O}$, and NE-11 (Hu *et al.* 2009, Ogawa *et al.* 2008). Each of these arose from various recombination events between PVY^{O} and PVY^{N} strains. $PVY^{N:O}$ is among the most common strains currently found in North America. It causes mild foliar symptoms in some potato varieties and tuber necrosis in a few varieties. Because the foliar symptoms of PVY^{N} and $PVY^{N:O}$ are mild on some varieties, inspectors cannot easily identify infected plants when certifying seed potato lots. Seed growers cannot easily identify and rogue infected plants as a means to reduce the level of virus inoculum in a field. As a result, PVY levels can quickly increase.

The current overall goal for most producers is to reduce the incidence of PVY. However, given that the tuber necrotic strains sometimes show mild foliar symptoms, accurate diagnosis of virus strains in the field is important for growers and pathologists to predict harvest yields. Because of the variety of symptoms caused by PVY strains (from minor to severe yield losses), plant pathologists need strain specific diagnostic techniques to monitor the threat to production fields and to make informed and useful disease management recommendations to growers. However, this is not always practical or achievable for several reasons. The presence of multiple PVY strains within lots of potato and even in the same plant, as well as their frequent recombinetion creates challenges for plant pathologists to accurately identify PVY strains and predict their impact on production. In addition, there is no single method currently available that distinguishes all strains. Serological techniques can distinguish between PVY^{O} and PVY^{N}, but do not differentiate N from NTN strains nor O from N:O strains (Karasev *et al.* 2010). Given that the necrotic strains PVY^{N} and PVY^{NTN} cause veinal necrosis on tobacco plants (Tribodet *et al.* 2005), a tobacco bioassay remains the best method to identify necrotic isolates, but cannot be performed on a large scale for disease diagnosis. Many primer sets used in RT-PCR assays do not detect all possible strains, and the cost of these assays is prohibitive for use in seed potato certification (Nie and Singh 2002). Thus in principle, accurate diagnosis involves a combination of tobacco bioassay, serological detection, and RT-PCR tests. Considering the costs and extensive assays needed to identify virus strains, it is impractical for growers with large production systems and seed potato certification agencies to focus on the accurate diagnosis of a particular PVY strain or the management of individual PVY strains.

Potyviruses, such as PVY and PVA, are spread in a non-persistent manner by numerous aphid species and there are chemical and cultural control methods used to limit insects as a means to limit virus infection. Since virus acquisition and spread take only seconds, systemic insecticides are not effective for stopping the spread of either virus (Nauen and Denholm 2005). Insecticides effectively control potato colonizing aphids (Parker *et al.* 2006), thus the most agronomically important aphid vectors for potato potyviruses are species that transiently associate with plants as they forage on potato in search for suitable host plants. The most important aphid species for PVY and PVA transmission vary among locations and years since the aphid species moving through a crop is affected by weather patterns, environmental conditions, and neighboring crops (Raccah 1986). Low temperatures affect aphid survival and therefore cold winters can impact the spread of aphid populations and onset of viral diseases in the spring. However, climate warming is affecting aphid populations. The warmer winter temperatures favor aphid populations, but the hotter summers may be threatening. Thus, as seasonal climates change, we expect to see changes in the patterns of vector and disease spread (Hazell *et al.* 2010a, 2010b). Aphid populations

can also be affected by the landscape structure surrounding potato fields. Researchers have shown that the sizes of bordering grassy fields can affect predation of potato pests (Werling and Gratton 2010). Since some aphid species preferentially land on field edges, surrounding fields of susceptible potatoes with either resistant potato varieties or crops that are not hosts of PVY, such as winter wheat, can be effective. Furthermore, combining cultural practices to control the spread of PVY such as the use of mineral oil sprays, which irritate aphids and thus inhibit probing, with carefully selected crop border methods effectively reduces the incidence of PVY (Boiteau *et al.* 2009). Video recordings of insect movements show that mineral oil impedes stylet penetration and is an effective antifeedant treatment reducing the proportion of aphids that can transmit PVY (Powell *et al.* 1998). Two other cultural control methods used by seed potato growers to control aphid-borne viruses include use of green sprouting (chitting) causing plants to emerge and mature earlier, thus benefiting from mature plant resistance, and early vine kill to avoid late season aphid flights (Saucke and Doring 2004). Purchasing certified seed is also an important method to control PVY and PVA. Farmers growing potatoes for fresh use or processing rely on certified seed potatoes and rarely use other chemical or cultural control methods to limit virus spread.

In the mid-1990's, PVY^N was reported in Canada and subsequently PVY incidence in seed potatoes has increased in North America over the past decade. The Canadian government tried to eradicate PVY^N from seed producing areas in the 1990s through implementation of a necrotic virus management plan (USDA 2004). This plan was also infused with trade goals, since it slowed movement of seed potatoes from Canada into the US and other countries. Canadians soon found PVY^N eradiation to be impossible. Diagnostic, regulatory, and inspection problems surrounding the spread of PVY^N in Canada led to a $75 million, 15-year lawsuit involving around 180 growers.

An important component of the rising incidence of PVY in the 1990s was the popularity of several new russet varieties in the US that were susceptible to PVY. These varieties showed mild or no foliar symptoms, but served as large reservoirs for PVY, increasing the risk of virus spread to other varieties as well as providing more opportunities for virus mutation and recombination. In addition, changes in aphid populations, including the invasion of soybean aphid into the Midwest, may have driven the increase in PVY incidence (Davis *et al.* 2005). Added factors driving PVY incidence such as changes in rotation crops planted by potato growers as well as climate change influence local aphid populations, where aphids overwinter and how quickly they reach northern seed potato growing regions.

In 2002, tuber necrotic strains of PVY were first reported in the Pacific Northwest of the US. This triggered both a large survey of PVY strains in Canada and the US and a review of the necrotic potato virus management plan. The survey revealed that many PVY strains are present in the US (Piche *et al.* 2004). As previously mentioned, researchers found that no single immunoassay or RT-PCR test could classify a strain as a tuber necrotic strain. Therefore, rather than attempting to eradicate specific strains, potato growers are now using strategies to reduce levels of PVY inoculum in seed potato lots and the use of genetic plant resistance to PVY has become a higher priority. New protocols are being implemented by the seed potato certification programs in the US and Canada that are designed to reduce the incidence of PVY across North America. These changes include widespread use of post-harvest testing and shipping point inspections to eliminate lots with high incidence of PVY, and especially tuber necrosis strains of PVY. Furthermore, the revised Canada/US-Management Plan for potato viruses that cause tuber necrosis was expanded to include TRV, PMTV, and *Alfalfa mosaic virus*. This management plan is a result of an intersection of agriculture, science, and politics, and will not eradicate or even slow the spread of these viruses. Rather, it provides a mechanism to remove seed potato lots with high levels of tuber necrosis and a mechanism to preserve trade between the US and Canada.

While PVY is well controlled among commercial farms, it remains an important problem in seed production. PVY is the main reason for rejection of seed potato lots from certification and in some years, this virus can cause downgrading of more than half (and sometimes all) of the seed lots of susceptible varieties. This leads to shortages of certified seed potatoes, forcing farmers to change their plans and affecting their contracts and long-term relationships with customers. When seed lots are downgraded, commercial farmers will plant non-certified seed, which leads to unpredictable yields and quality. This also affects their ability to buy crop insurance and may cause the grower to lose status as a reliable potato grower. Seed certification programs in some states will occasionally raise the tolerable PVY incidence rate if numerous seed lots have been downgraded, but this typically leads to increased levels of PVY since inoculum levels will be high in the following year.

Although genetic resistance remains the best long-term strategy for combating viral disease of potato, most of the potato acreage grown in North America is susceptible to PVY since other disease resistance and processing characteristics remain more important than resistance. It is difficult for growers and seed potato certification inspectors to see PVY symptoms on these varieties, which include mainly 'Russet Norkotah', 'Silverton Russet', and 'Gem Russet' (Hane 1999, Rykbost *et al.* 1999). Now potato breeders are learning about the genetic backgrounds of the varieties that they plant (Mollov and Thill 2004) and have increased efforts to release varieties that are resistant to PVY, or that at least show typical symptoms of this virus.

Therefore, breeding for resistance to this virus is especially important. Three sources of ER to PVY exist in potato: Ry_{sto} from *S. stoloniferum* Schlechtd. et Bché, Ry_{chc} from *S. chacoense* Bitt. (Asama *et al.* 1982), and Ry_{adg} from *S. tuberosum* ssp. *andigena* Hawkes (Munoz *et al.* 1975). These three genes are considered different from one another since they map to different locations within their respective genomes. Resistance encoded by the Ry_{sto} gene has been incorporated into several cultivars developed through European breeding programs (Barker and Dale 2006, Ross 1986). Cleaved-amplified polymorphic sequence (CAPS) markers derived from the restriction fragment polymorphism loci GP122 were used to detect the Ry_{sto} gene in different germplasm sources (Valkonen *et al.* 2008). The $Ry\text{-}f_{sto}$ gene was also introgressed from *S. stoloniferum* and both the Ry_{sto} and $Ry\text{-}f_{sto}$ genes map to chromosome XII (Flis *et al.* 2005, Song *et al.* 2005, Valkonen *et al.* 2008). At first, Ry_{sto} and $Ry\text{-}f_{sto}$ were reported to be separate genes (Flis *et al.* 2005, Song *et al.* 2005, Witek *et al.* 2006); however, Valkonen *et al.* (2008) identified markers linked to both Ry_{sto} and $Ry\text{-}f_{sto}$ that have identical estimated genetic distances to the resistance gene, suggesting that these genes may be identical.

PVY resistance from *S. chacoense* has not been widely utilized in potato breeding although some cultivars with the Ry_{chc} gene have been developed (Hosaka *et al.* 2001, Matsuoka *et al.* 1995). Ry_{chc} maps to potato chromosome IX (Hosaka *et al.* 2001, Sato *et al.* 2006) and is located in a region with several NB-LRR genes with nucleotide similarity to the tomato *Sw-5* gene conferring resistance to tomato spotted wilt virus (D. Halterman and X. Cai, unpublished data, Brommonschenkel *et al.* 2000).

Resistance derived from *S. tuberosum* ssp. *andigena* confers broad spectrum ER to various PVY strains, including the recombinant strain $PVY^{N:O}$ (Gebhardt and Valkonen 2001, Munoz *et al.* 1975, Whitworth *et al.* 2009). Markers associated with Ry_{adg} place the gene on chromosome XI (Hämäläinen *et al.* 1998). These markers include two sequence characterized amplified regions (SCAR) (Kasai *et al.* 2000) and a CAPS (Sorri *et al.* 1999). Interestingly, the latter marker shares homology to the kinase motif of previously cloned NB-LRR protein *N*, which confers resistance to *Tobacco mosaic virus* (TMV) in *Nicotiana gluti-*

nosa, suggesting the possibility that the marker is part of the *R* gene itself (Hämäläinen *et al.* 1998, Sorri *et al.* 1999).

S. tuberosum cultivars are a source of genes conferring HR to PVY and are easily accessible to breeders as sources of resistance. The Ny_{tbr} gene is recognized by the common strain of PVY and is located on chromosome IV (Celebi-Toprak *et al.* 2002). Another PVY HR gene, named *Ny-1*, recognizes both common and necrotic strains of PVY (Szajko *et al.* 2008). *Ny-1* is located on chromosome IX in a region near the Ry_{chc} gene, suggesting that they both might be members of a larger *R* gene hotspot in this region of the genome (Szajko *et al.* 2008).

Resistance to PVA does not receive the same amount of interest as other potato viruses with respect to breeding for resistance. This is possibly due to the rare occurrences of PVA or difficulties in breeding for PVA resistance due to a lack of symptomology of infected plants. Hypersensitive resistance to PVA is derived from *S. tuberosum* ssp. *andigena* and cosegregates with Ry_{adg} on chromosome XI (Barker 1997, Hämäläinen *et al.* 2000). Another recessive gene, *ra*, is either linked to or allelic with Ry_{chc} and suppresses virus transport. The resistance phenotype mediated by *ra* does not include cell death, suggesting a mechanism disparate from ER or HR (Hämäläinen *et al.* 2000).

More than other pathogens, viruses depend upon host cell processes and as a result, mutations in genes encoding processes critical for virus replication can cause plants to acquire resistance to specific viruses. The translation initiation factor eIF4E is one such host factor and tomato (*Lycopersicon esculentum*) and pepper (*Capsicum annum*) plants with natural or induced mutations in this gene are resistant to a range of potyviruses, including PVY (Piron *et al.* 2010, Ruffel *et al.* 2002). Resistance can also be conferred by over-expressing appropriate alleles of eIF4E in susceptible plants (Kang *et al.* 2007). This exciting finding opens the door to identification of mutant potato lines that are resistant to the potyviruses PVY and PVA.

Aphid-transmitted polerovirus: PLRV

PLRV is a single-stranded positive-sense RNA virus with a monopartite linear genome just under 6-kb long. PLRV belongs to the genus *Polerovirus* in the family *Luteoviridae* (Taliansky *et al.* 2003). Like other poleroviruses, PLRV is restricted to the phloem and does not spread into leaf mesophyll or other foliar tissues. The young leaves of PRLV infected plants stand upright, may be red at the margins, and may be slightly pale. Both the upper and lower leaves roll and the lower leaves have a leathery texture that is characteristic of PLRV infection (Peters 1987). Late in the season, some strains of PRLV cause a disease called net necrosis, which is the selective damage and death to cells in the vascular tissues of the tuber. The symptoms of net necrosis include small brown speckles or strands of discolored tissue that start at the stem end and extend as far as half way through the tuber. Net necrosis can occur in the absence of foliar symptoms and can intensify during prolonged storage (Manzer *et al.* 1982). In various potato varieties, there are reductions in seed yields, numbers of stems per plant, and in the size and number of marketable tubers (Hamm 1999, Harper *et al.* 1975).

PLRV is transmitted by at least 10 species of aphids, in a persistent circulative manner, but green peach and potato aphid are the most important vectors of this virus for potato (Rouze-Jouan *et al.* 2001, Srinivasan *et al.* 2008). Upon acquisition, aphids can transmit PLRV for their entire life. PLRV is uniquely interesting because it has been shown to alter plant volatiles in a manner that promotes aphid feeding and reproduction and this appears to be an effective adaptation for optimizing virus spread in agricultural systems. PLRV infected plants emit volatiles that attract feeding aphids, unlike healthy plants, and this likely improves vector uptake and transmission (Alvarez *et al.* 2007). It has also been shown that PLRV infected plants are better hosts for reproductive *M. persicae* than healthy plants (Castle and Berger 1993). PLRV infected fields also influence aphid immigration and emigration. In controlled experiments, researchers have shown that the influence of PLRV on aphid behavior depends on disease progression and plant age (Eigenbrode *et al.* 2002, Werner *et al.* 2009). Winged (alate) aphids spread the virus for long distances between fields, and non-winged (apterous) aphids are important in plant-to-plant spread within a field (Taliansky *et al.* 2003). Aphid feeding introduces PLRV into the phloem tissue where the virus multiplies, spreads, and initiates disease. If infected plants produce tubers, these tubers will grow into symptomatic plants that produce little, but that serve as inoculum sources in the following year.

Interestingly, isolates of PLRV show little sequence divergence, and no strains have been entered into the classification indexes. Studies comparing geographic isolates from several continents have found few differences with the published sequences, thus geography and aphid vectors do not function as evolutionary pressures for PLRV. Diagnosis is therefore straightforward using DAS-ELISA or RT-PCR (Du 2006).

The most widely applied methods to effectively control PLRV include the use of insecticides and seed potato certification. Insecticide application suppresses aphid populations in the field, thereby preventing inoculation and net necrosis in tubers (Roosen *et al.* 1997). The recent and widespread use of systemic insecticides has greatly reduced PLRV incidence in North America. Seed potato certification officials now report only a few plants infected with PLRV. For example, no seed potato lots have been rejected in Wisconsin due to PLRV incidence for at least the past decade. There are several cultural methods to control PLRV that are not widely used, mostly because of the high costs for implementing these methods in large production systems. These include heat treatment, or even cryogenic treatment of micro-plants, as a means to eliminate PLRV from tissue culture generated plants (Wang *et al.* 2006). Whitewash sprays and reflective materials are effective for controlling aphid-transmitted PLRV as well as PVY (Marco 1986) in certain small plots. For small plots, covering plants with a white net or spraying them with mineral oil or zinc containing Loven, Yalbin, Dabak (Tapazol Co.), and Virol effectively reduces PLRV as well as PVY incidence. The whitewash sprays and netting increase leaf reflectivity making them less attractive to aphids (Marco 1986). Heat treatment of tubers can eliminate PLRV, but not PVY, PVA, PVS, PVX, or TRV (Kaiser 1980). Heat-treated tubers show better survival and progeny plants are virus-free.

Most potato varieties grown in the US are susceptible to PLRV (Corsini and Brown 2001) and losses due to planting PLRV infected seed can reduce yields by up to 80% (Bantarri *et al.* 1993). The polygenic nature of most PLRV resistance sources in potato has made breeding for resistance difficult (Barker *et al.* 1994, Jansky 2000, Swiezynski *et al.* 1990). A major QTL (PLRV.1) for PLRV resistance was identified in diploid populations containing *S. chacoense*, *S. yugasense*, and *S. tuberosum* germplasm and contributes 60% of the variance on chromosome XI (Marczewski *et al.* 2001). A second QTL (PLRV.4) was also identified on the same chromosome (Marczewski *et al.* 2004). There are some monogenic sources of resistance to PLRV (Barker and Solomon 1990, Brown and Thomas 1993). For example, breeding programs have recently included monogenic PLRV resistance derived from *S. etuberosum* Lindl., which is a non-tuberizing wild species of potato (Kelley *et al.* 2009, Novy *et al.* 2007, Novy *et al.* 2002). This resistance gene, Rlr_{etb}, was incorporated through somatic fusion between *S. etuberosum* and cultivated potato (Novy and Helgeson 1994). The resistance was mapped to within 13.6 cM of COSII marker C2_A1g42990 on chromosome IV (Gillen and Novy 2007, Kelley *et al.* 2009, Novy *et al.* 2002).

Mechanically transmitted potexvirus and carlaviruses: PVX, PVM and PVS

PVX is the type member of the *Potexvirus* genus, in contrast, PVS and PVM are little studied members of the *Carlavirus* genus; all are members of the family *Flexiviridae* (Martelli *et al.* 2009). Potexviruses and carlaviruses have monopartite genomes of roughly 8.5 to 9.5 kilobases. The linear arrangement of genes is similar with the viral replicase located near the 5′ end of the genome, followed by the movement and coat protein genes. Two important features unique to carlaviruses are the genes encoding papain-like cysteine protease and additional nucleic-acid-binding protein which are located at the 3′ end of the carlavirus genome (Adams *et al.* 2004). The genomes of both viruses are packaged into flexuous rod shaped particles.

Both PVS and PVX occur wherever potatoes are grown, however the common strains rarely cause symptoms on potato (Pourrahim *et al.* 2007, Salazar 2006). There are numerous strains of PVX and these are classified into four groups according to their reactions with dominant resistance genes *Nb* and *Nx* that are directly involved in strain-specific recognition of PVX in cultivated potato varieties (Valkonen *et al.* 1994). Thus PVX strains are defined based on the classic gene-for-gene resistance response. Group 1 strains (Roth1, XS, P551, NL1 and Scot10) cause HR in the presence of *Nb* or *Nx,* group 2 strains (EX, DY, CP2 and WS2) cause HR in the presence of *Nb*, group 3 strains (UK3, S, X3, S6111, XA, CPG, KP, CP and CT23) cause HR with *Nx*, and group 4 strains (HB and CP4) react with neither *Nb* or *Nx* (Malcuit *et al.* 2000, Valkonen *et al.* 1994). The viral coat protein is the determinant for resistance mediated by *Nx* and *Rx* (Bendahmane *et al.* 1995, Santa Cruz and Baulcombe 1995) while the TGBp1 protein is responsible for *Nb* mediated resistance (Malcuit *et al.* 1999). Unlike the PVY strains, recombination is not the primary mechanism for evolution of PVX strains (Malcuit *et al.* 2000). While recombination can be used to explain strains that have acquired *Nb* and *Nx* virulence determinants, phylogenetic analysis indicates that the virulence determinant located in TGBp1 and CP coding sequences reflect multiple acquisitions or even losses (Malcuit *et al.* 2000). PVX is only transmitted mechanically and typically causes a mild mosaic disease on the foliage (Franc and Banttari 1984, Franc and Banttari 2001). When PVX and PVY occur in the same plant they act synergistically, and cause "rugous mosaic disease" which is a severe disease that is not seen in plants infected with the individual viruses (Pruss *et al.* 1997, Vance *et al.* 1995). The basis for this synergy is the suppression of gene silencing by PVY that releases PVX from the plant immune system, allowing it to cause a more serious disease (Llave *et al.* 2000).

There are two recognized PVS strains, PVS^{O} (ordinary) and PVS^{A} (Andean) (Foster and Mills 1990). PVS^{O} occurs worldwide, is mechanically transmissible, and causes localized infection of *Chenopodium spp*. PVS^{A} was first detected in the Andean region of South America but since has been found in Europe, USA, and New Zealand (Cerovska and Filigarova 1995, Fletcher *et al.* 1996, Slack 1983). PVS^{A} is spread by aphids in a non-persistent manner (Wardrop *et al.* 1989), causes a more severe disease than PVS^{O} in potato, and causes systemic disease in *Chenopodium* spp. (Weidemann and Koenig 1990). Phylogenetic analysis of the coat 7K and 11K proteins was carried out from numerous isolates worldwide to try to understand the genetic relatedness of isolates classified as PVS^{O} or PVS^{A}. However, none of these studies have been able to correlate genetic differences with systemic invasion of *Chenopodium* spp. (Cox and Jones 2010).

There is very little known about PVM. A new strain of PVM called PVM-ID was reported in Idaho in 1998 (Cavileer *et al.* 1998). The coat protein sequence for PVM-ID is sufficiently different from published PVM sequence to warrant defining the isolate as a new strain (Cavileer *et al.* 1998).

Both PVS and PVX are easily transmitted during cutting of seed tubers before planting (Franc and Banttari 1984). PVS is considered to be mainly transmitted mechanically (Franc and Banttari 2001, Lambert *et al.* 2007), but there are some strains that are transmitted non-persistently by aphids (Wardrop *et al.* 1989). The role and mechanisms for aphid transmission require further investigation. PVS symptoms range from mild to severe mosaic on the foliage, but most potato varieties do not show symptoms. PVS occasionally causes 20% yield reductions.

The widespread use of vegetative propagation through tissue culture (**Fig. 1**) has minimized the presence of PVX and PVS in North American potato production. Since PVX-free plantlets serve as the basis for potato production, and the few early generation seed potato farms in Canada and the US are free of PVX, this virus is hard to find in seed potatoes. However, PVX still occurs on operations that do not routinely plant certified seed. Potato varieties are initiated into tissue culture and produce plantlets that are first tested for common viruses, and then treated with heat and anti-viral chemicals to eliminate any viruses that may be present (Zapata *et al.* 1995). Heating micro-plants to 42°C alongside treatment with salicylic acid for 4 weeks leads to high survival of PVX-free plants (Lopez-Delgado *et al.* 2004). Compounds such as melamine, 2-thiouracil, and ribavirin are useful for combating both PVS and PVX (Conrad 1991). Of the common viruses, PVS is the most difficult to eradicate from potato tissue culture plantlets and the basis for this remains unknown. In fact, the primary method for PVS control has been to ensure that potato tissue culture plantlets are PVS-free. Therefore, this is seen as the critical step for propagating PVS-free potatoes for seed certification. The titers of another carlavirus, *Potato virus M* (PVM), decline over time in tissue culture plantlets and this virus is typically eliminated in the absence of heat or antiviral compounds. Thus, the difficulties of eradicating PVS cannot be generalized among all carlaviruses.

The *Ns* gene, derived from *S. tuberosum* ssp. *andigena*, confers hypersensitive resistance to PVS and sources of the *Ns* gene have been used in European breeding programs (Marczewski *et al.* 1998). Potato plants containing the *Ns* gene are symptomless after inoculation with the virus. A SCAR marker associated with *Ns* has been mapped to a region on chromosome VIII that does not correlate with resistance to any other pathogens and thereby extends the coverage of virus *R* genes within the potato genome (Gebhardt and Valkonen 2001, Marczewski *et al.* 2002, Szajko *et al.* 2008).

Extreme resistance to PVX is conferred by Rx_{adg} (Bendahmane *et al.* 1997, van der Voort *et al.* 1999), *Rx1*, and *Rx2* (also known as Rx_{acl} from *S. acaule*) (Ritter *et al.* 1991). While both *Rx1* and Rx_{adg} are found on chromosome XII, they are considered different genes (Bendahmane *et al.* 1997). The *Rx2* gene is located on chromosome V (Ritter *et al.* 1991). Both *Rx1* and *Rx2* are linked to the *Gpa2* and *Gpa3* genes, respectively for resistance to *Globodera pallida* (van der Voort *et al.* 1999), suggesting an ancestral duplication of the loci on both chromosomes. To date, Rx_{adg} and *Rx2* are the only potato virus resistance genes that have been cloned (Bendahmane *et al.* 1999, Bendahmane *et al.* 2000). Both genes encode CC-NB-LRR proteins that exhibit the same specificity for the PVX coat protein (Bendahmane *et al.* 1995, Querci *et al.* 1995). Further molecular experiments using Rx_{adg} showed that it physically interacts with a Ran-GTPase-activating protein (RanGAP2), which is also required for ER to PVX (Sacco *et al.* 2007, Tameling and Baulcombe 2007).

Hypersensitive resistance to PVX has also been identified. The dominant *Nb* gene maps near the *Rx2* gene on chromosome V (De Jong *et al.* 1997b) and recognizes the 25-kDa PVX movement protein to elicit resistance (Malcuit *et al.* 1999). The Nx_{phu} gene derived from *S. phureja* is found on chromosome IX near the *Ny-1* and Ry_{adg} genes for PVY resistance (Tommiska *et al.* 1998). PVX HR genes such as Nc_{tbr}, Nx_{tbr}, and Nb_{tbr} are available in many cultivars

of potato and the Nx_{tbr} gene has provided useful resistance for many years (Barker and Dale 2006).

Soil-borne Tobraviruses and Pomoviruses: TRV and PMTV

TRV is the type member of the *Tobravirus* genus and PMTV is the type member of the *Pomovirus* genus. Both viruses have multipartite genomes packaged into rod-shaped particles. TRV rarely causes foliar symptoms in potato, even in plants with severe tuber symptoms. Foliar symptoms of PMTV develop when plants are grown from infected tubers and only occur when plants are grown at temperatures below 20°C (Carnegie *et al.* 2010). The most common symptom of PMTV is the development of aucuba patterns on the stems which consist of bright yellow blotches and ring or line patterns on lower or middle leaves. A less common secondary symptom consists of pale, V-shaped, chlorotic chevrons, usually on the leaflets of young upper leaves, and ultimately resulting in a distinct mosaic in the upper leaves. A third type of symptom consists of extreme shortening of internodes accompanied by crowding or bunching of foliage, described as a "mop-top". Some of the smaller leaves may have wavy or rolled margins and the overall effect is a dwarfed and bunched growth habit.

TRV is spread by stubby root nematodes (*Trichodorus* and *Paratrichodorus* species), which are widespread (Van Hoof 1968). Both the virus and the vector have very wide host ranges, thus once a field is infested with viruliferous nematodes, it is impossible to eliminate the virus from the field. The virus is controlled by planting resistant cultivars and treating fields with nematicides prior to or at planting (Ingham *et al.* 2007, Ingham *et al.* 2000, Weingartner *et al.* 1983). Treatment after planting is not effective and will not greatly impact virus spread. The stubby root nematode is often overlooked during routine nematode sampling, due to both its shape, which results in it being discarded during typical nematode extraction procedures, and its migratory nature in the soil. These nematodes stay just above the water line in the soil, so may be below the depth of the sampling probe. Thus, unlike with other nematodes, growers typically do not have information on the incidence of stubby root nematodes in their fields.

PMTV is vectored by the pathogen that causes powdery scab disease in potatoes, *Spongospora subterranea* f. sp. *subterranea*, which is present throughout the world (Kirk 2008). Temperature affects the success of transmission with greatest success at 12 to 20°C and little or no infection above 24°C (Carnegie *et al.* 2010). PMTV is not transmissible by aphids or other vectors, but it may be transmitted to some hosts by grafting or mechanical inoculations. PMTV is retained in *S. subterranea* spore balls, which are stable for many years in soil (Kirk 2008). The degree of transmission through seed tubers is variable, and in the absence of the vector, plants may become free of the virus after a few generations. However, since the vector is typically tuber-borne, this is not a common situation.

TRV and PMTV are discussed together, not only because they are soil-borne, but also because both viruses cause severe necrosis in tubers, known as spraing or corky ringspot. Necrotic arcs or brown spots are evident in tuber flesh that dries into cork-like tissue (**Fig. 2D**). The yield losses caused by these viruses are generally minor, but quality losses due to spraing can cause rejection of entire fields from supermarkets or processors. There are extreme situations where potato plants are infected with TRV with no apparent foliar symptoms but severe tuber symptoms. Thus, growers are surprised by the near total losses at harvest of a crop that appeared healthy. For both TRV and PMTV, the storage temperature and its fluctuation are important factors contributing to symptom expression in tubers. More symptoms occur with wound healing at 18°C followed by storage at 8°C than when wound healing occurs at 25°C (Ryden *et al.* 1994; Molgaard and Nielsen 1996). A colder storage temperature, 4°C, is preferable for seed potatoes to inhibit development of PMTV related spraing symptoms (Sandgren 1995). Tuber maturity also affects symptom development, with mature tubers being more susceptible (Molgaard and Nielsen 1996). Therefore storage regimes are designed for diagnosis of spraing and repeated cycles of temperature shifts are used as a screening technique.

TRV is frequently grouped with PMTV in European countries for regulatory efforts, as the tuber symptoms appear similar. PMTV was first reported in North America in shipments of potatoes that were being sent from the US to Canada and screened by the Canadian Food Inspection Agency in 2004 (Xu *et al.* 2004). Currently TRV and PMTV are covered by the necrotic virus management plan. In this plan, seed produced for commercial production can be shipped between the USA and Canada but federal inspectors must provide quality assurance that seed with less than 0.5% incidence of necrotic tubers will be used as seed potatoes (USDA 2004). Federal inspectors provide recertification of material moving from a production State or Canada. The difficulty lies in assessing whether tuber necrosis is due to a viral pathogen, and this relies on improved testing methodologies and adequate training of inspectors to identify the causes of internal necrosis (USDA 2004). Unfortunately, the distribution of TRV was thought to be limited in the USA to a few production areas in the western part of the country, the southeastern USA, and sporadically throughout the rest of the USA and Canada (Gieck *et al.* 2007). However, it has recently been reported in new areas in North Dakota, Michigan, Minnesota, and Wisconsin, and it appears to be present on seed potato farms, which is facilitating the spread of this virus (Gudmestad *et al.* 2008; Crosslin *et al.* 2010; David *et al.* 2010). The recent finds were due to severe outbreaks that caused total crop losses, highlighting the importance of this emerging problem. Thus, we can expect TRV and PMTV to continue to spread across North America.

Detection of viruses in soil is complicated both by the soil matrix, the uneven distribution of viruliferous nematodes and S. *subterranea* in soil, and the migratory nature of the stubby root nematode that vectors TRV. The lack of foliar symptoms of TRV and during primary infection of PMTV makes it difficult to control this disease through seed certification inspections. In addition, TRV RNA-1 is sometimes transmitted without RNA-2, resulting in a type of infection termed 'non-multiplying' since TRV RNA-1 can replicate in plants but cannot be spread by nematodes in the absence of RNA-2. Non-multiplying infections cause problems for virus identification since serological assays, which rely on the detection of coat protein, fail to detect TRV in these infections. Therefore, RT-PCR assays are a more reliable method for detection of TRV (Crosslin *et al.* 1999, Xu and Nie 2006). In contrast, some researchers have reported that ELISA is preferable for PMTV detection (Sokmen *et al.* 1998). RT-PCR assays with fluorescent probes have increased the sensitivity of virus detection making accurate diagnosis easier (Mumford *et al.* 2000). Baiting methods, which trap the PMTV vector *S. subterranea*, combined with RT-PCR, have also proven effective in detection of this virus (Nakayama *et al.* 2010). RT-PCR can also successfully detect TRV in its nematode vectors (Boutsika *et al.* 2004; Riga *et al.* 2009).

Resistance to TRV corky ringspot (CRS) disease is available in some cultivated varieties of potato (Brown *et al.* 2009, Brown *et al.* 2000, Dale *et al.* 2004, Dale *et al.* 2000, Harrison 1968, Richardson 1970, Shumaker *et al.* 1984, Weingartner and McSorley 1994). Parental materials containing resistance to CRS are fairly common choices for breeding materials as about 20% of all varieties contain some level of resistance (Brown *et al.* 2009). Harrison (1968) first described CRS resistance in British varieties and 'Bintje'. Since then, resistance has been identified in varieties from Europe and North America, including 'Multa' and 'Bintje' (Swiezynski *et al.* 1998), Poland, including 'Cisa' (Brown *et al.* 2000), and New Zealand, including 'Fianna' and 'Karaka' (Brown *et al.* 2009). Breeding for

resistance to TRV is complicated by the fact that inheritance is usually not simple (Brown *et al.* 2009). In fact, simple inheritance has been reported in only one potato cultivar, named 'Record' (Barker and Dale 2006) and a major QTL for resistance has been identified on potato chromosome IX (Khu *et al.* 2008). Potato varieties that are tolerant to TRV allow the virus to accumulate without producing the symptomatic 'corky' appearance in the tubers. This creates the opportunity for TRV to be introduced into virus-free sites if the vector nematodes are present (Xenophontos *et al.* 1998). Furthermore, breeders rely on the presence of spraing in the tubers as a visual assessment of virus resistance, or resistance to the vector. The existence of host tolerance resulting in a lack of tuber symptoms can be problematic for breeders (Dale and Solomon 1988). More recent evidence has indicated that CRS-resistant cultivars do not limit reproduction of the nematode vector, suggesting that plants are not nematode resistant and that the major component of resistance involves an interaction with the virus (Brown *et al.* 2000). Similarly, a lack of symptom expression in tubers correlates with a lack of TRV detection using RT-PCR, further supporting the conclusion that CRS-resistance is resistance to TRV infection (Brown *et al.* 2009). Resistance to PMTV has not been well studied at the mechanistic or genetic level and breeding for resistance to PMTV has mainly been focused on resistance to the vector (reviewed by Merz and Falloon 2009).

The role of seed potato certification in potato virus control

The history and progress of seed potato certification has been reviewed several times over the past decades (Callison *et al.* 1982, Gudmestad 1991, Leach 1938, Slack 1993, Trank 1991). Seed potato certification programs were first established in Europe and the idea was imported into North America in 1913. At the time, variety purity and the use of fewer well-characterized varieties were considered the primary goals, although third party inspection of seed potato quality and disease thresholds was also seen as important. Attempts, generally led by the customers of seed potato growers, have been made to federalize seed potato certification in the US since the 1920s. As a result, state-based potato grower associations were formed prior to 1913 in many regions, and these associations were responsible for setting up seed certification schemes. Currently, each state has different regulations and the certification agencies are managed by state departments of agriculture, universities, or grower groups, depending on the state. Today, federal standards are in place, but the agencies remain local. Along with the desire to ease export of seed potatoes, the effort to control necrotic strains of PVY was one of the main reasons for establishment of federal seed potato certification standards (Trank 1991). In North America, seed potato certification is based mainly on visual inspection of growing plants since it has historically been effective and the benefit of large-scale laboratory testing for pathogens is not worth the added cost. Thus, varieties that did not show easily recognized symptoms of important pathogens were discouraged (de Souza-Dias and Betti 2003). However, the evolution of new PVY strains (discussed previously) that elicit few symptoms regardless of the host genotype makes rouging for infected plants difficult. Therefore, certification agencies are now forced to expend time and funds towards screening for PVY infection using biochemical methods, such as ELISA (Trank 1991).

In many countries, vegetative propagation and seed potato certification programs (**Fig. 1**) have reduced or eliminated many significant tuber-borne pathogens, such as PVX and PLRV (Gudmestad 1991, Trank 1991). For example, a reported 12-year study conducted in Brazil showed that the availability and popularization of ELISA services to detect viruses in seed-potato production has enabled local producers to sell high quality seed-potatoes and reduce their dependence on imported seed-potatoes. This transition to local production has improved the private sector economy (de Souza-Dias and Betti 2003). PVX and PLRV, in particular, have limited host ranges, are only spread by potato colonizing aphids or mechanically, and do not survive in soil, thus they are effectively dealt with by eliminating seed potato lots carrying these pathogens and by effective insecticides (Johnson 2008). In contrast, the recent emergence of soil-borne viruses has been a challenge for seed potato certification programs (Johnson 2008). TRV and PMTV are not yet present in all production fields, but are widespread enough that there is less interest by the potato industry to impose quarantines or enforce geographic information system mapping of contaminated fields. Therefore, we predict that these soil-borne viruses are likely to become significant problems over the next few decades.

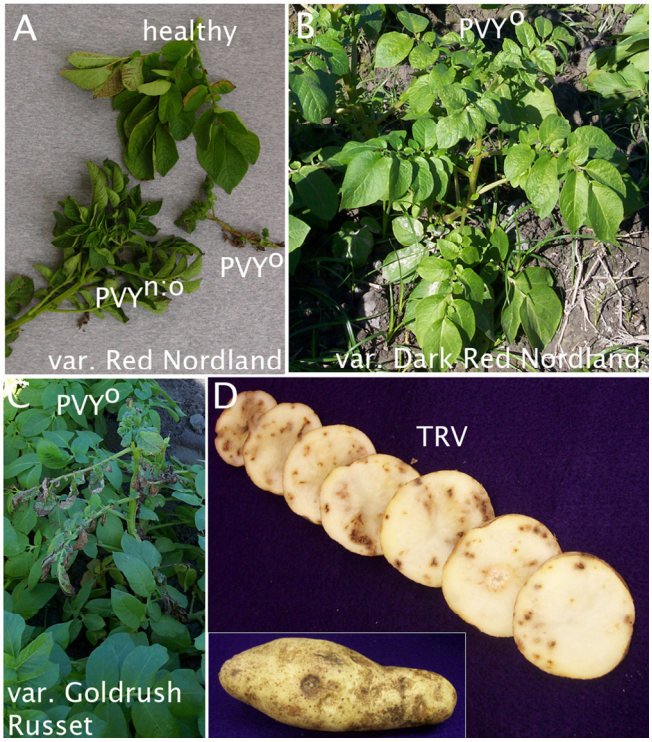

Fig. 2 Images of PVY- and TRV-infected potato plants. (**A**) Three branches of 'Red Nordland' variety shows a typical healthy set of leaves at the top. PVY^{O}-infected plant on the right is severely symptomatic. The branch at the bottom shows the mild foliar symptoms typical of PVY$^{N:O}$. (**B**) The 'Dark Red Nordland' shows mild PVY symptoms. The top of the plant shows symptoms and the bottom is healthy. (**C**) Typical PVY^{O}-infected 'Goldrush Russet' variety shows necrosis on the foliage. The top has mosaic symptoms. (**D**) TRV-infected potato tubers. The spraing is rarely found in arcs and concentric circles as often reported. The inset shows tuber distortions that are common among TRV-infected potatoes.

Generally, seed potatoes are divided into two main classes, one is considered suitable for planting on farms that raise seed potatoes and the other is suitable for planting on farms that raise potatoes for table stock or processing (Gagnon *et al.* 2007). PVY incidence in seed lots is one of the main characteristics used to determine which class a lot of seed potatoes falls into; it is the most common reason for down-grading seed potato lots in North America. For example, in Wisconsin, PVY is the only virus that has caused seed potatoes to be down-graded in class for at least the past decade (Genger and Charkowski 2007). The names used to describe the different classes of seed, subcategories in each class, and virus incidence thresholds for each seed class vary by seed potato certification agency. There is a zero tolerance for a virus in tissue culture and in greenhouses and this is insured by visual inspection and mandatory laboratory testing for growers of certified seed potatoes (**Fig. 1**). A virus incidence of 0.5 to 2% is allowed for lots destined for use on a seed potato farm. Seed potato lots with

virus incidence of more than 5 to 10% are considered to have too high of an incidence for use as planting stock on any farm (Genger and Charkowski 2007). Currently, these percentages are based mainly on visual inspection, not laboratory testing. The cost of extensive laboratory testing, while providing more accurate data, is prohibitively expensive.

CONCLUSIONS

Potato viruses have been intensively studied due to the significance of the host as a food crop and the effect that the diseases can have on yield and marketability. Great efforts have been made to integrate genetic resistance into cultivated varieties and stop virus spread through the use of certified seed. The biology and transmission of the majority of potato viruses are well characterized and detection methods, while sometimes expensive, are efficient at detecting even minute amounts of the pathogen. While detection, certification, and breeding efforts have limited the spread of viral diseases in potato in many cases, new strains of existing viruses and new viruses altogether continue to cause problems in developed agricultural systems. In these cases, controlling the insect, nematode, or protozoan vectors becomes critical. In developing countries, the adoption of reliable seed certification systems can have a dramatic effect on potato yield. In both cases, a better understanding of host resistance mechanisms and the incorporation of resistance into cultivated varieties using traditional breeding or biotechnological approaches provides the best long-term strategy for combating viral diseases.

ACKNOWLEDGEMENTS

This work was supported by the Oklahoma Center for Advancement of Science and Technology (OCAST) PSB09-028.

REFERENCES

Adams MJ, Antoniw JF, Bar-Joseph M, Brunt AA, Candresse T, Foster GD, Martelli GP, Milne RG, Zavriev SK, Fauquet CM (2004) The new plant virus family *Flexiviridae* and assessment of molecular criteria for species demarcation. *Archives of Virology* **149**, 1045-1060

Agindotan B, Perry KL (2007) Macroarray detection of plant RNA viruses using randomly primed and amplified complementary DNAs from infected plants. *Phytopathology* **97**, 119-127

Agindotan B, Perry KL (2008) Macroarray detection of eleven potato-infecting viruses and *Potato spindle tuber viroid*. *Plant Disease* **92**, 730-740

Agindotan B, Shiel PJ, Berger PH (2007) Simultaneous detection of potato viruses, PLRV, PVA, PVX and PVY from dormant potato tubers by TaqMan real-time RT-PCR. *Journal of Virological Methods* **142**, 1-9

Alvarez AE, Garzo E, Verbeek M, Vosman B, Dicke M, Tjallingii WF (2007) Infection of potato plants with potato leafroll virus changes attraction and feeding behaviour of *Myzus persicae*. *Entomologia Experimentalis et Applicata* **125**, 135-144

Ares X, Calamante G, Cabral S, Lodge J, Hemenway P, Beachy RN, Mentaberry A (1998) Transgenic plants expressing *Potato virus X* ORF2 protein (p24) are resistant to *Tobacco mosaic virus* and Ob tobamoviruses. *Journal of Virology* **72**, 731-738

Asama K, Ito H, Murakami N, Itoh T (1982) New potato variety "Konafubuki". *Bulletin of the Hokkaido Prefectural Agricultural Experiment Station* **48**, 75-84

Baldauf PM, Gray SM, Perry KL (2006) Biological and serological properties of *Potato virus Y* isolates in northeastern United States potato. *Plant Disease* **90**, 559-566

Bantarri EE, Ellis PJ, Khurana SMP (1993) Management of diseases caused by viruses and virus-like pathogens. In: Rowe RC (Ed) *Potato Health Management*, APS Press, St. Paul, MN, pp 127-133

Barker H (1997) Extreme resistance to *Potato virus V* in clones of *Solanum tuberosum* that are also resistant to *Potato viruses Y* and *A*: Evidence for a locus conferring broad-spectrum potyvirus resistance. *Theoretical and Applied Genetics* **95**, 1258-1262

Barker H, Dale MFB (2006) Resistance to viruses in potato. In: Lobenstein G, Carr JP (Eds) *Natural Resistance Mechanisms of Plants to Viruses*, Springer, Dordrecht, The Netherlands, pp 341-366

Barker H, Reavy B, McGeachy KD (1998) High level of resistance in potato to *Potato mop-top virus* induced by transformation with the coat protein gene. *European Journal of Plant Pathology* **103**, 737-740

Barker H, Solomon RM (1990) Evidence of simple genetic control in potato of ability to restrict *Potato leaf roll virus* concentration in leaves. *Theoretical and Applied Genetics* **80**, 188-192

Barker H, Solomon-Blackburn RM, McNicol JW, Bradshaw JE (1994) Resistance to *Potato leaf roll virus* multiplication in potato is under major gene control. *Theoretical and Applied Genetics* **88**, 754-758

Bendahmane A, Kanyuka K, Baulcombe DC (1999) The *Rx* gene from potato controls separate virus resistance and cell death responses. *Plant Cell* **11**, 781-792

Bendahmane A, Kohm BA, Dedi C, Baulcombe DC (1995) The coat protein of *Potato virus X* is a strain-specific elicitor of *Rx1*-mediated virus resistance in potato. *Plant Journal* **8**, 933-941

Bendahmane A, Querci M, Kanyuka K, Baulcombe DC (2000) *Agrobacterium* transient expression system as a tool for the isolation of disease resistance genes: Application to the *Rx2* locus in potato. *Plant Journal* **21**, 73-81

Bendahmane M, Fitchen JH, Zhang GM, Beachy RN (1997) Studies of coat protein-mediated resistance to tobacco mosaic tobamovirus: Correlation between assembly of mutant coat proteins and resistance. *Journal of Virology* **71**, 7942-7950

Bohl W, Nolte P, Thornton M (2000) *Potato Seed Management: Seed Certification and Selection*, University of Idaho Cooperative Extension System, Moscow, ID

Boiteau G, Singh M, Lavoie J (2009) Crop border and mineral oil sprays used in combination as physical control methods of the aphid-transmitted *Potato virus Y* in potato. *Pest Management Science* **65**, 255-259

Boutsika K, Phillips MS, MacFarlane SA, Brown DJF, Holeva RC, Blok VC (2004) Molecular diagnostics of some trichodorid nematodes and associated *Tobacco rattle virus*. *Plant Pathology* **53**, 110-116

Brommonschenkel SH, Frary A, Tanksley SD (2000) The broad-spectrum tospovirus resistance gene *Sw-5* of tomato is a homolog of the root-knot nematode resistance gene *Mi*. *Molecular Plant-Microbe Interactions* **13**, 1130-1138

Brown C, Mojtahedi H, Crosslin J, James S, Charlton B, Novy R, Love S, Vales M, Hamm P (2009) Characterization of resistance to corky ringspot disease in potato: A case for resistance to infection by *Tobacco rattle virus*. *American Journal of Potato Research* **86**, 49-55

Brown C, Mojtahedi H, Santo G, Hamm P, Pavek J, Corsini D, Love S, Crosslin J, Thomas P (2000) Potato germplasm resistant to corky ringspot disease. *American Journal of Potato Research* **77**, 23-27

Brown CR, Thomas PE (1993) Resistance to *Potato leafroll virus* derived from *Solanum chacoense*: Characterization and inheritance. *Euphytica* **74**, 51-57

Bukovinszki A, Diveki Z, Csanyi M, Palkovics L, Balazs E (2007) Engineering resistance to PVY in different potato cultivars in a marker-free transformation system using a 'shooter mutant' *A. tumefaciens*. *Plant Cell Reports* **26**, 459-465

Callison BL, Harrington J, Douglas D (1982) Seed potato certification: Its purpose, capabilities, and limitations. *American Potato Journal* **59**, 231-2236

Carnegie SF, Davey T, Saddler GS (2010) Effect of temperature on the transmission of *Potato mop-top virus* from seed tuber and by its vector, *Spongospora subterranea*. *Plant Pathology* **59**, 22-30

Castle SJ, Berger PH (1993) Rates of growth and increase of *Myzus persicae* on virus-infected potatoes according to type of virus-vector relationship. *Entomologia Experimentalis et Applicata* **69**, 51-60

Cavileer T, Clarke R, Corsini D, Berger PH (1998) A new strain of potato carlavirus M. *Plant Disease* **82**, 98-102

Celebi-Toprak F, Slack SA, Jahn MM (2002) A new gene, Ny_{tbr}, for hypersensitivity to potato virus Y from *Solanum tuberosum* maps to chromosome IV. *Theoretical and Applied Genetics* **104**, 669-674

Cerovska N, Filigarova M (1995) Specific detection of the Andean strain of *Potato virus S* by monoclonal antibodies. *Annals of Applied Biology* **127**, 87-93

Chung BY-W, Miller WA, Atkins JF, Firth AE (2008) An overlapping essential gene in the *Potyviridae*. *Proceedings of the National Academy of Sciences USA* **105**, 5807-5902

Coffin R, Moorehead S, Dawson D (1997) Potato research and extension from private industry: Potato production, genetically engineered varieties, pest control and fertility regimes. *Canadian Journal of Agricultural Economics* **45**, 461-469

Collard B, Mackill D (2008) Marker-assisted selection: an approach for precision plant breeding in the twenty-first century. *Philosophical Transactions of the Royal Society B* **363**, 557-572

Conrad PL (1991) *Potato virus S*-free plants obtained using antiviral compounds and nodal segment culture of potato. *American Potato Journal* **68**, 507-513

Corsini DL, Brown CR (2001) Important potato cultivars. In: Loebenstein G, Berger PH, Brunt AA, Lawson RH (Eds) *Virus and Virus-like Diseases of Potatoes, Production of Seed Potatoes*, Kluwer Academic Publishers, Dordrecht, The Netherlands, pp 39-52

Cox BA, Jones RAC (2010) Genetic variability in the coat protein gene of *Potato virus S* isolates and distinguishing its biologically distinct strains. *Archives of Virology* **155**, 1163-1169

Crosslin JM, Hamm PB, Kirk WW, Hammond RW (2010) Complete genomic sequence of a *Tobacco rattle virus* isolate from Michigan-grown potatoes.

Archives of Virology **155**, 621-625

Crosslin JM, Thomas PE, Brown CR (1999) Distribution of *Tobacco rattle virus* in tubers of resistant and susceptible potatoes and systemic movement of virus into daughter plants. *American Journal of Potato Research* **76**, 191-197

Cutler CG, Ramanaidu K, Astatkie T, Isman MB (2009) Green peach aphid, *Myzus persicae* (*Hemiptera: Aphididae*), reproduction during exposure to sublethal concentrations of imidacloprid and azadirachtin. *Pest Management Science* **65**, 205-209

Dale MFB, Robinson DJ, Todd D (2004) Effects of systemic infections with *Tobacco rattle virus* on agronomic and quality traits of a range of potato cultivars. *Plant Pathology* **53**, 788-793

Dale MFB, Robinson DJ, Wynne Griffiths D, Todd D, Bain H (2000) Effects of tuber-borne M-type strain of *Tobacco rattle virus* on yield and quality attributes of potato tubers of the cultivar Wilja. *European Journal of Plant Pathology* **106**, 275-282

Dale MFB, Solomon RM (1988) A glasshouse test to assess the sensitivity of potato cultivars to *Tobacco rattle virus*. *Annals of Applied Biology* **112**, 225-229

David N, Mallik I, Gudmestad NC (2010) First report of *Tobacco rattle virus* associated with corky ringspot in potatoes grown in North Dakota. *Plant Disease* **94**, 130

Davis JA, Radcliffe EB, Ragsdale DW (2005) Soybean aphid, *Aphis glycines* Matsumura, a new vector of *Potato virus Y* in potato. *American Journal of Potato Research* **82**, 197-201

De Jong W, Forsyth A, Leister D, Gebhardt C, Baulcombe DC (1997a) A potato hypersensitive resistance gene against *Potato virus X* maps to a resistance gene cluster on chromosome 5. *Theoretical and Applied Genetics* **95**, 246-252

De Jong W, Mise K, Chu A, Ahlquist P (1997b) Effects of coat protein mutations and reduced movement protein expression on infection spread by *Cowpea chlorotic mottle virus* and its hybrid derivatives. *Virology* **232**, 167-173

de Souza-Dias JAC, Betti JA (2003) A 12-year review of ELISA monitoring of major potato viruses in dormant seed-tubers in Brazil. *Acta Horticulturae* **619**, 153-159

Du Z (2006) Optimization and application of a multiplex RT-PCR system for simultaneous detection of five potato viruses using 18S rRNA as an internal control. *Plant Disease* **90**, 185-189

Dziewonska MA, Ostrowska K (1977) Necrotic reaction to *Potato virus M* in *Solanum stoloniferum* and *S. megistacrolobum*. *Journal of Phytopathology* **88**, 172-179

Ehrenfeld N, Romano E, Serrano C, Arce-Johnson P (2004) Replicase mediated resistance against *Potato leafroll virus* in potato Desiree plants. *Biological Research* **37**, 71-82

Eigenbrode SD, Ding H, Shiel P, Berger PH (2002) Volatiles from potato plants infected with *Potato leafroll virus* attract and arrest the virus vector, *Myzus persicae* (Homoptera: Aphididae). *Proceedings of the Royal Society B* **269**, 455-460

Fletcher JD, Lewthwaite SL, Boddington HJ, Nott HM, Wood RJ (1996) Virus disease surveys of ware potato crops, Franklin County, North Island, New Zealand. *New Zealand Journal of Crop and Horticultural Science* **24**, 7-12

Flis B, Hermig J, Strzelczyk-Zyta D, Gebhardt C, Marezewski W (2005) The *Ry-f_{sto}* gene from *Solanum stoloniferum* for extreme resistance to *Potato virus Y* maps to potato chromosome XII and is diagnosed by PCR marker $GP122_{718}$ in PVY resistant potato cultivars. *Molecular Breeding* **15**, 95-101

Foster GD, Mills PR (1990) Detection of strains of *Potato virus S* by nucleic-acid spot hybridization (Nash). *Potato Research* **33**, 487-495

Franc G, Banttari E (1984) The transmission of *Potato virus S* by the cutting knife and retention time of infectious PVS on common surfaces. *American Potato Journal* **61**, 253-260

Franc G, Banttari E (2001) Mechanically transmissible viruses of potato. In: Loebenstein G, Berger P, Brunt A, Lawson R (Eds) *Virus and Virus-like Diseases of Potatoes, Production of Seed Potatoes*, Kluwer Academic Publishers, The Netherlands, pp 159-175

Gagnon R, Drouin M, Peters D (2007) Canadian potato situation and trends 2006-2007. *Horticulture and Special Crops Division*, pp 1-36

Gargouri-Bouzid R, Jaoua L, Rouis S, Saidi MN, Bouaziz D, Ellouz R (2006) PVY-resistant transgenic potato plants expressing an anti-NIa protein scFv antibody. *Molecular Biotechnology* **33**, 133-140

Gebhardt C, Valkonen JP (2001) Organization of genes controlling disease resistance in the potato genome. *Annual Review of Phytopathology* **39**, 79-102

Genger RK, Charkowski AO (2007) *Certified Seed Potatoes-Health Insurance for Your Potato Crop*, Wisconsin Seed Potato Certification Program, Madison, WI

Gieck S, David N, Hamm P (2007) Delayed emergence, stem distortion, stunting, and foliar symptoms associated with *Tobacco rattle virus* and *Paratrichodorus allius* in potatoes grown in the pacific northwest. *Plant Health Progress* doi:10.1094/PHP-2007-01-BR

Gillen AM, Novy RG (2007) Molecular characterization of the progeny of *Solanum etuberosum* identifies a genomic region associated with resistance to *Potato leafroll virus*. *Euphytica* **155**, 403-415

Greenberg JT, Yao N (2004) The role and regulation of programmed cell death in plant-pathogen interactions. *Cellular Microbiology* **6**, 201-211

Gudmestad NC (1991) A historical perspective to pathogen testing in seed potato certification. *American Potato Journal* **68**, 99-102

Gudmestad NC, Mallik I, Pasche JS, Crosslin JM (2008) First report of *Tobacco rattle virus* causing corky ringspot in potatoes grown in Minnesota and Wisconsin. *Plant Disease* **92**, 1254

Hämäläinen JH, Kekarainen T, Gebhardt C, Watanabe KN, Valkonen JP (2000) Recessive and dominant genes interfere with the vascular transport of *Potato virus A* in diploid potatoes. *Molecular Plant-Microbe Interactions* **13**, 402-412

Hämäläinen JH, Sorri VA, Watanabe KN, Gebhardt C, Valkonen JPT (1998) Molecular examination of a chromosome region that controls resistance to *Potato Y* and *A* potyviruses in potato. *Theoretical and Applied Genetics* **96**, 1036-1043

Hamm P (1999) Effects of seedborne *Potato leafroll virus* on Russet Norkotah potato. *Plant Disease* **83**, 1122-1124

Hane DC (1999) Effects of seedborne *Potato virus Y* infection in two potato cultivars expressing mild disease symptoms. *Plant Disease* **83**, 43-45

Harper F, Nelson G, Pittman U (1975) Relationship between leafroll symptoms and yield of Netted Gem potato. *Phytopathology* **65**, 1244-1249

Harrison B (1968) Reactions of some old and new British potato cultivars to *Tobacco rattle virus*. *Potato Research* **11**, 165-176

Hazell SP, Groutides C, Neve BP, Blackburn TM, Bale JS (2010a) A comparison of low temperature tolerance traits between closely related aphids from the tropics, temperate zone, and arctic. *Journal of Insect Physiology* **56**, 115-122

Hazell SP, Neve BP, Groutides C, Douglas AE, Blackburn TM, Bale JS (2010b) Hyperthermic aphids: Insights into behaviour and mortality. *Journal of Insect Physiology* **56**, 123-131

Heath MC (2000) Hypersensitive response-related death. *Plant Molecular Biology* **44**, 321-334

Hosaka K, Hosaka Y, Mori M, Maida T, Matsunaga H (2001) Detection of a simplex RAPD marker linked to resistance to *Potato virus Y* in a tetraploid potato. *American Journal of Potato Research* **78**, 191-196

Hu X, Karasev AV, Brown CJ, Lorenzen JH (2009) Sequence characteristics of *Potato virus Y* recombinants. *Journal of General Virology* **90**, 3033-3041

Ingham RE, Hamm PB, Baune M, Merrifield KJ (2007) Control of *Paratrichodorus allius* and corky ringspot disease in potato with shank-injected metam sodium. *Journal of Nematology* **39**, 258-262

Ingham RE, Hamm PB, Willams RE, Swanson WH (2000) Control of *Paratrichodorus allius* and corky ringspot disease of potato in the Columbia Basin of Oregon. *Journal of Nematology* **32**, 566-575

Inoue-Nagata AK, Fonseca EN, Lobo TOTA, De Avila AC, Monte DC (2001) Analysis of the nucleotide sequence of the coat protein and the 3' untranslated region of two Brazilian *Potato virus Y* isolates. *Fitopatologia Brasileira* **26**, 45-52

Jansky SH (2000) Breeding for disease resistance in potato. In: Janick J (Ed) *Plant Breeding Reviews*, John Wiley & Sons Inc., New York, NY, pp 69-155

Johnson DA (2008) *Potato Health Management*, APS Press, St. Paul, MN, 261 pp

Kaiser WJ (1980) Use of thermotherapy to free potato tubers of *Alfalfa mosaic, Potato leaf roll*, and *Tomato black ring viruses*. *Phytopathology* **70**, 1119-1122

Kang BC, Yeam I, Li HX, Perez KW, Jahn MM (2007) Ectopic expression of a recessive resistance gene generates dominant potyvirus resistance in plants. *Plant Biotechnology Journal* **5**, 526-536

Karasev A, Nikolaeva O, Hu X, Sielaff Z, Whitworth J, Lorenzen J, Gray SM (2010) Serological properties of ordinary and necrotic isolates of *Potato virus Y*: A case study of PVY^N misidentification. *American Journal of Potato Research* **87**, 1-9

Karasev AV, Meacham T, Hu X, Whitworth J, Gray SM, Olsen N, Nolte P (2008) Identification of *Potato virus Y* strains associated with tuber damage during a recent virus outbreak in potato in Idaho. *Plant Disease* **92**, 1371

Kasai K, Morikawa Y, Sorri VA, Valkonen JPT, Gebhardt C, Watanbe KN (2000) Development of SCAR markers to the PVY resistance gene *Ry_{adg}* based on a common feature of plant disease resistance genes. *Genome* **42**, 1-8

Kawchuk LM, Martin RR, McPherson J (1991) Sense and anti-sense RNA-mediated resistance to *Potato leafroll virus* in Russet Burbank potato plants. *Molecular Plant-Microbe Interactions* **4**, 247-253

Kelley K, Whitworth J, Novy R (2009) Mapping of the potato leafroll virus resistance gene, *Rlr_{etb}*, from *Solanum etuberosum* identifies interchromosomal translocations among its E-genome chromosomes 4 and 9 relative to the A-genome of *Solanum* L. sect. *Petota*. *Molecular Breeding* **23**, 489-500

Khu D-M, Lorenzen J, Hackett C, Love S (2008) Interval mapping of quantitative trait loci for corky ringspot disease resistance in a tetraploid population of potato (*Solanum tuberosum* subsp. *tuberosum*). *American Journal of Potato Research* **85**, 129-139

Khurana S (2004) Potato viruses and their management. *Diseases of Fruits and Vegetables* **2**, 389-440

Kirk HG (2008) Mop-top virus, relationship to its vector. *American Journal of Potato Research* **85**, 261-265

Knutson K, Eastman P, Hoyman W, Jones E, MacLaghlan D, Picha B, Spencer E (1967) 1966 report of potato seed certification and nomenclature

committee. *American Journal of Potato Research* **44**, 255-256
Lambert S, Hay F, Pethybridge S, Wilson C (2007) Spatiotemporal spread of *Potato virus S* and *Potato virus X* in seed potato in Tasmania, Australia. *Plant Health Progress* doi:10.1094/PHP-2007-0726-07-RS
Lawson EC, Weiss JD, Thomas PE, Kaniewski WK (2001) NewLeaf Plus® Russet Burbank potatoes: Replicase-mediated resistance to *Potato leafroll virus*. *Molecular Breeding* **7**, 1-12
Leach JG (1938) The biological basis for certification of seed potatoes. *American Potato Journal* **15**, 117-130
Llave C, Kasschau KD, Carrington JC (2000) Virus-encoded suppressor of posttranscriptional gene silencing targets a maintenance step in the silencing pathway. *Proceedings of the National Academy of Sciences USA* **97**, 13401-13406
Lopez-Delgado H, Mora-Herrera ME, Zavaleta-Mancera HA, Cadena-Hinojosa M, Scott IM (2004) Salicylic acid enhances heat tolerance and *Potato virus X* (PVX) elimination during thermotherapy of potato microplants. *American Journal of Potato Research* **81**, 171-176
Lu R, Martin-Hernandez AM, Peart JR, Malcuit I, Baulcombe DC (2003) Virus-induced gene silencing in plants. *Methods* **30**, 296-303
Malcuit I, de Jong W, Baulcombe DC, Shields DC, Kavanagh TA (2000) Acquisition of multiple virulence/avirulence determinants by *Potato virus X* (PVX) has occurred through convergent evolution rather than through recombination. *Virus Genes* **20**, 165-172
Malcuit I, Marano MR, Kavanagh TA, De Jong W, Forsyth A, Baulcombe DC (1999) The 25-kDa movement protein of PVX elicits *Nb*-mediated hypersensitive cell death in potato. *Molecular Plant-Microbe Interactions* **12**, 536-543
Manzer F, Merriam D, Storch R, Simpson G (1982) Effect of time of inoculation with *Potato leafroll virus* on development of net necrosis and stem-end browning in potato tubers. *American Potato Journal* **59**, 337-349
Marco S (1986) Incidence of aphid-transmitted virus-infections reduced by whitewash sprays on plants. *Phytopathology* **76**, 1344-1348
Marczewski W, Flis B, Syller J, Schafer-Pregl R, Gebhardt C (2001) A major quantitative trait locus for resistance to *Potato leafroll virus* is located in a resistance hotspot on potato chromosome XI and is tightly linked to *N*-gene-like markers. *Molecular Plant-Microbe Interactions* **14**, 1420-1425
Marczewski W, Flis B, Syller J, Strzelczyk-Żyta D, Hennig J, Gebhardt C (2004) Two allelic or tightly linked genetic factors at the *PLRV.4* locus on potato chromosome XI control resistance to *Potato leafroll virus* accumulation. *Theoretical and Applied Genetics* **109**, 1604-1609
Marczewski W, Hennig J, Gebhardt C (2002) The *Potato virus S* resistance gene *Ns* maps to potato chromosome VIII. *Theoretical and Applied Genetics* **105**, 564-567
Marczewski W, Ostrowska K, Zimnoch-Guzowska E (1998) Identification of RAPD markers linked to the *Ns* locus in potato. *Plant Breeding* **117**, 88-90
Marczewski W, Strzelczyk-Żyta D, Hennig J, Witek K, Gebhardt C (2006) Potato chromosomes IX and XI carry genes for resistance to *Potato virus M*. *Theoretical and Applied Genetics* **112**, 1232-1238
Martelli GP, Adams MJ, Kreuze JF, Dolja VV (2009) Family *Flexiviridae*: a case study in virion and genome plasticity. *Annual Review of Phytopathology* **45**, 73-100
Matsuoka M, Tamaoki M, Tada Y, Fuyjimura T, Tagiri A, Yamamoto N, Kanomurakami Y (1995) Expression of rice *Osh1* gene is localized in developing vascular strands and its ectopic expression in transgenic rice causes altered morphology of leaf. *Plant Cell Reports* **14**, 555-559
Melander M, Lee M, Sandgren M (2001) Reduction of *Potato mop-top virus* accumulation and incidence in tubers of potato transformed with a modified triple gene block gene of PMTV. *Molecular Breeding* **8**, 197-206
Merz U, Falloon R (2009) Review: Powdery scab of potato—increased knowledge of pathogen biology and disease epidemiology for effective disease management. *Potato Research* **52**, 17-37
Molgaard JP, Nielsen SL (1996) Influence of post harvest temperature treatments, storage period and harvest date on development of spraing caused by *Tobacco rattle virus* and *Potato mop-top virus*. *Potato Research* **39**, 571-579
Mollov DS, Thill CA (2004) Evidence of *Potato virus Y* asymptomatic clones in diploid and tetraploid potato-breeding populations. *American Potato Journal* **81**, 317-326
Mortimer-Jones SM, Jones MGK, Jones RAC, Thomson G, Dwyer GI (2009) A single tube, quantitative real-time RT-PCR assay that detects four potato viruses simultaneously *Journal of Virological Methods* **161**, 289-296
Mowry TM (2005) Insecticidal reduction of *Potato leafroll virus* transmission by *Myzus persicae*. *Annals of Applied Biology* **146**, 81-88
Mumford RA, Walsh K, Barker I, Boonham N (2000) Detection of *Potato mop top virus* and *Tobacco rattle virus* using a multiplex real-time fluorescent reverse-transcription polymerase chain reaction assay. *Phytopathology* **90**, 448-453
Munoz F, Plaisted R, Thurston H (1975) Resistance to *Potato virus Y* in *Solanum tuberosum* spp. *andigena*. *American Journal of Potato Research* **52**, 107-115
Nakayama T, Maoka T, Hataya T, Shimizu M, Fuwa H, Tsuda S, Mori M (2010) Diagnosis of *Potato mop-top virus* in soil using bait plant bioassay and RT-PCR-microplate hybridization. *American Journal of Potato Research* **87**, 218-225
Nauen R, Denholm I (2005) Resistance of insect pests to neonicotinoid insecticides: Current status and future prospects. *Archives of Insect Biochemistry and Physiology* **58**, 200-215
Nie X, Singh RP (2002) A new approach for the simultaneous differentiation of biological and geographical strains of *Potato virus Y* by uniplex and multiplex RT-PCR. *Journal of Virological Methods* **104**, 41-54
Nolte P, Miller JS, Geary BD, Corsini DL (2003) Disease mangement. In: Stark JC, Love SL (Eds) *Potato Production Systems*, University of Idaho Extension, USA, pp 153-183
Novy R, Gillen A, Whitworth J (2007) Characterization of the expression and inheritance of *Potato leafroll virus* (PLRV) and *Potato virus Y* (PVY) resistance in three generations of germplasm derived from *Solanum etuberosum*. *Theoretical and Applied Genetics* **114**, 1161-1172
Novy R, Nasruddin A, Ragsdale D, Radcliffe E (2002) Genetic resistances to *Potato leafroll virus, Potato virus Y*, and green peach aphid in progeny of *Solanum etuberosum*. *American Journal of Potato Research* **79**, 9-18
Novy R, Helgeson JP (1994) Somatic hybrids between *Solanum etuberosum* and diploid, tuber bearing *Solanum* clones. *Theoretical and Applied Genetics* **89**, 775-782
Ogawa T, Tomitaka Y, Nakagawa A, Ohshima K (2008) Genetic structure of a population of *Potato virus Y* inducing potato tuber necrotic ringspot disease in Japan; Comparison with North American and European populations. *Virus Research* **131**, 199-212
Parker WE, Howard JJ, Foster SP, Denholm I (2006) The effect of insecticide application sequences on the control and insecticide resistance status of the peach-potato aphid, *Myzus persicae* (*Hemiptera:Aphididae*), on field crops of potato. *Pest Management Science* **62**, 307-315
Peters D (1987) Spread of viruses in potato crops. In: de Bokx J, van der Want J (Eds) *Viruses of Potatoes and Seed-Potato Production*, Pudoc, Wageningen, Netherlands, pp 126-145
Piche LM, Singh RP, Nie X, Gudmestad NC (2004) Diversity among *Potato virus Y* isolates obtained from potatoes grown in the United States. *Phytopathology* **94**, 1368-1375
Piron F, Nicolai M, Minoia S, Piednoir E, Moretti A, Salgues A, Zamir D, Caranta C, Bendahmane A (2010) An induced mutation in tomato eIF4E leads to immunity to two potyviruses. *PLoS One* **5**, e11313
Pourrahim R, Farzadfar S, Golnaraghi A, Ahoonmanesh A (2007) Incidence and distribution of important viral pathogens in some Iranian potato fields. *Plant Disease* **91**, 609-615
Powell G, Hardie J, Pickett JA (1998) The effects of antifeedant compounds and mineral oil on stylet penetration and transmission of *Potato virus Y* by *Myzus persicae* (Sulz.) (*Hom., Aphididae*). *Journal of Applied Entomology* **122**, 331-333
Pruss G, Ge X, Shi XM, Carrington JC, Bowman Vance V (1997) Plant viral synergism: The potyviral genome encodes a broad-range pathogenicity enhancer that transactivates replication of heterologous viruses. *Plant Cell* **9**, 859-868
Querci M, Baulcombe DC, Goldbach RW, Salazar LF (1995) Analysis of the resistance-breaking determinant of *Potato virus X* (PVX) strain HB on different potato genotypes expressing extreme resistance to PVX. *Phytopathology* **85**, 1003-1010
Raccah B (1986) Nonpersistent viruses: Epidemiology and control. In: Maramorosch K, Murphy FA, Aaron JS (Eds) *Advances in Virus Research*, Academic Press, pp 387-429
Radcliffe EB, Ragsdale DW (2002) Aphid-transmitted potato viruses: The importance of understanding vector biology. *American Journal of Potato Research* **79**, 353-386
Richardson DE (1970) The assessment of varietal reactions to spraing caused by *Tobacco rattle virus*. *Journal of the National Institute of Agricultural Botany* **4**, 16-18
Riga E, Larsen R, Eastwell K, Guerra N, Guerra L, Crosslin JM (2009) Rapid detection of *Tobacco rattle tobravirus* in viruliferous *Paratrichodorus allius* from greenhouse and field specimens. *Journal of Nematology* **41**, 60-63
Ritter E, Debener T, Barone A, Salamini F, Gebhardt C (1991) RFLP mapping on potato chromosomes of two genes controlling extreme resistance to *Potato virus X* (PVX). *Molecular and General Genetics* **227**, 81-85
Roosen J, Huffaker RG, Folwell RJ, Marsh TL, Mittelhammer RC (1997) The relation of *Potato leaf roll virus* net necrosis in potato tubers to the interval between planting and inoculation. *Crop Protection* **16**, 533-539
Ross H (1986) Potato breeding – Problems and perspectives. *Journal of Plant Breeding* **13 (Supplement 13)**, 132
Rouze-Jouan J, Terradot L, Pasquer F, Tanguy S, Giblot Ducray-Bourdin DD (2001) The passage of *Potato leafroll virus* through *Myzus persicae* gut membrane regulates transmission efficiency. *Journal of General Virology* **82**, 17-23
Ruffel S, Dussault MH, Palloix A, Moury B, Bendahmane A, Robaglia C, Caranta C (2002) A natural recessive resistance gene against *Potato virus Y* in pepper corresponds to the eukaryotic initiation factor 4E (eIF4E). *Plant Journal* **32**, 1067-1075
Ryazantsev DY, Zavriev SK (2009) An efficient diagnostic method for the identification of potato viral pathogens. *Molecular Biology* **43**, 515-523
Ryden K, Sandgren M, Hurtado S (1994) Development during storage of

spraing symptoms in potato-tubers infected with *Tobacco rattle virus*. *Potato Research* **37**, 99-102

Rykbost KA, Hane DC, Hamm PB, Voss R, Kirby D (1999) Effects of seed-borne *Potato virus Y* on Russet Norkotah performance. *American Journal of Potato Research* **75**, 91-96

Sacco MA, Mansoor S, Moffett P (2007) A RanGAP protein physically interacts with the NB-LRR protein Rx, and is required for *Rx*-mediated viral resistance. *The Plant Journal* **52**, 82-93

Salazar L (2006) Emerging and re-emerging potato diseases in the Andes. *Potato Research* **49**, 43-47

Sandgren M (1995) *Potato mop-top virus* (PMTV): Distribution in Sweden, development of symptoms during storage and cultivar trials in field and glasshouse. *Potato Research* **38**, 379-389

Santa Cruz S, Baulcombe D (1995) Analysis of *Potato virus X* coat protein genes in relation to resistance conferred by the genes *Nx, Nb* and *Rx1* of potato. *Journal of General Virology* **76**, 2057-2061

Santala J, Samuilova O, Hannukkala A, Latvala S, Kortemaa H, Beuch U, Kvarnheden A, Persson P, Topp K, Orstad K, Spetz C, Nielsen SL, Kirk HG, Budziszewska M, Wieczorek P, Obrepalska-Steplowska A, Pospieszny H, Kryszczuk A, Sztangret-Wisniewska J, Yin Z, Chrzanowska M, Zimnoch-Guzowska E, Jackeviciene E, Taluntyte L, Pupola N, Mihailova J, Lielmane I, Jarvekulg L, Kotkas K, Rogozina E, Sozonov A, Tikhonovich I, Horn P, Broer I, Kuusiene S, Staniulis J, Uth JG, Adam G, Valkonen JPT (2010) Detection, distribution and control of *Potato mop-top virus*, a soil-borne virus, in northern Europe. *Annals of Applied Biology* **157**, 163-178

Sato M, Nishikawa K, Komura K, Hosaka K (2006) *Potato virus Y* resistance gene, Ry_{chc}, mapped to the distal end of potato chromosome 9. *Euphytica* **149**, 367-372

Saucke H, Doring TF (2004) *Potato virus Y* reduction by straw mulch in organic potatoes. *Annals of Applied Biology* **144**, 347-355

Shumaker J, Weingartner C, Hensel D, Webb R (1984) Promising russet-skin potato cultivars for north Florida. *Soil and Crop Science Society of Florida* **43**, 166-169

Singh VK, Wood SM, Knowles VL, Plaxton WC (2003) Phosphite accelerates programmed cell death in phosphate-starved oilseed rape (*Brassica napus*) suspension cell cultures. *Planta* **218**, 233-239

Slack SA (1983) Identification of an isolate of the Andean strain of *Potato virus S* in North America. *Plant Disease* **67**, 786-789

Slack SA (1993) Seed certification and seed improvement programs. In: Rowe RC (Ed) *Potato Health Management*, APS Press, St Paul MN

Sokmen MA, Barker H, Torrance L (1998) Factors affecting the detection of *Potato mop-top virus* in potato tubers and improvement of test procedures for more reliable assays. *Annals of Applied Biology* **133**, 55-63

Solomon-Blackburn RM, Barker H (2001) A review of host major-gene resistance to *Potato viruses X, Y, A* and *V* in potato: Genes, genetics and mapped locations. *Heredity* **86**, 8-16

Song Y-S, Hepting L, Schweizer G, Hartl L, Wenzel G, Schwarzfischer A (2005) Mapping of extreme resistance to PVY (Ry_{sto}) on chromosome XII using anther-culture-derived primary dihaploid potato lines. *Theoretical and Applied Genetics* **111**, 879-887

Sorri VA, Watanabe KN, Valkonen JPT (1999) Predicted kinase-3a motif of a resistance gene analogue as a unique marker for virus resistance. *Theoretical and Applied Genetics* **99**, 164-170

Srinivasan R, Alvarez JM, Bosque-Perez NA, Eigenbrode SD, Novy RG (2008) Effect of an alternate weed host, hairy nightshade, *Solanum sarrachoides*, on the biology of the two most important *Potato leafroll virus* (*Luteoviridae: Polerovirus*) vectors, *Myzus persicae* and *Macrosiphum euphorbiae* (*Aphididae: Homoptera*). *Environmental Entomology* **37**, 592-600

Stuible H-P, Kombrink E (2004) The hypersensitive response and its role in disease resistance. In: Punja Z (Ed) *Fungal Disease Resistance in Plants – Biochemistry, Molecular Biology, and Genetic Engineering*, Haworth Press, Binghamton, NY, pp 57-92

Swiezynski KM, Dziewonska M, Ostrowska K (1990) Inheritance of the *Potato leafroll virus* (PLRV) in the potato. In: MacKerron D, Edmond H, Hall D, Kirkman M, Lang R, Mackay G, McRae D, Oxley J (Eds) *Abstracts of the 11th Triennial Conference of the European Association for Potato Research*, 8-13 July 1990, Edinburgh, UK, pp 538-539

Szajko K, Chrzanowska M, Strzelczyk-Zyta D, Zagórska H, Marczewski W (2008) Endonuclease restriction of SCAR amplicons SC811 is required to identify *Ns*-false-positive markers in PVS-susceptible potato cultivars. *Journal of Applied Genetics* **49**, 45-47

Tacke E, Salamini F, Rohde W (1996) Genetic engineering of potato for broad-spectrum protection against virus infection. *Nature Biotechnology* **14**, 1597-1601

Taliansky M, Mayo MA, Barker H (2003) *Potato leafroll virus*: A classic pathogen shows some new tricks. *Molecular Plant Pathology* **4**, 81-89

Tameling WIL, Baulcombe DC (2007) Physical association of the NB-LRR resistance protein Rx with a Ran GTPase-activating protein is required for extreme resistance to *Potato virus X*. *Plant Cell* **19**, 1682-1694

Tenorio J, Franco Y, Chuquillanqui C, Owens RA, Salazar LF (2006) Reaction of potato varieties to *Potato mop-top virus* infection in the Andes. *American Journal of Potato Research* **83**, 423-431

Thill CA, Mollov DS (2004) Evidence of *Potato virus Y* asymptomatic clones in diploid and tetraploid potato-breeding populations. *American Journal of Potato Research* **81**, 317-326

Tommiska TJ, Hämäläinen JH, Watanabe KN, Valkonen JPT (1998) Mapping of the gene Nx_{phu} that controls hypersensitive resistance to *Potato virus X* in *Solanum phureja* IvP35. *Theoretical and Applied Genetics* **96**, 840-843

Trank W (1991) Progress in seed potato certification 1913-1991. *American Potato Journal* **68**, 243-244

Tribodet M, Glais L, Kerlan C, Jacquot E (2005) Characterization of *Potato virus Y* (PVY) molecular determinants involved in the vein necrosis symptom induced by PVYN isolates in infected *Nicotiana tabacum* cv. Xanthi. *Journal of General Virology* **86**, 2101-2105

Unruh T, Willett L (2008) Survey for resistance to four insecticides in *Myzus persicae* clones from peach trees and weeds in south-central Washington. *Journal of Economic Entomology* **101**, 1919-1926

Urcuqui-Inchima S, Haenni A-L, Bernadi F (2001) Potyvirus proteins: A wealth of functions. *Virus Research* **74**, 157-175

USDA (2004) Canada/US-Management plan for potato viruses that cause tuber necrosis (Agriculture UDo, Service CFI eds). Riverdale, MD and Ottawa, Ontario: USDA, pp. 1-12

Valkonen JPT, Slack SA, Plaisted RL (1994) Use of the virus-strain group concept to characterize the resistance to PVX and PVY(O) in the potato cv Allegany. *American Potato Journal* **71**, 507-516

Valkonen JPT, Wiegmann K, Hämäläinen JH, Marczewski W, Watanabe KN (2008) Evidence for utility of the same PCR-based markers for selection of extreme resistance to *Potato virus Y* controlled by Ry_{sto} of *Solanum stoloniferum* derived from different sources. *Annals of Applied Biology* **152**, 121-130

van der Voort JR, Kanyuka K, van der Vossen E, Bendahmane A, Mooijman P, Klein-Lankhorst R, Stiekema W, Baulcombe D, Bakker J (1999) Tight physical linkage of the nematode resistance gene *Gpa2* and the virus resistance gene *Rx* on a single segment introgressed from the wild species *Solanum tuberosum* subsp. *andigena* CPC 1673 into cultivated potato. *Molecular Plant-Microbe Interactions* **12**, 197-206

Van Hoof H (1968) Transmission of *Tobacco rattle virus* by Trichodorus species. *Nematologica* **14**, 20-24

Vance VB, Berger PH, Carrington JC, Hunt AG, Shi XM (1995) 5' proximal potyviral sequences mediate *Potato virus X*/potyviral synergistic disease in transgenic tobacco. *Virology* **206**, 583-590

Vazquez Rovere C, Asurmendi S, Hopp HE (2001) Transgenic resistance in potato plants expressing *Potato leaf roll virus* (PLRV) replicase gene sequences is RNA-mediated and suggests the involvement of post-transcriptional gene silencing. *Archives of Virology* **146**, 1337-1353

Volkov YG, Kakareka NN, Balabanova LA, Kozlovskaya ZN, Sapotsky MV (2009) Characterization of a novel far-eastern *Potato virus Y* isolates. *Plant Pathology Journal* **8**, 62-67

Wang Q, Liu Y, Xie Y, You M (2006) Cryotherapy of potato shoot tips for efficient elimination of *Potato leaf roll virus* (PLRV) and *Potato virus Y* (PVY). *Potato Research* **49**, 119-129

Wardrop E, Gray A, Singh R, Peterson J (1989) Aphid transmission of *Potato virus S*. *American Potato Journal* **66**, 449-459

Weidemann HL (1988) Importance and control of *Potato virus* Y^N (PVY^N) in seed potato production. *Potato Research* **31**, 85-94

Weidemann HL, Koenig R (1990) Differentiation of isolates of *Potato virus S* which infect *Chenopodium quinoa* systemically by means of quantitative cDNA hybridization tests. *Zeitschrift Fur Pflanzenkrankheiten Und Pflanzenschutz-Journal of Plant Diseases and Protection* **97**, 323-327

Weingartner DP, McSorley R (1994) Management of nematodes and soil-borne pathogens in sub tropical potato production. In: Zehnder G, Powerlson M, Janssen R, Raman K (Eds) *Advances in Potato Pest Biology and Management*, APS Press, Minneapolis, MN, pp 202-213

Weingartner DP, Shumaker JR, Smart GC, Jr. (1983) Why soil fumigation fails to control potato corky ringspot disease in Florida. *Plant Disease* **67**, 130-134

Werling BP, Gratton C (2010) Local and broadscale landscape structure differentially impact predation of two potato pests. *Ecological Applications* **20**, 1114-1125

Werner BJ, Mowry TM, Bosque-Perez NA, Ding HJ, Eigenbrode SD (2009) Changes in green peach aphid responses to *Potato leafroll virus*-induced volatiles emitted during disease progression. *Environmental Entomology* **38**, 1429-1438

Whitworth J, Novy R, Hall D, Crosslin J, Brown C (2009) Characterization of broad spectrum *Potato virus Y* resistance in a *Solanum tuberosum* ssp. *andigena*-derived population and select breeding clones using molecular markers, grafting, and field inoculations. *American Journal of Potato Research* **86**, 286-296

Whitworth J, Nolte P, McIntosh C, Davidson R (2006) Effect of *Potato virus Y* on yield of three potato cultivars grown under different nitrogen levels. *Plant Disease* **90**, 73-76

Witek K, Strzelczyk-Zyta D, Hennig J, Marczewski W (2006) A multiplex PCR approach to simultaneously genotype potato towards the resistance alleles $Ry\text{-}f_{sto}$ and *Ns*. *Molecular Breeding* **18**, 273-275

Xenophontos S, Robinson D, Dale M, Brown D (1998) Evidence for persis-

tent, symptomless infection of some potato cultivars with tobacco rattle virus. *Potato Research* **41**, 255-265

Xu H, DeHaan T-L, De Boer S (2004) Detection and confirmation of *Potato mop-top virus* in potatoes produced in the United States and Canada. *Plant Disease* **88**, 363-367

Xu H, Nie J (2006) Molecular detection and identification of potato isolates of *Tobacco rattle virus*. *Canadian Journal of Plant Pathology* **28**, 271-279

Zapata C, Miller JCJ, Smith RA (1995) An *in vitro* procedure to eradicate *Potato viruses X*, *Y*, and *S* from Russet Norkotah and two of its strains. *In Vitro Cellular and Developmental Biology – Plant* **31**, 153-159