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Review

Top 10 plant viruses in molecular plant pathology

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SUMMARY

Many scientists, if not all, feel that their particular plant virus should appear in any list of the most important plant viruses. However, to our knowledge, no such list exists. The aim of this review was to survey all plant virologists with an association with *Molecular Plant Pathology* and ask them to nominate which plant viruses they would place in a 'Top 10' based on scientific/economic importance. The survey generated more than 250 votes from the international community, and allowed the generation of a Top 10 plant virus list for *Molecular Plant Pathology*. The Top 10 list includes, in rank order, (1) *Tobacco mosaic virus*, (2) *Tomato spotted wilt virus*, (3) *Tomato yellow leaf curl virus*, (4) *Cucumber mosaic virus*, (5) *Potato virus Y*, (6) *Cauliflower mosaic virus*, (7) *African cassava mosaic virus*, (8) *Plum pox virus*, (9) *Brome mosaic virus* and (10) *Potato virus X*, with honourable mentions for viruses just missing out on the Top 10, including *Citrus tristeza virus*, *Barley yellow dwarf virus*, *Potato leafroll virus* and *Tomato bushy stunt virus*. This review article presents a short review on each virus of the Top 10 list and its importance, with the intent of initiating discussion and debate amongst the plant virology community, as well as laying down a benchmark, as it will be interesting to see in future years how perceptions change and which viruses enter and leave the Top 10.

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INTRODUCTION

Many papers, reviews and grant applications claim that a particular plant virus is of huge importance, and this is probably rightly so. *Molecular Plant Pathology* considered which viruses would appear in a 'Top 10' list of plant viruses based on their perceived importance, scientifically or economically, from the views of the contributors to the journal.

To achieve this, all authors, reviewers, editorial board members and senior editors of *Molecular Plant Pathology* were contacted and asked to nominate three viruses that they would expect to see in a list of the most scientifically/economically important plant viruses.

The survey generated more than 250 votes from the international community, and allowed the generation of a Top 10 plant virus list for *Molecular Plant Pathology* (see Table 1).

Those viruses making a strong appearance on the basis of their scientific importance include: (1) *Tobacco mosaic virus* (TMV), (4) *Cucumber mosaic virus* (CMV), (6) *Cauliflower mosaic virus* (CaMV), (9) *Brome mosaic virus* (BMV) and (10) *Potato virus X* (PVX). It is perhaps justified that TMV and CMV are the two highest placed in terms of scientific importance, as a search of the ISI WEB of Science database in 2011 for papers with these viruses in their titles yielded counts of 3636 (TMV) and 1258 (CMV) versus counts for the other viruses (BMV, PVX and CaMV) of 400–600 for each.

Although many of these viruses still cause significant problems in terms of economic losses in a wide range of crops, it is

Table 1 Top 10 plant viruses. The table represents the ranked list of plant viruses voted for by plant virologists associated with *Molecular Plant Pathology*.

Rank	Virus	Author of virus description
1	*Tobacco mosaic virus* (TMV)	Karen-Beth G. Scholthof
2	*Tomato spotted wilt virus* (TSWV)	Scott Adkins
3	*Tomato yellow leaf curl virus* (TYLCV)	Henryk Czosnek
4	*Cucumber mosaic virus* (CMV)	Peter Palukaitis
5	*Potato virus Y* (PVY)	Emmanuel Jacquot
6	*Cauliflower mosaic virus* (CaMV)	Thomas Hohn and Barbara Hohn
7	*African cassava mosaic virus* (ACMV)	Keith Saunders
8	*Plum pox virus* (PPV)	Thierry Candresse
9	*Brome mosaic virus* (BMV)	Paul Ahlquist
10	*Potato virus X* (PVX)	Cynthia Hemenway

their use as scientific tools that have placed them in high roles of importance for scientists. It is interesting to note that, for viruses such as PVX, the picture has evolved: whilst starting off as a major problem causing significant losses, certification schemes and breeding programmes have acted to reduce its impact; moreover, the initial interest from scientists has led to what is now an excellent model system, not only in terms of the virology of PVX, but also with regard to plant–virus interactions.

The majority of viruses are single-stranded, positive-sense RNA viruses, although other forms of nucleic acid genomes are represented, e.g. double-stranded DNA (dsDNA), represented by CaMV, placed for scientific interest, with unusual translation strategies, the use of reverse transcription in replication, and continued interest and application of CaMV promoters for plant molecular biology studies and transgenic crop applications.

The single-stranded DNA viruses of the *Geminiviridae* are represented by two viruses in the Top 10, namely *Tomato yellow leaf curl virus* (TYLCV) and *African cassava mosaic virus* (ACMV), both having huge economic importance representing billions of US dollars of losses, much of which is exacerbated by efficient transmission via whitefly vectors. In the case of ACMV (and related species), the annual losses are now estimated at US$1.9–2.7 billion, with the cassava disease pandemic in East and Central Africa causing severe hardship and problems. In future years, when another survey of the Top 10 is carried out, it will be interesting to see whether *Cassava brown streak virus* (CBSV, *Potyviridae*), the causal agent of cassava brown streak disease, makes an appearance, as it is clearly emerging as the most serious challenge to cassava production (Monger *et al*., 2001a, b; Yadav *et al*., 2011).

Representing a further form of vector transmission by thrips, and indeed a nucleic acid genome with negative and ambisense single-stranded RNAs, is *Tomato spotted wilt virus* (TSWV), in the Top 10 at position 2, nominated for scientific as well as economic importance. With a worldwide distribution and a broad host range in many economically important crops, TSWV not only raises interest through economic losses, but also through its intriguing biology, at it replicates not only within the plant, but also within the thrips vectors.

The final two entries in the Top 10 at positions 5 and 8, *Potato virus Y* (PVY) and *Plum pox virus* (PPV), respectively, are both from one of the largest families of plant viruses, the *Potyviridae*, also containing many of the most economically significant viruses. Both viruses have a worldwide distribution, and are efficiently transmitted by aphids, making them difficult to control. PPV is the most serious viral disease of stone fruit crops, with control measures costing billions of US dollars over recent years. PVY, also transmitted by aphids, shows further problems created by the wide range of isolates with highly variable degrees of virulence, and although the hugely important crop of potato remains a primary source of concern and of crop losses, PVY also causes significant damage in tobacco, tomato and pepper.

Although the aim of this review article was to identify the Top 10 most important plant viruses according to contributors to *Molecular Plant Pathology*, we are very much aware that importance and priorities can vary locally across continents and disciplines. We are also aware that not all viruses can make it into any Top 10, for obvious numerical reasons, although such viruses may still be regarded as hugely important. We therefore felt it appropriate to make honourable mentions for viruses just missing out on the Top 10 list, including *Citrus tristeza virus* (Moreno *et al*., 2008), *Barley yellow dwarf virus* (Miller *et al*., 2002), *Potato leafroll virus* (Taliansky *et al*., 2003) and *Tomato bushy stunt virus* (Yamamura and Scholthof, 2005), all clearly important.

This review contains brief descriptions, with illustrative figures, of the Top 10 viruses, which will introduce the reader to each of them and provide some key references for further reading. Overall, the review hopes to trigger discussion and debate amongst the plant virology community, as well as to lay down a benchmark, as it will be interesting to see how perceptions change in future years and which viruses enter and leave the list.

1. *TOBACCO MOSAIC VIRUS* (TMV)

Tobacco mosaic virus (TMV) has been voted as the most important plant virus in this poll of the plant virology community. TMV continues to be an important teaching system for the classroom (Scholthof, 2000), and has developed and maintained its status as a model system for more than 110 years, as a result of a plethora of scientific studies initiated from a need to understand how to control TMV-induced disease on tobacco (Scholthof, 2004) (Fig. 1).

Martinus Beijerinck was the first to define TMV as a small infectious entity in 1898 (reviewed in Scholthof *et al*., 1999). His findings were confirmed and recapitulated by others in the early 20th century, yet it was Henry A. Allard of the US Department of Agriculture (USDA) who performed very forward thinking and careful experiments to demonstrate that the mosaic disease of tobacco was not a physiological effect or an enzyme—it was an infection, a virus (Allard, 1916). Research remained at almost a standstill until the work of three central figures in virology: Helen Purdy Beale and Francis O. Holmes of the Boyce Thompson Institute for Plant Research, and Howard H. McKinney of the USDA (Scholthof, 2004, 2011; Scholthof and Peterson, 2006; Scholthof *et al*., 1999). They introduced the now universal tools of serology, local lesion assay/resistance genes and cross-protection, respectively. In some sense, the rest can be considered commentary or refinements of their findings.

TMV also had a direct role in at least two Nobel Prizes (Creager, 2002; Klug, 2010), and many 'firsts': the first plant virus RNA sequenced, the first defined movement protein (MP), the first demonstration of the efficacy of transgenic coat protein (CP) expression for protection from infection, the first plant breeding and molecular evidence of a gene-for-gene resistance interaction and, more recently, the first proof-of-principle platform for both nanodevices and the expression of therapeutic monoclonal antibodies and other pharmaceutically relevant proteins (Baker *et al*., 1997; Abel *et al*., 1986; Scholthof *et al*., 1999; Liu *et al*., 2010).

What has determined the success of using TMV? Early on, the driving interest was economic, in that tobacco was an enormously profitable crop. Therefore, in the early 20th century, determining the cause of infection, detection and subsequent prevention were the impetus. However, the interest in TMV soon extended beyond the practical plant pathology findings. TMV became a source of deep scientific curiosity to understand the physicochemical nature of the virus, which was determined by Wendell Stanley, a co-worker of Holmes and collaborator with Beale (Creager, 2002). Stanley was greatly influenced by enzymologists and thus defined TMV incorrectly as a protein, which was quickly rectified by F. C. Bawden and N. W. Pirie (Bawden, 1956; Harrison and Wilson, 1999), who realized that it was a ribonucleoprotein (RNP). Soon, thereafter, the first electron micrographs made TMV a visible entity (Fig. 2). More exemplary work followed in the second half of the 20th century showing: (i) that the RNA alone was infectious; (ii) that the structure determined by X-ray fibre diffraction resolved the RNA–protein interactions; (iii) that there was a discrete region on the virus for the initiation of encapsidation; (iv) that triplet codons encoded specific amino acids; (v) the definition of the virus sequence and open reading frames (ORFs); and (vi) a biologically active cDNA clone (reviewed in Scholthof *et al*., 1999). This led directly to our understanding of replication and the paradigm-shifting findings that the MP bound to the TMV RNA to form thin threads of RNP that could traffic through plasmodesmata (Citovsky *et al*., 1992; Citovsky and Zambryski, 1993; Scholthof, 2005), leading another generation of scientists to a new understanding of viruses. Similarly, the expression of TMV CP in plants has resulted in the commercial production of transgenic plants for virus cross-protection and the realization that this and variants of such methods are effective for other plant–virus systems. TMV has also been an agent of discovery with the isolation of the host *N*-gene and ongoing investigations of the molecular mechanisms of its actions (Harries *et al*., 2008; Kobayashi *et al*., 2010). More recently, the utility of TMV has been extended, as it has been employed to develop new concepts for computer data storage, to extend our knowledge of the virus structure and carriers of small molecules, and to refine our understanding of the local ecology and fitness of mechanically transmitted viruses (Kendall *et al*., 2007; Sacristan *et al*., 2011; Steinmetz *et al*., 2008; Tseng *et al*., 2006). Moreover, plant biology is benefitting greatly from TMV, which has pointed the way to the elaboration of functional host–virus interactions, including the mechanics of cell-to-cell movement through the plasmodesmata and RNP trafficking from the nucleus to the cytosol (Amari *et al*., 2010; Harries *et al*., 2009; Hofmann *et al*., 2009; Kathiria *et al*., 2010; Komarova *et al*., 2010; Ruggenthaler *et al*., 2009). The future looks bright for our 'favourite' virus.

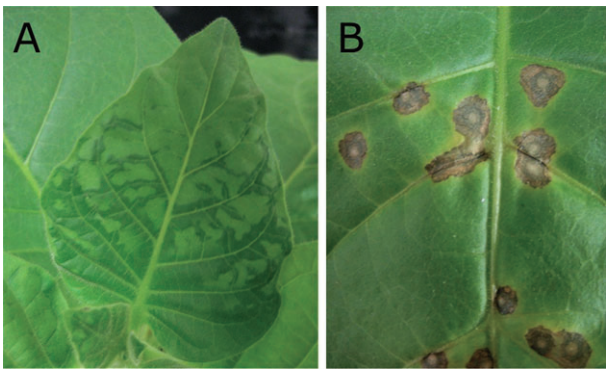

Fig. 1 *Tobacco mosaic virus* (TMV). (A) Systemic infection of *Nicotiana tabacum* cv. Turk plants showing TMV-associated mosaic. (B) Necrotic local lesions on *N. tabacum* cv. Glurk leaf, demonstrating Holmes' *N*-gene resistance following inoculation with TMV.

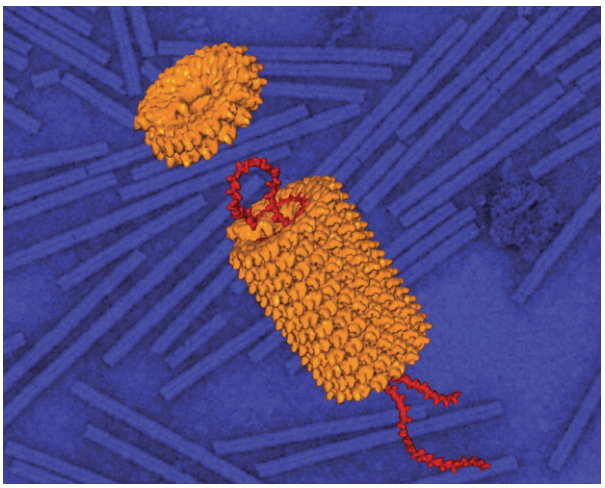

Fig. 2 *Tobacco mosaic virus* (TMV) particles and encapsidation. Foreground: schematic diagram showing TMV protein aggregate binding to the RNA origin-of-assembly loop; additional aggregates then bind to the initial complex, pulling the 5′ end of the RNA up through the hole in the middle of the growing virus particle. Background: negative-stain electron micrograph of TMV virions. (Photomontage courtesy of Amy Kendall and Gerald Stubbs, Vanderbilt University, Nashville, TN, USA.)

2. *TOMATO SPOTTED WILT VIRUS* (TSWV)

The first description of the 'spotted wilt' disease of tomato occurred in 1915 in Australia (Brittlebank, 1915). The disease was later shown to be transmitted by thrips (Pittman, 1927) and caused by a virus, which was named *Tomato spotted wilt virus* (TSWV) (Samuel *et al*., 1930). Although the virus was soon reported in many other countries, the more recent worldwide dispersal of Western flower thrips (*Frankliniella occidentalis*), the major vector of TSWV, led to the re-emergence of TSWV as a major agricultural pest in the 1980s with worldwide losses estimated to be in excess of US$1 billion annually by 1994 (Goldbach and Peters, 1994). The continuing economic importance of TSWV is a result of: (i) its worldwide distribution and wide host range (>800 plant species), including tomato, pepper, lettuce, peanut and chrysanthemum; (ii) the significant crop losses resulting from infection; and (iii) the difficulty in managing the thrip vectors, and hence the virus (reviewed in Adkins, 2000; Pappu *et al*., 2009). TSWV causes variable symptoms, including necrotic/chlorotic rings and flecking on leaves, stems and fruits, with early infections leading to one-sided growth, drooping leaves reminiscent of vascular wilt, stunting or death (Fig. 3). Later infections produce unmarketable fruit with striking chlorotic/necrotic ringspots that often appear only when the fruit reaches full colour (reviewed in Chiemsombat and Adkins, 2006). Novel integrated management strategies have been developed for TSWV because the complex vector–virus relationship and the rapidity of transmission limit the effectiveness of insecticides (reviewed in Funderburk, 2009).

TSWV also garners attention for its fascinating biology that challenges the management of both virus and vector. In the 1980s, it was first observed that TSWV resembled viruses within the family *Bunyaviridae* (Milne and Francki, 1984), a large group of mostly arthropod-transmitted, vertebrate-infecting viruses (Nichol *et al*., 2005). Subsequent molecular studies of TSWV supported the creation of the genus *Tospovirus* (named for TSWV, the type and only original member) within the family *Bunyaviridae* (reviewed in Whitfield *et al*., 2005). Later study and characterization of similar viruses, some of which had been classified previously as TSWV isolates (e.g. de Haan *et al*., 1992; Law and Moyer, 1990), placed ~20 species (accepted and tentative) in the genus *Tospovirus* today. TSWV and more recently described tospoviruses are unique among plant viruses in that virions are enveloped in a host-derived membrane studded with two viral glycoproteins (Fig. 4), and contain one negative-sense and two ambisense single-stranded RNAs encapsidated in multiple copies of the viral nucleocapsid protein. The details of TSWV biology are reviewed elsewhere (e.g. Chiemsombat and Adkins, 2006; Whitfield *et al*., 2005), but two aspects are sufficiently novel to mention: (i) virions contain the viral RNA-dependent RNA polymerase which uses host cell mRNAs to prime viral transcription via cap-snatching (Plotch *et al*., 1981); and (ii) thrips can only transmit TSWV if acquired as larvae, although both larvae and adults are able to transmit (reviewed in Whitfield *et al*., 2005).

TSWV replicates in its thrip vectors (Ullman *et al*., 1993; Wijkamp *et al*., 1993), making thrips both vectors and mobile hosts for the virus, and suggesting that TSWV and other tospoviruses may have evolved from thrip-infecting species to thrip- and plant-infecting species (Goldbach and Peters, 1994). Nearly a century after its first report, and following 30 years of intense molecular study, TSWV remains one of the 10 most economically destructive and scientifically challenging plant viruses.

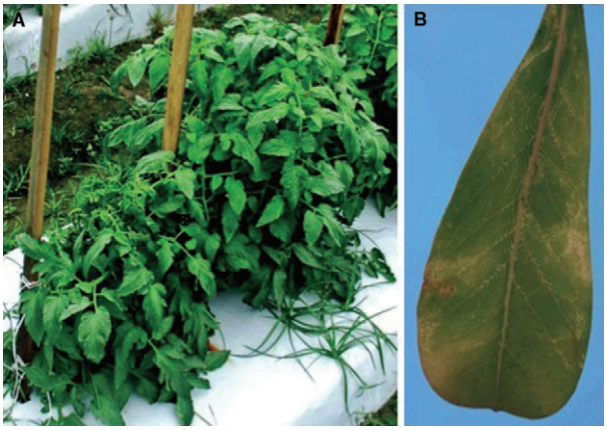

Fig. 3 *Tomato spotted wilt virus* (TSWV) symptoms. (A) Stunted tomato plant (foreground) as a result of TSWV infection at an early stage of growth. Noninfected tomato plant (background) is shown for comparison. (B) Ring/line patterns on desert rose (*Adenium obesum*) leaf from plant infected with TSWV.

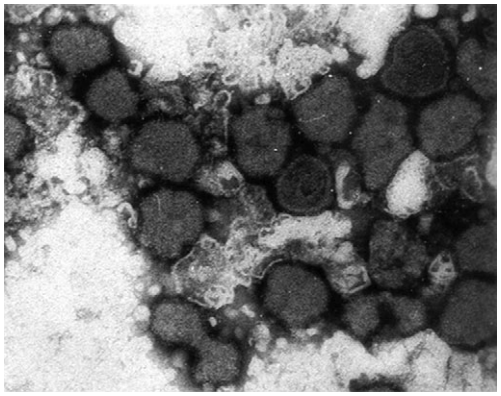

Fig. 4 Transmission electron micrograph of isolated *Tomato spotted wilt virus* (TSWV) virions. Nonfixed virion preparation stained with 1% (w/v) methylamine tungstate.

3. *TOMATO YELLOW LEAF CURL VIRUS* (TYLCV)

Tomato yellow leaf curl virus (TYLCV) causes one of the most devastating emerging diseases of tomato worldwide (Czosnek, 2007). The virus (genus *Begomovirus*, family *Geminiviridae*) is transmitted by the whitefly *Bemisia tabaci*. From the early 1960s, TYLCV has quickly spread from the Eastern Mediterranean Basin to the entire Middle East, Central Asia, North and West Africa, southeastern Europe, the Caribbean islands, southeastern USA, Mexico, the Southern Indian Ocean islands and Japan (Lefeuvre *et al*., 2010). In severely affected regions, crops may be totally lost (Picó *et al*., 1996). The tremendous economic impact of TYLCV and the swift spread of TYLCV disease worldwide have triggered a large body of research tackling many aspects of the viral disease over the last 30 years: molecular biology, plant–virus–vector relationship, epidemiology, disease management and breeding for resistance.

TYLCV was the first begomovirus shown to possess a single genomic component (monopartite), contrary to the begomoviruses known so far, such as ACMV (see virus 7), which has two genomic components (bipartite). Like all geminiviruses, TYLCV has an ~20 nm × 30 nm particle of twinned morphology (Fig. 5A). Its circular single-stranded DNA genome (2787 nucleotides) is enveloped in a capsid consisting of two joined incomplete icosahedra of 22 capsomeres, each containing five units of a 260-amino-acid CP (30.3 kDa). The TYLCV genome (Fig. 5B) comprises two ORFs: V1 encodes CP, and V2 encodes a movement-like protein (MP) with suppressor of RNA silencing properties. The genome complementary sense comprises four ORFs: C1 encodes a replication-associated protein (Rep), C2 a transcriptional activator protein (TrAP), C3 a replication enhancer protein (REn), and C4 a symptom and movement determinant (Díaz-Pendón *et al*., 2010). The viral DNA replicates in the nuclei of infected cells according to a rolling circle mechanism, using its own encoded proteins and the host cell machinery. Sequencing and phylogenetic analyses have shown that TYLCV includes a complex of more than 10 virus species and their strains (Lefeuvre *et al*., 2010). Recombination between viruses/strains may be a major driver of TYLCV diversification (García-Andrés *et al*., 2007).

The rapid spread of the viral disease is caused by whitefly pressure (Fig. 6A) and by high transmission efficacy. A single whitefly is able to inoculate a plant following a 15-min acquisition period and a 15-min inoculation period. In the field, inoculation can occur immediately after transplantation. Infected seedlings will remain stunted and will not yield fruits (Fig. 6B). Apart from whiteflies, TYLCV can be transmitted by grafting, by agroinoculation and by DNA-coated particle bombardment. It is not seed transmitted. The relationships between begomoviruses and whiteflies are complex. TYLCV is transmitted by *B. tabaci* in a circulative manner. TYLCV and some related viruses influence several features of insect pathogens: they affect *B. tabaci* longevity and fertility and are sometimes transovarially transmitted; they affect the whitefly transcriptome, activating the expression of genes related to the whitefly immune response (Luan *et al*., 2011).

TYLCV management is usually attempted by controlling whitefly populations with frequent insecticide sprays. However, chemical control has been a difficult task because of the rapid emergence of resistance to most insecticides (Horowitz *et al*., 2005). Breeding tomatoes resistant to TYLCV started in the mid-1970s and several commercial varieties with adequate resistance have been released (Fig. 6C). Breeding involved introgression of resistance found in accessions of several wild tomato species (e.g. *Solanum chilense*, *S. peruvianum*, *S. pimpinellifolium* and *S. habrochaites*) into the domesticated tomato (*S. lycopersicum*). Several loci tightly linked to TYLCV resistance, coined *Ty-1* to *Ty-5*, have been mapped to the tomato chromosomes (Anbinder *et al*., 2009). A variety of transgenic strategies have also been devised on the basis of the pathogen-derived resistance concept, which involves the expression of functional as well as dysfunctional viral genes (Shepherd *et al*., 2009). RNA-mediated virus resistance based on antisense RNA and post-translational gene silencing is efficient, but highly sequence dependent (Noris *et al*., 2004; Zrachya *et al*., 2007).

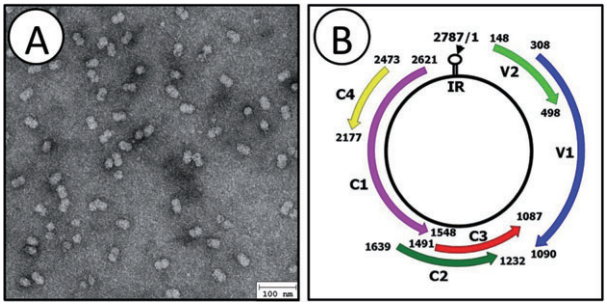

Fig. 5 (A) Geminate *Tomato yellow leaf curl virus* (TYLCV) particles. (B) Genome organization of TYLCV. The single-stranded virion DNA comprises 2787 nucleotides. Open reading frames (ORFs) of virion-sense and complementary-sense strand polarity are designated (V) and (C), respectively. ORFs are represented by arrows; numbers indicate first and last nucleotides of each ORF. The conserved inverted repeat flanking the conserved sequence TAATATT/AC is symbolized by a stem-loop; an arrowhead indicates the cleaving position of replication-associated protein (Rep) in the TAATATT/AC loop; A at the cutting site (/) is nucleotide number one, by definition.

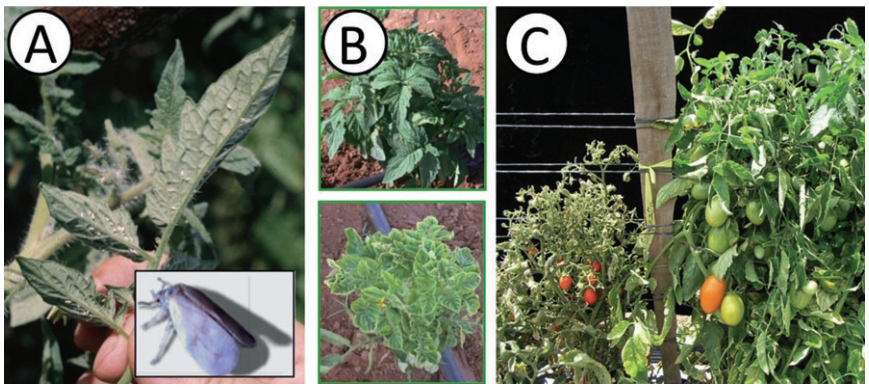

Fig. 6 (A) Numerous whiteflies on a tomato leaf. (B) Top panel, noninfected tomato plant; bottom panel, typical *Tomato yellow leaf curl virus* (TYLCV) disease on a tomato plant. (C) Infected susceptible (left) and resistant (right) tomato lines bred for resistance to begomoviruses.

4. *CUCUMBER MOSAIC VIRUS* (CMV)

Cucumber mosaic virus (CMV) is the type member of the genus *Cucumovirus* in the family *Bromoviridae*. CMV particles are icosahedral in shape and 29 nm in diameter (Fig. 7A), each consisting of 180 subunits of a single CP of ~24 kDa and one of the genomic RNAs. Based on their nucleic acid sequence similarity, CMV strains can be divided broadly into two major subgroups, designated I and II, with subgroup I strains divided into two (A and B) or more additional subgroups. The CMV genome contains five genes, expressed from either the three genomic RNAs or two subgenomic RNAs (Fig. 7B). The 1a and 2a proteins are involved in virus replication, which occurs on tonoplast membranes, whereas the 2b protein is an RNA silencing suppressor, an antagonist of other host defence mechanisms and a viral recombination effector protein. The 3a protein and CP are essential for both cell-to-cell and long-distance movement, processes affected by all of the CMV-encoded proteins. Protein 2b and CP are expressed from subgenomic RNAs, designated RNA 4A and RNA 4, respectively. RNA 4 is packaged together with RNA 3, whereas the packaging arrangements for RNA 4A are not known, except that it is only packaged by subgroup II CMV strains. RNA 5, which is also packaged only by subgroup II strains of CMV, corresponds to the 3′ nontranslated region of RNAs 2 and 3. Its function is not known. (reviewed by Palukaitis and García-Arenal, 2003; Palukaitis *et al*., 1992).

CMV has been studied extensively at the molecular level, with many of the results regarding translation and replication paralleling observations made with BMV. The nature of various CMV–plant interactions is beginning to become clear. CMV also supports satellite RNAs of *c*. 330–390 nucleotides, some of which induce a lethal necrosis in tomato, with a few inducing chlorosis in tobacco, tomato or pepper, but most satellite RNAs attenuate CMV-induced symptoms on most hosts tested. CMV interacts synergistically with potyviruses, tobamoviruses and PVX in solanaceous plants, as well as with potyviruses in cucurbit hosts (reviewed by Palukaitis and García-Arenal, 2003; Palukaitis *et al*., 1992).

The mosaic disease caused by CMV was first described in 1916 and, over the years, this virus has been found to infect many crop species. Prior to the availability of molecular diagnostic techniques and because of differences between strains in the types of symptoms induced and the host range, CMV has often been misidentified as a new virus, leading to at least 43 aliases for CMV (Kaper and Waterworth, 1981). Unlike other members of the family *Bromoviridae*, the strains of CMV have a very broad, collective host range, infecting more than 1200 plant species in over 100 families, including fruit crops, vegetables and ornamentals, both monocots and eudicots. CMV particles are transmitted in a stylet-borne, nonpersistent manner by more than 80 species of aphid in 33 genera, and many symptomless, overwintering weed hosts have been described. Seed transmission of CMV also occurs in many weeds, although with frequencies ranging from <1% to 50% (Palukaitis and García-Arenal, 2003; Palukaitis *et al*., 1992). Together, these factors have contributed to the success of CMV as a pathogen and its effects on crop losses. Although losses in crop yields vary from year to year in different locations and are difficult to quantify, especially when mixed infections are involved, some values for direct effects on crop losses have been reported, e.g. 25%–50% of tomato in China (Tien and Wu, 1991), and 60% of melon and up to 80% of pepper in Spain (Avilla *et al*., 1997; Luis-Arteaga *et al*., 1998). When a necrogenic satellite RNA was present, the recorded losses in Spain and Italy were 80% of tomato plants in 70% of the growing regions, with losses of 100% in some regions (Gallitelli, 2000; Jordá *et al*., 1992). Each year, further hosts of CMV and new diseases are described. Increased aphid activity in northern temperate regions may lead to further epidemics, especially as many control measures are not very effective. CMV is also becoming of increased importance in tropical and subtropical regions, especially where mixed cropping is undertaken. Control of CMV in the field by controlling its aphid vector is not very effective, although resistance genes have been utilized in several instances. However, many of these genes are for tolerance and others can be overcome by different strains of CMV. Pathogen-derived resistance offers the best hope for durable resistance to CMV, but currently this approach is not politically popular (reviewed by Palukaitis and García-Arenal, 2003; Palukaitis *et al*., 1992).

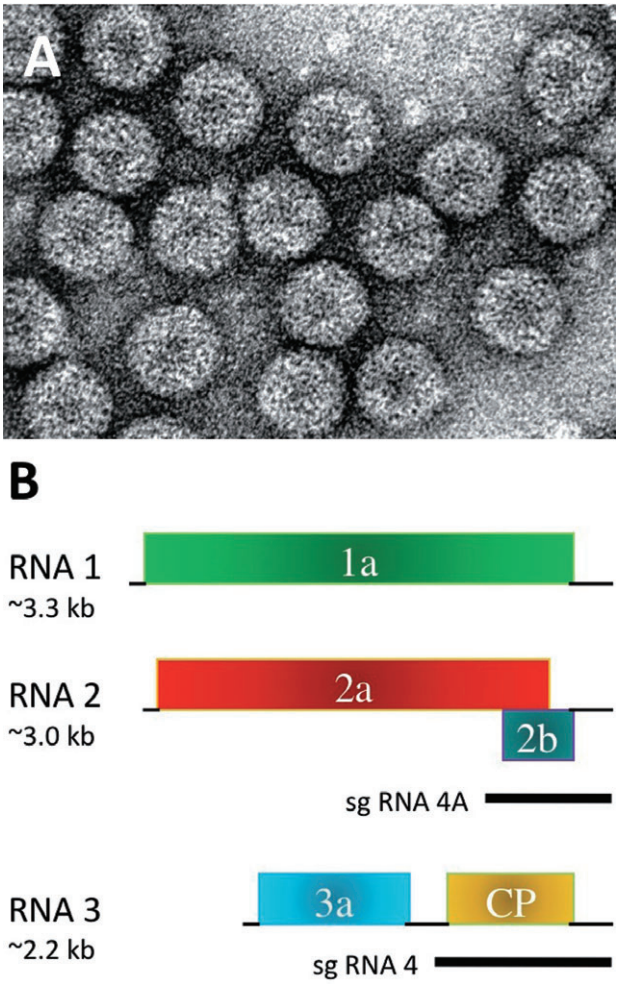

Fig. 7 *Cucumber mosaic virus* (CMV). (A) Negatively stained isometric particles of 29 nm in diameter. (B) Genome organization.

5. *POTATO VIRUS Y* (PVY)

Potato virus Y (PVY, *Potyvirus*; Kerlan and Moury, 2008) possesses a filamentous and flexuous particle (Fig. 8A). The ss(+)RNA genome of approximately 9.7 kb encodes a large ORF and a short ORF (PIPO) embedded within the large ORF (Fig. 8B; Chung *et al*., 2008; Urcuqui-Inchima *et al*., 2001). A viral genome-linked protein (VPg) is covalently attached to the 5′ end of the RNA and a poly(A)$_n$ tail is present at the 3′ end (Shukla *et al*., 1994). PVY is transmitted by more than 40 aphid species (e.g. *Myzus persicae*) in a nonpersistent manner (Radcliffe and Ragsdale, 2002; Sigvald, 1984). Described for the first time in the 1930s (Smith, 1931), it infects a wide host range mainly within the Solanaceae, and is distributed worldwide (Valkonen, 2007). Isolates of PVY species are highly variable at the biological, serological and molecular levels. Thus, groups (e.g. PVY^O and PVY^N; Singh *et al*., 2008) have been proposed according to the symptoms induced during infection (Fig. 8C–F). PVY^O isolates induce mosaic on tobacco and potato, and leaf drop on potato. PVY^N isolates are responsible for the partial/total leaf necrosis of infected hosts. In the 1980s, variants (e.g. PVY^{NTN} able to induce potato tuber necrosis; Beczner *et al*., 1984) were described in potato. PVY has been known for many decades as a threat to seed, ware and processed potatoes (De Bokx and Cuperus, 1987; Loebenstein *et al*., 2001). Potato is the fourth most important food crop in the world, with a yield of 315 million tons in 2006 (http://www.potato2008.org), and a continuous progression (4.5% per year) of the world production of tubers. As a result of a lack of efficient resistance to PVY isolates inducing leaf/tuber necrotic symptoms in cultivated varieties and the plant-to-plant transmission of isolates through daughter tubers, the control strategy used to reduce the incidence of PVY is mainly based on certification of seed production. However, in spite of the latest improvements in detection and molecular characterization methods (Kogovsek *et al*., 2008; Lorenzen *et al*., 2006; Schubert *et al*., 2007; Rolland *et al*., 2008), routinely applied procedures are unable to accurately characterize isolates responsible for tuber necroses. Consequently, there is no efficient means to manage the risks of epidemics caused by emerging necrotic variants. In the current context of a highly competitive international potato market worth several billion Euros, the weaknesses of both our knowledge of the PVY–host interactions involved in the induction of necrosis symptoms and diagnostic tools have led to a situation in which necrotic PVY isolates are still potentially responsible for huge agronomic and economic losses.

PVY is also a destructive virus in tobacco crops, causing height reductions and modifying the chemical composition (e.g. nicotine content; Verrier *et al*., 2001) of cured leaves. Other crop species affected by PVY include pepper, where infection rates of 100% have been observed, and tomato, where emerging PVY strains cause serious damage to yields and fruit quality. Finally, crops with lower economic impacts have also been shown to be strongly affected by PVY [e.g. petunia in Europe (Boonham *et al*., 1999; and synergistically with TMV (Spence *et al*., 2001)].

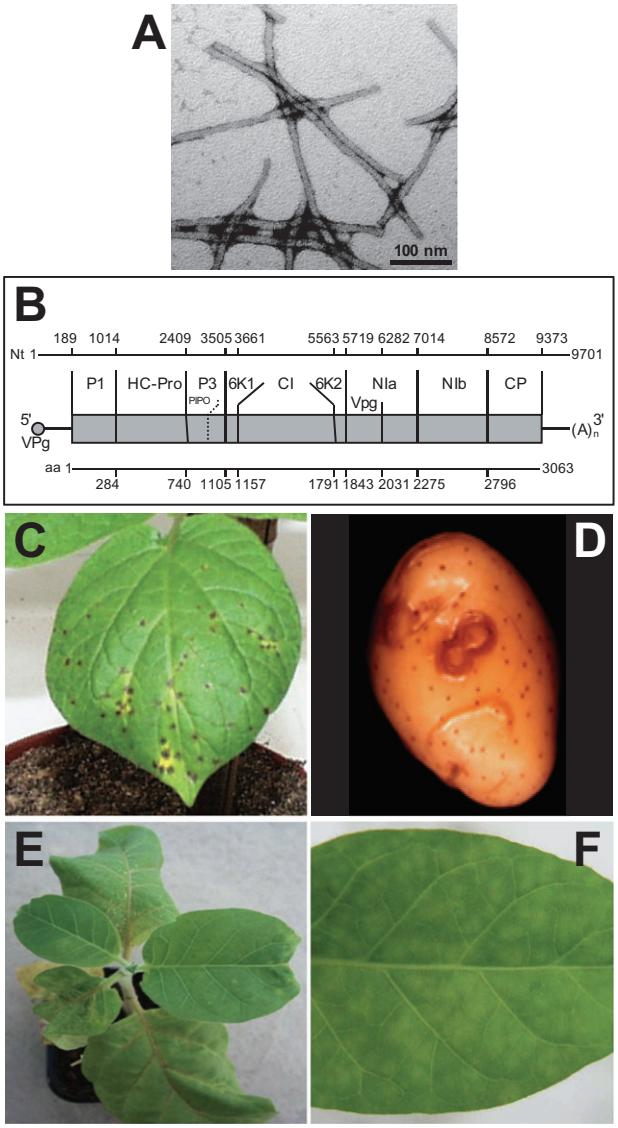

Fig. 8 Electron micrograph of negatively stained, purified, *Potato virus Y* (PVY) particles (A), organization of the PVY genome (B) and symptoms on *Solanum tuberosum* (C, D) and *Nicotiana tabacum* (E, F). The viral RNA in (B) is illustrated by a thin line with the viral genome-linked protein (VPg) (grey circle) and poly-A tail ((A)$_n$) attached at the 5′ and 3′ ends, respectively. The grey box corresponds to the large open reading frame (ORF); the names of the different proteins are listed. The scales [nucleotide (Nt) and amino acid (aa)] are according to isolate PVY^N-605 (Jakab *et al*., 1997; GenBank accession no. X97895). Local lesions (C) and tuber necrosis (D) on *S. tuberosum*. Vein necrosis (E) and mosaic (F) on *N. tabacum*. Copyrights ©: (A) NIB-INRA, M. Tušek (NIB); (C)–(F) INRA, L. Glais.

6. *CAULIFLOWER MOSAIC VIRUS* (CaMV)

For several reasons, CaMV has become a focus of intense interest in plant virology: (i) it was the first plant virus identified as a DNA virus; (ii) it is the first plant virus whose genome has been sequenced; and (iii) it is the first plant virus shown to be replicated by reverse transcription. In addition, this virus is unique in using a particular mode of translation. Further, it uses fascinating strategies to suppress the defence systems implemented by its hosts.

It is remarkable that the beginning of CaMV research started with the discovery of inclusion bodies containing virus-like particles in infected plants (Brierly, 1933; Goldstein, 1927). Subsequently, CaMV particles were isolated from *Brassica* plants containing such inclusion bodies (Rubio-Huertos, 1950; Tomkins, 1937). It was somewhat of a sensation that this virus turned out to contain DNA (Shepherd *et al*., 1970) as, at that time, plant viruses were believed to be RNA viruses in general. However, the incorporation of labelled thymidine into the inclusion bodies (i.e. into DNA) had already been observed previously (Kamei *et al*., 1969).

These discoveries ignited intensive research on CaMV: the sequence of the virus became available as one of the first sequenced genomes (Franck *et al*., 1980), and it was confirmed by cloning the genome in an infectious form (Lebeurier *et al*., 1980). Information from this sequence allowed conclusions to be drawn about the ORFs used by this virus for its life cycle, as well as *cis*-elements of prime importance for viral performance. These included promoters and polyadenylation sites, as well as a site for the binding of the primer-t-RNA. Subsequently, virus-derived 35S, 19S and 8S mRNA species were isolated (Covey and Hull, 1981).

Virion DNA versions were identified as circular double-stranded and sometimes knotted varieties (Ménissier *et al*., 1983), thus pointing to reverse transcription-mediated replication, which ultimately leads to the viral genome containing three single-stranded overlaps, leftovers from reverse transcription Pfeiffer and Hohn, 1983). In nuclei, however, CaMV DNA exists as a minichromosome, i.e. supercoiled circular double-stranded DNA covered with histones (Ménissier *et al*., 1983; Fig. 9).

CaMV is a pararetrovirus, and does not use, as do true retroviruses, genomic integration as part of its life cycle. Viral genomic 35S RNA is terminally redundant (as in retroviruses) as it bypasses the polyadenylation signal on first encounter (Sanfaçon and Hohn, 1990). The promoter is fully active in the absence of any viral factor. In addition, because of its composition of separate tissue-specific enhancer elements, it is active in a tissue-independent manner (Benfey *et al*., 1990). It is this reason, together with its strength and mostly constitutive nature, that makes the 35S promoter a universal tool in transgenesis.

The 35S RNA is not only the template for reverse transcription, but also serves as the main RNA for translation. Unusual for eukaryotic mRNAs, it is polycistronic. Translation is accomplished with the help of the transactivation/viroplasmin protein (TAV) which binds to the translation machinery and prevents the ribosomes from falling off after translation of each individual ORF (Bonneville *et al*., 1989; Park *et al*., 2001). Another unusual translation strategy involves shunting of the highly structured 600-nucleotide-long leader of the 35S RNA by scanning ribosomes (Fütterer *et al*., 1993).

One after the other of the viral proteins was identified, with the exception of ORF VII, which is dispensable. MOV is the MP forming tubular structures across the cell walls through which virus particles are transported (Perbal *et al*., 1993). ITF is the insect transmission factor, mediating the binding of virus particles to the aphid stylet (Uzest *et al*., 2007). The virion-associated protein (VAP) associates loosely with the virion (Hoh *et al*., 2010), binds both MOV and ITF proteins, and is required for their action (Stavolone *et al*., 2005). The viral CP GAG (name borrowed from retrovirus 'group-specific antigen') packages viral DNA into an icosahedral particle which, on entry into a cell, uses a nuclear targeting signal of the GAG protein in order to gain entry into the nucleus (Leclerc *et al*., 1999). Replication is accomplished by the POL polyprotein, which is related to the retrovirus POL (Toh *et al*., 1983) and is cleaved by its own protease (Torruella *et al*., 1989) into reverse transcriptase/RNaseH and protease. The multitasking TAV is translated from the 19S RNA, which is driven by a separate promoter; it is a structural element, forming the inclusion bodies required for virus assembly (Kobayashi and Hohn, 2003), and the mediator of polycistronic translation (Bonneville *et al*., 1989). In addition, it represents an avirulence factor recognized by the plant innate immunity system (Kobayashi and Hohn, 2004). Last, but not least, the TAV protein has been shown to act as a silencing suppressor interfering with the RDR6/DCL4/DRB4 silencing pathway (Haas *et al*., 2008; Shivaprasad *et al*., 2008). CaMV gives rise to massive amounts of small interfering viral RNAs of 21, 22 and 24 nucleotides, the majority stemming from the 8S dsRNA (Moissiard and Voinnet, 2006; Blevins *et al*., 2006). The latter do not restrict viral replication, but may serve as a decoy diverting the silencing machinery from viral promoter and coding regions (Blevins *et al*., 2011).

The analysis of CaMV has not only allowed insights into the principal requirements of 'life', but has also taught us lessons about plant biology in general.

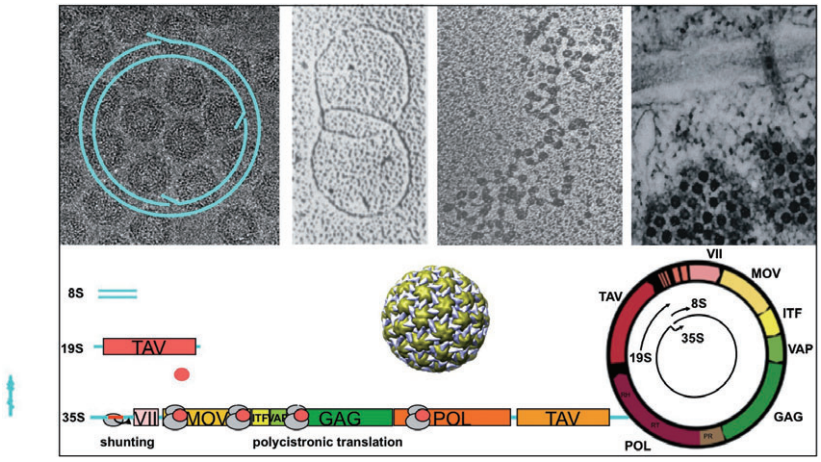

Fig. 9 *Cauliflower mosaic virus* (CaMV). Top row, left to right: virion double-stranded DNA (dsDNA) with single-strand overlaps (background virus particles: diameter, 50 nm); knotted DNA; minichromosome (from Ménissier *et al*., 1983); electron micrograph: section through infected cells showing inclusion body harbouring virus particles and a tubular structure bridging two cells transporting a virus particle. Bottom left: the three types of RNA: 8S, 19S and 35S. The process of shunting and transactivation/viroplasmin protein (TAV)-guided polycistronic translation of the open reading frames (ORFs) is shown. Centre: particle showing the icosahedral arrangement of the GAG (name borrowed from retrovirus 'group-specific antigen') (yellow) and virion-associated (VAP) (blue) proteins (from Hoh *et al*., 2010). Bottom right: map of CaMV showing the ORFs and RNAs. ITF, insect transmission factor.

7. *AFRICAN CASSAVA MOSAIC VIRUS* (ACMV)

African cassava mosaic virus (ACMV), a geminivirus, is the causative agent of cassava mosaic disease. Virus symptoms in the infected host plant vary from mild to very severe and can, in some instances, result in the total devastation of the crop (Fig. 10). Consequently, this disease has become a constraint for cassava cultivation and its exploitation. Initially recorded as cassava latent virus and, subsequently, as African cassava mosaic virus, there are now seven distinct African species and two species prevalent on the Indian subcontinent, all of which are defined primarily by their nucleotide sequence (Fauquet *et al*., 2008). The cassava-infecting geminiviruses display intense recombination events accounting for their rapid molecular diversification, and hence their ability to respond to changes in the environment (Patil and Fauquet, 2009). The recent cassava mosaic disease pandemic affected many countries in East and Central Africa and, by 2005, was responsible for an estimated economic loss of between some US$1.9–2.7 billion (Legg *et al*., 2006). Despite the large-scale cultivation of cassava in many South-East Asian countries and in South and Central America, the cassava-infecting geminiviruses have only been reported from Africa and the Indian subcontinent. The absence of the disease in some of the major cassava-growing regions of the world could be a result of the fact that the polyphagous whitefly, *B. tabaci* biotype B, responsible for the transmission of the virus, is not able to colonize cassava effectively in these parts of the world.

Grown in Africa by farmers, often on marginal land, cassava (*Manihot esculenta*) is vital for both food security and income generation. Although tolerant to drought and productive on poor soils, cassava is propagated vegetatively by stem cuttings and, consequently, the adaptation and introduction of new improved varieties with desired disease and virus resistance properties have been slow. Cassava root, the food source of the crop, is bulky and is readily perishable but, nevertheless, forms the staple food for nearly 80% of the African population (FSN, 2009). Although providing mainly carbohydrates, cassava is deficient in protein. Diets need to be supplemented with other food sources, such as vegetables, legume and cereal grains. The increased growth of cereal crops, adapted for local conditions in many communities, presents a threat to future cassava development. Less research and development has been devoted to cassava than to other staple crops, such as rice, maize and wheat (FAO, 2008). This apparent lack of scientific interest in cassava has contributed to its uneven cultivation and, consequently, has allowed for the progression of cassava mosaic disease to the detriment of large human populations.

ACMV was one of the first geminiviruses to be molecularly characterized. The analyses revealed the presence of two similarly sized single-stranded DNA molecules, each containing an identical nucleotide sequence of approximately 200 nucleotides, or common region, from which divergent viral transcription occurs (Fig. 11). The common region also possesses the sequences necessary for the initiation and termination of rolling circle replication, the mechanism by which these viruses replicate (Hanley-Bowdoin *et al*., 1999). Consequently, both DNAs are required for infectivity and thereby satisfy Koch's postulate. In subsequent years, following much research in many laboratories, the functions of its many ORFs have been identified. DNA-A possesses the genes necessary for virus replication and encapsidation of viral nucleic acid. The second DNA, DNA-B, encodes the functions required for the movement of the viral genome from the nucleus, the location of viral replication, into the cytoplasm and, subsequently, to noninfected cells to propagate the infection (Hull, 2002). ACMV research is now concerned with what factors govern the host range of the virus, resulting in its differing and varied symptomatology, virus gene regulation and transcription, the functionality of the geminivirus promoter region and an understanding of what role, if any, RNA interference may play in these activities.

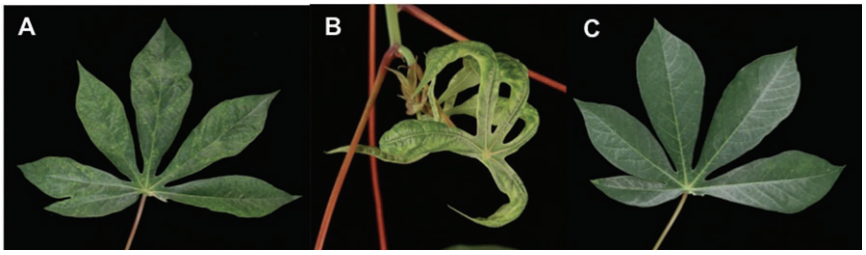

Fig. 10 Varied symptoms of disease caused by *African cassava mosaic virus* (ACMV) in cassava: (A) mild; (B) severe; (C) noninfected.

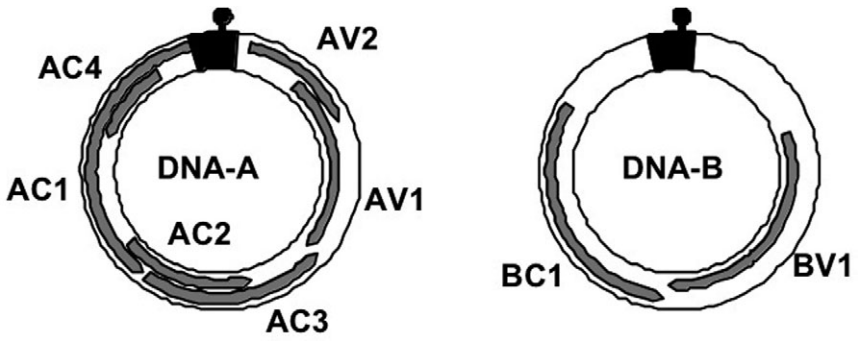

Fig. 11 Genomic components of *African cassava mosaic virus* (ACMV). DNA-A encodes six open reading frames: AV1, coat protein; AV2, precoat protein; AC1, replication-associated protein; AC2, transcriptional activator protein; AC3, replication enhancer protein; AC4, silencing suppressor protein. DNA-B encodes two genes required for plant virus movement: BC1, movement protein (cell to cell); BV1, nuclear shuttle protein. C, complementary-sense open reading frames (ORFs); V, virus-sense ORFs. Black box, location of the nucleotide sequence common to both genomic components. Light grey boxes designate ORFs.

8. *PLUM POX VIRUS* (PPV)

Plum pox potyvirus (PPV) causes Sharka, the most important viral disease of stone fruit crops (Fig. 12) (Cambra *et al*., 2006; Garcia and Cambra, 2007). Five factors contribute to this situation, the first three shared with many potyviruses: (i) efficient transmission by numerous aphid species, leading to rapid epidemic spread that is difficult to control; (ii) symptom severity, which may result in 100% production losses in the most susceptible varieties; (iii) vegetatively propagated hosts providing for efficient dissemination over local and global scales; (iv) a general susceptibility of hosts, with very few resistance sources identified, which has largely frustrated international resistance breeding efforts (Dicenta *et al*., 2000); and (v) a quarantine or regulated status in most producing regions, with ensuing high surveillance and eradication costs, but a limited effectiveness at preventing PPV entry into unaffected regions. Although PPV was limited to Europe for most of the 20th century, the past 20 years have seen its discovery in Africa, South and North America, and Asia (Candresse and Cambra, 2006), so that almost all major production areas are now affected to varying degrees. Costly eradication or control efforts exist in many countries. Although successful in a few cases, these efforts have generally slowed the progression of PPV, but not stopped it. The combined costs of Sharka disease and control efforts have been evaluated at US$10 billion over the past 30 years worldwide (Cambra *et al*., 2006).

This economic impact explains why PPV is among the best studied potyviruses, despite the technical constraints imposed by the woody nature of its natural hosts. Over the years, PPV has been one of the plant viruses for which novel detection techniques have first become available, including enzyme-linked immunosorbent analysis (ELISA), polymerase chain reaction (PCR) and more novel techniques (Pasquini *et al*., 2008; Schneider *et al*., 2004; Varga and James, 2006; Voller *et al*., 1976; Wetzel *et al*., 1991). PPV variability has also been studied extensively. A large effort of the EU-funded SharCo consortium (http://www.sharco.eu/sharco/) recently generated sequence information for over 800 field isolates, possibly making PPV the potyvirus for which information is available for the largest number of isolates.

PPV is also among the best studied potyviruses for plant–virus interactions, and is one of the few for which an Arabidopsis-based pathosystem is available (Fig. 13) (Decroocq *et al*., 2006; Sicard *et al*., 2008), making it very attractive for the genetic identification of host susceptibility genes. PPV has also been at the forefront of the study of two potyviral proteins, the CI helicase (Fernández *et al*., 1995, 1997; Gómez de Cedrón *et al*., 2006; Jiménez *et al*., 2006) and the CP, in particular for the analysis of its glycosylation by the host (Chen *et al*., 2005; Fernández-Fernández *et al*., 2002; de Jesús Pérez *et al*., 2006; Kim *et al*., 2011).

Efforts towards the development of PPV-resistant transgenics have also been particularly active, culminating with the validation through field trials of the resistance of the HoneySweet transgenic plum (Capote *et al*., 2008; Hily *et al*., 2004; Scorza and Ravelonandro, 2006) and its deregulation in the USA, making it one of the few virus-resistant transgenic crops developed to marketability.

The fight against PPV is ongoing and the interest in PPV will not diminish in the coming years. Efforts to better understand and model its epidemic spread to improve eradication or control efforts, on the one hand, and to understand its interactions with its hosts in order to develop PPV-resistant *Prunus* through a translational research effort, on the other, are likely to be key future themes.

Fig. 12 Severe symptoms of *Plum pox virus* (PPV) infection on plums of the Pozegaca type.

Fig. 13 Symptoms of *Plum pox virus* (PPV) infection in *Arabidopsis thaliana* (ecotype Ler). The plant on the left is a noninfected control.

9. *BROME MOSAIC VIRUS* (BMV)

Brome mosaic virus (BMV) is a positive-strand RNA virus that primarily infects grasses, including cereals. BMV virions (Fig. 14) consist of approximately 30-nm, nonenveloped, T = 3 capsids surrounding three separately encapsidated genomic RNAs (Fig. 15). RNA1 and RNA2 encode the two BMV RNA replication factors, the highly multifunctional, membrane-targeting 1a methyltransferase/helicase protein and the 2a polymerase. RNA3 encodes the viral MP and, via subgenomic RNA4, the CP.

Although rarely causing significant crop losses, BMV is very successful in nature, being distributed throughout much of the world. BMV has also been successful in the laboratory. Its high yield, genetic and biochemical tractability and other features have long attracted diverse researchers whose contributions have made BMV a broadly useful model for viral gene expression, RNA replication, RNA recombination, encapsidation, virus–host interactions and other processes too numerous to discuss fully here. Many BMV results have proved to be relevant across and beyond plant viruses and, in some cases, beyond positive-strand RNA viruses.

Among other findings, BMV RNAs were the subject of important early translation studies (Shih and Kaesberg, 1973). These included the first definition of a eukaryotic ribosome binding site, revealing the linkage of eukaryotic translation initiation to mRNA 5′ ends (Dasgupta *et al*., 1975). BMV also produced the first eukaryotic viral RNA-dependent RNA polymerase extract with marked specificity for its cognate viral RNAs (Hardy *et al*., 1979), facilitating many studies of RNA synthesis. Early sequencing of the BMV, TMV and Sindbis virus genomes revealed unexpected conservation of multiple domains in their RNA replication proteins, establishing the concept of viral superfamilies that spanned diverse host kingdoms and virion morphologies (Haseloff *et al*., 1984).

BMV was the first plant RNA virus for which designed infectious transcripts were engineered from cloned viral cDNA (Ahlquist *et al*., 1984). This allowed recombinant DNA manipulation of the viral genome for mechanistic studies and other goals, and has since been used *in vitro* and *in vivo* for reverse genetics of many other RNA viruses. Complementing these abilities, later demonstrations that BMV proteins direct virus-specific RNA replication, transcription, encapsidation and recombination in the genetic model yeast *Saccharomyces cerevisiae* have facilitated the identification and study of viral and host functions in many infection processes (Janda and Ahlquist, 1993; Krol *et al*., 1999; Kushner *et al*., 2003). Yeast and plant studies have shown, for example, that BMV induces new membrane-bounded mini-organelles for RNA replication. Multiple features of the structure, assembly and function of these replication complexes (Schwartz *et al*., 2002) and the tRNA-like 3′ ends of BMV genomic RNAs (Shih *et al*., 1974; Weiner and Maizels, 1987) suggest that positive-strand RNA viruses, retroviruses and dsRNA viruses arose from a common ancestor (Ahlquist, 2006).

Other notable BMV studies include, but are not limited to, the first demonstration of plant viral RNA recombination (Bujarski and Kaesberg, 1986) and detailed analyses of promoter function (Kao, 2002), encapsidation (Rao, 2006), nanotechnology applications (Chen *et al*., 2006) and infection movement and host specificity (Kaido *et al*., 2007). BMV was also the first RNA virus engineered to express a foreign gene and continues to be used to direct gene expression, silencing, etc. (Ding *et al*., 2006; French *et al*., 1986; Mori *et al*., 2001). Through such established and new approaches, BMV will continue to advance our understanding, control and practical applications of viruses and virus–host interactions.

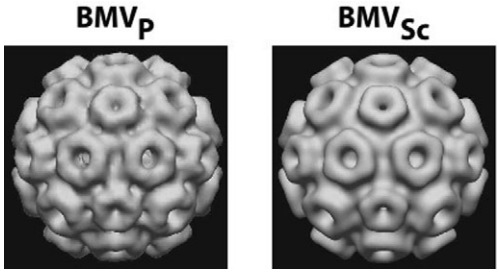

Fig. 14 Cryo-electron microscope three-dimensional image reconstructions of *Brome mosaic virus* (BMV) virions from BMV-infected barley plants (BMV$_P$, left panel) and from *Saccharomyces cerevisiae* yeast cells expressing BMV coat protein and BMV genomic RNA2 as an encapsidation substrate (BMV$_{Sc}$, right panel). From Krol *et al*. (1999).

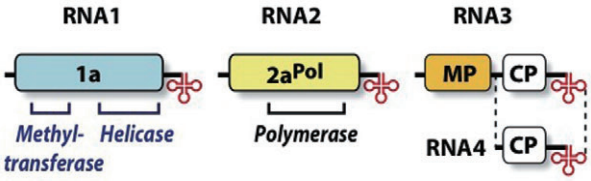

Fig. 15 Schematic diagram of the *Brome mosaic virus* (BMV) genome, showing genomic RNA1 (3.2 kb), RNA2 (2.9 kb) and RNA3 (2.1 kb), plus subgenomic RNA4. The shaded boxes indicate the open reading frames for RNA replication proteins 1a and 2a^{Pol}, the movement protein (MP) and coat protein (CP). Brackets indicate the conserved RNA capping methyltransferase and NTPase/helicase domains in 1a and the RNA polymerase domain in 2a^{Pol}. The red cloverleaf structures represent the aminoacylatable tRNA-like regions at the 3′ ends of RNAs 1–4.

10. *POTATO VIRUS X* (PVX)

Potato virus X (PVX), the type member of the genus *Potexvirus* in the family *Flexiviridae*, was described in 1931 as the 'X virus of potato' (Adams *et al*., 2004; Smith, 1931). Although the significance of PVX infections to crop yields has been mitigated through seed certification programmes (Jones *et al*., 1981), studies on PVX structure, replication and spread have advanced our understanding of viral gene expression, virus–host interactions, gene silencing and the utilization of PVX-based vectors for diverse agricultural and biomedical applications.

The flexuous nature of the rod-shaped virion of PVX (Fig. 16) and the *Flexiviridae* has been a challenge for the determination of the structure of this class of viruses. Nevertheless, biochemical and biophysical approaches have provided models indicating that the ~515 nm × 13 nm particle is composed of a single-stranded genomic RNA of ~6400 nucleotides, encapsidated by ~1270 identical 25-kDa CP subunits in a helical arrangement (Kendall *et al*., 2008). Assembly and disassembly studies have also been important for the reconstitution of particles *in vitro* and for providing insights into particle remodelling for translation (Atabekov *et al*., 2007; Goodman *et al*., 1976). The substantial surface area of the rod-shaped particle and the location of the CP N-terminus at the surface have enabled the development of PVX-based biocatalysis, nanoparticle delivery and epitope display (Baratova *et al*., 1992; Carette *et al*., 2007; Cruz *et al*., 1996; Grasso and Luca, 2010; Steinmetz *et al*., 2010).

Analyses of PVX genome expression have defined *cis*- and *trans*-acting functions for potexvirus replication (Batten *et al*., 2003; Verchot-Lubicz *et al*., 2007, 2010). The capped and polyadenylated genomic RNA (Fig. 17) encodes replicase for viral RNA synthesis, triple gene block (TGB) proteins for virus cell-to-cell movement and CP that functions in assembly, cell-to-cell movement and as an elicitor for Rx-mediated PVX resistance. Replicase translated from the genome synthesizes minus- and plus-strand copies of the viral RNA and subgenomic RNAs that are templates for translation of the TGB proteins and CP. A unique feature of PVX replication is that, in addition to requirements for localized *cis*-acting elements and structures on the viral RNA (Kim and Hemenway, 1999; Miller *et al*., 1998; Pillai-Nair *et al*., 2003), long-distance interactions between terminal sequences and complementary, conserved internal regulatory elements that span distances up to ~4400 nucleotides are essential for all RNA synthesis (Hu *et al*., 2007).

Functional studies of PVX TGB proteins and their interactions with CP, replicase, viral RNA, host endomembranes, actin cytoskeleton and plasmodesmata indicate a complex interplay of virus–host interactions during virus movement (Verchot-Lubicz *et al*., 2010). The multifunctional PVX TGBp1 regulates the plasmodesmata size exclusion limit and traffics virions, CP/RNA RNP or single-tailed particles across the plasmodesmata; this process is facilitated by TGBp2 and TGBp3 (Angell *et al*., 1996; Karpova *et al*., 2006; Lough *et al*., 1998; Lough *et al*., 2000; Santa Cruz *et al*., 1998; Verchot-Lubicz *et al*., 2010). TGBp1-mediated suppression of silencing is also required for movement (Bayne *et al*., 2005; Voinnet *et al*., 2000), and the ability of TGBp1 to remodel virions and promote translation may be important during and after transport (Atabekov *et al*., 2000).

The development of PVX-based vectors for expression in transgenic plants was central to the discovery of RNA silencing (Baulcombe, 1996a), suppression of silencing (Anandalakshmi *et al*., 1998; Marathe *et al*., 2000) and mechanisms of pathogen-derived resistance in transgenic plants (Baulcombe, 1996b). The PVX amplicon and amplicon-plus vectors (Angell and Baulcombe, 1997; Mallory *et al*., 2002), together with various iterations of PVX-based vectors, have been used extensively for expression/silencing studies in diverse systems and have been proven to be valuable platforms for molecular farming (Canizares *et al*., 2005; Fischer *et al*., 2004).

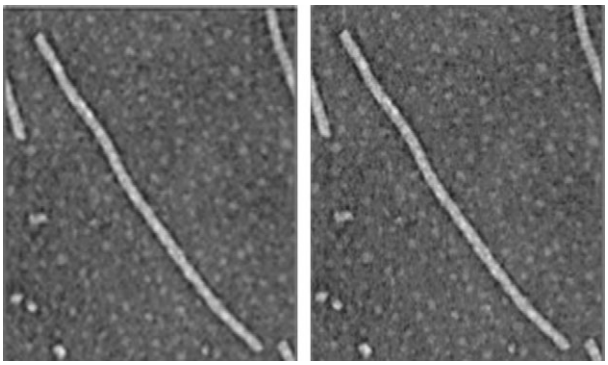

Fig. 16 Electron micrograph of the *Potato virus X* (PVX) particle.

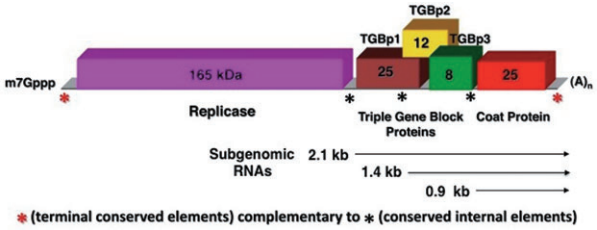

Fig. 17 *Potato virus X* (PVX) genome. Interacting *cis*-acting elements near the termini of viral RNA and complementary internal conserved elements are marked with red and black asterisks, respectively.

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