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POTATO VIRUS Y SYMPOSIUM

Genetic Diversity of *Potato virus Y* Complex

Alexander V. Karasev · Stewart M. Gray

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Abstract *Potato virus Y* (PVY) has emerged as a significant problem in all potato-producing areas, including North America. PVY exists as a complex of strains producing a range of disease symptoms in various potato cultivars leading to yield reduction, and some of these strains are known to affect tuber quality. In the past 30 years, significant changes in PVY strains circulating in potato crops have been observed in Europe, and more recently in North America, with an increased incidence of PVY strains associated with potato tuber damage. Different models have been proposed to explain these changes, including spread of new recombinants, enhanced vector transmission of certain strains, or introduction of new potato varieties. Here, we analyze the current knowledge of PVY genetic diversity with an emphasis on PVY strains common in North America. Multiple types of PVY genome recombinants with links to specific symptoms in potato varieties are described and discussed. Different approaches to distinguish PVY strains are reviewed and compared, including biological and laboratory methods.

Resumen El virus Y de la papa (PVY) ha emergido como un problema significativo en todas las áreas de producción de papa, incluyendo Norte América. El PVY existe como un complejo de variantes produciendo una gran amplitud de síntomas de la enfermedad en diversas variedades de papa, lo que ha conducido a reducción de rendimientos, y se sabe que algunas de estas variantes afectan la calidad del tubérculo. Se han observado en los últimos 30 años cambios significativos en las variantes del PVY circulando en los cultivos de papa en Europa, y más recientemente en Norte América, con un aumento en la incidencia de variantes del PVY asociadas con daño en el tubérculo. Se han propuesto diferentes modelos para explicar estos cambios, incluyendo la dispersión de nuevos recombinantes, aumento en la transmisión por vectores de ciertas variantes, o la introducción de nuevas variedades de papa. Aquí analizamos el conocimiento actual de la diversidad genética del PVY con énfasis en las variantes comunes de Norte América. Se describen y discuten múltiples tipos de recombinantes del genomio del PVY asociados a síntomas específicos en variedades de papa. Se revisan y se comparan diferentes enfoques para distinguir variantes del PVY, incluyendo métodos biológicos y de laboratorio.

Keywords PVY · Strains · Classification

Introduction

Potato virus Y (PVY) has emerged in recent years as one of the most important pathogens in potato, seriously affecting yield (Hane and Hamm 1999; Nolte et al. 2004) and quality of tubers by inducing potato tuber necrotic ringspot disease (PTNRD) in susceptible cultivars (Beczner et al. 1984; Le Romancer et al. 1994; Gray et al. 2010). The virus can be transmitted from plant to plant mechanically, however, in nature it is transmitted via vegetative propagation by seed tubers, or non-persistently by aphids (Kerlan 2006). Recombinant strains of PVY had become established in North America in 1990s and spread in both Canada and United States (Baldauf et al. 2006; Crosslin et al. 2002, 2005, 2006; Gray et al. 2010; Karasev et al. 2008; McDonald and Singh 1996).

PVY is a positive-sense, single-stranded RNA virus which belongs to the large family *Potyviridae*, genus *Potyvirus*; it has ca. 9.7-kb genome with poly(A) tail at the 3′-terminus and

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A. V. Karasev (✉)
Department of PSES, University of Idaho, Moscow, ID 83844, USA
e-mail: akarasev@uidaho.edu

S. M. Gray
USDA-ARS, Cornell University, Ithaca, NY 14853, USA

a covalently-linked VPg protein attached to the 5′-terminus (Adams et al. 2011). PVY expresses its genome as a single large polyprotein which is subsequently cleaved by three virus-specific proteases into 10 mature proteins (Fig. 1). PVY has elongated, helical particles composed of genomic ssRNA and one major species of 29-kDa capsid protein (CP) (Adams et al. 2011). Minor amounts of another PVY protein, HC-Pro, were found present in virus particles, in addition to CP and VPg that is covalently attached to the PVY RNA genome (Torrance et al. 2006).

Genetic Classification of PVY Strain Groups

PVY exists as a complex of strains or strain groups which can be distinguished by reactions towards a series of resistance genes in potato (Cockerham 1970; de Bokx and Huttinga 1981; Kerlan 2006; Singh et al. 2008). A group of strains eliciting hypersensitive resistance (HR) response in the background of *Ny* gene was named PVY^{O}, and those eliciting HR in the background of *Nc* gene were named PVY^{C} strains (Table 1). Existence of an additional resistance gene, *Nz*, was postulated based on HR reactions of a PVY strain group PVY^{Z} that did not elicit HR in either *Ny* or *Nc* genetic backgrounds but elicited HR in cultivars like Maris Bard and Pentland Ivory carrying this putative *Nz* gene (Jones 1990; Singh et al. 2008). Isolates that could overcome *Ny*, *Nc*, and *Nz* genes, and did not elicit HR towards any of these potato *N* genes could be further distinguished based on their reactions in tobacco (Table 1)—those inducing vein necrosis in tobacco were classified into PVY^{N} strain (Jones 1990; Singh et al. 2008), and those inducing only mosaic were classified into PVY^{E} strain (Kerlan et al. 1999; Singh et al. 2008). Of these five original, historic PVY strain groups, only PVY^{N} induced vein necrosis in tobacco (Table 1), and hence was named "necrotic" PVY strain (Cockerham 1970; de Bokx and Huttinga 1981; Boonham et al. 2002). It is important to note that this genetic classification scheme (Table 1) includes two strain groups, PVY^{O} and PVY^{N}, that are well-characterized molecularly, through serology, RT-PCR, and whole genome sequencing, but also two other strain groups, PVY^{Z} and PVY^{E}, whose molecular properties have not been studied until very recently (Kerlan et al. 2011; Galvino-Costa et al. 2012).

Molecular Classification of PVY Recombinants

Complete genome sequences are known for non-recombinant isolates from PVY^{O}, PVY^{C}, and PVY^{N} strain groups (Robaglia et al. 1989; Turpen 1989; Singh and Singh 1996; Lorenzen et al. 2006a; Fomicheva et al. 2009; Dullemans et al. 2011; Karasev et al. 2011). In addition to these main, parental genomes, multiple recombinant PVY genomes have been discovered, built of segments of PVY^{O} and PVY^{N} sequences spliced in various combinations (Thole et al. 1993; Jakab et al. 1997; Glais et al. 1998, 2002a, b; Boonham et al. 2002; Nie and Singh 2003b; Lorenzen et al. 2006a; Chikh Ali et al. 2007, 2008; Schubert et al. 2007; Hu et al. 2009b, 2011; Karasev et al. 2011; Galvino-Costa et al. 2012). At least nine recombination patterns were revealed with PVY^{O} and PVY^{N} identified as parents, based on sequence analysis of multiple PVY genomes known to date (Hu et al. 2009a). These recombinant patterns had between 1 to 4 recombinant junctions (RJs), most of which were relatively conserved in their position in the PVY genome, especially in the P1, HC-Pro, and VPg-NIa regions (Hu et al. 2009a).

The three most common recombinant patterns characteristic of PVY^{NTN}, $PVY^{N:O}$, and PVY^{N-Wi} are presented on Fig. 1, with positions of the corresponding RJs indicated. All four RJs indicated on Fig. 1 were tested for possible use as a differentiating tool for typing PVY recombinants, and several primer sets exploiting single nucleotide polymorphism around these RJs are now available to type PVY recombinants

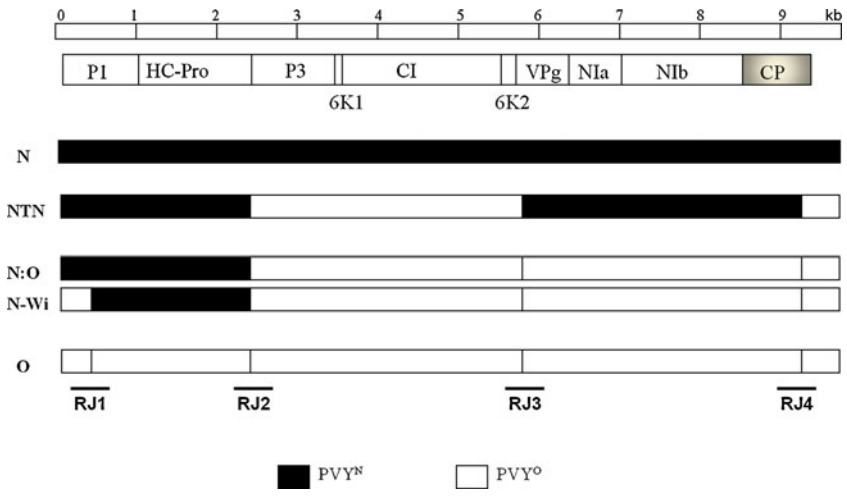

Fig. 1 Schematic diagram of the main types of PVY recombinants found in Europe and North America—PVY^{NTN}, $PVY^{N:O}$, and PVY^{N-Wi}. Different shading marks origin of genome fragments either from PVY^{N} (*black*) or PVY^{O} (*white*) parental sequences. Positions of recombinant junctions, RJ 1 to 4, often used to distinguish PVY recombinants are labeled below the diagram. The ten PVY genes are drawn above the diagram approximately to scale

Table 1 Genetic classification of the PVY strains, with serological and molecular properties of the main genetic strains of PVY. The data compiled according to Singh et al. (2008), Dullemans et al. (2011), Kerlan et al. (2011), and Galvino-Costa et al. (2012)

	PVY^{O}	PVY^{C}	PVY^{Z}	PVY^{N}	PVY^{E}
Desiree, Pentland Crown (***Ny**:nc:nz*)	HR	s	s	s	s
King Edward (*ny:**Nc**:nz*)	s	HR	s	s	s
Maris Bard, Pentland Ivory (***Ny**:**Nc**:**Nz***)	HR	HR	HR	s	s
Tobacco	mos	mos	mos	VN	mos
Serotype	O	O	N	N	N
Genome type	NR	NR	R	NR	R

HR hypersensitive resistance; *s* susceptible, *mos* mosaic; *VN* vein necrosis, *O* serotype PVY^{O}; *N* serotype PVY^{N}, *NR* non-recombinant; *R* recombinant

in a uniplex or multiplex RT-PCR formats (Nie and Singh 2002, 2003a; Boonham et al. 2002; Lorenzen et al. 2006b; Rigotti and Gugerli 2007; Chikh Ali et al. 2010). Other non-recombinant, PVY^{NA-N} (Nie and Singh 2003b), and recombinant genomes, e.g. NE-11 (Lorenzen et al. 2008), significantly different from PVY^{O} and PVY^{N} had also been reported. Most of these recombinant PVY variants were only studied by laboratory methods, and the only biological characterization conducted on some of them was tobacco test (Table 2). Interestingly, almost all PVY recombinants induced vein necrosis in tobacco, and thus would have been typed as PVY^{N} if only this tobacco test had been used for PVY strain typing (see Table 2). Because of their recombinant nature, some PVY strain groups, like $PVY^{N:O}$ and PVY^{N-Wi}, possessed PVY^{O} serotype and PVY^{N} symptomatology on tobacco, creating difficulties in correct differentiation of a PVY isolate. The recombinant PVY^{NTN} strain group (Table 2) attracted the most attention, since necrotic tuber reaction, i.e. the PTNRD syndrome, was associated with this recombinant type (Beczner et al. 1984; Le Romancer et al. 1994; Glais et al. 2002b). Thus, multiple recombinant isolates of PVY are now classified based largely on genome properties of the isolates—the number and position of their RJs, which may be determined by RT-PCR assays or through genome sequencing. Tobacco reaction is not very useful for differentiating PVY recombinants, since most of them induce vein necrosis in tobacco (Table 2). Serology alone, based on PVY^{O} and PVY^{N} serotypes identified by monoclonal antibodies, also has limited usefulness in differentiating recombinant isolates of PVY, since it cannot distinguish $PVY^{N:O/N-Wi}$ from non-recombinant PVY^{O}, or PVY^{NTN} from non-recombinant PVY^{N} (Table 2). Notably, very few of the recombinant isolates were tested on potato indicators (see Table 1), to determine their genetic type (for exceptions see Barker et al. 2009; Kerlan et al. 2011; Galvino-Costa et al. 2012), and consequently these two classifications, genetic and molecular, remain separate, creating confusions and making it difficult to navigate through the complexities of the PVY strain biology.

Merging Genetic and Molecular Classifications

Two factors made a seemingly minor, technical problem of merging the two systems of PVY strain classification quite a difficult endeavor. The first factor was related to a somewhat narrow group of researchers using potato indicators for typing PVY strains—these indicators essentially represented old European cultivars that were not universally available in every country with a PVY problem. To mitigate the problem of indicators' availability, tobacco became the biological indicator of choice where potato indicators were not available. The logic behind this biological indicator change was to focus PVY strain detection on the "necrotic", PVY^{N} strain group (Boonham et al. 2002) which could overcome all *N* resistance genes in potato (see Table 1). Unfortunately, almost all recombinants induced vein necrosis in tobacco, and were thus indistinguishable from PVY^{N} (Table 2). The second factor was related to fast changes in the composition of PVY isolates circulating in potato in Western Europe during late 1980s and early 1990s; the profound effect of

Table 2 Characteristics of the PVY strain groups classified based on their genome properties. Data compiled according to Singh et al. (2008), Lorenzen et al. (2006a, 2008), Barker et al. (2009), Kerlan et al. (2011), and Galvino-Costa et al. (2012)

	PVY^{O}	PVY^{C}	PVY^{N}	PVY^{E}	PVY^{N-Wi}	$PVY^{N:O}$	PVY^{NTN}	PVY^{NA-N}	NE-11
Tobacco	mos	mos	VN	mos	VN	VN	VN	VN	VN
Serology	O	O	N	N	O	O	N	N	N
Genome	NR	NR	NR	R	R	R	R	NR	R
Number of RJs	0	0	0	4+	2	1	3–4	0	2+
N gene triggered	*Ny*	*Nc*	none	none	?	?	*Nz*	?	?

mos mosaic; *VN* vein necrosis

O serotype PVY^{O}; *N* serotype PVY^{N}

NR non-recombinant; *R* recombinant

RJ recombinant junction

these changes became obvious only later, when reliable molecular tools for PVY strain identification were developed (Nie and Singh 2002, 2003a; Boonham et al. 2002; Lorenzen et al. 2006b; Rigotti and Gugerli 2007; Chikh Ali et al. 2010). Specifically, certain types of genetically characterized PVY strains, like PVY^{C} and PVY^{N}, were largely displaced by new, recombinant types, like $PVY^{N\text{-}Wi}$, $PVY^{N:O}$, and PVY^{NTN}, which were not characterized genetically (Blanchard et al. 2008; Kerlan and Moury 2008). Some of the recombinants, like PVY^{NTN}, were initially noticed only because of their association with a new disease in potato, PTNRD (Beczner et al. 1984; Le Romancer et al. 1994). Throughout 1990s and 2000s, the main focus of the PVY differentiation was on molecular tools to distinguish various types of PVY recombinants. Attempts were made to apply these new molecular tools to type the old, historical PVY strains, like PVY^{Z} and PVY^{E} (Kerlan et al. 1999; Singh 2009; Kehoe and Jones 2011), but these were not very successful because the old isolates lost their infectivity in long-term storage. Some data retrieved for the non-infectious material with original PVY^{Z} pointed at N-type serology (Singh 2009) or to PVY^{O}-type capsid protein sequence present (Kerlan et al. 1999; Kehoe and Jones 2011), and were, thus, contradictory. A search for PVY^{Z} and PVY^{E} isolates among field isolates of PVY was the best strategy to characterize these historical strains.

In 2008, a seminal paper was published (Singh et al. 2008) that drew attention to the lack of biological characterization of PVY recombinants in potato cultivars with defined genetic background. The problem became very practical—PTNRD was clearly not confined to only PVY^{NTN} recombinants, and new, specific tools to identify PTNRD-inducing PVY isolates were needed (Singh et al. 2008; Gray et al. 2010). Molecular and serological tools were useful to distinguish between various recombinant types of PVY, but they could not identify whether a particular recombinant, or a particular isolate would induce PTNRD in a particular potato cultivar.

This discussion paper (Singh et al. 2008) renewed interest in genetic classification of PVY strains, and prompted a series of publications addressing genetic typing of PVY recombinants on potato indicators. Hence, significant progress was made on biological characterization of PVY recombinants since 2008 (Barker et al. 2009; Kerlan et al. 2011; Galvino-Costa et al. 2012). This progress was based on biological characterization of PVY isolates on potato indicators and on concomitant whole genome sequencing. Importantly, molecular make-up for two genetic strains of PVY was determined, for PVY^{Z} (Kerlan et al. 2011) and PVY^{E} (Galvino-Costa et al. 2012). Both PVY^{Z} and PVY^{E} genomes turned out to be recombinant (Figs. 1, 2), which is consistent with the timing of their initial collection and characterization in the 1980s and early 1990s (Jones 1990; Kerlan et al. 1999). PVY^{E} represented a new recombinant type associated with PTNRD (Galvino-Costa et al. 2012), with parental genomes identified as PVY^{NTN} and PVY-NE 11, recombinants themselves (Lorenzen et al. 2006a, 2008). PVY^{Z}, on the other hand, was found to have a well-known PVY^{NTN} genome long known to be associated with PTNRD (Hu et al. 2009b; Kerlan et al. 2011).

This identification of PVY^{Z} genome as having a typical PVY^{NTN} recombinant structure came as a surprise, and prompted a study on interactions of ordinary PVY^{NTN} isolates with the *Nz* resistance gene, present in cultivar Maris Bard (Kerlan et al. 2011; see Table 1). Subsequent biological characterization of four additional PVY^{NTN} isolates on cultivars Desiree, King Edward, and Maris Bard indicated that two of them induced typical HR, and two—severe local and systemic HR-like necrosis in Maris Bard, while producing no HR or systemic necrosis in both Desiree and King Edward (Kerlan et al. 2011; Galvino-Costa et al. 2012). A similar reaction was reported by Barker et al. (2009) for a PVY^{NTN} isolate PVY^{NTN}-Slo in Maris Bard, i.e. local and systemic HR, while no HR on Desiree, Pentland Crown, King Edward, and Duke of York. Thus, it appears that both PVY^{Z} and PVY^{NTN} strain groups elicit HR in cultivars harboring *Nz* gene (Kerlan et al. 2011), and the only difference between PVY^{Z} and PVY^{NTN} isolates is their symptoms in tobacco: PVY^{Z} induces mosaic and vein clearing, while PVY^{NTN} induces vein necrosis (Tables 1, 2). Consequently, Kerlan et al. (2011) proposed that PVY^{Z} and PVY^{NTN} are biologically very close or identical in potato and should be named PVY^{Z}-NTN. In an amended genetic classification, tobacco symptoms were suggested to be subordinate to symptoms in potato indicators (Kerlan et al. 2011). Hence two of the five original, historic strains of PVY were found to have recombinant genomes (see Table 1).

Diversity Within Strain Groups

In the course of the 2004–2006, U.S.-wide survey of seed potato crops, more than 4,000 PVY positive samples were typed using biological reaction on tobacco, serological profiling with PVY specific monoclonal antibodies, and RT-PCR profiling using multiplex assay identifying the two most prominent recombinant junctions in the PVY genome (Gray et al. 2010). An unusual sub-group of isolates was revealed during this survey, which was assigned to the PVY^{O} strain based on the absence of recombinant junctions (RT-PCR assay), failure to induce vein necrosis in tobacco, and positive reaction to PVY^{O}-specific monoclonal antibody MAb2 (Ellis et al. 1996; Karasev et al. 2010; Gray et al. 2010). Nevertheless, this sub-group of isolates was distinguished from other, ordinary PVY^{O} isolates by a distinct serological marker, i.e. positive reaction towards one PVY^{N}-specific monoclonal antibody, 1F5 (Karasev et al. 2010).

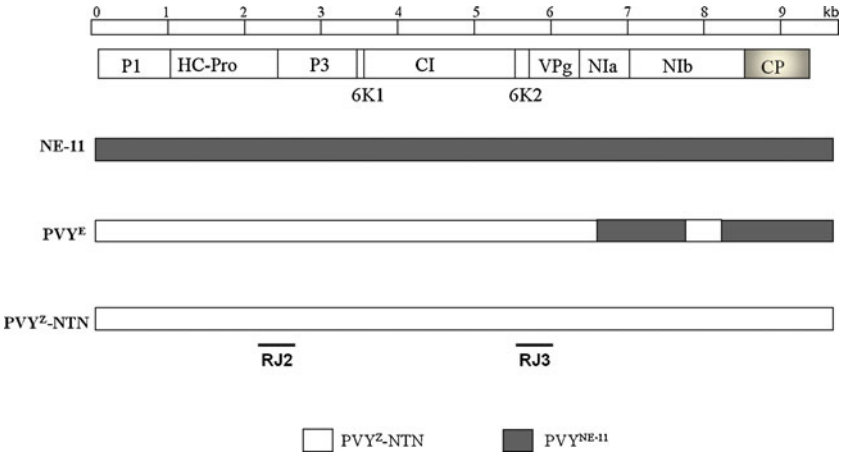

Fig. 2 Schematic diagram of a rare recombinant type of PVY, PVY^{E}, with genome composed of two parental genomes, PVY NE-11 and PVY^{Z}-NTN (Galvino-Costa et al. 2012). Positions of the two recombinant junctions, RJ2 and RJ3, characteristic of PVY^{Z}-NTN are labeled below the diagram. Note that if only RJ2 and RJ3 are probed using a multiplex RT-PCR assay (Lorenzen et al. 2006b), PVY^{E} will be mistaken for PVY^{Z}-NTN

This sub-group was named PVY^{O}-O5 (Karasev et al. 2010), and subsequently clearly identified as belonging to the PVY^{O} strain group through whole genome sequencing conducted on multiple, more than 30 isolates displaying this unusual serological marker (Karasev et al. 2011). Remarkably, almost all PVY^{O}-O5 isolates grouped into a distinct, separate evolutionary lineage when whole genomes were subjected to phylogenetic analysis, suggesting that this serological marker reflected a more profound diversity within the PVY^{O} strain (Karasev et al. 2011). Isolates belonging to this PVY^{O}-O5 lineage were initially found in the U.S. (Gray et al. 2010; Karasev et al. 2010), but later identified in Canada (Nie et al. 2011), and in Australia (Kehoe and Jones 2011). Although biologically PVY^{O}-O5 isolates were found similar to ordinary PVY^{O}, they demonstrated substantial differences in certain biological features, notably in their interaction with the *Ny* gene in potato—reaction against PVY^{O}-O5 was much more severe than against PVY^{O} in cultivars bearing *Ny* gene (Karasev et al. 2011). It is important to point out that PVY^{O}-O5 was found ecologically expanding during the 3-year PVY survey (Gray et al. 2010), and thus hypothesized to have certain evolutionary advantages over the ordinary PVY^{O} isolates (Karasev et al. 2010).

Similarly the $PVY^{N:O/N-Wi}$ strain can be divided into the two groups $PVY^{N:O}$ and PVY^{N-Wi} (sometimes referred to as the a and b subgroups) (Lorenzen et al. 2006a) by the presence of an extra recombination junction (Fig. 1). Additionally, these groups can be further subdivided on biological properties. The U.S./Canadian survey (Gray et al. 2010) identified significant numbers of variants within the $PVY^{N:O/N-Wi}$ strain that did not cause necrosis in tobacco. Furthermore, the isolates within all variant groups differ in their ability to cause PTNRD. The diversity of isolates within the $PVY^{N:O/N-Wi}$ appears to be significant, although the spatial and temporal characteristics of distribution are unknown since recent surveys have not generally used the biological tests that can separate the variant groups and molecular characterization has not been completed.

Concluding Remarks

The biological and genetic diversity of PVY is vast and it is clear the research community will continue to struggle with taxonomic schemes until the molecular determinants regulating the biological and genetic properties of the various strain groups and variant groups within strains can be better identified. This should then lead to improved diagnostic capabilities that can more efficiently categorize PVY isolates into a meaningful and understandable taxonomy. Nevertheless, this is likely to be a continually moving target given the changes is strain composition in recent times. The PVY^{N} strain has all but disappeared from potato in the U.S. and Canada, with only a handful of isolates being identified in surveys of the national seed potato crop since 2004. The predominance of the PVY^{O} strain also appears to be waning in many of the potato production regions of the U.S. and similar findings have been reported from other geographic regions. The recombinant $PVY^{N:O/N-Wi}$ and PVY^{NTN} are becoming prevalent in potato production and the reasons are not fully understood. In general, the recombinant strains tend to induce foliar symptoms that are milder that the PVY^{O} strain. This has compromised the efficiency of seed certification inspections and on-farm rogueing operations and perhaps has led to increased virus incidence in seed stocks. Also contributing is a practice of some seed certification schemes to allow a higher tolerance from mild mosaic than severe mosaic that can also lead to a selection of recombinant types of PVY. Regardless of the reasons, the recombinant strains of PVY are on the increase and they have the potential to significantly increase the consequences of PVY on the potato crop due to the ability of some of these strains/variants to cause PTNRD. Currently PVY is a major disease concern of seed growers, if the PTNRD strains of PVY continue to increase and become endemic, PVY could become a major disease concern for the entire potato industry, growers and processors, as well as consumers.

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