Christophe Lacomme · Laurent Glais
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Potato virus Y: biodiversity, pathogenicity, epidemiology and management

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Preface

Plant viruses are very important pathogens causing significant direct and indirect losses to crop production and threaten global food sustainability. Increase in human population and balancing the demand for more sustainable ways of crop and food production, while maintaining crop productivity and quality, pose continuous challenges to scientists, agronomists and farmers worldwide. Over the past decades, considerable progress has been made in the understanding of the molecular basis of plant pathogen interactions, epidemiology of diseases and their causal agents and the deployment of this knowledge to design suitable control and management methods. Plants have the ability to defend themselves against most types of pathogens including viruses. Breeding programs have successfully introgressed resistance genes in numerous plant species such as potato to provide means to minimise the impact of viruses. However, as for any biological entities, the continuous evolution of pathogens, in particular viruses such as *Potato virus Y* (PVY), to escape host defence mechanisms and to adapt to different environments represents a constant threat. Potato was recently ranked as the fourth most important crop in the world and the most important non-grain crop, while PVY was identified as one of the top ten most important pathogens due to its economic impact in all potato-growing areas worldwide. In 2009, the international "PVY-Wide" network was created with the aim to share and disseminate knowledge on different aspects of PVY research focussing on the PVY-potato pathosystem and on the interactions of PVY with other solanaceous and non-solanaceous plant species. This informal network initially comprised 26 laboratories from 20 countries and has expanded over the years to include up to 40 laboratories. The participants are from different types of organisations including academia, agricultural research organisations, plant health organisations, laboratories involved in certification schemes mainly on seed potato production and private companies involved in pathogen diagnostics, breeding, and so on. The objectives of this book is to review and disseminate information communicated by colleagues of the PVY-Wide network (including yet unpublished and many other published data) on PVY research worldwide spanning the past few decades, to report the most up-to-date research outputs of basic and of more applied nature and to identify knowledge gaps with the view to stimulate future research.

This book should appeal to plant virologists, plant pathologists and the broad diagnostic, breeding and agronomical industries. The nine chapters of the book cover the essential aspects of PVY research including structure-function and diversity of the PVY genome, plant responses, evolution, diagnostic, epidemiology and transmission, control and management, resistance and the interactions of PVY with other plant species. The editors and authors of this book are indebted to all our colleagues of the PVY-Wide network as well as colleagues from the European Association of Potato Research Virology section for their input. Finally, last but by no means least, we would like to thank our colleague Stuart Carnegie for his contribution, valuable comments and suggestions.

Edinburgh, UK
Le Rheu, France
March 2017

Christophe Lacomme
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Chapter 1
General Characteristics of *Potato virus Y* (PVY) and Its Impact on Potato Production: An Overview

Christophe Lacomme and Emmanuel Jacquot

Abstract Diseases caused by plant viruses can have significant and devastating impacts on many cultivated crops worldwide. The impact of disease caused by a virus depends on the virus species, strains, type of inoculum, host plant characteristics, vector pressure, climatic conditions, trade, changes in agricultural landscape and intensive production practices. Viruses affect plants by causing a large variety of symptoms such as alteration of shape, pigmentation, necrosis on different parts of the plant, thus affecting plant development. In most of the cases, these lead to a decrease in crop yield and quality. There are numerous viruses that affect potato; among them, *Potato virus Y* is considered to be one of the ten most important plant viruses of crops, because of its worldwide distribution and economic impact. Some PVY isolates are able to cause potato ringspot necrotic disease in infected tubers rendering them unmarketable. Understanding the genetic diversity and molecular biology of PVY is essential to understand its infectious cycle, epidemiology and developing efficient methods of control and management for the virus itself and its vector. In spite of an ever-increasing wealth of data in these topics, several major scientific challenges remain in understanding the molecular nature of the interaction between PVY, its hosts, aphid vector in different environments and the epidemiology of PVY. This and following chapters will present the context and current state of our knowledge for these different topics and attempt to provide some answers to these important questions.

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1 Introduction

Changes in the agricultural landscape, crop management, crop intensification, introduction of foreign plant material via increased trade and climate change favor the emergence of infectious diseases of plants (Fargette et al. 2006). Plant viruses, as causal agents of diseases, can have significant and devastating impacts on many cultivated crops worldwide. These impacts depend, among other parameters, on virus inoculum, host plant characteristics (genotype and development stage), vector pressure and climatic conditions (Anderson et al. 2004). Most viruses affect plants by altering their development, causing in most of the cases a range of symptoms such as alteration of shape, modification of pigmentation, elicitation of necrosis on leaves, fruit or tubers, and reduction in plant growth. These different symptoms lead to a decreased crop yield and/or crop quality. These effects can, however, vary greatly for each virus/host combination, and it is not uncommon for crop losses to be either moderate or dramatically high, as exemplified by tomato spotted wilt virus disease on lettuce in USA generating losses of 30–90% (Sherwood et al. 2003) and tomato yellow leaf curl disease on common bean and tomato reducing yield up to 80% (Anderson et al. 2004). Occurrences of virus diseases can sometimes spread over large areas within a relatively short timeframe, as it was the case of bunchy top virus disease that was introduced in Australia in 1913, wiping out banana production in New South Wales by 1927 (Magee 1927, reviewed in Smith et al. 1998). Epidemics of virus disease in a new ecological niche, especially in a suitable environment, can often be very difficult to control and regular outbreaks are likely to occur. The *Groundnut rosette virus* is a good example of pathogen associated to regular outbreaks as more than 15 epidemics of this plant virus were reported on groundnut since the beginning of the twentieth century with losses up to £200 million in sub-Saharan Africa (Sastry and Zitter 2014).

In order to develop effective virus management strategies, it is necessary to diagnose accurately the virus(es) associated with the disease and to understand the disease life cycle of etiological agents (Sastry and Zitter 2014). An accurate assessment of agronomical impacts of a virus disease will require further knowledge on its epidemiology by studying the dynamics and distribution of the disease in hosts and alternative hosts (including wild plants) that act as reservoir of inoculum (Sastry and Zitter 2014). The agronomical impact of a virus depends on the intended use and economic importance of its host plants (grown either as ornamental, staple crop, or cash crop). In 2013, potato was ranked as the fourth most important crop in the world behind corn, wheat, and rice and was ranked the most important non-grain crop with an annual production of over 364 million tons. The importance of potatoes as a staple food worldwide has increased in the past few decades (World Potato Statistics 2015). There has been a dramatic increase in production and demand in Asia, Africa, and Latin America. For the first time in 2005, the developing world's production exceeded that of the developed world. This trend is continuing and reached 52% of global potato output in 2013. China and India are now the greatest producers of potato with about 96 and 45 million tons in 2013, respectively (Table 1.1).

Table 1.1 Top 25 potato producing countries in 2013

Rank	Country	Potato production [tons]
1	China	95,987,500
2	India	45,343,600
3	Russian Federation	30,199,100
4	Ukraine	22,258,600
5	The United States of America	19,843,900
6	Germany	9,669,700
7	Bangladesh	8,603,000
8	France	6,975,000
9	The Netherlands	6,801,000
10	Poland	6,334,200
11	Belarus	5,913,710
12	The United Kingdom	5,580,000
13	Iran (Islamic Republic of)	5,560,000
14	Algeria	4,928,030
15	Egypt	4,800,000
16	Canada	4,620,000
17	Peru	4,570,670
18	Malawi	4,535,960
19	Turkey	3,948,000
20	Pakistan	3,802,200
21	Brazil	3,553,770
22	Belgium	3,479,600
23	Kazakhstan	3,343,600
24	Romania	3,289,720
25	Nepal	2,690,420

Source: World Potato Statistics, FAOSTAT, 2014

2 Viruses Infecting Potato

The importance of viruses as agents of infectious disease of plants was emphasized by Anderson et al. (2004). Viruses represent almost half (47%) of emerging infectious diseases surveyed between 1996 and 2002. A virus can infect many different plant species and a single plant can be infected by many different virus species, strains or isolates. Viruses are submicroscopic obligate intracellular parasites living and replicating in host cells. With some rare exceptions, viruses are assembled into particles made of a nucleic acid core that can be of different nature (see Table 1.2) and encapsidated into a matrix essentially composed of coat protein (CP) and in some cases, additional viral-encoded "accessory" proteins facilitating virus movement and/or transmission. Many diseases of potato are caused by viruses and can be transmitted to succeeding crops through infected seed tubers. Virus disease leads to an ongoing decline in health of a propagated crop, which in early descriptions was generically reported as "degeneration". These pathological phenotypes were further distinguished by the names of leaf roll, mosaic and streak (reviewed by Salaman 1949).

Table 1.2 List of viruses infecting potato and their prevalence in cultivated potato. (*tentative species)

Acronym	Species	Family/(Subfamily)/Genus	Type of genome	Geographical distribution	Transmission/Vector
AMV	*Alfalfa mosaic virus*	*Bromoviridae/Alfamovirus*	ssRNA+	Worldwide	Aphids
APLV	*Andean potato latent virus*	*Tymoviridae/Tymovirus*	ssRNA+	South America	TPS, Beetles.
APMoV	*Andean potato mottle virus*	*Secoviridae/Comovirinae/Comovirus*	ssRNA+	Latin America	Contact, Beetles
AVB	*Arracha virus B*	*Secoviridae/Comovirinae/Cheravirus*	ssRNA+	South America	TPS
BCTV	*Beet curly top virus*	*Geminiviridae/Curtovirus*	ssDNA	Worldwide (arid regions)	Leafhoppers
CMV	*Cucumber mosaic virus*	*Bromoviridae/Cucumovirus*	ssRNA+	Worldwide (rare)	Aphids
EMDV	*Eggplant mottle dwarf nucleorhabdovirus*	*Rhabdoviridae/Nucleorhabdovirus*	ssRNA−	Iran	Aphids
GBNV	*Groundnut bud necrosis virus*	*Bunyaviridae/Tospovirus*	ssRNA−	India	Thrips
GRSV	*Groundnut ringspot virus*	*Bunyaviridae/Tospovirus*	ssRNA−	Argentina	Thrips
INSV	*Impatiens necrotic spot virus*	*Bunyaviridae/Tospovirus*	ssRNA−	Iran	Thrips
PAMV	*Potato aucuba mosaic virus*	*Alphaflexiviridae/Potexvirus*	ssRNA+	Worldwide (uncommon)	Contact, Aphids
PBRSV	*Potato black ringspot virus*	*Secoviridae/Comovirin ae/Nepovirus*	ssRNA+	Peru, others Andean countries?	TPS, Contact, Nematodes?
PLRV	*Potato leaf roll virus*	*Luteoviridae/Polerovirus*	ssRNA+	Worldwide	Aphids
PMTV	*Potato mop top virus*	*Virgaviridae/Pomovirus*	ssRNA+	Europe, America, Asia	Fungi *(Spongospora subterranea*)
PotLV	*Potato latent virus*	*Betaflexiviridae/Quinvirina e/Carlavirus*	ssRNA+	North America	Aphids
PVA	*Potato virus A*	*Potyviridae/Potyvirus*	ssRNA+	Worldwide	Aphids

PVH	*Potato virus H*	*Betaflexiviridae/Quinvirinae/Carlavirus*	ssRNA+	China	Aphids?
PVM	*Potato virus M*	*Betaflexiviridae/Quinvirinae/Carlavirus*	ssRNA+	Worldwide	Aphids
PVP	*Potato virus P*	*Betaflexiviridae/Quinvirinae/Carlavirus*	ssRNA+	South America	Aphids
PVS	*Potato virus S*	*Betaflexiviridae/Quinvirinae/Carlavirus*	ssRNA+	Worldwide	Aphids
PVT	*Potato virus T*	*Betaflexiviridae/Trivirinae/Tepovirus*	ssRNA+	South America	Contact, TPS, Pollen
PVU	*Potato virus U*	*Secoviridae/Comovirinae/Nepovirus*	ssRNA+	Peru	TPS, Contact, Nematodes?
PVV	*Potato virus V*	*Potyviridae/Potyvirus*	ssRNA+	Worldwide	Aphids
PVX	*Potato virus X*	*Alphaflexiviridae/Potexvirus*	ssRNA+	Worldwide	Contact
PVY	*Potato virus Y*	*Potyviridae/Potyvirus*	ssRNA+	Worldwide	Aphids
PYDV	*Potato yellow dwarf nucleorhabdovirus*	*Rhabdoviridae/Nucleorhabdovirus*	ssRNA−	North America	Leafhoppers
PYMV	*Potato yellow mosaic virus*	*Geminiviridae/Begomovirus*	ssDNA	Carribean, Latin America	Whiteflies
PYV	*Potato yellowing virus	*Bromoviridae/Ilarvirus*	ssRNA+	South America	TPS, Aphids
PYVV	*Potato yellow vein virus*	*Closteroviridae/Crinivirus*	ssRNA+	South America	Whiteflies
SALCV	*Solanum apical leaf curl virus	*Geminiviridae/Begomovirus*	ssDNA	South America (Peru)	?
SoMV	*Sowbane mosaic virus*	Unassigned/*Sobemovirus*	ssRNA+	Worldwide (rare)	?
TBRV	*Tomato black ring virus*	*Secoviridae/Comovirinae/Nepovirus*	ssRNA+	Europe, Asia	TPS, Nematodes
TCSV	*Tobacco chlorotic spot virus*	*Bunyaviridae/Tospovirus*	ssRNA−	Argentina/Brazil?	Thrips
ToMoTV	*Tomato mottle Taino virus*	*Geminiviridae/Begomovirus*	ssDNA	Cuba	Whiteflies
ToMV	*Tomato mosaic virus*	*Virgaviridae/Tobamovirus*	ssRNA+	Hungary	Contact
TMV	*Tobacco mosaic virus*	*Virgaviridae/Tobamovirus*	ssRNA+	Worldwide (rare)	Contact
TNV	*Tobacco necrosis virus A*	*Tombusviridae/Alphanecrovirus*	ssRNA+	Europe, South America, Tunisia	Fungi *(Olpidium brassicacae*)

(continued)

Table 1.2 (continued)

Acronym	Species	Family/(Subfamily)/Genus	Type of genome	Geographical distribution	Transmission/Vector
ToLCNDV	*Tomato leaf curl New Delhi virus*	*Geminiviridae/Begomovirus*	ssDNA	India	Whiteflies
ToYVSV	*Tomato yellow vein streak virus*	*Geminiviridae/Begomovirus*	ssDNA	South America (Brazil, Argentina?)	Whiteflies
TRSV	*Tobacco ringspot virus*	*Secoviridae/Comovirinae/Nepovirus*	ssRNA+	Worldwide (except Europe)	TPS, Nematodes
TRV	*Tobacco rattle virus*	*Virgaviridae/Tobravirus*	ssRNA+	Worldwide	Free living nematodes
TSV	*Tobacco streak virus*	*Bromoviridae/Ilarv rus*	ssRNA+	Worldwide	?
TSWV	*Tomato spotted wilt tospovirus*	*Bunyaviridae/Tospovirus*	ssRNA−	Worldwide (hot climates)	Thrips
WPMV	*Wild potato mosaic virus*	*Potyviridae/Potyvirus*	ssRNA+	South America (Peru)	Aphids

Adapted from Jeffries (1998), Valkonen (2007) and updated according to ICTV (2015). TPS: true potato seed. (+)ssRNA, single stranded RNA positive sense; (−)ssRNA, single stranded RNA negative sense; ssDNA: single stranded DNA

2.1 General Properties and Disease Symptoms Caused by Viruses Infecting Potato

Cultivated potatoes can be infected naturally by at least 39 viruses that are classified into 13 families (Table 1.2). The incidence, impact and geographical distribution of these virus species are extremely variable and largely depend on the occurrence of vectors, climatic conditions and management of host crops/plant species.

The foliar symptoms caused by potato viruses include leaf rolling, mosaic (severe or mild), stunting, rugosity, chlorosis, mottling and necrosis (Fig. 1.1). Many potato diseases are often complexes of related or unrelated viruses causing a specific type of foliar symptom (Fig. 1.1). However, symptom severity caused by a single virus isolate can significantly vary between cultivars (Fig. 1.1d, e). In some cases of mixed infections (*e.g*. potex- and luteovirus, potex- and potyviruses, Barker 1987; Vance 1991; Pruss et al. 1997), symptoms can be even more severe than those associated with single infections (Fig. 1.1c).

In addition to foliar symptoms, some viruses can also cause symptoms in tubers, appearing usually as internal and/or external (superficial) necrosis, ringspots and growth cracks (Fig. 1.2). In some cases, tuber necrosis is only observed in specific interactions between one virus and a potato cultivar. Indeed, PLRV infection can elicit tuber net necrosis in the cultivar Russet Burbank (Douglas and Pavek 1972). On the other hand, potato tuber necrotic ringspot disease (PTNRD) (Fig. 1.2f, g, h and i) caused by some *Potato virus Y* (PVY) isolates can be observed in a relatively wide range of potato cultivars. PTNRD caused serious losses in potato crops in several central European countries (Slovenia, Hungary and Germany) and the Middle East (Lebanon) in the 1980s–1990s (Le Romancer et al. 1994). The impact of the disease was dramatic (*i.e*. 18,000 ha or 60% of potato area with more than 50% of frequency of necrotic tubers were reported) and was largely associated with the emergence of PVY isolates with tuber necrotic properties and by a large proportion of acreage in these countries being occupied by a small number of potato cultivars (*e.g*. cvs. Igor, Lola, Monalisa, Rosalie and Hela) which were susceptible to PTNRD (Le Romancer and Nedellec 1997) (see Chaps. 3 and 5). Several studies have shown that PTNRD development depends on the potato genotype, virus genotype and particularly environmental conditions (optimal conditions for PTNRD expression are 20°C during both crop growth and storage). Consequently, PTNRD may develop only in a small proportion of infected tubers if the environmental conditions are less than optimal. In addition, some cultivars such as Spunta, Thalassa and Maris Piper either do not develop PTNRD or only develop relatively mild PTNRD symptoms, while susceptible cultivars such as Hermes, Igor, Lola, Nadine, Nicola, Pentland Crown, and Romano are prone to severe PTNRD development (Le Romancer and Nedellec 1997). Information relating to genetic resources against PVY present in potato germplasm and to potato-virus interactions are presented in Chaps. 2, 3 and 8.

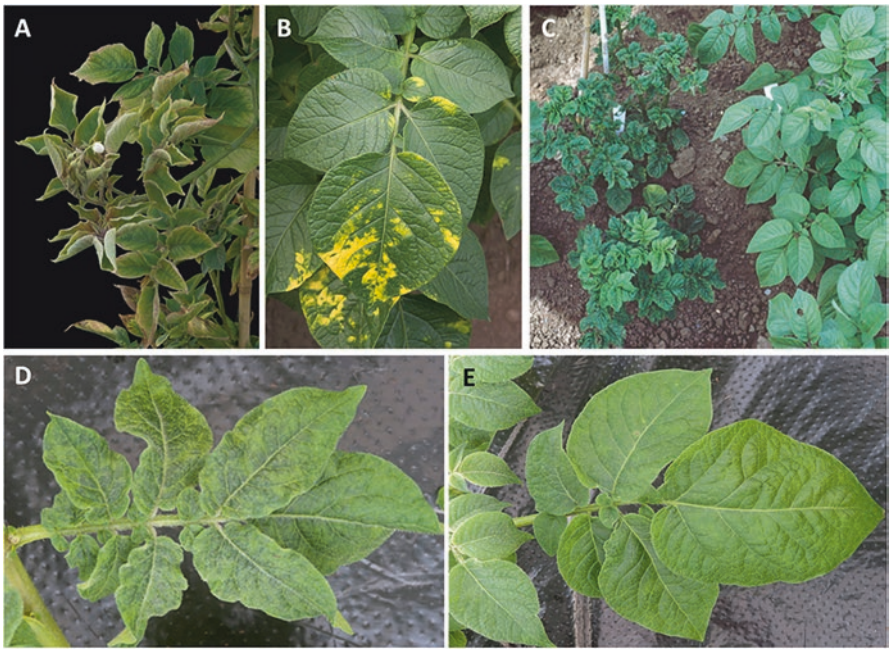


Fig. 1.1 Examples of foliar symptoms caused by some viruses infecting potato. (**a**): Leaf roll caused by *Potato leaf roll virus* (PLRV). (**b**): Mottling caused by *Potato mop top virus* (PMTV). (**c**): Severe mosaic on cv. Red Pontiac caused by *Potato virus A* (PVA) and *Potato virus X* (PVX) mixed infection (stunting and rugosity). D and E: Range of symptoms caused by *Potato virus Y* (PVY) on different potato cultivars. Severe mosaic (leaf distortion and well-defined chlorotic patches) on cv King Edward (**d**) and mild mosaic (mild mottle, not well-defined chlorotic patches) on cv Pentland Crown (**e**) elicited by the same PVY isolate. Photos are courtesy of SASA (Crown copyright©, UK)

2.2 Potato virus Y

2.2.1 Economic Impact

Potato virus Y (PVY) was considered to be one of the ten most important plant viruses of crops, because of its worldwide distribution and economic impacts (Scholthof et al. 2011). PVY is the most important virus infecting potatoes due to its worldwide prevalence, and being the main cause of crop degeneration (both yield and quality) (De Bokx and Huttinga 1981). Some PVY isolates are able to cause PTNRD in infected tubers (see Chap. 3) rendering them unmarketable and thus reducing the marketable yield of tubers. PVY is also a major threat for tobacco and pepper crops and, to a lesser extent, for tomato and eggplant productions (Bhat et al. 1999; Aramburu et al. 2006; Mascia et al. 2010) (see Chap. 9).

Because of its important economic impact, extensive programs have been developed to control PVY epidemics by applying prophylactic measures, controlling aphid vectors and breeding for resistance in potato cultivars. Losses due to virus

Fig. 1.2 Examples of tuber symptoms caused by some viruses infecting potato. (**a**): Net necrosis caused by PLRV on cv. Russet Burbank (Photo courtesy of SA Slack©). (**b**): Spraing on cv. Bute caused by PMTV. (**c**): Spraing and internal necrosis on cv. Habibi caused by PMTV. (**d**): Spraing on cv. Valor caused by *Tobacco rattle virus* (TRV). (**e**): Growth cracks caused by PVA on cv. Estima. (**f**–**g**): Potato tuber necrotic ringspot disease (PTNRD) symptoms caused by PVY (circular ringspot with sunken necrotic skin) on cv. Nadine. (**h**): Severe PTNRD symptoms on cv. Nadine caused by PVY. (**i**): Isolated PTNRD blisters on cv. Maris Piper caused by PVY. Unless specified photos are courtesy of SASA ©Crown copyright (UK)

diseases are not only restricted to direct losses of plant products but are also associated with indirect financial losses such as increased production costs (*e.g*. breeding, training and machinery), cost of control and management of disease (virus control, certification, inspection, virus testing and management tools) and sometimes social and environmental costs (loss of resources, cultural change and contamination of the environment). It has been reported that both direct and indirect estimated losses incurred to PVY to be about $34 million per year for the Idaho state (USA) economy (McIntosh and O'Connell 2014). It was estimated that for each 1% increase of PVY incidence in seed crops (cvs. Russet Burbank and Russet Norkotah), this could result in a reduction of yield of about 180 kg per hectare representing a gross revenue loss of about $18 per hectare (Nolte et al. 2004).

The greatest losses associated to PVY are experienced when the seed tubers being planted are already infected (secondary infection) (De Bokx and van der Want 1987; Whitworth et al. 2006). When plants become infected from virus in seed tubers, reductions in their tuber yield can range from 10 up to 80% in very extreme cases (De Bokx and van der Want 1987). A study of more than 30 cultivars grown in pots demonstrated yield reductions between 50% and 85% compared with plants

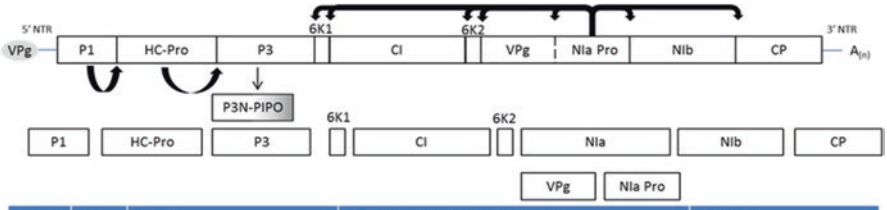
derived from uninfected seed tubers (reviewed in Valkonen 2007). However, yield losses in field grown potato crops with low incidences of PVY are often less marked because neighboring healthy plants have a competitive advantage for space and nutrients and, therefore, compensate for affected plants with an increase in tuber yield. A yield of reduction of 10–15% would be expected for an incidence of PVY-infected seed tubers of 30% in Spain (Valkonen 2007); while in a separate study, yield losses from a crop with 10–20% PVY-infected seed tubers in Finland were found to be negligible (Kurppa and Hassi 1989). However, although crop yield may be only minimally affected, tuber growth can be altered resulting in a wide range of tuber size, grade and shape, thus affecting marketability.

2.2.2 Demarcation Between Virus Genera and Virus Species

Potato virus Y is a member of the *Potyvirus* genus and one of eight genera (*Brambyvirus*, *Bymovirus*, *Ipomovirus*, *Macluravirus*, *Poacevirus*, *Potyvirus, Rymovirus* and *Tritimovirus*) belonging to the family *Potyviridae.* The *Potyviridae* family is the second largest plant virus family after *Geminiviridae*, encompassing about 30% of all described plant viruses (ICTV 2015, Berger et al. 2005). *Potyvirus* is one of the largest genus of plant viruses with 162 virus species currently identified (ICTV 2015). Potyviruses share similar properties in relation to their mode of transmission (aphid transmitted in a non-persistent manner) and genome relatedness. Adams et al. (2005a, b) defined criteria for the demarcation of species and set a threshold of 75–76% of nucleotide identity and 81–82% of amino acid identity, for which higher values represent comparisons between full genome sequences of different isolates of the same species. Alternatively, sequence comparison of the CI gene (RNA helicase, see below) most accurately reflects analysis of the full potyviral genome, suggesting that the RNA helicase is best suited for taxonomic studies when it comes to discrimination between virus genera and species (Adams et al. 2005a).

2.2.3 Genome Structure

Potato virus Y, as for other members of the *Potyvirus* genus, have rod shaped, flexuous particles (about 700 nm in length, 11–13 nm in diameter, helix pitch 3.4–3.5 nm) (Fig. 1.4a) encapsidating the viral RNA with multiple copies (2000 units) of a single coat protein (CP) of 30 kDa. The genome of PVY is a positive (+)-sense single stranded RNA of approximately 9700 nucleotides in length, linked at its 5′ end to a viral protein genome-linked (VPg) and ending with a poly-A tail at its 3′ end (Fig. 1.3). The PVY genome contains one open reading frame (ORF) which is translated as a large polyprotein (about 340–370 kDa), that is then cleaved into 10 (multi) functional proteins (Fig. 1.3): P1, HC-Pro, P3, 6K1, CI, 6K2, VPg, NIa, NIb and CP (reviewed in Danci et al. 2009). More recently, an additional protein P3N-PIPO (Pretty Interesting Potyvirus ORF) has been identified in potyviruses. P3N-PIPO is generated by either a ribosomal slippage creating a +2 frameshift within the P3 ORF



Protein	Size	Function	Cellular Location / Interaction / Comments	References
P1	35 kDa	Serine proteinase. Cleave its own terminus. Stimulate genome amplification. Stimulate HC-Pro silencing suppression activity. Stabilise CP.	Interacts with nucleic acids and binds RNA. Interacts with chloroplastic Rieske Fe/S proteins..	Verchot et al (1991), Verchot & Carrington (1995) Anandalakshmi et al (1998), Shi et al (2007)
HC-Pro	52 kDa	Helper-component proteinase. Cysteine protease. Cleave its own terminus. Required for aphid transmission. Involved in viral replication and pathogenicity. Silencing suppressor activity.	Localise at the ends of virions. Interacts with virus-encoded proteins CP, CI, P1, VPg, NIa. Binds RNA. Interacts with a large number of host proteins (calmodulin-related protein, small RNA methyltransferase, proteasome components, microtubule associated protein, *etc*....). PTK motif interacts with DAG motif of CP to promote binding to aphids mouthparts.	Carrington et al (1989), Dunoyer et al (2014), Kasshau & Carrington (2001), Revers & Garcia (2015), Blanc et al (1997), Torrance et al (2006), Valli et al (2014)
P3, 6K1, P3N-PIPO	50 kDa 6 kDa 25 kDa	P3: required for viral replication, pathogenicity and symptomatology. 6K1: exact function unknown. Role in potyviral infection. P3N-PIPO: required for viral cell-to-cell movement.	P3 associate with cylindrical inclusion formed by CI and with nuclear inclusion formed by NIa and NIb. Localize to endoplasmic reticulum membranes, Golgi apparatus and replication vesicles. Interacts with host Rubisco subunits. PIPO is believed to promote movement of CI and virions/RNA-CP complexes through plasmodesmata.	Guo et al (2001), Rodriguez Cerezo et al (1993), Langenberg & Zhang (1997), Lin et al (2011), Hong et al (2007), Wei et al (2010), Chung et al (2008), White (2015), Ollspert et al (2015), Rodamilans et al (2015),
CI	71 kDa	Forms the Cylindrical cytoplasmic Inclusion protein and pinwheels. RNA helicase. Cell-to-cell movement.	NTP binding, NTPase and ATPase activities. Acts in conjunction with P3N-PIPO. Associated with end of virions, may provide motor function to help virus to traffic through plasmodesmata. Interacts with eIF4E, chloroplast PSI, dsRNA-dependant Protein Kinase inhibitor.	Sorel et al (2014), Fernandez et al (1997), Wei et al (2010), Rodriguez Cerezo et al (1997), Gabrenaite et al (2008), Tavert et al (2012), Jimenez et al (2006), Bilgin et al (2003).
6K2	6 kDa	Membrane anchoring to ER-types membranes.	Crystalline inclusions in cytoplasm and nuclei. Associates with NIa. Strong association with ER vesicles, anchoring of the viral replication complex to ER membranes.	Leonard et al (2004), Beauchemin et al (2007) Schaad et al (1997)
VPg	21 kDa	Virus genome-linked protein. Initiation of replication complex. Nuclear Inclusion protein.	Interacts with host proteins (eukaryotic translation initiation factor eIF4E and eIF(iso)4E, ...) to initiate replication. Interacts with most of potyviral proteins including 6K2 to associate with vesicular ER-membranes	Wittman et al (1997), Leonard et al (2000), Beauchemin et al (2007), Jiang & Laliberte (2011),
NIa-Pro	27 kDa	Small Nuclear Inclusion protein A. Serine-like protease activity. Main potyviral protease : cleave P3-6K1-CI-6K2-VPg-NIa-NIb-CP . Prime RNA synthesis. Required for systemic infection.	Aggregates in the nucleus and cytoplasm to form nuclear inclusion bodies. Binds RNA on its own or as a NIa-6K2 complex. DNAse activity putatively involved in host gene expression regulation.	Adams et al (2005b), Anindya & Savithri (2004).
NIb	58 kDa	Large Nuclear Inclusion protein B. RNA-dependant RNA polymerase (RNA replicase). Viral replication.	Aggregates in the nucleus to form nuclear inclusion bodies. Interacts with NIa. Interacts with host proteins eEF1A, PABP, Hsc70-3, SCE1 to form functional replication complexes. and NIb nucleocytoplasmic transport	Kassanis (1939), Hong & Hunt (1996), Anindya et al (2005), Dufresne et al (2008), Xiong & Wang (2013)
CP	30 kDa	Coat Protein. Role in viral RNA encapsidation, virus movement, aphid transmission and regulation of viral RNA amplification.	In virions CP subunits arranged as helix wrapping genomic RNA. DAG motif at the CP N-terminus interacts with the PTK motif of the HC-pro to promote binding to aphids mouthparts. Interacts with NIb to promote genome amplification. Interacts with hosts Rubisco large subunit.	Lopez et al (2009), Carrington et al (1998), Rojas et al (1997), Blanc et al (1997), Feki et al (2005),

Fig. 1.3 Genomic map of a representative member of Potyvirus genus. Cleavage sites of the potyviral polyprotein by viral-encoded proteases are indicated (bold arrows) and the generated mature proteins are presented below the genomic map. The main characteristics of potyviral proteins are summarized in the table (reviewed in Revers and Garcia 2015)

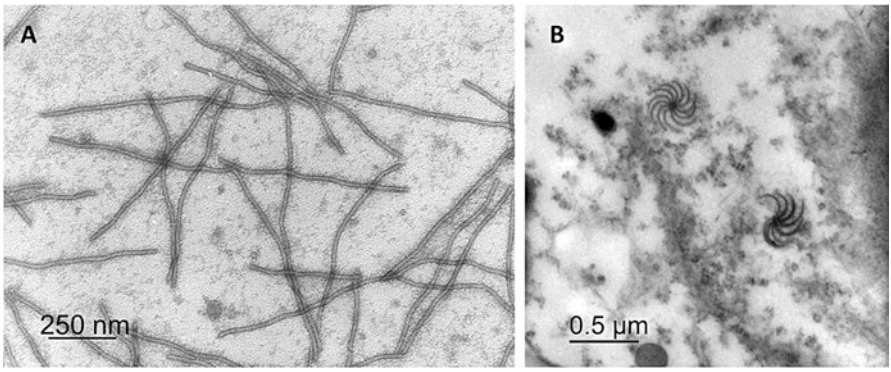


Fig. 1.4 Electron microscopy micrographs of PVY particles (virions) (**a**) and "pinwheels" structures (**b**) in infected plant cells. Courtesy of C. Kerlan (INRA, France) and M.T. Znidaric (NIB, Slovenia)

(Wei et al. 2010) or incorporating an additional nucleotide through slippage of the viral RNA-dependent RNA polymerase at a highly conserved $G_{1\text{-}2}A_{6\text{-}7}$ motif at the 5′ end of PIPO sequence (Olspert et al. 2015, Rodamilans et al. 2015, White 2015).

These viral-encoded proteins display a remarkable degree of multiplicity of functions and are often associated with specific subcellular compartments (chloroplasts, Endoplasmic Reticulum and Golgi apparatus). Viral proteins interact with numerous other viral-encoded proteins and, in some cases, with host proteins which will allow potyviruses to perform all basic viral functions and complete their life cycle (Fig. 1.5). The function of these proteins is summarized in Fig. 1.3 and their roles will be discussed in the following chapters.

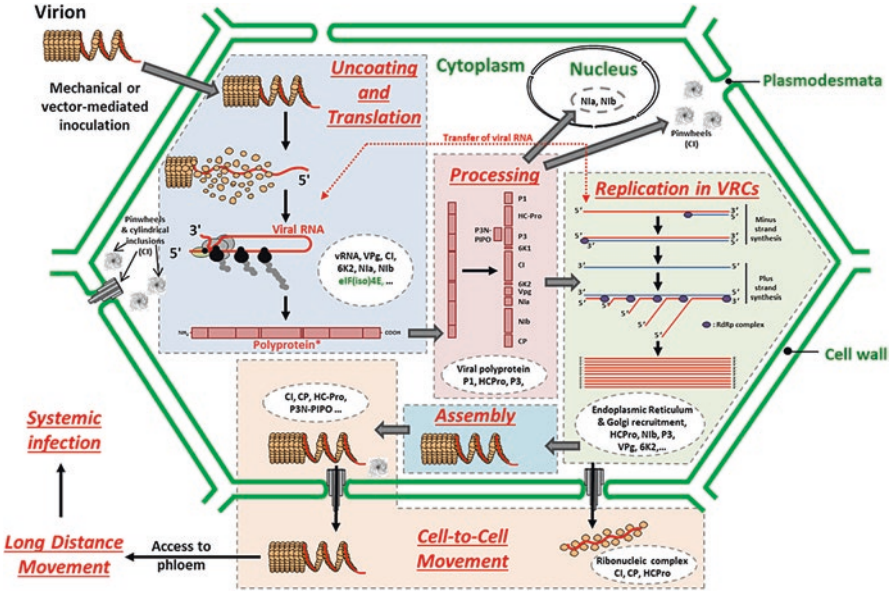


Fig. 1.5 Schematic representation of the major steps in potyvirus infection (adapted from Ivanov et al. 2014). Once virions have infected a plant cell (*i.e.* through aphid feeding or mechanical inoculation), uncoating occurs and expose the viral genomic RNA (5′-3′ molecule illustrated in red) which is then recruited by the host translation machinery (eIF(iso)4E, ribosomes, *etc*...) to synthesize the viral proteins. Processing and maturation of proteins occur and Viral Replication Complex (VRCs) are generated. VRCs are associated to the membrane of host organelles (endoplasmic reticulum, Golgi and vesicles; not illustrated). Replication is initiated by synthesizing a (−) ssRNA (5′-3′ molecule illustrated in blue) by the viral replicase using the (+) ssRNA as a template. The newly synthesized (−) ssRNA is in turn used as a template by the viral replicase to produce numerous (+) ssRNA, which are either encapsidated to produce new virions or recruited by the viral and host proteins to form ribonucleic complexes. Virions and/or ribonucleic complexes recruit the host cytoskeleton (not illustrated) and are transported into the neighboring cells through plasmodesmata via the coordinated action of viral and host proteins. (*): illustration of translation products are simplified as the polyprotein leading to the production of P3N-PIPO is not illustrated

3 Major Properties of PVY

During PVY infection, host cells undergo cytopathological changes that can be observed by electron microscopy. PVY virions (Fig. 1.4a) have been observed to be associated with plasmodesmata (see Sect. 3.2), endoplasmic reticulum (ER) and Golgi apparatus. PVY induce typical cellular inclusion know as cylindrical inclusion (CI) bodies and "pinwheels" structures (Fig. 1.4b). As obligate cellular parasites, viruses must highjack host cellular components in order to perform basic viral functions such as replication, local-systemic movement, transmission and inhibition of host defense mechanisms. These events involve complex molecular mechanisms regulated by host and viral proteins, leading to extensive host gene expression reprogramming events (see Chap. 2) that are spatially and temporally closely associated (for recent reviews on the molecular biology of potyviruses see Ivanov et al. 2014 and Revers and Garcia 2015).

3.1 Replication

After a viral particle has entered a cell through either probing by an aphid vector or by wounding of an epidermal cell, uncoating of the viral particle occurs exposing the viral RNA that will recruit ribosomes and host factors to initiate translation of the viral genes and replication (Fig. 1.5). The (+) sense single stranded (ss)RNA is copied into a complementary (−) sense single RNA strand which in turn is used as a template to synthesize new (+) strands by the action of the RNA-dependent RNA polymerase and RNA helicase. The (+) ssRNA molecules produced during the replication process will then be encapsidated to form new virions (Figs. 1.4a and 1.5).

3.2 Local and Systemic Movement

Once encapsidated, PVY virions move within the initial cell to reach plasmodesmata (PD, symplastic pores between cells), which they cross to enter the neighboring cell and initiate another cycle of replication. A wealth of data supports a direct role for CI (RNA helicase) and CP in the cell-to-cell movement of the viral RNA through plasmodesmata (PD) where CI could form conical structures that facilitate the movement of virions (or CP-coated ribonucleic particles) across the cell wall (Roberts et al. 1998, reviewed in Sorel et al. 2014). The coordinated action of P3N-PIPO (in anchoring the ribonucleic complex to PD), CP and HC-Pro (in increasing the size exclusion limit of PDs) and host proteins will promote virus movement (Fig. 1.5 and Wei et al. 2010).

Successive intra and intercellular movements will occur until the virions reach phloem vessels to be transported throughout the plant in sap from source tissues

(*e.g*. tubers and leaves) to sink tissues (*e.g*. newly formed leaves, stems, roots and tubers). This long distance phloem-mediated movement of virus is tightly regulated, because rate of virus translocation can be reduced or blocked in specific tissues/organs depending on the developmental stage of host plant (Revers and Garcia 2015).

3.3 Transmission

To survive in their environment, viruses can be transmitted either from the infected plant to the progeny (i) through propagation (*i.e*. seeds or storage organs such as tubers), and hence could be present in the next crop generation (vertical transmission) or (ii) mechanically or by means of a vertebrate or invertebrate vector such as animals, insect, fungi or bacteria (horizontal transmission). Aphids are the most common vector of plant viruses accounting for more than 60% of viruses transmitted by invertebrates (reviewed in Wilson 2014 and in Katis et al. 2007). Aphid-mediated transmissions mainly occur in persistent or non-persistent modes.

In the persistent mode of transmission, virus is acquired by an aphid feeding on phloem sap, ingested and internalized by the vector. The virus then either replicates (propagative) or do not (circulative) inside the aphid (Katis et al. 2007). The acquisition of virus can last several hours and is often specific (*e.g*. PLRV transmission is essentially performed by the peach-potato colonizing aphid *Myzus persicae* Sulzer). In persistent transmission, a latency period occurs during which time the virus invades the salivary glands before it can be transmitted to a new host.

Non-persistent transmission (non-circulative) is the most common mode of transmission of plant viruses. PVY is transmitted by aphids in a non-persistent manner (see Chap. 5). The helper component protein (HC-Pro) facilitates the binding of virus particles to the aphid's stylet during brief periods of probing of an infected leaf. Subsequently, this virus is transferred mechanically to a new host during further feeding. Unlike persistent transmission, non-persistently transmitted viruses are acquired in less than a minute, do not need latency period and can be transmitted almost immediately after acquisition. However, aphids lose their infectivity rapidly following subsequent feeding. The association between aphid vector and virus is believed to be relatively non-specific, with a wide range of virus species-strains being able to be transmitted with different efficiency by a wide range of aphid species (see Chap. 6).

In addition to persistent and non-persistent transmission mode, semi-persistent transmission has been described for some viruses (*e.g*. *Citrus tristeza virus* – CTV). Semi-persistent transmission requires periods of acquisition and inoculation longer than non-persistent mode, but does not include a latency period as described for persistent transmission. Usually semi-persistent transmission is efficient about 15 min after acquisition period (Katis et al. 2007).

All types of interactions between aphid, plant and virus involve very complex molecular mechanisms which regulate a wide array of events (*e.g.* virus retention-infection efficiency, suppression of host defense mechanisms, virus-vector host range [for a review, see Giordanengo et al. 2010] and virus-induced chemical changes in infected plants) that impact on aphid behavior and performance (for recent reviews, see Eigenbrode and Bosque-Perez 2016; Fereres 2016). These characteristics of transmission have major implications for the epidemiology and management of viruses and their vectors worldwide. These aspects will be addressed in Chaps. 6 and 7.

4 Current and Future Challenges in PVY Research and Management

For the past 20 years, the vast majority of viral infections in cultivated potato are mainly caused by PVY. In spite of strict management and prophylactic measures, recombinant PVY variants have become prevalent in most of the potato-growing areas worldwide (see Chaps. 3 and 4). To provide assurance of the quality of seed potatoes being planted, seed potato certification schemes have been established to produce seed potatoes containing as limited as practicable amounts of viruses, including PVY (see Chap. 7). However, controlling and managing non-persistent viruses remain an ongoing challenge (Gray et al. 2010, Karasev and Gray 2013). Chemical control of the aphid vectors by insecticides is not effective for non-persistent viruses (Kirchner et al. 2014). Consequently, efficient control of PVY requires the development of different researches programs addressing the following topics: (i) the identification of host resistance genes and the consequences of their deployment (Chap. 8), (ii) the characterization of PVY diversity and pathogenicity (Chaps. 2, 3 and 4), (iii) the epidemiology of PVY in different environments (Chap. 6), (iv) the innovation in diagnosis methods and their deployment (Chap. 5) and (v) the development of suitable control measures and crop management (Chap. 7).

In spite of an increasing wealth of data, several major scientific challenges remain in understanding the complexity of the interaction between PVY with its host(s), aphids vector in their environment, and more broadly, of other vector-borne virus diseases in plants. The current challenges and questions for the scientific community include the following: (i) Why some PVY variants/biotypes are becoming more prevalent in some geographical area? (ii) What is the biological significance of the genomic variability of PVY? (iii) What are the genetic and molecular bases of PVY/host/vector interactions? (iv) How diverse is the epidemiology of PVY in various ecological niches? and (v) Can we integrate environmental and epidemiological data to develop accurate predictive model(s) of PVY incidence? The following chapters will present the current state of our knowledge in these different topics and attempt to provide some answers to these important questions.

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