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Brief Review

Discussion paper: The naming of *Potato virus Y* strains infecting potato

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Summary

Potato virus Y (PVY) strain groups are based on host response and resistance gene interactions. The strain groups PVY^O, PVY^C and PVY^N are well established for the isolates infecting potato in the field. A switch in the emphasis from host response to nucleotide sequence differences in the virus genomes, detection of isolates recombining sequences of different strains, and the need to recognize isolates that cause necrotic symptoms in potato tubers have led to the assignment of new acronyms, especially to isolates of the PVY^N strain group. This discussion paper proposes that any newly found isolates should be described within the context of the original strain groups based on the original methods of distinguishing strains (i.e., tobacco and potato assays involving use of 'differential' potato cultivars). Additionally, sequence characterization of the complete genomes of isolates is highly recommended. However, it is acceptable to amend the names of PVY isolates with additional, specific codes to show that the isolate differs at the molecular, serological or phenotypic level from the typical strains within a strain group. The new isolates should preferably not be named using geographical, cultivar, or place-association designations. Since many new variants of PVY are being discovered, any new static classification system will be meaningless for the time being. A more systematic investigation and characterization of PVY from potato at the biological and molecular levels should eventually result in a biologically meaningful genetic strain concept.

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Introduction

Potato virus Y (PVY, species *Potato virus Y*) is the type member of genus *Potyvirus*, the largest group of plant viruses, containing 128 approved and 89 tentative species [30]. According to potato specialists in most parts of the world, PVY is currently

considered to be economically the most harmful virus in cultivated potatoes, as the relative significance of potato leaf roll virus (PLRV) has decreased in most countries (reviewed in 93). Australia is exceptional in that PVY has never caused significant problems to potato production there, but PLRV still does. At the same time, previously unknown strains of PVY have evolved in or spread to new geographical areas [10, 45, 56]. The emergence of molecular techniques and a switch in the emphasis from host response to nucleotide sequence differences in characterization of PVY isolates has led to the assignment of new acronyms to PVY isolates and strains. This paper analyzes the reasons behind the names currently used with PVY strain groups and strains. Its purpose is to stimulate discussion over the naming of PVY strains to help overcome the current confusion in PVY strain and isolate nomenclature. It is proposed that more systematic accumulation of biological data on host responses, especially those corresponding to the criteria of the established strain group concept for PVY from potato, and sequence data covering the whole genome of each isolate will be needed.

Historical background of alphabetized nomenclature for potato viruses

In the infancy of plant virology, distinct disease symptoms caused by viruses infecting cultivated potato (*Solanum tuberosum* L.) were the driving force in naming the different viruses. At that time, the causative virus entities were not known and so could not be included in the virus names. Attempts were made [71] to standardize the ''degeneration diseases'' (virus diseases) by growing ''pure cultures of diseases'' in different potato cultivars and observing the characteristic symptoms that developed. Thus, *common*, *interveinal*, *marginal*, *crinkle*, *severe* or *rugose* and *simple* mosaic diseases were all described for potatoes. The method of inoculation used initially was grafting but the development of needle inoculation and the use of 'filter hosts' prompted several groups of researchers to dissect the *rugose* mosaic complex, one of the most damaging diseases in potatoes at the time. Two studies [47, 91] separated two constituents of the *rugose* mosaic into an aphid-transmitted virus and a sap-transmitted mottle virus. The same year, for the sake of clarity, Smith [82] separated and named the two component viruses as Y and X, respectively, based on the transmission mode and the host reaction in tobacco (*Nicotiana tabacum* L.). Component X, which caused double concentric rings with a central spot in tobacco, was referred to as ringspot, while component Y represented the aphid-borne virus, which in tobacco caused a darkening of the green colour of the tissues along the veins (vein banding). The X and Y components became the origin of the *Potato virus X* (PVX) and *Potato virus Y* (PVY) names used thereafter. These subsequently became the type members of two viral 'genera' (*Potexvirus* and *Potyvirus*, respectively) in the current viral taxonomy [30]. Taken together, in the early years of potato virology, PVY was referred to with at least the following names: potato virus 20, potato virus C, potato acropetal necrosis virus, potato leafdrop streak virus, potato severe mosaic virus, potato stipple streak virus, potato streak virus, potato veinal necrosis virus, Solanum virus 2, tobacco veinal banding virus, and tobacco veinal necrosis virus.

The tradition of potato virus nomenclature using single alphabet letters continued and, because they caused distinct symptoms in potato, other viruses were called by different alphabetized names. For example, *crinkle mosaic* became potato virus A (PVA) [60], *foliar necrosis* became potato virus D (PVD) [7], *top-necrosis* viruses became potato virus B (PVB) and potato virus C (PVC) [8], *pseudo-net-necrosis* became potato virus F (PVF) [21], and *aucuba mosaic* became potato virus G (PVG) [21]. In the 1950s, *interveinal mosaic* was named potato virus M (PVM) [5] and the viruses described earlier as potato virus E (PVE) [27] and potato virus K (PVK) [48] were shown to be isolates of PVM. An additional virus previously considered to belong to the PVM group was assigned the name of potato virus S (S stands for Van Slogteren, who first discovered this virus) [76]. Several of these earlier names for viruses are no longer in use. For example, the viruses first described as potato virus B and potato virus C were soon shown to be strain groups of PVX and PVY, respectively.

Early delineation of PVY strains based on the resistance gene interaction in potato

In the early days, it was observed that PVY caused a variety of symptoms with differing intensity depending on the potato cultivar infected [9, 22]. Thus, there were distinct types of virus grouped purely by their distinctive symptoms expressed in different potato cultivars. PVY, the forerunner of the alphabetized nomenclature, was subjected to a second wave of alphabetized superscript-nomenclature as its strains were divided into distinct strain groups based especially on their ability or inability to induce top necrosis when graft-inoculated to differential potato cultivars or systemic necrosis in tobacco following sap-inoculation.

Early studies on virus resistance in cultivated potatoes showed the inheritance of dominant, monogenic resistance against some potato viruses [16, 22, 39, 42, 87]. Thus, the use of differential potato cultivars enabled the viruses to be distinguished on the basis of a hypersensitive resistance response (HR) visualized by development of necrotic local lesions in inoculated leaves with or without systemic necrosis, and systemic necrosis in graft-inoculated plants. For example, PVA was distinct because *top necrosis* following infection with PVA corresponded to the gene *Na*, potato virus B to *Nb*, potato virus C (= PVY^C) to *Nc*, PVY (= PVY^O) to *Ny*, PVX to *Nx*, etc. When these genes were first found and named, it was not realized that potato virus C and PVY were different strain groups of the same virus, which was also the case with potato virus B and PVX [94]. Thus, the original potato virus C, which induced top necrosis in differential cultivars that responded without top necrosis to the original PVY, was renamed as the PVY^C strain group [22]. The original virus Y [9] and 'old' Y strain [2] were named PVY^O, where 'O' stands for 'ordinary' [25]. However, in some papers PVY^O has been referred to as 'strain normal (Y^N)' [38, 58].

Some strains of PVY failed to elicit HR genes in potato and hence overcame the resistance. However, they induced systemic necrosis in tobacco (*N. tabacum*) and so were named as PVY^N [25] (see more details below). A group of strains that did not induce necrosis in tobacco and did not fit into either PVY^C or PVY^O based on necrotic reactions in differential potato cultivars was called strain group PVY^Z [42].

Some potato cultivars also contain resistance genes that are effective against all strains of PVY. These dominant genes for extreme resistance (inhibition of virus accumulation in infected tissues) come from *S. tuberosum* subsp. *andigena* (gene Ry_{adg}) or the wild species *S. stoloniferum* (Ry_{sto}) [23, 70, 75, 94]. While these genes are not useful for differentiation of PVY strains and therefore not considered further here, they are very important in breeding for PVY resistance. These PVY resistance genes have been mapped to chromosomes XI and XII, respectively, and molecular marker-assisted selection for both genes is possible [33, 98]. Gene *Ny* for HR to PVY^O has been mapped to chrosome IV [17], but the chromosomal positions of genes *Nc* and the proposed gene *Nz* [42] are not known. Hence, the genes for HR cannot be detected using molecular markers and need to be identified by inoculating plants with PVY isolates representing the different strain groups.

PVY strains from pepper, tomato and tobacco

PVY is an important pathogen not only in potato but also in other solanaceous crops such as pepper (*Capsicum* spp.), tomato (*Solanum lycopersicum* L.) and tobacco. Isolates of PVY infecting pepper in the field are often unable to cause systemic infection in potato [34, 74]. Likewise, some isolates of PVY from potato can show limited ability to infect pepper plants [32, 74; Gray, unpublished data]. It was suggested that typical pepper isolates of PVY could be distinguished from those identified in potato by the milder symptoms they cause in tobacco (cv. Xanthi) [74]. The biological differences between potato and pepper isolates are also reflected by overall differences in the viral sequences. In addition, because of systematic differences in the coat protein (CP)-encoding sequences, the potato and pepper strains of PVY can be distinguished with monoclonal antibodies to the CP [84] and restriction or phylogenetic analyses of the CP sequences [74]. However, many isolates from pepper are related to PVY^C from potato [29, 74; Schubert, un-

published]. Furthermore, while showing variability in biological properties, based on their CP sequences, the pepper isolates of PVY seem to constitute a single genetic strain [74]. In contrast, PVY^{C} strain group isolates from potato are a homogenous pathotype but exist as two genetic strains revealed by analysis of the CP-encoding sequences [11].

Isolates of PVY infecting pepper in the field were originally divided into three main groups (pathotypes) based on their ability to overcome the recessive resistance genes $pvr2^{1}$ and $pvr2^{2}$ in *Capsicum annuum* L. Isolates that are unable to overcome these genes and can infect only genotypes lacking them belong to pathotype 0. Isolates that overcome $pvr2^{1}$ belong to pathotype (0, 1), and those which overcome both resistance genes ($pvr2^{1}$ and $pvr2^{2}$) belong to pathotype (0, 1, 2) [34]. However, $pvr2^{1}$ or $pvr2^{2}$ were subsequently found to be allelic and the same locus to contain additional recessive alleles of the PVY resistance gene (reviewed in 3). The *pvr2* alleles that do not confer resistance to PVY have been designated as $pvr2^{+}$ [59]. The recessive resistance controlled by the *pvr2* alleles is not associated with an induced response to infection. This is in contrast to interactions of PVY with the dominant potato resistance genes *Ny* and *Nc* specific to PVY^{O} and PVY^{C}, respectively. There are also additional recessive and dominant genes for resistance to PVY in *Capsicum* spp. [3, 51], but the aforementioned pathotype concept based on the *pvr2* alleles is still in use. PVY may cause necrotic local lesions, systemic vein necrosis and top necrosis in pepper, which is not currently considered a resistance response [3, 29] but which it might well be.

Interaction with the four *pvr2* alleles ($pvr2^{+}$, $pvr2^{1}$, $pvr2^{2}$ and $pvr2^{3}$), of which all but $pvr2^{+}$ confer resistance to PVY, is controlled by a 23-amino-acid-long region in the viral genome-linked protein (VPg) [4]. The *pvr2* alleles, in turn, are sequence variants of the translation initiation factor 4E (eIF4E). Mutations in VPg and eIF4E interfere with the interaction of these proteins. Consequently, the virus fails to accumulate in inoculated tissues and complete its infection cycle [4, 77]. Because the viral and host genes and the specific features of their sequences involved in the PVY-*pvr2* interactions have been identified, it should be possible to use the VPg sequence to place an isolate to the respective pathotype. Indeed, knowledge on sequence variability of the VPg region and the virulence of the viral variants in pepper genotypes carrying different *pvr2* alleles distinguishes a total of eight pathotypes instead of the original three [4].

Potato and pepper as hosts seem to be selective for PVY strains. While PVY isolates from these two species may not be readily exchangeable, it seems that tomato and tobacco can be infected with most PVY isolates from potato and pepper [1, 88; and Refs. therein]. It has been difficult to define criteria by which PVY strains from tomato could be placed to groups that would correspond to biological differences or pathotypes similar to those described for potato and pepper strains. For example, the monoclonal antibodies used to detect strains O, N and C among potato isolates of PVY do not provide a meaningful grouping of tomato isolates in terms of biological differences [1]. The *pot-1* gene introgressed from *Lycopersicon hirsutum* to tomato confers resistance to PVY and tobacco etch virus and, similar to *pvr2* alleles in pepper, mutations in the VPg of PVY overcome resistance [59]. However, this information and the VPg sequences have not yet been used for grouping tomato isolates to pathotypes.

PVY isolates from tobacco can be placed to three strains based on the necrotic symptoms they cause in tobacco plants [37]. Isolates of strain $M^{S}N^{R}$ [37; also referred to as MsNr or MN in the literature] induce necrosis only in tobacco plants that carry the dominant root-knot nematode resistance gene *Rk*. In contrast, isolates of strain $M^{S}M^{R}$ cause mosaic symptoms and strain $N^{S}N^{R}$ necrotic symptoms, regardless of the *Rk* gene [37]. The linkage of nematode resistance controlled by *Rk* and the necrotic response to the PVY strain $M^{S}N^{R}$ is so tight that inoculation of detached tobacco leaves with PVY-$M^{S}N^{R}$ is suitable for use in screening tobacco breeding lines for nematode resistance [100]. There is evidence that the replicase (NIb) of PVY determines the necrotic response in the presence of *Rk*, but this awaits confirmation by mutational analysis

of an infectious clone of PVY [31]. ''Partial'' resistance to some isolates of PVY is conferred by the recessive gene *va* and some other genes in tobacco. These genes can also be used for grouping PVY isolates (for further information, see Descriptions of Plant Viruses no. 414 at www.dpvweb.net). However, there is little information as to how PVY isolates belonging to the three strain groups defined by their reactions to *Rk* would respond to the genes *Ny*, *Nc* or the proposed *Nz* in potato plants. Phylogenetic analysis of coat protein (CP) sequences indicates that the few M^SN^R and N^SN^R isolates studied so far are most closely related to PVY^C [14, 54; Tian et al. unpublished].

Isolates of PVY from tobacco can infect potato plants systemically with a few exceptions [56], and as already mentioned, PVY isolates from potato infect tobacco plants systemically. Induction of local and systemic veinal necrosis in leaves and sometimes stem necrosis, in contrast to the leaf mosaic symptoms, divides the PVY isolates from potato into two strain groups or pathotypes in tobacco. Grouping of isolates based on phylogenetic analysis of the CP-encoding sequences correlates well with the necrotic and mosaic symptom phenotypes of the isolates [19, 53, 95]. Another genomic region which encodes the helper-component proteinase (HC-Pro) contains important determinants for the necrotic phenotype observed in tobacco [90], but they may not be the only determinants needed to induce necrosis [78].

PVY^N strain group

In the 1940–50s, a variant of PVY was detected in potatoes in many countries in South America and Europe and was referred to by various names. It caused veinal necrosis in tobacco leaves and mild mottle symptoms in most potatoes and was referred to as 'necroses das nervuras' [67], '*veinal necrosis virus*' [9], the *tobacco vein browning* strain [2], ''*Rippenbräune*'' strain Y^R [66], *tobacco veinal* Y^N [25], and '*necrotic*' Y^R [38, 49]. Keller and Münster [44] designated Y^N to describe the *tobacco veinal necrosis* strain, although the only reference they cited was Nienhaus [66], which used the acronym Y^N to refer the *normal* strain of PVY. When PVY^N was discovered in the U.S.A. in two *Solanum* samples from Bolivia, the authors tagged a descriptive name of *tobacco veinal necrosis* strain of PVY (PVY-TVN) [43]. The PVY^N isolates induced necrosis in tobacco but did not induce necrosis in the presence of the genes *Nc* or *Ny* in potato cultivars. Hence, the ability to overcome these two genes for HR in potato seems to be linked with the ability to induce necrotic symptoms in tobacco. However, there is little information as to whether all PVY isolates from tobacco that cause necrotic symptoms in this host are able to overcome the aforementioned HR genes in potato.

In the 1980s, additional isolates of PVY^N were found, some of which were associated with potato tuber necrotic ringspot disease (PTNRD) [10, 45, 50, 86]. These isolates, first called PVY^{NN} [45], were given the acronym PVY^{NTN} [52]. Another group of PVY^N isolates characterized by differences in virulence in potato was reported in Poland and named $PVY^{N\text{-}Wi}$ based on their detection in potato cv. 'Wilga' [20].

A primary characteristic of PVY^{NTN} isolates is the production of PTNRD, yet this feature is highly variable. For example, the infection of PVY^{NTN} is not always accompanied by the development of necrotic rings on the tubers despite exhibiting 50–70% infection in the field (e.g., in cvs 'Mona Lisa' and 'Rosalie') [10]. Similarly, cvs 'Ágata', 'Achat', 'Atlantic, 'Asterix', 'Baraka Manjke' and 'Vivaldi' in Brazil (A.C. Avila, pers. com.) or 'Nicola', 'Linda', 'Belldonna' and 'Nadine' in Germany (Schubert, unpublished) exhibit tuber necrosis symptoms under field conditions, but not in all infected tubers. Even among the 11 isolates defined using molecular techniques as PVY^{NTN}, only one exhibited PTNRD symptoms in highly susceptible cv. 'Nadine' under greenhouse condition [99]. On the other hand, some PVY^N isolates which were isolated from symptomless tubers and not known to cause PTNRD in the field induced tuber necrosis in greenhouse experiments [15]. Thus, a reliable and sensitive biological assay for these strains is necessary, and the conditions responsible for reliable induction of tuber symptoms have yet to be defined. Furthermore, we do not yet understand which viral sequences or domains are actually re-

sponsible for a tuber necrosis phenotype, which hampers molecular detection of PVY^{NTN} isolates. The problem is increased by observations that even isolates of PVY^{N-Wi} type may cause tuber necrosis on sensitive cultivars [6, 69, 78]. Similarly, isolates of the PVY^{N} originating from New Zealand and showing the typical non-recombinant molecular structure of PVY^{N} strains can induce tuber necrosis in highly sensitive cultivars such as 'Nadine' [78].

Molecular virology and assignment of complex acronyms

With the discovery of a PVY^{NTN} isolates in several European countries [10, 45, 50, 86] and the emergence of PVY^{N} strains in North America [56, 79], the stage was set for another wave of altered nomenclature – this one based on the serological and molecular characterization of the virus. By this time, partial and complete nucleotide sequences of PVY RNA [35, 41, 54, 61–65, 73, 80, 89] were available in public databases and cloning and sequencing methodology was becoming a commonly used tool in plant virology. As a result, pathological, serological and molecular features were considered in the nomenclature of PVY^{N} isolates. For example, some PVY^{NTN} isolates characterized at the molecular level were found to be recombinants of PVY^{O} and PVY^{N} in the CP-encoding region [13, 14, 35, 72] and later were shown to display additional recombination junctions in the HC-Pro and nuclear inclusion protein a (NIa)- and b (NIb)-encoding regions [36].

Further isolates were identified from North America, Denmark, New Zealand, Germany, Poland and Japan, which were associated with PTNRD but which had no recombination junctions within the coat protein gene [14, 68, 78]. The PVY^{NTN} isolates not detected by a primer set developed against PVY^{NTN} from Europe [96] were referred to as North American PVY^{NTN} [97] or NA-PVY^{NTN} [62]. Additional PVY^{N} variants, which serologically reacted with PVY^{O}-specific monoclonal antibodies but caused veinal necrotic symptoms in tobacco plants (similar to PVY^{N-Wi}) [20] were reported in Canada [57] (isolates referred I-136 and I-L56) and in Spain (isolate 17) [12]. They were subsequently found to be present in most potato-cultivating countries. Such isolates were considered to be recombinants [35] and later were demonstrated to have one recombination junction on the basis of RT-PCR and termed $PVY^{N:O}$ [64, 65] or one-to-two recombination junctions based on RT-PCR-RFLP analyses [36]. Some rare isolates

Table 1. The commonly described isolates, strain groups, synonyms and definitions of *Potato virus Y*

Proposed strain name	Strain group	Synonymous codes	Definition	Refs.
PVY^{O}	PVY^{O}	PBY^{O5}	Common or ordinary strain group, isolates elicit the gene *Ny*	[6, 25, 27]
PVY^{N}	PVY^{N}	PVY^{EU-N}, PVY^{NA-N}, NA-PVY^{N}, PVY^{R}, PVY^{-TVN}	Tobacco veinal necrosis strain group, isolates not known to cause PTNRD	[10, 25, 38, 54, 58, 62, 66, 97]
PVY^{NTN}	PVY^{N}	EU-PVY^{NTN}, Eu-PVY^{NTN}, PVY^{EU-NTN}, PVY^{NN}, PVY^{NA-NTN}, NA-PVY^{NTN}	PVY^{N} isolates able to cause PTNRD	[24, 45, 52, 54]
PVY^{N-Wi}	PVY^{N}	$PVY^{N-Wilga}$, PVY^{N-W}, PVYN-Wi-P, $PVY^{N:O}$	Recombinant isolates, phenotypically PVY^{N} but serologically PVY^{O}	[12, 20, 36, 46, 62, 64]
PVY^{C}	PVY^{C}	PVY^{C1}, PVY^{C2}	Strain group C, isolates elicit the gene *Nc*	[11, 22]
PVY^{Z}	PVY^{Z}		Strain group Z, isolates elicit the proposed gene *Nz*	[42]
PVY^{E}	PVY^{E}	PVY^{ZE}	Strain group E, isolates do not elicit *Ny*, *Nc* or *Nz* and do not cause necrosis in tobacco	[46]

of $PVY^{N\text{-}Wi}$ type may have four recombination junctions [78].

The Spanish PVY isolates 18 and 32 were amplified by a PVY^{N}-specific primer pair, and the amplified region revealed a similar restriction pattern to that of PVY^{N} [12]. However, the two isolates reacted serologically to PVY^{O}-specific antibodies and did not cause veinal necrosis symptoms in tobacco [12]. These isolates were able to overcome the HR genes, *Ny*, *Nc* and the proposed *Nz* in the potato cultivar 'Maris Bard'. They were described as PVY^{ZE}, i.e., variants of PVY^{Z} [46]. However, since *Nz* did not recognize these isolates, they do not, by definition, belong to PVY^{Z}. To avoid confusion over the use of "Z" in their name, we therefore propose that the distinct PVY strain group they represent be renamed PVY^{E}. Examples of the different names used for PVY^{N} isolates, including the recently used acronyms for isolates described in North America [24, 54, 55], are listed in Table 1.

Situation analysis of PVY isolates and their nomenclature

From the foregoing description of PVY nomenclature it is apparent that three aspects of virus research have propelled the nomenclature of the PVY strain groups in potato. The first approach was based on host response. Both phenotypic differences (mosaic or necrotic symptoms in tobacco) and resistance gene interaction (elicitation of specific HR genes) influenced the assigning of PVY strain groups PVY^{O}, PVY^{C}, PVY^{N} and PVY^{Z} [26, 42]. Appearance of isolates with the potato tuber necrotic ringspot phenotype within the PVY^{N} strain group created a need to distinguish the two putative pathotypes of this strain group using serological and molecular methods. Finally, molecular analysis of PVY genomes revealed an increasing number of isolates with recombined genomes of PVY strains that belong to different strain groups based on host responses or serological criteria. Detection of recombinants has fuelled the use of additional acronyms (Table 1).

One strain group, PVY^{O5} [6, 28], was suggested purely on the basis of serological reaction with monoclonal antibodies. However, serological detection of the PVY strain groups based on monoclonal antibodies is not yet fully established. The epitopes (amino acid residues or their combinations in CP) responsible for recognition of the different strain groups (PVY^{O}, PVY^{N}, PVY^{C}, PVY^{Z}, PVY^{E}) by the monoclonal antibodies (MAbs) used have not been experimentally determined. However, in a recent study, the amino acid residue Gly29 was found to be the key determinant for detection with a MAb of 24 PVY isolates which do not induce necrosis in tobacco, whereas the 28 isolates of strain group N, including NTN, had a Glu residue at this position and were not detected [19]. Because there is no evidence that CP is responsible for the mosaic or veinal necrosis phenotype of PVY in tobacco or recognition of PVY by the aforementioned resistance genes, the risk remains that use of serological criteria alone will be erroneous regarding the strain group assignment of a PVY isolate.

A putative molecular determinant consisting of two amino acids in HC-Pro exists for the necrotic phenotype of PVY in tobacco [40, 90], but what remains unanswered is whether all PVY^{N} isolates have these two amino acid changes and whether these amino acids are solely responsible for the phenotype [78]. Clearly there is no one diagnostic tool or method that reliably distinguishes all PVY isolates with similar phenotypes. Ultimately, it will be possible to answer these questions, but currently there is no complete answer as exceptions remain [78].

The importance of the strain group concept

Identification of potato isolates of PVY to strain groups or pathotypes on the basis of phenotypic reactions in differential potato cultivars carrying specific resistance genes (*Ny*, *Nc* or the proposed *Nz*) has been well justified because it is important in breeding for resistance. The pathotypes of PVY isolates from pepper are also defined based on the virus-resistance gene interaction for the same reason. The genetic pathotype-resistance gene concept for pepper isolates was recently converted to a molecular model by identification of the viral avirulence determinant (VPg) and the resistance gene (eIF4E) [4, 59]. The same thing remains to be

Table 2. Resistance specificity and responses of the differential potato cultivars and tobacco plants (*Nicotiana tabacum*) to strains of *Potato virus Y*[1]

Differentials	PVY strain group reaction					PVY-specific resistance genes[2]
	PVY^C	PVY^O	PVY^N	PVY^Z	PVY^E	
'King Edward'	HR	s	s	s	s	*Nc*:*ny*:*nz*
'Pentland Crown' or 'Desiree'	s	HR	s	s	s	*nc*:*Ny*:*nz*
'Pentland Ivory' or 'Maris Bard'	HR	HR	s	HR	s	*Nc*:*Ny*:*Nz*
Tobacco (e.g., cvs. Samsun or Xanthi)	s	s	VN	s	s	–[3]

[1] Data from Refs. [23], [42] and [94]. *HR* Hypersensitive resistance response (localized or systemic); *s* susceptible (systemic infection, no necrosis); *VN* local and systemic veinal necrosis.
[2] *Nc*, *Ny* and the proposed *Nz* are dominant genes that confer strain-group-specific HR to PVY.
[3] Tobacco cultivars that contain the gene *Rk* for resistance to the root-knot nematode [37] are not suitable to use.

achieved with the potato strains of PVY and the corresponding genes for HR.

Linking the important phenotypic traits to molecular signatures will be possible following further accumulation of reliable data on PVY isolates, both concerning their strain group assignment using 'differential' potato cultivars that have well-defined pedigree information (Table 2) and determination of complete genome sequences. The risk associated with using less characterized potato cultivars lies in the additional or different virus resistance genes they may contain. For example, potato virus V (PVV) was originally misdiagnosed as PVY^C because the differential cultivar with *Nc* also contained the gene *Nv* for HR to PVV [42, 85]. Wild potato species and the cultivated species other than *S. tuberosum* contain resistance genes that have different (broader) recognition specificities than the genes *Ny*, *Nc* or the proposed *Nz*. For example, some accessions of the wild species *S. chacoense*, *S. sparsipilum*, *S. stoloniferum* and *S. sucrense* [92] and *S. tuberosum* subsp. *andigena* [81] express HR to PVY^N. The gene or genes of *S. demissium* introgressed to potato clone 'A6', which is used to index potato viruses, control(s) HR to PVY^O, PVY^C and PVY^N [23]. The gene Ry_{sto} from *S. stoloniferum* responds to all PVY strains but confers extreme resistance or necrotic responses of different severities depending on the potato genetic background to which it has been introgressed [75]. Therefore, the use of wild species germplasm in breeding programs may introduce unexpected responses to the different PVY strain groups, including PVY^N [23, 75, 92], in new cultivars.

The tobacco cultivars carrying the *Rk* gene are unsuitable for the strain group identification of PVY isolates from potato because these cultivars will react with necrotic symptoms to certain PVY isolates that would otherwise cause only mosaic symptoms [37]. Using them causes the risk that isolates of PVY^O, PVY^C, PVY^Z and PVY^E will be erratically considered deviant isolates or putative recombinants of two strains due to elicitation of genes *Ny*, *Nc* or the proposed *Nz*, respectively, and, additionally, induction of necrosis in tobacco.

Certain potato cultivars such as Hermes, Nadine, Nicola and Yukon Gold [6, 15; Schubert and Kerlan, unpublished] are particularly prone to develop tuber necrosis symptoms with PVY isolates designated as PVY^{NTN}. While these cultivars can be recommended for use as indicators for detection of isolates that can induce tuber necrosis, it is equally important also to test the isolates in tobacco and the 'differential potato cultivars'. After doing this, the isolates can be firmly related to the strain group concept, and it is possible to reveal whether they express recombined traits of more than a single strain group. Currently, PVY^{NTN} isolates are considered to belong to the PVY^N strain group because of the uncertainties in confirming that a PVY^N isolate would not cause tuber necrosis in any cultivar and under any environmental conditions, as discussed above. Necrosis induced in potato tubers, and sometimes also in other parts of the plants, by the isolates of this

strain group may be an HR-like response, but the host genes controlling it are not known, which hampers the establishment of strain group criteria similar to those of PVY^O, PVY^C and PVY^Z.

It is vital also to establish that no other viruses are present in mixed infection with PVY before 'differential' potato cultivars are used to characterize an isolate, as many cultivars including those proposed in Table 2 for use contain genes for HR to several viruses [42, 83, 94]. Furthermore, isolates from different strain groups of PVY may co-infect potato plants in the field, which can be detected because the mixture of strains will elicit necrotic responses in a higher number of the differential varieties than expected for any single strain (Table 2). Finally, it is important to emphasize that the strain group classification discussed here is applicable only to PVY isolates obtained from potato. As mentioned above, PVY isolates from other host species such as pepper often show reduced virulence on potato [34, 74] and cannot be reliably classified using the 'differential' potato cultivars.

There is already evidence that extensive analysis of PVY sequences and their use to study phylogenetic relationships results in a grouping of isolates that corresponds to the PVY strain group concept defined as described above. The grouping can be further refined by identifying points of recombination and taking the information obtained into consideration [55]. To predict the phenotypic traits based on sequence information requires identification of the genomic regions of PVY responsible for specific host responses, which in turn requires critical studies using experimentally made chimeric viruses [59, 90]. Eventually, the current PVY strain concept may be converted to a biologically meaningful genetic strain concept.

Suggestions for the future naming of PVY strains infecting potato

1. Considering the recent trends and historical findings in PVY research discussed above, it is too early to propose that PVY strain identification in potato be based on molecular (sequence or serological) information. It is tempting to test a new isolate initially through sequencing because it is a relatively quick and straightforward procedure. However, although generation of more sequence data on new isolates is very important, definitive strain group assignment for potato isolates can only be provided using inoculation to 'differential' potato cultivars (Table 2) and tobacco plants. Because the exact parts or motifs of the PVY genome responsible for the various phenotypic features, most importantly those recognized by *Ny* and *Nc* gene and the suspected *Nz* gene have not yet been identified, it is impossible to predict the strain group accurately solely based only on genomic sequence variation. However, sequence data could be used in the future once the sequence motifs responsible for change in phenotype are understood.

2. It is suggested that if a nucleotide difference (even recombination) does not alter the biology of the virus (i.e., what it does to the potato and tobacco plant), then it does not justify designation of a new 'strain' descriptor. Indeed, we propose reduction of nomenclature to the names indicated in Table 1. With advanced methods for detecting recombination points in viral genomes, the number of known recombinant PVY isolates will continue to increase [18]. For example, the *NTN* designation is currently meaningful only when the ability of the isolate to cause tuber necrosis has been demonstrated experimentally. Once the molecular determinant(s) for tuber necrosis is/are identified, then the *NTN* designation could be applied on a molecular basis and any difference so identified. This is not yet the case.

However, it is acceptable to amend the names of PVY isolates with additional, specific codes to show that the isolate somehow differs, at the molecular or phenotypic level, from the typical strains in a strain group. For example, some recombinant strains of PVY^N react with antibodies to PVY^O but not PVY^N, which deviates from the typical, expected serological behavior of PVY^N and is of diagnostic importance. Therefore, we propose that these isolates be named $PVY^{N\text{-}Wi}$ within the PVY^N strain group (Table 1). Similarly, there might be recombinant isolates that belong to strain groups PVY^O, PVY^C or PVY^Z but are found to react with antibodies to

PVY^N. Such isolates would deserve to be considered a strain in the strain groups PVY^O, PVY^C or PVY^Z, respectively.

3. It is recommended that isolates should not be named after the countries in which they are first found. The increase in global trade, which moves plant material, including potatoes, over long distances is likely to mean that country or location descriptors soon become meaningless as isolates become established in new locations, either in nearby countries or on different continents. From a policy or international trade perspectives, these designators can be very confusing and are a potential deterrent to the international potato trade.

4. Nucleotide sequence information is of limited value unless it can be linked to a stable phenotype that has relevance (in this case to the potato industry). It is suggested through this proposal that virologists all over the world working with PVY isolates from potato would follow the aforementioned principles in the naming of new isolates.

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