Journal of Biology, Agriculture and Healthcare
ISSN 2224-3208 (Paper) ISSN 2225-093X (Online)
Vol.5, No.9, 2015 

# A Review Paper on Potato Virus Y (PVY) Biology, Economic Importance and its Managements

Binyam Tsedaley
College of Agriculture and Veterinary Medicine, Jimma University, P.O. Box 307, Jimma, Ethiopia
* E-mail of the corresponding author: binyamtsedaley@gmail.

**Abstract**
PVY is distributed worldwide in potato growing areas, tobacco growing areas and outdoor crops of tomato and pepper in warm countries. The different strains of PVY develops different symptoms on different parts of the host plant species. The PVY have a host range of 495 species in 72 genera of 31 families. Potato isolates have historically been divided into three main strain groups. The different strains of PVY have different stability under thermal inactivation point, dilution end-point and longevity *in vitro*. PVY have ssRNA nucleotide and two proteins (VPg and coat protein). The genomic RNA of PVY is positive sense and approximately 9700 kb in length excluding the poly(A) tail. PVY is spread from plant to plant either by aphids, mechanical means or by contact. In the family *Aphidinae*, *Myzus persicae* is clearly the most important vector of PVY given that it is widespread and with high transmission efficiency. Potato virus Y belongs to a group of the most important potato viruses infecting the potato, tobacco, pepper and tomato. PVY is the most important viral pathogen in potato worldwide and can cause yield loss of 10-100% and 39-75% on tobacco. The two main types of resistance in potato are extreme resistance and hyper-sensitive resistance. The generation of resistant cultivars is considered the most economic and environmentally acceptable way of controlling viral diseases in potato. Vector control plays an important role in management of PVY. Controlling the aphid plays a great role in the management of *Potato virus Y*. Since PVY is among the most important viral diseases of potato and that cause significant yield loss, so understanding its biology and developing an efficient management strategy is very important. In the real world there is no singly effective management of several important diseases, on behalf of this, searching a good integrated management approach is very crucial.
**Keywords:** Coat Protein; Hyper-sensitive Resistance; *Myzus persicae*; Potato; ssRNA; Vector control

## 1. INTRODUCTION

Potato (*Solanum tuberosum* L.) is one of mankind's most valuable food crops (FAO, 2004). It is the most important vegetable crop in terms of quantities produced and consumed worldwide (FAO, 2005). In volume of production it ranks fourth in the world after wheat (*Triticum aestivum* L.), rice (*Oryza sativa* L.), and maize (*Zea mays* L.) (Bowen, 2003). Potato is a highly recommended food security crop that can help shield low-income countries from the risks posed by rising international food prices (FAO, 2008). Potato has become an important staple and cash crop in the highlands of Sub-Saharan Africa. Because of its relatively short growing season (3-4 months) (Nyankanga *et al*., 2004). However, Potato production is treated by both biotic and abiotic factors. Potato is prone to more than a hundred diseases caused either by bacteria, fungi, viruses or mycoplasms (Paul, 1992). *Potato virus Y* (PVY) is a plant pathogenic virus of the family Potyviridae, and one of the most important plant viruses affecting potato production (Wikipedia, 2013). PVY was first recognised in potato in 1931 (Smith, 1931) as a member of a group of pathogens associated with potato degeneration, a disorder known since the 18$^{th}$ century. It has been one of the most studied plant viruses. It was the type species of the former Potyvirus group (Harrison *et al*., 1971).

PVY in potato has received a lot of attention in recent years, and indeed this virus is the most economically important disease problem in seed potatoes in many areas of the world. The virus is responsible for decreases in yield and quality, but the main issue in seed potato production is a requirement for strict virus tolerance limits for certified seed. High levels of PVY have been responsible for many seed lots being rejected as certified seed, resulting in a significant reduction in crop value (Gray *et al*., 2010). Measures to reduce losses caused by viral infections are both limited and expensive. But the spread of the virus can be controlled by several different methods including chemical protection from virus vectors, elimination of the sources of infection and by breeding new varieties with extreme resistance (Kang *et al*., 2005). The generation of resistant cultivars is considered the most economic and environmentally acceptable way of controlling viral diseases in potato (Solomon-Blackburn and Barker, 2001). With this regard the paper stands to review the general biology, economic importance and its management options of *Potato virus Y* (PVY).

## 2. HISTORY OF PVY

Since its identification, PVY has been seen as a complex of different isolates (Smith, 1931). *Potato virus C* (PVC), recognized as a PVY isolate (Bawden, 1943) when identified in the 1930s (Bawden, 1936), was the first of a strain group later named PVYC. Another strain group, now designated PVYN (De Bokx, 1961), was found

 

first in 1935 in a tobacco plant growing close to experimental potato plants (Smith and Dennis, 1940), then in Peruvian and Bolivian potato cultivars in 1941-1942 (Silberschmidt, 1960). This strain group was associated with severe epidemics in potato and tobacco crops in Europe in the 1950s (Weidemann, 1988). PVY was recognized in the 1950s as damaging to tomato crops in Australia and in South and North America (Simons, 1959), and to pepper crops in Florida (Simons, 1959). Due to the emergence of necrotic isolates in the 1970s and 1980s, PVY has become a major threat to both of these crops. Last, PVY has emerged as a severe problem in trailing petunias (Boonham *et al*., 1999).

Several steps in PVY history can be recognized, *e.g.* the emergence of the $PVY^N$ strain in potato and tobacco, of $PVY^N$ variants in potato, and of pathotypes breaking recessive resistance genes in tobacco and in pepper. Both $PVY^O$ and $PVY^N$ strains are thought to originate from the Andean countries where they were probably adapted to infection of various wild *Solanum* species in their natural habitats, maybe in separate regions (Jones, 1981). $PVY^O$ and $PVY^N$ could have diverged from a common viral ancestor that has followed two different evolutionary paths (Glais *et al*., 2002b), maybe on different hosts (either *Solanum* sp. or *Nicotiana* sp.). Indeed, from data regarding the biological and genetic specificity of pepper PVY isolates, it was inferred that the host seems to be an important factor in PVY evolution and that the strain specialization in the virus could be an effect of co-evolution with the plant (Romero *et al*., 2001).

Advances in knowledge of genomic diversity of PVY have provided more and more insights into PVY evolution. Links between mutation or recombination events in the genome and evolution of PVY have been thoroughly studied (Matousek *et al*., 2000; Glais *et al*., 2002a; Nie and Singh, 2003b). From phylogenetic analysis based on the P1 protein gene and the full genomic RNA sequence, it was suggested that North American-$PVY^N$ (NA-$PVY^N$) and European-$PVY^N$ (Eu-$PVY^N$) evolved in parallel, and that NA-$PVY^{NTN}$ originated from NA-$PVY^N$ by mutations, whereas Eu-$PVY^{NTN}$ evolved from Eu-$PVY^N$ and $PVY^O$ by recombination (Nie and Singh, 2003b).

## 3. MAIN DISEASE OF PVY

In potato, PVY causes a severe disease called mosaic or rugose mosaic. Symptoms are variable depending on viral strain, host cultivar, climatic conditions, and whether it is a primary infection (inoculation by aphid vectors) or secondary infection (when mother tuber is infected) (Draper *et al*., 2002). Symptoms in the aerial parts of the plants consist of a mild to severe mottle, often associated with distortion (crinkling) of the leaves. Yellowing and necrosis (vein necrosis and necrotic spots) frequently occur in the lower leaves. Symptoms also include collapse and dropping of intermediate leaves (leaf drop), which remain clinging to the stem. Secondarily infected plants are dwarfed and brittle, with crinkled and puckered leaves. Necrosis in tubers may occur in numerous cultivars (Beemster and De Bokx, 1987). The potato tuber necrotic ringspot disease, described in the 1980's (Beczner *et al*., 1984), has now spread worldwide.

PVY usually causes mild mottling in tobacco, tomato and pepper. However a large range of various symptoms may be observed and many strains induce necrosis. The veinal necrosis disease in tobacco, formerly described as associated with tobacco veinal necrosis virus, is caused by the $PVY^N$ strain. Occurrence of necrotic symptoms in tobacco crops may be associated with unbalanced nutrition, notably magnesium deficiency (Delon *et al*., 1997). Necrotic patterns in tomato plants include veinal necroses and necrotic spots on leaves and sometimes necrotic streaks on petioles and stems (Gebre-Slassie *et al*., 1985).

Isolates belonging to $PVY^N$ induce veinal necrosis on *N. tabacum cv. Xanthi* leaves and very mild mottling, with only occasional necrotic leaves on potato. $PVY^O$ isolates induce only mottling and mosaic symptoms on tobacco and mild to severe mosaic and leaf drop on potato. Finally, $PVY^C$ isolates induce stripple streak symptoms on some cultivars of potato (Jacquot *et al*., 2005). $PVY^N$ and $PVY^O$ isolates are responsible for high yield losses in potato and tobacco; corresponding to losses of up to 40–70% in the case of $PVY^O$ infecting potato (Van der Zaag, 1987) and 14–59% in PVY-infected tobacco crops (Latore *et al*., 1984). These yield losses can reach 100% in the case of veinal necrosis disease induced by $PVY^N$ isolates in tobacco. Given these facts, the efficient detection and identification of necrotic and non-necrotic PVY isolates in tobacco and potato crops has always been an important scientific challenge (Jacquot *et al*., 2005).

$PVY^C$ is a non-aphid transmissible strain of PVY, induces a hypersensitive response in the presence of the *Nc* gene and can cause stipple streak or mild mosaic patterns in a wide range of cultivars (Visser, 2012). $PVY^N$ was first described as “tobacco veinal necrosis” strain in the USA from samples originating from Bolivia (Kahn and Monroe, 1963). $PVY^N$ induces mild mottling and occasional leaf veinal necrosis in potato plants and may cause mild to slight damage of tubers in some instances (Crosslin *et a*l., 2005). $PVY^O$ (“ordinary” strain), which induces a hypersensitive response in the presence of the *Ny* gene, was first described in 1931 and is for the most part limited to foliar symptoms inducing mild to severe mosaic patterns on leaves (Smith, 1931). Both $PVY^N$ and $PVY^O$ have been identified in South Africa (Visser and Bellstedt, 2009). PVYNTN (NTN stands for “N-tuber necrotic”) is a genetic recombinant of PVYN and PVYO which was first described in Hungary in 1984 (Beczner *et al*., 1984). $PVY^{NTN}$ can result in mild to severe leaf symptoms including mild mottling and leaf

 

veinal necrosis but it is the necrotic flecking and potato tuber necrotic ringspot disease (PTNRD) this strain induces which is of greatest concern for seed and table growers (Visser, 2012).

## 4. SYMPTOMOLOGY AND HOST RANGE OF PVY

### 4.1. Symptom

The extent of symptom development is dependent on the interaction of viral proteins with molecular machinery belonging to the host plant, the level of expression of viral genes using host plant proteins and the subsequent spread of the newly formed virus particles throughout the plant (Visser, 2012). Studies have shown that plants are least susceptible when senescing but more susceptible before flowering during vegetative growth (DiFonzo *et a*l., 1994). On *Solanum tuberosum* (Potato) it shows, mild to severe leaf mosaic, or streak or 'leaf-drop streak' (leaves either fall from the plant or remain suspended, often giving a bare stem with leaves at the tip) with vein necrosis or 'stipple-streak' (Beemster and Rozendaal, 1972). Primary infection by necrotic strains ($PVY^{-N}$) often causes necrotic rings or spots and mild to very mild mottle late in the season. Secondary symptoms are usually more obvious as a severe mosaic early in the season. Some necrotic strains ($PVY^{-NTN}$) cause a damaging disease with symptoms on the tubers, known as potato tuber necrotic ring disease. Primary symptoms are usually present following storage, however, secondary infection may produce symptoms on lifting (Beczner *et al*., 1984). Infection with ordinary strain ($PVY-^{O}$) causes necrosis, mottling or yellowing of leaflets, leaf drop and death. Infection with $PVY-^{C}$ may cause necrosis, mottling, crinkle or 'stipple-streak', and necrosis may occur on tubers. The differences between similar viruses and strains are often uncertain because of the diversity of potato cultivars and virus strains, and the effect of climatic conditions (de Bokx and Piron, 1977). On other species like Capsicum spp, It shows mild leaf mottling symptom and Nicotiana spp. it shows mild mottle with ordinary strains; severe veinal necrosis with necrotic strains, which may cause complete loss of crop. On tomato it also shows mild leaf mottling; severe in mixed infections (Edwardson, 1974). Descriptors: Whole plant: dwarfing. Leaves: necrotic areas; abnormal colors; abnormal patterns; abnormal forms; abnormal leaf fall. Stems: discoloration of bark. Vegetative organs: surface lesions or discoloration.

### 4.2. Host Range of PVY

Potato virus Y (PVY) is the type member of the genus Potyvirus and infects several important *solanaceous* crops, including potato, tomato, pepper, and tobacco (De Bokx and Huttinga, 1981; Shukla *et al*., 1994). PVY has a wide host range, naturally infecting plants in more than nine families, including 14 genera of the Solanaceae, such as pepper, tomato, eggplant, and tobacco (Kerlan, 2006). The natural host range is wide and comprises up to nine families including important crops such as pepper (*Capsicum* spp.), potato (*Solanum tuberosum* ssp. *tuberosum*), tobacco (*Nicotiana* spp.), tomato (*Lycopersicon esculentum*), some ornamental plants (*Dahlia* and *Petunia* spp.) and many weeds (*Datura* spp., *Physalis* spp., *Solanum dulcamara* and *S. nigrum*) (Jeffries, 1998). It has been reported that PVY infects natural and commonly occurring host plants and weeds such as Chicory (*Cichorium intybus*), Horseweed (*Conyza canadensis*), Prickly lettuce (*Lactuca serriola*), Dandelion (*Taraxacum officinale*), Shepherd's purse (Capsella *bursa-pastoris*), Common lambsquarters (*Chenopodium album*), Buckhorn plantain (*Plantago lanceolata*), Bittersweet nightshade (*Solanum dulcamara*) and Black nightshade (*Solanum nigrum*) in central Europe (Kaliciak and Syller, 2009). The experimental host range is reported to comprise 495 species in 72 genera of 31 families including 287 species in 14 genera of the *Solanaceae* (among which 141 *Solanum* species and 70 *Nicotiana* species), 28 species of *Amaranthaceae*, 25 species of *Leguminosae*, 20 species of *Chenopodiaceae*, and 11 species of *Compositae* (Edwardson and Christie, 1997).

## 5. GEOGRAPHICAL DISTRIBUTION OF PVY

PVY is common in most potato production areas around the world, and there is increasing recognition of various strain types (Gray *et al*., 2010). PVY is distributed worldwide in potato growing areas, in tobacco growing areas and in outdoor crops of tomato and pepper in warm countries. In potato crops, the $PVY^{O}$ strain occurs worldwide (Jeffries, 1998). The $PVY^{N}$ strain has been recorded in South America, Europe, Africa, Asia (Weidemann, 1988a) and New Zealand (Flecher, 1989), whereas it is a quarantine pathogen in Canada (Ellis *et al*., 1997) and USA, with localized outbreaks (Singh *et al*., 1993). The $PVY^{C}$ strain, known in Europe, North America, India, South Africa, Australia, New Zealand and Ecuador (Jeffries, 1998), is probably more prevalent than commonly accepted. The $PVY^{NTN}$ variant has been identified in most potato-growing countries around the world, including USA (McDonald And Singh, 1996; Nie and Singh *et al*., 2003b) and Japan (Ohshima *et al*., 2000). The $PVY^{N}$-W variant has been reported in a few other countries including France and Spain (Blancno-Urgoiti *et al*., 1998a). $PVY^{N:O}$ was reported in Canada (Manitoba) and USA (Minnesota, Montana, North Dakota) (Singh *et al*., 2003a).

## 6. STRAIN OF PVY

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. $PVY^{O}$ does not typically cause tuber necrosis, but causes significant yield reduction (Halterman *et al*., 2012). $PVY^{N}$ 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 $PVYN^{:O}$) 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 (Halterman *et al*., 2012).

Potato isolates have historically been divided into three main strain groups: $PVY^{O}$, $PVY^{N}$ and $PVY^{C}$ according to symptoms induced in *N. tabacum* cv Samsun, *S. tuberosum* ssp. *tuberosum* and *Physalis floridana* (De Bokx and Huttinga, 1981). $PVY^{O}$ and $PVY^{C}$ are separated on the basis of hypersensitive reactions in potato cultivars bearing different resistance genes, namely $Ny_{tbr}$ and *Nc* for $PVY^{O}$ and $PVY^{C}$, respectively (Cockerham, 1970). $PVY^{N}$ differs from $PVY^{O}$ and $PVY^{C}$ in causing a severe veinal necrosis reaction in tobacco, but elicits a hypersensitive response in few if any potato cultivars (Valkonen, 1997). The virus formerly called Potato virus C (PVC) (non-aphid transmissible) has been demonstrated to be a PVY strain (Cockerham, 1943; Bawden and Kassanis, 1947), and is included in $PVY^{C}$.

In the past, a fourth group called $PVY^{An}$ (Horvath 1967b) was described which included particular potato and tomato isolates. Many other variants have been reported, including: isolates that do not induce necrosis in *S. demissum* A6 (De Bokx *et al*., 1975; Thompson *et al*., 1987); $PVY^{Z}$ and $PVY^{ZE}$, which are particular pathotypes found in Britain, Spain and France that overcome the $Ny_{tbr}$ and *Nc* genes (Jones, 1990; Blancno-Urgoiti *et al*., 1998a; Kerlan *et al*., 1999); $PVY^{NTN}$, which is characterised by its necrotic properties in potato tubers (Le Romancer *et al*., 1994); $PVY^{N}$-$^{W}$ (Glais *et al*., 1998) and $PVY^{N-O}$ which share properties with both $PVY^{N}$ and $PVY^{O}$ (Singh *et al*., 2008). PVY isolates from tobacco can be placed to three strains based on the necrotic symptoms they cause in tobacco plants (Gooding and Tolin, 1973). Isolates of strain $M^{S}N^{R}$; also referred to as $M^{S}N^{R}$ 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 (Gooding and Tolin, 1973). 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 (Yi *et al*., 1998).

PVY isolates infecting pepper have been classified into three pathotypes designated PVY-0, PVY-1 and PVY-1-2 according to their ability to overcome resistance genes (*vy1*, *vy2*) present in pepper cultivars Bastidon, Yolo Y and Florida VR-2 (Gebre-Selassie *et al*., 1985). 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 $pvr^{21}$ and $pvr^{22}$ in *Capsicum annuum* L. (Singh *et al*., 2008). Isolates that are unable to overcome these genes and can infect only genotypes lacking them belong to pathotype 0. Isolates that overcome $pvr^{21}$ belong to pathotype (0, 1), and those which overcome both resistance genes ($pvr^{21}$ and $pvr^{22}$) belong to pathotype (0, 1, 2) (Gebre-Selassie *et al*., 1985). Molecular classifications based on genome polymorphism, either considering the whole RNA or more frequently a single region, distributed PVY isolates into two or three main clusters (Van der Vligt *et al*., 1993). Three genetically defined strains were established on the basis of RFLP typing of the coat protein gene: $PVY^{O}$, $PVY^{N}$ and $PVY^{NP}$ (non-potato), this last including isolates from pepper, tobacco and *Datura* species (Blancno-Urgoiti *et al*., 1996). By using the same typing, $PVY^{C}$ isolates were recently characterised as a homogeneous pathotype but divided into two genetically distinct strains designated $PVY^{C1}$ and $PVY^{C2}$, $PVY^{C1}$ isolates being included in the $PVY^{NP}$ cluster whereas $PVY^{C2}$ was found to be separate from the other PVY groups or subgroups described so far (Blancno-Urgoiti *et al*., 1998b).

$PVY^{N}W$, $PVY^{N:O}$ and the majority of $PVY^{NTN}$ isolates are recombinants whose genome displays $PVY^{O}$-type segments and $PVY^{N}$-type segments (Revers *et al*., 1996; Glais *et al*., 1998; Boonham *et al*., 1999). $PVY^{N}W$ and $PVY^{N:O}$ isolates induce a vein necrosis reaction in tobacco and possess a $PVY^{O}$-type coat protein (McDonald and Singh, 1996; Glais *et al*., 1998; Nie and Singh, 2003a). All $PVY^{N}W$ isolates tested were shown to display one or two recombination breakpoints (Glais *et al*., 2002a). Most $PVY^{NTN}$ isolates (*i.e.* inducing potato tuber necrosis under natural conditions) display recombination breakpoints in the coat protein gene and two other genomic regions (HC-Pro/P3 and CI/6K2) (Revers *et al*., 1996; Glais *et al*., 1998; Boonham *et al*., 1999). However many tuber necrosis-inducing isolates, including one North-American $PVY^{NTN}$ isolate (Nie and Singh, 2003b), possess a $PVY^{N}$-type genome without any recombination breakpoint (Boonham *et al*., 22002b. North-American (NA)- and European (EU)-$PVY^{NTN}$ isolates were separated on the basis of phylogeny using the P1 and 5' non-translated regions (Nie and Singh, 2002a)

On a genomic level $PVY^{Z}$ shares the three recombination junctions of $PVY^{NTN}$ (Kerlan *et al*., 2011). Recently a new variant of PVY was identified and the isolate classified NE-11 (Lorenzen *et al*., 2008). NE-11

 

was previously classified as $PVY^{NA-NTN}$ but further investigations have revealed that this classification was incorrect. Close scrutiny of the NE-11 genome revealed that the sequence of the first 2000 nucleotides is highly similar to that of $PVY^{N}$. This fragment is followed by a fragment of approximately 600 nucleotides of which the sequence resembles that of $PVY^{NA-NTN}$. The rest of the NE-11 genome resembles $PVY^{N}$ while the biggest part of the CP gene region has no similarity towards that of any of the known PVY strains (Lorenzen *et al*., 2008). The NE-11 isolate has been described as being able to induce mosaic symptoms on leaves as well as mild PTNRD (Lorenzen *et a*l., 2008). $PVY^{E}$, a strain of PVY of Brazilian origin, was identified on basis of its ability not to elicit the response genes Ny, Nc and Nz in potatoes and its inability to induce necrosis in tobacco (Galvino-Costa *et al*., 2011).

## 7. CALCIFICATION /TAXONOMY OF PVY

The virus classified in to family Potyviridea, Genus; Potyvirus and species *Potato virus* Y. Genomic variability of PVY has been extensively studied (Glais *et al*., 2002b) leading to the classification of PVY isolates into two, three or four clusters (O, N, NP and C2), in rather good agreement with classical PVY strain definitions based on host range and symptomatology. However most classifications obtained from phylogenetic analyses probably rely on neutral markers and correlate only partially with biological classifications. For instance, $PVY^{C}$ isolates were split into two genetic lineages, though the classical $PVY^{C}$ strain is a single pathotype (Blancno-Urgoiti *et al*., 1998b). In contrast, pepper-infecting PVY isolates were shown to be a single genetic lineage containing several pathotypes (Romero *et al*., 2001). There are many exceptions, such as reference isolates $PVY^{N}$-Fr and PVY $M^{S}M^{R}$ (a tobacco isolate), which were grouped with the potato $PVY^{O}$ isolates (Blancno-Urgoiti *et al*., 1996). Recombination events may also result in flawed if not false molecular classifications based on the capsid protein alone, as was shown for the $PVY^{N}$-W and $PVY^{N:O}$ isolates (Glais *et al*., 1998) and a pepper isolate. Lastly, the exact positions and outlines of different groups of isolates have yet to be precisely defined, as for instance the exact relationship between the potato ($PVY^{N}$) and tobacco ($N^{S}N^{R}$, $M^{S}N^{R}$) necrotic strains (McDonald and Kristjansson, 1993), or the border between tuber-necrosing ($PVY^{NTN}$) and non-necrosing $PVY^{N}$ isolates. A recombination breakpoint in the coat protein gene was thought to correlate with the potato tuber necrotic ringspot phenotype induced by the $PVY^{NTN}$ isolates (Revers *et al*., 1996; Glais *et al*., 1998; Boonham *et al*., 1999), but it is not present in all isolates associated with PTNRD (Boonham *et al*., 2002b).

Differentiation and consequently detection of the various groups of isolates are often uncertain. For instance, numerous RT-PCR methods have been published for detection of $PVY^{NTN}$ isolates (Weidemann and Maiss, 1996; Glais *et al*., 1996; Weilgunny and Singh, 1998; Boonham *et al*., 2002a; Nie and Singh, 2002b,2003a), but they are often outdated or even unreliable, being based on small numbers of isolates or inadequately characterised isolates and also relying on gene sequences not proved to be responsible for inducing tuber necrosis. Bioassays, in which PVY isolates are inoculated to sensitive potato cultivars, could be another means to discriminate $PVY^{NTN}$ from the rest of $PVY^{N}$. However, the reliability of such bioassays when carried out under artificial conditions is also in doubt as shown by some experiments (McDonald and Singh, 1996).

## 8. TRANSMISSION OF PVY

PVY is spread from plant to plant by aphids, mechanical means or by contact. the discretion of these transmission means are presented as follows:

### 8.1. Transmission by Vectors

All of the PVYs are transmitted in nature by numerous species of aphids; the current total is more than 50 species that are able to transmit PVY with varying efficiency (Ragsdale *et al*., 2001). Aphids transmit PVY in a non-persistent manner which requires acquisition and inoculation times of less than one minute (Bradley, 1954), allowing many species of aphids that are only casual visitors to potato plants ample opportunity to either acquire or transmit the virus. There are data that indicate some of the PVY strains may be more efficiently transmitted by some aphid vectors than by others (Basky and Almasi, 2005; Cervantes and Alvarez, 2008; Fereres *et al*., 1993), although transmission efficiency will differ among virus isolates within a strain and aphid populations within a species (Verbeek *et al*., 2010). Once the plant foliage is inoculated by aphids, virus is translocated to tubers; although the efficiency of this translocation can vary among cultivars (Gray *et al*., 2010).

In potato crops in most areas and seasons, *Myzus persicae* is clearly the most important vector given that it is widespread and that its efficiency of transmission is high. Efficiency of transmission by other vector species is comparatively low or very low (Sigvald, 1985), but despite this low efficiency, some species are noteworthy vectors of PVY, either species that colonise potatoes such as *A. nasturtii* (in Central and Eastern Europe), *Macrosiphum euphorbiae* and *Aulacorthum solani* or species that visit but rarely colonise potatoes. 22 such visiting species were quoted by and some of them (*R. padi*, *A. pisum*, *B. helichrysi*, *Metopolophium dirhodum*, *C. aegopodii*) are possibly involved in epidemics due to PVY (Weidemann, 1988). Studies on the relative importance of different aphid species as PVY vectors in Southern England proved that *B. helichrysi* and

 

*Myzus persicae* accounted for about half of all observed transmissions, but that *Phorodon humili* and many *Aphis* species also played a significant role (Harrington and Gibson, 1989).

PVY is transmitted in a non-persistent manner which means brief acquisition and inoculation periods (a few seconds or minutes for acquisition, a few seconds for inoculation). There is no discernible latent period. Acquisition and inoculation involve stylet penetrations into the epidermal cell layer of the plants and occur when stylets puncture plant cell membranes, though the possibility for the virus to be acquired and inoculated via broken plasmodesmata is not totally excluded (Powell, 1992). Retention of the virus in aphids in most cases lasts not more than one or two hours. However, longer retention periods were reported, with significant differences according to the viral strain and the aphid vector species: up to 4 hours and 8 hours in *Myzus persicae* and *Phorodon humuli*, respectively (Van Hoof, 1980). Such a long retention period may explain why $PVY^N$ isolates can be transmitted over rather long distances (Van Hoof, 1980). The virus does not pass through the moult. Prior starvation of the aphids increases the efficiency of transmission though it does not affect the occurrence of electrically-recorded membrane punctures during acquisition access (Powell, 1998). The efficiency of transmission depends on many other factors including the nature of the source and test plants (Van Hoof, 1980), the virus concentration in the source plant (De Bokx *et al*., 1978), the mature-plant resistance in potato (Sigvarld,1985), the environmental conditions and the viral strain, $PVY^N$ isolates being better transmitted than other PVY isolates (Proeseler and Weidling, 1975). Interference between PVY strains during aphid transmission has been reported: transmission of $PVY^O$ decreased when aphids had previously or subsequently fed on $PVY^N$-infected source plants (Katis *et al*., 1986).

HC-Pro has been implicated in the transmission of Potyviruses by acting as a link between the virus and the aphid mouthpart (Wang *et al*., 1998). For this reason the HC-Pro can be described as a regulator of transmission. The capacity of different species of aphids to transmit PVY has been correlated with the ability of the HC-Pro/virus complex to interact with the aphid stylet (Wang *et al*., 1998). Sequence comparisons of HCs of aphid-transmissible ($PVY^N$) and non-transmissible (PVC) isolates revealed two amino acid substitutions specific to the non-transmissible isolate: Lys to Glu at position 50 in the N-terminal cysteine-rich, metal-binding region, and Ile to Val at position 225 in the middle of the HC sequence (Thornbury *et al*., 1990). The N-terminal domain of PVY CP is also involved in aphid transmissibility. This domain contains the DAG triplet found in all PVY aphid transmissible isolates examined (Shukla *et al*., 1991), and also in the non-aphid transmissible isolate PVY-18 (Shukla *et al*., 1988c). Unlike *Tobacco vein mottling virus*, PVY isolates having the sequence DAGE are aphid-transmissible (Shukla *et al*., 1994).

### 8.2. Transmission by Mechanical Means

Mechanical transmission generally occurs when an infected plant and an adjacent healthy plant are wounded by wind or human activity (such as operation from equipment in field). The wounds of an infected plant sap that contains the virus, and the wound of a nearby healthy plant may take in some of that virus when the two plants touch. Mechanical seed cutting can also spread PVY, which is why it is always best to sanitize seed cutting equipment before using it for another variety or seed lot (Schramm *et al*., 2011).

## 9. SEROLOGICAL RELATIONSHIP WITH OTHERS

### 9.1. Serology of PVY

PVY is strongly immunogenic (Shukla *et al*., 1994). Antisera with precipitin titres of 1/4096 have been readily obtained. Monoclonal antibodies have been produced in mice immunised with purified virus preparations (Gugerli and Fries, 1983; Rose *et al*., 1987; Ohshima *et al*., 1990; Sanz *et al*., 1990; Singh *et al*., 1993; Ellis *et al*., 1996). Antisera and monoclonal antibodies have also been produced against synthetic peptides (Ohshima *et al*., 1992; Ounouna *et al*., 2002). Recombinant antibodies (single chain Fv antibody fragments) have been obtained by using phage display antibody technology (Boonham and Barker, 1998). Antibodies have also been generated by DNA-based immunization of rabbits (Hinrichs *et al*., 1997). ELISA is in current usage to detect the virus from leaves or other organs such as potato tubers according to the basic double antibody sandwich (DAS) protocol (Boonham *et al*., 2002b). Antigen coated plate-ELISA and triple antibody sandwich-ELISA are also frequently used. ELISA testing has been found to be unsatisfactory on dormant tubers (De Bokx and Huttinga, 1981) and more reliable when tuber dormancy is broken by Rindite treatment. DAS-ELISA has been used for PVY detection in the aphid *M. persicae* (Carlebach *et al*., 1982). ISEM was used for studying antigenic relationships between different PVY strains (Gebre-Slassie *et al*., 1985).

### 9.2. Relationships with Others

PVY is distantly serologically related to two other potato potyviruses, *Potato virus A* (PVA) and Potato virus V (PVV) (Fribourg & Nakashima, 1984), and to other distinct potyviruses such as: Bean common mosaic virus, Bidens mottle virus, Beet mosaic virus, Bean yellow mosaic virus, Celery mosaic virus, Henbane mosaic virus, Lettuce mosaic virus, Maize dwarf mosaic virus, Plum pox virus, Papaya ringspot virus, Pepper veinal mottle

 

virus, Passion fruit woodiness virus, Pokeweed mosaic virus, Tobacco etch virus (TEV), Turnip mosaic virus and Watermelon mosaic virus (De Bokx and Huttinga, 1981). PVY is also distantly related to several but not all isolates of Pepper mottle virus (PepMoV) (Shukla *et al*., 1994). PepMoV, first described as an atypical isolate of PVY has been shown to be a distinct species since the two viruses differ from each other in many properties (Purcifull and Hibert, 1992), including host range, lack of cross-protection, type of cytoplasmic inclusions and genomic sequences (Shukla *et al*., 1994). Most of the serological relationships with other potyviruses are due to common epitopes located in the conserved core region of the PVY coat protein (Shukla *et al*., 1992), the relationship to TEV being an exception (Shukla *et al*., 1989). Phylogenetic comparisons indicated that PVY, PepMoV, PVV, Pepper yellow mosaic virus, Pepper severe mosaic virus, Wild potato mosaic virus and Peru tomato virus constitute a group distinguishable from other potyviruses including PVA (Spetz *et al*., 2003).

**10. Stability of PVY**

Thermal inactivation point (10 min) in tobacco sap has been estimated to vary from 50°C for an isolate from tomato (Clark and Hill, 1978) to 74°C for a $PVY^{An}$ isolate. It was also recorded to be 56-72°C for $PVY^{O}$, 64°C for $PVY^{N}$, and 58-60°C for $PVY^{C}$. Dilution end-point is $1 \times 10^{-4}$ to $2 \times 10^{-6}$ for $PVY^{O}$, $2 \times 10^{-6}$ for $PVY^{N}$, and $2 \times 10^{-1}$ to $1 \times 10^{-4}$ for $PVY^{C}$ (Horvath, 1967). Longevity *in vitro* (18-22°C) was recorded from one day (Clark and Hill, 1978) to 50 days (Klinkowski and Schmelzer, 1957). It was reported to be 18-31 days for $PVY^{O}$, 21-27 days for $PVY^{N}$, and 15-18 days for $PVY^{C}$ (Horvath, 1967). Infectivity in sap from tobacco leaf tissue containing 1% sodium azide is preserved at 25°C for 4 weeks (Goding and Tsakiridis, 1971), and is not changed by treatment with diethyl ether. Leaves are the best virus-infected material to store. PVY in potato or tobacco foliage can be stored effectively at -18°C. For long-term preservation, PVY-infected samples, dried and stored over calcium chloride at 4°C, can remain infective for 15 years (De Bokx, 1987), though inoculation from such material may sometimes be unsuccessful. PVY can also be preserved long-term by storing clarified virus-containing sap in or over liquid nitrogen (De Wijis and Suda-Bachman, 1979). Antigenic properties can be retained for one year in freeze-dried crude extracts from infected *N. tabacum* plants (Purcifull *et al*., 1975).

**11. PURIFICATION OF PVY**

Most purification methods currently used are improvements of formerly described methods: Delgado-Sanchez and Grogan (1966), Damirdagh and Shepherd (1970), Huttinga (1973), Shepard *et al*. (1974), Gnutova and Krylov (1975b) modified by Rose and Hubbard (1986), Moghal & Francki (1976) modified by Sanz *et al*. (1990), Leiser and Richter (1978), Gugerli (1978), Hammond and Lawson (1988). modified by Chandelier *et al*. (2001). $Na_2$-EDTA, urea, citrate and Triton X-100 have been used to prevent aggregation of the particles. Clarification has been done with chloroform, diethyl ether or carbon tetrachloride, singly or in combination. Further purification has been achieved by ammonium sulphate precipitation, ultracentrifugation through a layer of sucrose, and caesium chloride or sucrose gradient centrifugation (Huttinga and Maat, 1987). Yields in mg/kg of infected leaves varied from 9 to 23 (Makkouk and Gumpf, 1974) and 46 to 116 Hammond and Lawson (1988).

Good results can be obtained using the method reported by Leiser and Richter (1978) with a final centrifugation stage in caesium chloride (Kerlan, unpublished). Homogenize 100 g leaf tissue (*N. tabacum* cv Xanthi) in 300ml 0.5 M citrate buffer, pH 7.4, containing 5 mM $Na_2$-EDTA and 15 mM sodium DIECA. Filter the homogenate through muslin and centrifuge for 15 min at 4360 g (6000 rpm). Add Triton X-100 to a final concentration of 3% (v/v) and stir for 30 min in a cold room. Centrifuge for 2 hours at 31 000 g. Resuspend the pellets in 10 mM citrate buffer, pH 7.4, containing 1M urea and 0.1 % 2-mercaptoethanol. After keeping overnight at 4-6°C, centrifuge for 15 min at 4360 g. Layer the supernatant fluid over a cushion of 20% (w/v) sucrose and centrifuge for 2 h at 50,000 g. Resuspend the pellets in 5mM sodium borate buffer, pH 8. Layer aliquots (0.3 ml/tube) over a CsCl gradient (0.47 g/ml) in 5mM sodium borate buffer, pH 8, and centrifuge for 5 h at 110,000 g (40,000 rpm in Beckman rotor 70.1 Ti) at 12°C. Recover the virus band identified by spectrophotometric analysis of the gradient fractions at 254 nm, dialyze against 5mM sodium borate buffer, pH 8, overnight at 4-6°C and centrifuge for 3 hours at 146,000 g (40,000 rpm) at 4°C.

**12. MORPHOLOGY/PHYSICOCHEMICAL PROPERTIES OF POTATO VIRUS Y**

**12.1. Morphology of PVY**

Virus particles are long, flexuous, non-enveloped filaments, with a clear modal length of 684 nm in purified preparations (Delgado-Sanchez and Grogan, 1966) or 730 nm in leaf-dip preparations, and 11 nm wide. Particles have a helical construction with a pitch of 3.3 nm (Varma *et al*., 1968). Virions contain 5.4-6.4% ribonucleic acid (Leiser and Richter, 1978) and 93.6-94.6% protein. The genome consists of a unipartite single-stranded RNA. Total genome size is 9704 nucleotides (Robaglia *et al*., 1989). The 5' terminus of RNA has a genome-linked protein (VPg). Infectivity retained when deproteinized with phenol or detergent. Poly A region present at 3' end, but not essential for infectivity.

 

### 12.2. Particle Composition PVY

#### 12.2.1. Nucleic acid

The nucleic acid of PVY is a single-stranded linear RNA with a sedimentation coefficient of 25*S* (Makkouk and Gumpf, 1974) to 39*S* (Makkouk and Gumpf, 1975), and a molecular weight of 3.1 x $10^6$ (Makkouk and Gumpf, 1974) to 3.2 x $10^6$ (Hinostroza-Orihuela, 1975). It has a VPg at the 5' terminus and a polyA sequence at the 3' terminus. No subgenomic RNA is produced. The percentage of RNA in particles is from 5.4 to 6.4% (Stace-Smith and Tremaine, 1970; Makkouk and Gumpf, 1974;Leiser and Richter, 1978).

#### 12.2.2. Protein of PVY

Protein content in the particle is about 94%. Only two proteins, VPg and coat protein (CP), are detected in viral particles. CP molecular weight was calculated to be 29.95 kDa (Shukla *et al*., 1986). The coat protein consists of 267 amino acid residues, except for that of isolate PVY 18, which has a deletion at position 25 (Shukla *et al*., 1988c). Complete and partial amino acid sequences of the CP have been obtained (Shukla *et al*., 1986, 1988a, 1988c, 1994). Around 200 CP sequences are available in databases. N-terminal residues of the CP were not blocked in any of the five PVY isolates studied (Shukla *et al*., 1986, 1988c). No lipid or other components have been detected in the particles.

## 13. GENOME ORGANIZATION OF POTATO VIRUS Y

The genomic RNA of PVY is positive sense and approximately 9700 kb in length excluding the poly(A) tail (Shukla *et al*., 1994). As in all members of the picorna-like super-group, it is expressed as a large polyprotein precursor of 3063 amino acids for a $PVY^N$ isolate (Robaglia *et al*., 1989), 3061 amino acids for a $PVY^{NTN}$ isolate and 3061 amino acids for a $PVY^O$ isolate (Singh and Singh, 1996). This is subsequently cleaved by proteases to yield nine functional proteins including those involved in RNA replication and other non-structural proteins (Shukla *et al*., 1994). In the 5'-3' direction, the nine proteins are referred as P1 (first protein), HC or HC-Pro (helper component protein), P3 (third protein), 6K1 (first 6 kDa protein), CI (cytoplasmic inclusion protein), 6K2 (second 6 kDa protein), NIa (small nuclear inclusion protein), NIb (large nuclear inclusion protein), and CP (coat protein).

There are two distal non-coding regions. The length of the 5' non-coding region is 184 nt (Robaglia *et al*., 1989). This includes blocks of sequences conserved in potyviruses, referred as the "potybox", box "a" (a 9 nt sequence, part of the potybox) and box "b". PVY has a potybox motif (UCAACACAACAU), in which 11/12 nucleotides match the consensus sequence, and a perfect copy of the consensus sequence motif of box b (UCAAGCAA). The potybox starts 12 nt from the 5'end and is separated from box b by a 39 nt sequence (Shukla *et al*., 1994). The 5' non-coding region is involved in initiation of translation (Levis and Astier-Manifacier, 1993). The length of the 3' non-coding region is variable: 326 nt (Van der Vlugt *et al*., 1989), 329-332 nt (Van der Vlugt *et al*., 1993), 333 nt (Rosner and Raccah, 1988).

Table 1. The lengths (in number of amino acid residues) and positions of each of the gene products in the polyprotein on the basis of the sequence of the PVYN strain are as follows:

| Gene Product | Length (Number of amino acid residues) | Positions |
|---|---|---|
| P1 | 284 AA | 1-284 |
| HC | 456 AA | 285-740 |
| P3 | 365 AA | 741 – 1105 |
| 6K1 | 52 AA | 1106- 1157 |
| CI | 634 AA | 1158 - 1791 |
| 6K2 | 52 AA | 1792 - 1843 |
| NIa – VPg | 188 AA | 1844 - 2031 |
| NIa – Pro | 244 AA | 2032 - 2275 |
| NIb | 521 AA | 2276 – 2796 |
| CP | 267 AA | 2797 – 3063 |

Source, Robaglia *et al*., 1989

NIa has a two-domain structure where the N-terminal domain is the genome-linked protein VPg. VPg is attached to the 5'end of the RNA via a phosphate ester linkage to $Y^{60}$ in the conserved sequence motif NMY which is present in PVY NIa (Robaglia *et al*., 1989; Shukla *et al*., 1994). CP can be divided into three regions: a) a surface-exposed N-terminus of 30 amino acids, variable in length and sequence in different potyviruses; b) a core of 218 amino acids, highly conserved among potyviruses; and c) a surface-exposed C-terminus of 19 amino acids (Shukla and Ward, 1989). The N-terminal and C-terminal regions are not required for virus assembly. Trypsin digestion of particles removes these termini, but leaves a fully assembled particle (composed of the core region) that appears indistinguishable from untreated native particles by electron microscopy and is still infective

 

(Shukla *et al*., 1988b).Polyprotein processing as for all potyviruses involves three proteinases: NIa, HC and P3. The NIa proteinase cleavage sites were identified in the sequence of the $PVY^N$ strain (Robaglia *et al*., 1989). RNA synthesis is believed to occur in the cytoplasm. The entire RNA genome is copied. The replication complex comprises the proteins NIb, CI and VPg and possibly involves the proteins 6K1 and 6K2. The NIb protein is believed to be the RNA-dependent RNA polymerase since it contains the consensus sequence motif GDD found in viral RNA-dependant RNA polymerases, this motif being located at residues 2628-2630 (Robaglia *et al*., 1989).

As for all potyviruses, two proteins, HC and CP, are required for aphid transmission of PVY. The N-terminal third of the HC protein contains a cluster of cysteine residues, similar to the consensus sequence of metal-binding sites in nucleic acid binding proteins (Robaglia *et al*., 1989). Indeed, the HC of PVY expressed in *Escherichia coli* possesses nucleic acid binding-activity which could be involved in cell-to-cell movement of the virus or in RNA replication (Robaglia *et al*., 1989). The N-terminal half of HC (amino acids 1 to 228) is capable of self-interaction, the 83 N-terminal residues being sufficient. Mutations in the conserved His and two Cys residues within the Cys-rich region of PVY HC-proteinase (amino acids 23 to 56) were reported to reduce self interaction (Urcuqui-Inchima *et al*., 1999a, 1999b). HC was shown to be involved in PVY accumulation in tobacco and to be a suppressor of post-transcriptional gene silencing (PTGS) (Brigneti *et al*., 1998). The HC protein, but not the 5' and 3' non-coding regions, P1 or CP, may be involved in induction of vein necrosis in tobacco (*N. tabacum* cv Xanthi) (Glais et al., 1998, 2002). VPg was shown to be involved in breaking resistance conferred by the gene *va* in tobacco (Masuta *et al*., 1999), by the gene *pot-1* in tomato (Moury *et al*., 2004) and by the gene *pvr2* in pepper (Moury *et al*., 2004). NIa was shown to interact with the resistance gene *Ry* in potato (Mestre *et al*., 2000), its protease activity however being not sufficient for elicitation of *Ry* (Mestre *et al*., 2003). NIb was found to be the elicitor of a veinal necrosis/hypersensitive response in root-knot nematode resistant tobacco cultivars (Fellers *et al*., 2002).

## 14. ECONOMIC IMPORTANCE OF PVY

Potato virus Y belongs to a group of the most important potato viruses infecting the potato (*Solanum tuberosum*, L.). Potyviruses constitute the largest and agronomically most important group of plant viruses known, causing devastating diseases in many crop plants (Hollings and Brunt, 1981). Potato virus Y (PVY) is the type member of the group, and strains of PVY cause diseases in tobacco, potato, pepper and tomato (De Bokx and Huttinga, 1981). Potato Virus Y (PVY) is one of the most important virus diseases of potato and is one of the leading causes of rejection of seed lots from certification programs (Ragsdale, *et al*., 2002). Losses from this disease include direct yield losses of 10-80% depending on the variety and losses in product quality (Hane and Hamm, 1999; German, 2001).

Potato virus Y (PVY) is the most important viral pathogen in potato worldwide (German, 2001; Kogovšek *et al*., 2010). Yield losses of up to 40% have been documented (Nolte *et al*., 2004). Most strains of PVY cause only mild mottling in pepper, tobacco and tomato. Some strains, however, can cause yield losses of 10-100% in potato (de Bokx and Huttinga, 1981); it is still a major virus of potatoes because it spreads so easily and can greatly decrease yield. Combined with other potato viruses such as potato A potyvirus, potato X potexvirus and potato S carlavirus, it can be particularly damaging, sometimes destroying the entire crop. In tobacco, yield losses of 39-75% can occur, although PVY-N strains can cause complete crop loss depending on strain, cultivar and time of infection. Infection of potato plants by PVY may result in 10% yield loss to total crop failure (Warren *et al*., 2005). The magnitude of this loss is dependent on time of infection, viral load within infected plants, the cultivar susceptibility or resistance, the strain of virus infecting the plants as well as environmental conditions (Visser, 2012). PVY is an important virus of potatoes as it spreads easily and can decrease yield greatly when the incidence is high, the cultivar sensitive and the strain virulent. Potential losses are even greater when PVY is present in combined infection with other potato viruses. If $PVY^O$ became established in WA, yield loss in potatoes would be expected to be minor due to control via the seed certification scheme. $PVY^O$ usually causes only mild mottling in capsicum, tobacco and tomato, so yield losses are minor in them, but $PVY^N$ causes severe necrosis in tobacco, killing the infected plant (Jones, 2003).

## 15. RESISTANT SOURCE OF PVY

Major dominant genes that confer PVY resistance have been identified in wild *Solanum* relatives and in cultivated Solanum tuberosum Andigenum Group and Chilotanum Group germplasm (Solomon-Blackburn and Barker, 2001; Valkonen, 1997). The two main types of resistance in potato are extreme resistance (ER), which protects against all strains of PVY by suppressing virus accumulation in infected cells, and hyper- sensitive resistance (HR). ER has been identified in the wild potato species S. chacoense, S. hougasii, S. stoloniferum, and the S. tuberosum Andigenum Group (Cockerham, 1943; Munoz *et al*., 1975; Sato *et al*., 2006), while HR to PVY ordinary (O) strain group ($PVY^O$) has been reported in *S. chacoense, S. demissum, S. megistacrolobum, S. polyadenium, S. sparsipilum, S. stoloniferum,* and the *S. tuberosum* Andigenum and Chilotanum Groups

 

(Celebi-Toprak *et al*., 2002; Cockerham, 1970; Valkonen, 1997). Resistance in other Solanum spp. has been identified but the physiological basis is not known. For example, high levels of resistance have been reported in the non-tuber-bearing species S. etuberosum (Novy and Helgeso, 1994; Novy *et al*., 2002; Valkonen *et al*., 1992).

Natural genes for resistance to the main viral diseases of potato can be found in wild potato species (*Solanum* spp.) and can be integrated into the genome of cultivated potato (Valkonen, 1994). The dominant gene $Ry_{sto}$ from *S. stoloniferum* that was introduced in a number of potato cultivars more than 30 years ago (Ross, 1986). This gene is effective against all potato virus Y (PVY) isolates (Fernandez-Northcote, 1983) including the tuber necrosis causing isolate $PVY^{NTN}$ (Chrzanowska, 1995; Dolnicar, 1995; Le Romancer & Nedellec, 1997). To date, no breakdown of resistance has been observed. $Ry_{sto}$ or a closely linked gene may also confer extreme resistance to other potyviruses including potato virus A (PVA) (Cockerham, 1970), potato virus V (PVV) and tobacco etch virus (TEV) (Hinrichs *et al*., 1997).

Plant–Ppotyvirus interactions constitute an interesting model to investigate recessive resistance because an unusually high frequency of occurrence of genes conferring recessive resistance to potyviruses has been observed (40% versus 20% for resistance against other viruses) (Provvidenti and Hampton, 1992). $pvr^2$-mediated resistance of pepper (Capsicum spp.) to potato virus Y (PVY) falls into this type of resistance. The $pvr^2$ resistance locus consists of two alleles, $pvr2^1$and $pvr2^2$ (Kyle and Palloix, 1997): $pvr2^1$ is effective only against PVY-0, while $pvr2^2$ is effective against PVY-0 and -1 but is overcome by PVY-1,2. Further studies revealed another characteristic feature of potyvirus resistance genes: resistance is often not restricted to a single potyvirus. Thus, $pvr2^2$ is also associated with resistance to a second potyvirus, the TEV. Whether a single locus or two tightly linked genes controls PVY and TEV resistance remains to be determined (Ruffel *et al*., 2002).

Resistance to PVY was achieved via the ectopic expression of several viral proteins: the coat protein (CP), the RNA-dependent RNA polymerase (Nib), the nuclear inclusion proteins gene (Nia) and P1 proteinase gene in sense and antisense orientation as well as with a heterologous sequence of lettuce mosaic virus. Resistance was not always very strong (Kaniewski *et al*., 1990; Lawson *et al*., 1990), often strain-specific (Farinelli *et al*., 1992; Maki-Valkama *et al*. 2000a; Maki-Valkama *et al*., 2000b; Pehu *et al*. 1995) and protection appeared almost always in only a few of the transgenic lines generated. In retrospect, attempts at expressing virus-derived sense or antisense sequences can be interpreted such that they actually initiated posttranscriptional gene silencing (PTGS) via accidental formation of dsRNA (Dougherty *et al*., 1994; Dougherty and Parks 1995; Vaucheret *et al*., 2001; Waterhouse *et al*., 1998). In one case, the presence of the protein (p17, movement protein) was reported necessary for resistance (Tacke *et al*., 1996) and therefore other mechanisms, e.g. an inhibition of normal viral protein function, cannot be completely ruled out. PTGS, or simply RNA silencing, is a sequence-specific mRNA degradation mechanism in plants that does not affect transcription, at least not initially. It is believed that this mechanism evolved to protect plants against viruses and transposons (Voinnet, 2001).

## 16. MANAGEMENT OF PVY

Measures to reduce losses caused by viral infections are both limited and expensive. But the spread of the virus can be controlled by several different methods including chemical protection from virus vectors, elimination of the sources of infection and by breeding new varieties with extreme resistance (Kang *et al*., 2005). The generation of resistant cultivars is considered the most economic and environmentally acceptable way of controlling viral diseases in potato (Solomon-Blackburn and Barker, 2001). Use of insecticides is largely ineffective in the control of aphid-borne spread of PVY (de Bokx and Huttinga, 1981). The main control methods currently in use are listed below.

### 16.1. Cultural Control

The following methods can be used to control PVY infection: avoidance of infection, e.g. growing crops at higher altitude, where vectors are absent; not growing crops in the proximity of established crops; and destroying haulms of seed potato crops before maturity, to prevent late spread.

### 16.2. Host-Plant Resistance

The generation of resistant cultivars is considered the most economic and environmentally acceptable way of controlling viral diseases in potato (Solomon-Blackburn and Barker 2001). Genes for PVY resistance have been identified in close relatives of potato, pepper, tobacco and tomato, and have been used to develop resistance using classical breeding methods (Provvidenti and Hampton, 1992). More recently, potato plants genetically modified to express viral sequences have been shown to have a high degree of resistance, although as yet these have not found widespread usage (Lawson *et al*., 1990; Kaniewski *et al*., 1990).

### 16.3. Vector Control

Management practices that can help to reduce the spread of PVY to some degree include the application of crop

 

oil to foliage on a regular basis, especially just before and during periods of peak aphid activity (Nolte *et al*., 2004) . The use of reflective surfaces and sticky insect traps can reduce virus spread by the aphid vectors (Loebenstein and Raccah, 1980). The use of mineral oils may also be partially effective.

**REFERENCES**

Basky, Z., and Almasi, A. 2005. Differences in aphid transmissibility and translocation between PVYN and PVYO isolates. *J. Pestic. Sci.* 78:67-75.

Bradley, R. H. E., 1954. Studies of the mechanism of transmission of potato virus Y by the green peach aphid, *Myzus persicae* (Sulz.). *Can. J. Zool.* 32:64-73.

Bawden, F., 1934. Studies on a virus causing foliar necrosis of the potato. *Proc Royal Soc (London) Ser B* 116: 375–395.

Bawden F., 1936. The viruses causing top necrosis (acronecrosis) of the potato. *Ann Appl Biol* 23:487–497.

Blanco-Urgoiti B, Sa ´nchez F, Perez de San Roman C, Dopazo J, Ponz F., 1998a. Potato virus Y group C isolates are a homogenous pathotype but two different genetic strains. *J Gen Virol* 79: 2037–2042

Beczner L, Horvath H, Romahanyi I, Forster H, 1984. Studues on the etiology of tuber necrotic ringspot disease in potato. *Potato Research*, 27:339-352.

Beemster ABR, de Bokx JA. 1987. Survey of properties and symptoms. in:Viruses of Potatoes and Seed Potato Production. page: 48-113. Clark MF, Adams AN. 1977. Characteristics of the micro plate method of Enzyme-Linked Immunosrobent Assay for the detection of plant viruses. *J gene virol*. 34: 475-483.

Beemster ABR, and Rozendaal A, 1972. In: de Bokx JA, ed. Viruses of Potatoes and Seed-Potato Production. Wageningen: PUDOC.

Blanco-Urgoiti B, Tribodet M, Leclere S, Ponz F, Perez de San Roman C, Legorburee FJ, Kerlan C, 1998b. Characterization of potato virus Y (PVY) isolates from seed potato batches. Situation of the NTN, Wilga and Z isolates. *Eur J Plant Pathol* 104: 811–819.

Bradley, R.H.E., 1954. Studies of the mechanism of transmission of potato virus Y by the green peach aphid, Myzus persicae (Sulz.). *Can. J. Zool.* 32:64-73.

Boonham, N., Barker,I., 1998. Strain-specific recombinant antibodies to potato virus Y potyvirus. *J. Virol. Methods* 74 (2), 193–199.

Boonham, N., M. Hims, I. Barker, N. Spence, 1999. Potato virus Y from petunia can cause symptoms of potato tuber necrotic ringspot disease (PTNRD). *Eur J Plant Pathol* 105: 617–621

Boonham N, Walsh K, Hims M, North J, Barker I., 2002a. Biological and sequence comparisons of Potato virus Y isolates associated with potato tuber necrotic ringspot disease. *Plant Pathol* 51: 117–126.

Boonham, N., Walsh, K., Preston, S., North, J., Smith, P. & Barker, I. 2002b. The detection of tuber necrotic isolates of Potato Virus Y, and the accurate discrimiation of $PVY^{O}$, $PVY^{N}$ and $PVY^{C}$ strains using RT-PCR. *Journal of Virological Methods*, 102, 103-112.

Bowen, W.T., 2003. Water productivity and potato cultivation. p 229-238. In J.W. Kijne, R. Barker and D. Molden (Eds.) Water Productivity in Agriculture: Limits and Opportunities for Improvement. CAB International, 2003. Available on line at: http://www.iwmi.cgiar.org/pubs/Book/CA_ CABI-Series/Water_Productivity/Protected/0 851996698ch14.pdf/

Brigneti G., Voinnet O., Li W.X., Ji L.H., Ding S.W. and Baulcombe D.C. 1998. Viral pathogenicity determinants are suppressors of transgene silencing in Nicotiana benthamiana. *EMBO J*. 17: 6739–46.

Carlebach R., Raccah B., Loebesntein G., 1982. Detection of Potato virus Y in the aphid *Myzus persicae* by enzyme-linked immunosorbent assay (ELISA). *Ann Appl Biol* 101, 511-516.

Celebi-Toprak, F., Slack, S. A., and Jahn, M. M. 2002. A new gene, Nytbr, for hypersensitivity to Potato virus Y from Solanum tuberosum maps to chromosome IV. *Theor. Appl. Genet*. 104:669-674.

Cervantes, F.A. and J.M Alvarez, 2008. Role of hairy nightshadeSolanum sarrachoides (Sendtner) in the transmission of Potato virus Y (PVY) strains by aphids and study of different PVY strains reaction on Solanum tuberosum (Linnaeus). (Abstr.) *Phytopathology* 98:S190.

Chandelier, Dubois, Baden, De Leener, Warmon, Remacle and Lepoivre, 2001. *Journal of Virological Methods* 91: 99.

Chrzanowska, M., 1995. The reaction of Polish potato cultivars to $PVY^{NTN}$. *Potato Research* 38, 434-435.

Cockerham, G. 1943. Potato breeding for virus resistance. *Ann. Appl. Biol.* 30:105-108.

Cockerham, G. 1970. Genetical studies on resistance to potato viruses X and Y. *Heredity* 25:309-348.

Crosslin, J. M., Hamm, P. B., Shiel, P. J., Hane, D. C., Brown, C. R. & Berger, P. H., 2005. Serological and molecular detection of tobacco veinal necrosis isolates of Potato virus Y ($PVY^{N}$) from potatoes grown in the western United States. *American Journal of Potato Research*, 82, 263-269.

De Bokx, JA., 1961. Waardplanten van het aardappel- YN-virus. *T Pl-ziekten* 67: 333–342.

De Bokx, 1987. In: J.A. De Bokx & J.P.H. Van der Want, *Viruses of potatoes and seed-potato production,* 2nd edition, p.58, ed. Wageningen: Pudoc.

 

De Bokx JA, Huttinga H, 1981. Potato virus Y. CMI/AAB Descriptions of Plant Viruses No 242. Wellesbourne, UK: Association of Applied Biologists, 6 pp.

Delgado-Sanchez S, Grogan RG, 1966. Purification and properties of potato virus Y. *Phytopathology*, 56:1397.

De Wijs & Suda-Bachman, 1979. *Netherlands Journal of Plant Pathology* 85: 23.

Difonzo, C. D., Ragsdale, D. W. & Radcliffe, E. B., 1994. Susceptibility to Potato leafroll virus in potato: Effects of cultivar, plant Age at inoculation, and inoculation pressure on tuber infection. *Plant Disease*, 78, 1173-1177.

Dolnicar, P., 1995. The segregation for extreme resistance in progeny infected with different isolates of PVYn strain. *Potato Research* 38, 434.

Dougherty W.G., Lindbo J.A., Smith H.A., Parks T.D., Swaney S. and Proebsting W.M. 1994. RNA-mediated virus resistance in transgenic plants: exploitation of a cellular pathway possibly involved in RNA degradation. *Mol. Plant Microbe Interact*. 7:544–52.

Dougherty W.G. and Parks T.D. 1995. Transgenes and gene suppression: telling us something new? *Curr. Opin. Cell Biol.* 7:399–405.

Draper, Pasche & Gudmestad, *American Journal of Potato Research* 79: 155, 2002.

Edwardson JR, 1974. Some properties of potato virus Y group. Forida Agricultural Experiment Station Monograph, No. 4, 398 pp.

Edwardson and Christie, 1997. *Florida Agricultural Experiment Station Monograph Series* 18-II: 467.

Ellis, P., Stace-Smith, R., Bowler, G., Mackenzie, D.J., 1996. Production of monoclonal antibodies for detection and identification of strains of potato virus Y. *Can. J. Plant Pathol.* 18, 64–70.

Ellis, P., Stace-Smith, R., and de Villiers, G. 1997. Identification and geographic distribution of serotypes of potato virus Y. *Plant Dis*. 81:481-484.

FAO, 2004. Agricultural data. Production and Indices Data Crop Primary. http://www.fao.org/ 05 /6 /2013.

FAO, 2005. FAOSTAT Agricultural Data. Agricultural production, crops, primary. Available at http://faostat.fao.org/faostat/collections?subset=agriculture Accessed on 10 February 2005; verified on 17 March 2005. United Nations Food and Agriculture Organization.

FAO (Food and Agriculture Organitiobn), 2008. Potato World: Africa—International Year of the Potato 2008. http://www.potato2008.org/en/world/africa.html. Date of accession: 08/6/2013.

Fellers, J.P, D. Tremblay, M.F. Handest and S.A. Lommel, 2002. The *Potato virus Y* $M^{S}N^{R}$ Nib-replicase is the elicitor of a veinal necrosis-hypersensitive response in root knot nematode resistant tobacco. *Molecular Plant Pathology* 3, 145-152.

Fereres, A., Perez, P., Gemeno, C., and F. Ponz, 1993. Transmission of Spanish pepper-PVY and potato-PVY isolates by aphid (Homoptera, Aph- ididae) vectors: Epidemiologic implications. *Environ. Entomol*. 22:1260-1265.

Fernandez-Northcote, E. N., 1983. Prospects for stability of resistance to potato virus Y. In Research for the Potato in the Year 2000. Proceedings of the International Congress in Celebration of the Tenth Anniversary of the International Potato Center, Lima, Peru, 22-27 February, 1982, p. 82. Edited by W. J. Hooker. Peru: International Potato Center (CIP).

Fletcher, J. D. 1989. Potato virus YN - Host range and incidence in seed potato crops in New Zealand. N.Z. *J. Crop Hortic. Sci*. 17:259-263.

Fribourg, C.E. & Nakashima, J., 1984. Characterization of a new potyvirus from potato. *Phytopathology* 74, 1363-1369.

Galvino-Costa, S.B.F., Dos Reis Figueira, A., Camargos, V.V., Geraldino, P.S., Hu, X.J., Nikolaeva, O.V., Kerlan, C. & Karasev, A.V., 2011. A novel type of Potato virus Y recombinant genome, determined for the genetic strain PVYE. *Plant Pathology*, 1-11.

Gebre-Selassie K, Marchoux G, Delecolle B, and E. Pochard, 1985. Variability naturelle de souches du virus Y de la pomme de terre dams les cultures de piment du sudest de la France. Characte erisation et classification en pathotypes. *Agronomie* 5: 621–630

German, T. L., 2001. Potato virus Y. Pages 69-71. in: Compendium of Potato Diseases W. R. Stevenson, R. Loria, G. D. Franc, and D. P. Weingartner, eds. American Phytopathological Society, St. Paul, MN.

Glais L, Tribodet M, Gauthier JP, Astier-Manifacier S, Robaglia C, Kerlan C., 1998. RFLP mapping of the whole genome of potato viral isolates representative of different biological groups of potato virus Y. *Arch Virol* 143: 2077–2091.

Glais L, Tribodet M, Kerlan C., 2002. Genomic variability in potato potyvirus Y (PVY): evidence that $PVY^{NW}$ and $PVY^{NTN}$ variants are single to multiple recombinants between PVYO and $PVY^{N}$ isolates. *Arch Virol* 147: 363–378

Gnutova and Krylov, 1975b. *Acta Phytopathologica Academiae Scientiarum Hungaricae* 10: 203.

Gooding GV. and SA. Tolin, 1973. Strains of potato virus Y affecting flue-cured tobacco in the southeastern United States. *Plant Dis Rep*, 57: 200–204.

 

Gray, S., S. De Boer, J. Lorenzen, A. Karasev, J. Whitworth, P. Nolte, R. Singh, A. Boucher and H. Xu, 2010. Potato Virus Y. An Evolution Concern for Potato Crops in the United States and Canada. *The American Phytopathological Society*, 10.1094.

Halterman, D., A. Charkowski, and J. Verchot, 2012. Potato, Viruses, and Seed Certification in the USA to Provide Healthy Propagated Tubers. Pest Technology Global Science Books.

Hammond and Lawson, 1988. *Journal of Virological Methods* 20: 203.

Harrington, R. and R.W. Gibson, 1989. Transmission of potato virus Y by aphids trapped in potato crops in southern England. *Potato Res*. 32, 167-174.

Harrison, B.O., Finch, L.T., Gibbs, A.J., Hollings, M., Shepherd, R.J., Valenta, V. and C. Wetter, 1971. Sixteen groups of plato viruses. *Virology* 45, 356-363.

Hinrichs, J., Berger, S. and Buchenauer, H. 1997. Susceptibility of potato cultivars to the N and O strain of potato virus Y (PVY) and other potyviruses. Zeitschrift fu X r Panzenkrankheiten und Panzenschutz 104, 193-199.

Hollings, I. & Brunt, A. A., 1981. In Handbook of Plant Virus Infections, pp. 732-777. Edited by E. Kurstack. Amsterdam: Elsevier/North-Holland.

Huttinga and Maat, 1987. In: De Bokx J.A. and J.P.H. Van der Want, Wageningen: *Viruses of potatoes and seed-potato production*, 2nd edition, p.25, ed. Pudoc.

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.

Jacquot, E., M. Tribodet, F. Croizat, V. Balme-Sinibaldi and C. Kerlan, 2005. A single nucleotide polymorphism-based technique for specific characterization of YO and YN isolates of Potato virus Y (PVY). *Journal of Virological Methods* 125 , 83–93

Jeffries, C.K. 1998. Potato. FAO/IPGRI Technical Guidelines for the Safe Movement of Germplasm No. 19. Rome, Italy: Food and Agriculture Organization of the United Nations / International Plant Genetic Resource Institute.

Jones, R.A.C. 1981. The ecology of viruses infecting wild and cultivated potatoes in the Andean region of South America. In: Pests, Pathogens and Vegetation (ed. Tresh, J.M.), Pitman Books Ltd., London. pp. 89-105.

Jones RAC., 1990. Strain group specific and virus specific hypersensitive reactions to infection with potyviruses in potato cultivars. *Ann Appl Biol* 117:93–105.

Jones, R., S. Kumar and A. Mackie, 2003. Potato Virus Y. Fact sheet, State of Western Australia. ISSN: 1443-7783.

Kahn, R. P. & Monroe, R. O. 1963. Detection of the tobacco veinal necrosis strain of the Potato virus Y in Solanum cardenasii and S. andigenum introduced into the United States. *Phytopathology* 53, 1356-1359.

Kang , B.C., I. Yeam, and M.M. Jahn, 2005. Genetics of plant virus resistance. Ann Rev *Phytopathol* 43:581–621.

Kaniewski W, Lawson C, Sammons B, Haley L, Hart J, Delannay X, Tumer NE, 1990. Field resistance of transgenic Russet Burbank potato to effects of infection by potato virus X and potato virus Y. *Bio/Technology*, 8:750-754; 20.

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.

Katis, N., J. M. Carpenter and R. W. Gibson, 1986. Interference between potyviruses during aphid transmission. *Plant Pathology*, 35: 152-157.

Kerlan, C. 2006. Potato Virus Y. CMI/AAB Descriptions of Plant Viruses 414.

Kerlan C, Tribodet M, Glais L, Guillet M., 1999. Variability of potato virus Y in potato crops in France. *J Phytopathol* 147: 643–651.

Kerlan, C., Nikolaeva, O.V., Hu, X., Meacham, T., Gray, S.M. & Karasev, A.V., 2011. Identification of the molecular make-up of the Potato virus Y strain PVY(Z): Genetic typing of $PVY^{(Z)-NTN}$. *Phytopathology* 101, 1052-1160.

Klinkowski & Schmelzer, 1957. *Phytopathologische Zeitschrift* 28: 285

Kogovšek, P., Pompe-Novak, M., Baebler, Š., Rotter, A., Gow, L., Gruden, K. D., Foster, G., Boonham, N., and Ravnikar, M., 2010. Aggressive and mild Potato virus Y isolates trigger different specific responses in susceptible potato plants. *Plant Pathol*. 59:1121-1132.

Latore, B.A., Flores, V., Marholz, G., 1984. Effect of potato virus Y on growth, yield and chemical composition of flue-cured tobacco in Chile. *Plant Dis*. 68, 884–886.

Lawson C, Kaniewski W, Haley L, Rozman R, Newell C, Sanders P, Tumer NE, 1990. Engineering resistance to mixed virus infection in a commercial potato cultivar: resistance to potato virus X and potato virus Y in

 

transgenic Russet Burbank. *Bio-Technology*, 8(2):127-134.

Leiser, R.M. and J. Richter, 1978. Purification and some characteristics of potato virus Y. *Archiv fur Phytopathologie und Pflanzenschutz*, 14(6):337-350.

Le Romancer, M. and Nedellec, M., 1997. Effect of plant genotype, virus isolate and temperature on the expression of the potato tuber necrotic ringspot disease (PTNRD). Plant *Pathology* 46, 104-111.

Le Romancer M, Kerlan C, and M. Nedellec, 1994. Biological characterization of various geographical isolates of potato virus Y inducing superficial necrosis on potato tubers. *Plant Pathol* 43: 138–144

Loebenstein G, and Raccah B, 1980. Control of non-persistently transmitted aphid-borne viruses. Phytoparasitica, 8:221-235.

Lawson G., Kaniewski W., Haley L., Rozman R., Newell C., Sanders P. and Tumer N.E. 1990. Engineering resistance to mixed virus infection in a commercial potato cultivar: resistance to potato virus X and potato virus Y in transgenic Russet Bur- bank. *Biotechnology* 8: 1277–134.

Maki-Valkama T., Pehu T., Santala A., Valkonen J.P., Koivu K., Lehto K. and E. Pehu, 2000a. High level of resistance to potato virus Y expressing P1 sequence in antisense orientation in transgenic potato. *Molecular Breeding* 6: 95–104.

Maki-Valkama T., Valkonen J.P., Kreuze J.F. and Pehu E. 2000b. Transgenic resistance to $PVY^{O}$ associated with post-transcrip-tional silencing of P1 transgene is overcome by $PVY^{N}$ strains that carry highly homologous P1 sequences and recover transgene expression at infection. Mol. *Plant Microbe Interact*. 13:366–73.

Makkouk, K.M. and D.J. Gumpf, 1974.Isolation and Properties of Potato Virus Y Ribonuclic Acid. *Phytopathology* 64: 1115-1118.

Masuta, C., M. Nishimura, H. Morishita and T. Hataya, 1999. A single amino acide change in viral genome-associated protein of potato virus Y correlates with resistance breeding in "virgin A mutant" tobacco. *Phytopathology* 89, 118-123.

Matousek, J., Ptacek, J., Dedic, P., Schubert, J., 2000. Analysis of variability of P1 gene region of N strain of potato virus Y using temperaturegradient gel electrophoresis and DNA heteroduplex analysis. *Acta Virol*. 44 (1), 41–46.

McDonald JG. and Kristjansson GT., 1993. Properties of strains of potato virus YN in North America. *Plant Dis* 77: 87–89

McDonald JG, Singh RP., 1996. Host range, symptomlogy and serology of isolates of potato virus Y(PVY) that shared properties with the $PVY^{N}$ and $PVY^{O}$ strain groups. Am Potato J 73: 309–315.

Menzies, J.D., & Griebel, G.E., 1967. Survival and saprophytic growth of *verticillium dahliae* in uncropped soil. *Phytopathology*, *57*, 703–709.

Mestre, P., Brigneti, G., Baulcombe, D.C., 2000. An Ry-mediated resistance response in potato requires the intact active site of the NIa proteinase from potato virus Y. *J. Plant* 23, 653– 661.

Mestre, P., Brigneti, G., Durrant, M.C., Baulcombe, D.C., 2003. Potato virus Y NIa protease activity is not sufficient for elicitation of Ry-mediated disease resistance in potato. *J. Plant* 36, 755– 761.

Milne, R.G., 1988. The Plant Viruses-4. In: Milne, R.G. (Ed.), The Filamentous Plant Viruses. Plenum Press, New York, London.

Munoz, R. J., Plaisted, R. L., and Thurston, H. D., 1975. Resistance to potato virus Y in Solanum tuberosum ssp. andigena. *J. Am. Potato* 52:107-115.

Moury B,Morel C, Johansen E, Guilbaud L, Souche S, Ayme V, Caranta C, Palloix A, Jacquemond M., 2004. Mutations in Potato virus Y genome-linked protein determine virulence towards recessive resistances in Capsicum annuum and Lycopersicon hirsutum. *Mol Plant Microbe Interact* 17: 322–329

Nie X, Singh RP., 2003a. Evolution on North American $PVY^{NTN}$ strains Tu 660 from local PVYN by mutation rather than recombination. Virus Genes 26: 39–47.

Nie X, Singh RP., 2003b. Specific differentiation of recombinant PVYN:O and PVYNTN isolates by multiplex RT-PCR. *J Virol Meth* 113: 69–77.

Nie X. and R.P. Singh, 2003a. Evolution on North American $PVY^{NTN}$ strains Tu 660 from local $PVY^{N}$ by mutation rather than recombination. *Virus Genes* 26: 39–47.

Nolte, P., Whitworth, J. L., Thornton, M. K., and McIntosh, C. S. 2004. Effect of seedborne Potato virus Y on performance of Russet Burbank, Russet Norkotah, and Shepody potato. *Plant Dis.* 88:248-252.

Novy, R. G., and Helgeson, J. P., 1994. Resistance to Potato virus Y in somatic hybrids between Solanum etuberosum and a S. tuberosum × S. berthaultii hybrid. *Theor. Appl. Genet*. 89:783-786.

Novy, R. G., Nasruddin, A., Ragsdale, D. W., and Radcliffe, E. B., 2002. Genetic resistances to potato leafroll virus, Potato virus Y, and green peach aphid in progeny of Solanum etuberosum. *Am. J. Potato Res.* 79:9-18.

Nyankanga RO, Wien HC, Olanya OM, Ojiambo PS. 2004. Farmers, cultural practices and management of potato late blight in Kenya highlands: Implications for development of integrated disease management.

 

*Int J Pest Man.* 50:135–144.

Ounouna, H., Kerlan, C., Lafaye, P., Loukili, M.J., ElGaaied, A., 2002. Production of monoclonal antibodies against synthetic peptides of the N-terminal region of Potato virus Y coat protein and their use in PVYstrain differentiation. *Plant Pathol.* 51, 487–494.

Oshima, K., Inoue, A.K., Ishikawa, Y., Shikata, E., Takashi, H., 1990. Production and application of monoclonal antibodies specific to ordirnary strain and necrotic strain of potato virus Y. Ann. Phytopathol. *Soc. Jpn.* 56, 508–514.

Ohshima, K., Sako, K., Hiraishi, C., Nakagawa, A., Matsuo, K., Ogawa, T., Shikata, E. and Sako, N. 2000. Potato tuber necrotic ringspot disease occurring in Japan: Its association with Potato Virus Y necrotic strain. *Plant Dis.:* 84:1109-1115.

Paul, H.,1992. The potato crop. The scientific basis for improvement. 2nd Eds. London. 403p.

Pehu T., Maki-Valkama T., Valkonen J.P., Koivu K., Lehto K. and Pehu E., 1995. Potato plants transformed with a potato virus Y P1 gene sequence are resistant to $PVY^{-O}$. *Am. Potato J.* 72: 523–532.

Powell, G., 1992. The effect of mineral oil on stylet activities and Potato virus Y transmission by aphids. *Entomol. Exp. Appl*. 63:237-242.

Powell, G., Hardie, J. and Pickett, J. A., 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 Enthomology* 122: 331-333.

Provvidenti R, Hampton RO, 1992. Sources of resistance to viruses in the Potyviridae. In: Barnett, OW. Potyvirus Taxonomy. New York, USA: *Springer (Archives of Virology [suppliment]* 5), 189-211.

Purcifull, D. and Hiebert, E. 1982. Tobacco etch virus- CMI/AAB Description of Plant Viruses. No. 258.

Ragsdale, D., Radcliffe, E., and diFonzo, C. D., 2001. Epidemiology and field control of PVY and PLRV. Pages 237-270. In: G. Loebenstein, P. H. Berger, A. A. Brunt, and R. H. Lawson, eds. Kluwer Academic, Dordrecht, Netherlands. Virus and virus-like diseases of potatoes and production of seed-potatoes.

Revers F, Le Gall O, Candresse T, Le Romancer M, Dunez J., 1996. Frequent occurrence of recombinant potyvirus isolates. *J Gen Virol* 77: 1953–1965

Robaglia C, Durand Tardif M, Tronchet M, Boudazin G, Astier Manifacier S, Casse Delbart F, 1989. Nucleotide sequence of potato virus Y (N strain) genomic RNA. *Journal of General Virology*, 70(4):935-947.

Romero A., Blanco-Urgoiti B., Soto M.J., Fereres A., F. Ponz, 2001. Characterization of typical pepper-isolates of PVY reveals multiple pathotypes within a single genetic strain. *Virus Res* 79: 71–80.

Ross, H., 1986. Potato breeding ± problems and perspectives. Advances in Plant Breeding 13.

Ruffel, S., M.H. Dussault, A. Palloix, B. Moury, A. Bendahmane, C. Robaglia and C.Caranta, 2002. A natural recessive resistance gene against potato virus Y in pepper corresponds to the eukaryotic initiation factor 4E (eIF4E). *The Plant Journal,*32, 1067–1075.

Sanz, A., Cambra, M., Perez de San Roman, C., Miguet, J.G., Cort´ es, E., Gorris, M.T., Vela, C., 1990. Preparation of additional monoclonal antibodies for detection and discrimination of potato virus Y isolates infecting potato. *Potato Res.* 33, 365–375.

Sato, M., Nishikawa, K., Komura, K., and Hosaka, K. 2006. Potato virus Y resistance gene, Rychc, mapped to the distal end of potato chromosome 9. *Euphytica* 149:367-372.

Schramm, S., K. Frost, A. Charkowski, S. Gray, A. Crochford and R.L. Groves, 2011. Management of Potato Virus Y (PVY) in Wisconsin Seed Potato Production. A3951, I-06-11.

Shukla, D.D., Inglis, A.S., McKern, N. & Gouge, T.H., 1986. Coat protein of potyviruses. 2. Amino acide sequence of coat protein of potato virus Y. *Virology* 152, 118-125.

Shukla, D.D. and Ward, C.W., 1989. Structure of Potyvirus coat protein and its application in the taxonomy of the Potyvirus group. *Advances in the Virus Research* 36, 273-314.

Shukla, D.D., Ward, C.W. and Brunt, A.A., 1994. The Potyviridae. C.A.B. International, Wallingford, UK.

Shukla, D.D., Jilka, J., Tosic, M. & Ford, R. E., 1989. A novel approach to serology of potyviruses involving afffinity-purified polyclonal antibodies directed towards virus-specific N termini of coat proteins. *Journal of General Virology* 70, 13-23.

Shukla, D.D., Jilka, J., Tosic, M. & Ford, R. E., 1988a. A novel approach to serology of potyviruses involving afffinity-purified polyclonal antibodies directed towards virus-specific N termini of coat proteins. Journal of General Virology (in press).

Shukla, D.D., Mckern, N.M., Gough, K.H., Tracy, S.L. & Letho, S.G., 1988b. Differentiation of potyviruses and their strains by high performance liquid chromatographic peptide profiling of coat proteins. *Journal of General Virology* 69, 493-502.

Shukla, D.D., McKern, N.M. & Ward, C.W., 1988c. Coat protein of potyviruses. 5. Symptomatology, serology and coat protein sequences of three strains of passion-fruit woodiness virus. Archives of Virology (in press).

Shukla, D.D., Toslc, M., Jilka, J., Ford, R. E., Toler, R. W. & Langham, M., 1989. Taxonomy of potyviruses

 

infecting maize, sorghum and sugarcane in Australia and the United States as determined by reactivities of polyclonal antibodies directed towards virus-specific N-termini of coat proteins. Phytopathology (in press).

Sigvald, R., 1984. The relative efficiency of some aphid species as vector of potato virus Y. *Potato Res.* 27, 285–290.

Sigvald, R., 1985. Mature plant resistance of potato plants against potato virus Y (PVY ) *Potato Res*, 28, 135-143.

Singh, R.P., Boucher, A., Somerville, T.H., Dhar, A.K., 1993. Selection of a monoclonal antibody to detect PVY-N and its use in ELISA and DIBA assays. *Canad. J. of Plant Pathol*., 15, 293-300.

Singh, R. P., McLaren, D. L., Nie, X., and Singh, M. 2003. Possible escape of a recombinant isolate of Potato virus Y by serological indexing and methods of its detection. *Plant Dis*. 87:679-685.

Singh, R.P., J.P.T.Valkonen, S. M. Gray, N. Boonham, R.A.C. Jones, C. Kerlan and J. Schubert, 2008. Discussion paper: The naming of Potato virus Y strains infecting potato. Netherlands, *Arch Virol*, 153: 1–13.

Singh, M. and R.P. Singh, 1996. Nucleotide sequence and genome organization of a Canadian isolate of the common strain of potato virus Y ($PVY^{O}$). *Can J Plant Pathol* 18: 209–224.

Silberschmidt K., 1960. Types of potato virus Y necrotic to tobacco history and recent observation. *Am Potato J* 37:151–159.

Simons, J. H. 1956. The pepper vein-banding mosaic virus in the Everglades area of south
Florida. *Phytopathology* 46, 53-57.

Smith, K.M., 1931. Composite nature of certain potato viruses of the mosaic group. *Nature* 127: 702.

Smith KM, Dennis RWG., 1940. Some notes on a suspected variant of solanum virus 2 (potato virus Y). *Ann Appl Biolog* 27:65–70.

Solomon-Blackburn, R. M., and 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.

Spetz C, Taboada AM, Darwich S, Ramsell J, Salazar LF, Valkonen JPT., 2003. Molecular resolution of a complex of potyviruses infecting solanaceous crops at the centre of origin in Peru. *J Gen Virol* 84: 2565–2578.

Urcuqui-Inchima, S., Walter, J., Drugeon, G., German-Retana, S., Haenni, A.L., Candresse, T., Bernardi, F. and O. Le Gall, 1999. Potyvirus helper component-proteinase self-interaction in the yeast two-hybrid system and delineation of the interaction domain involved. *Virology* 258, 95-99.

Valkonen, J.P.T., 1994. Natural genes and mechanisms for resistance to viruses in cultivated and wild potato species (Solanum spp.). *Plant Breeding* 112, 1-16.

Valkonen, J.P.T., 1997. Novel resistances to four potyviruses in tuber- bearing potato species, and temperature-sensitive expression of hyper- sensitive resistance to potato virus Y. *Ann. Appl. Biol*. 130:91-104.

Valkonen, J. P. T., Brigneti, G., Salazar, F., Pehu, E., and Gibson, R. W., 1992. Interactions of the Solanum spp. of the Etuberosa group and nine potato-infecting viruses and a viroid. Ann. *Appl. Biol*. 120:301-313.

Van der Vlugt RAA, Leunissen J, Goldbach R., 1993.Taxonomic relationships between distinct potato virus Y isolates based on detailed comparisons of the viral coat proteins and 30-nontranslated regions. *Arch Virol* 131: 361–375.

Van der Zaag, D.E., 1987. Yield reduction in relation to virus infection. In: De Bokx, J.A., van des Want, J.P.H. (Eds.), Viruses of Potatoes and Seed-Potato Production, second ed. Pudoc, Wageningen, pp. 149–150.

van Hoof, 1980. Aphid vectors of potato virus Y, Neth. *J. Pl Path* 86, 159-162.

Varma A, Gibbs AJ, Woods RD, Finch JT, 1968. Some observations on the structure of the filamentous particles of several plant viruses. *Journal of General Virology*, 2:107.

Vaucheret H., Beclin C. and Fagard M., 2001. Post-transcriptional gene silencing in plants. J. Cell Sci. 114: 3083–91.

Verbeek, M., Piron, P. G. M., Dullemans, A. M., Cuperus, C. and R.A.A. van der Vlugt, 2010. Determination of aphid transmission efficiencies for N, NTN and Wilga strains of Potato virus Y. *Ann. App. Biol*. 156:39-49.

Visser, J.C., 2012. A study of the strain evolution and recombination of South Africa isolates of *Potato virus Y.* Dissertation presented for the degree of Doctor of Philosophy.

Visser, J.C. & Bellstedt, D.U., 2009. An assessment of molecular variability and recombination patterns in South African isolates of Potato virus Y. *Archives of Virology*, 154, 1891-1900.

Voinnet O., 2001. RNA silencing as a plant immune system against viruses. *Trends Genet*. 17: 449–59.

Wang, R.Y., Powell, G., Hardie, J. & Pirone, T.P., 1998. Role of the helper component in vector-specific transmission of potyviruses. *Journal of General Virology*, 79 1519-1524.

Warren, M., Krüger, K. & Schoeman, A. S., 2005. Potato virus Y (PVY) and Potato leaf roll virus (PLRV): Literature review for potatoes South Africa. Department of Zoology and Entomology, Faculty of

 

Natural and Agricultural Sciences, University of Pretoria.

Weidemann HL., 1988. Importance and control of Potato virus YN (PVYN) in seed potato production. *Potato Research* 31, 85-94.

Weidemann H.L. and E. Maiss, 1996. Detection of the potato tuber necrotic ringspot strain of potato virus Y ($PVY^{NTN}$) by reverse transcription and immunocapture polymerase chain reaction. *J Plant Dis Protec* 103: 337–345

Weilguny H. and R.P. Singh, 1998. Separation of Slovenian isolates of PVYNTN from the North American isolates $PVY^{N}$ by a 3-primer PCR. *J Virol Methods* 71: 57–68.

Wikipedia, 2013. Potato virus Y. Available at <<http://en.wikipedia.org/wiki/Potato_virus_Y>>. Date of Access: 08/06/2013.

Yi H.Y., R.C. Rufty, E.A.Wernsman, M.C. Conkling, 1998. Mapping the root-knot nematode resistance gene (Rk) in tobacco with RAPD markers. *Plant Dis* 82: 1319–1322.

The IISTE is a pioneer in the Open-Access hosting service and academic event management. The aim of the firm is Accelerating Global Knowledge Sharing.

More information about the firm can be found on the homepage:
http://www.iiste.org

## CALL FOR JOURNAL PAPERS

There are more than 30 peer-reviewed academic journals hosted under the hosting platform.

**Prospective authors of journals can find the submission instruction on the following page:** http://www.iiste.org/journals/ All the journals articles are available online to the readers all over the world without financial, legal, or technical barriers other than those inseparable from gaining access to the internet itself. Paper version of the journals is also available upon request of readers and authors.

## MORE RESOURCES

Book publication information: http://www.iiste.org/book/

Academic conference: http://www.iiste.org/conference/upcoming-conferences-call-for-paper/

## IISTE Knowledge Sharing Partners

EBSCO, Index Copernicus, Ulrich's Periodicals Directory, JournalTOCS, PKP Open Archives Harvester, Bielefeld Academic Search Engine, Elektronische Zeitschriftenbibliothek EZB, Open J-Gate, OCLC WorldCat, Universe Digtial Library , NewJour, Google Scholar