Helical capsids of plant viruses: architecture with structural lability

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Capsids of numerous filamentous and rod-shaped plant viruses possess helical symmetry. In positive-stranded RNA viruses, helical capsids are typically composed of many identical subunits of the viral capsid protein (CP), encapsidating a molecule of viral genomic RNA. Current progress in structural studies of helical plant viruses has revealed differences between filamentous and rod-shaped viruses, both in structural folds of their CPs and in the interactions of CP molecules in their capsids. Many filamentous and rod-shaped viruses have functionally similar lateral inter-subunit contacts on the outer virion surface. Additionally, the extreme N-terminal CP region in filamentous viruses is intrinsically disordered. Taken together, the available data establish a link between the structural features of molecular interactions of CP molecules and the physical properties of helical virions ranging from rigidity to flexibility. Overall, the structure of helical plant viruses is significantly more labile than previously thought, often allowing structural transitions, remodelling and the existence of alternative structural forms of virions. These properties of virions are believed to be functionally significant at certain stages of the viral life cycle, such as during translational activation and cell-to-cell transport. In this review, we discuss structural and functional features of filamentous and rod-shaped virions, highlight their shared features and differences, and lay emphasis on the relationships between the molecular structure of viral capsids and their properties including virion shape, lability and capability of structural remodelling.

For instance, helical structures can be advantageous in terms of the size of packed RNA genomes. The size of RNA packed into icosahedral capsids is limited by their internal volume. In known icosahedral plant viruses, the largest RNAs packed in individual capsids are found in viruses of the family Tymoviridae and the genus Nepovirus. These viral RNAs are approximately 7.5 kb in length, which is close to the maximum packaging capacity of icosahedral capsids with \( T(P)=3 \), the highest triangulation number known for icosahedral capsids of plant viruses. In contrast, the helical organization of the capsid imposes no limits on the size of packed RNA genome, and the size of helical capsids is determined by the length of encapsidated RNA. Accordingly, helical capsids of plant viruses can contain longer RNAs than icosahedral capsids. For example, numerous viruses of the family Potyviridae, constituting about 25% of known plant viruses, have genomes of 8.5–12 kb in size, while the length of genomic RNA in viruses of the family Closteroviridae can be as big as 19.3 kb (Dolja et al., 2003).

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Introduction

Viral capsids – proteinaceous shells containing viral genomes – are generally symmetrical assemblies built according to either icosahedral or helical symmetry (Harrison, 2007). Three structurally dissimilar types of viral helical capsids have been studied so far: capsids of filamentous bacteriophages resembling, in structure and biogenesis, the cal capsids have been studied so far: capsids of filamentous son, 2007). Three structurally dissimilar types of viral heli-cal capsids among plant viruses can be explained by possible functional benefits provided by this type of capsid structure. These properties of virions are believed to be functionally significant at certain stages of the viral life cycle, such as during translational activation and cell-to-cell transport. In this review, we discuss structural and functional features of filamentous and rod-shaped virions, highlight their shared features and differences, and lay emphasis on the relationships between the molecular structure of viral capsids and their properties including virion shape, lability and capability of structural remodelling.

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**Tobacco mosaic virus** (TMV), 2130 molecules of viral CP encapsidate a single genomic RNA of 6395 nucleotides to give a rod-like virion 300 nm in length and 180 Å in diameter (Stubbs, 1999; Stubbs & Kendall, 2012). Helical capsids typically have a central channel disposed along the virion axis, and in TMV it has a diameter of about 40 Å. In the TMV virion, the CP molecules form a right-handed helix and each protein subunit binds to three nucleotides of the single-stranded viral RNA, and thus the RNA strand follows the protein helix at a low virion radius of about 40 Å (Stubbs & Kendall, 2012).

Two structural classes of helical plant viruses are currently distinguished: rod-shaped and flexuous filamentous viruses. In addition to the virion shape, these two classes of viruses differ in their capsid diameter, which ranges from 100 to 130 Å for filamentous virions and 180 to 220 Å for rod-shaped virions. Rod-shaped virions are found for viruses of the family Virgaviridae and the unassigned genus *Benyvirus*, while filamentous virions are seen for viruses of the families Alphaflexiviridae, Betaflexiviridae, Closteroviridae and Potyviridae. In terms of the number of described virus species, filamentous viruses are noticeably more abundant in nature than rod-shaped viruses (King et al., 2012), and this could reflect their higher evolutionary fitness in flowering plants currently dominating the biosphere.

In this review, we discuss structural and functional features of filamentous and rod-shaped virions, highlight their shared features and differences, and place special emphasis on recently revealed relationships between the molecular structure of viral capsids and their properties, including virion shape, lability and capability of structural remodelling.

### Progress in structural studies of helical viruses

Rod-shaped TMV remained the only plant virus with a resolved capsid structure for a long time (Holmes et al., 1975). X-ray crystallographic analysis of '20S disks' comprising 34 subunits of the TMV CP and serving as an intermediate in virus assembly, along with X-ray fibre diffraction studies of TMV virions, led to determination of the TMV structure at a 4 Å resolution (Bloomer et al., 1978; Stubbs & Warren, 1977). Further progress in TMV structural studies was made considerably later, by X-ray fibre diffraction analysis (resolution of 2.9 Å) and cryoelectron microscopy (4.6 Å) (Clare et al., 2010; Namba et al., 1989). At present, high-resolution structures are also available for several other rod-shaped viruses closely related to TMV, such as *Cucumber green mottle mosaic virus* (CGMMV), *Tobacco mild green mosaic virus*, *Odontoglossum ringspot virus* (ORSV) and *RiRgrass mosaic virus* (RMV) (Lobert et al., 1987; Pattanayek & Stubbs, 1992; Planchart, 1995; Wang et al., 1997), which are assigned (together with TMV) to the genus *Tobamovirus*. These viruses appeared to be very similar to TMV in their structure. Therefore, until recently, high-resolution data were obtained only for structurally similar rod-shaped tobamoviruses.

Filamentous viruses, due to their flexuous virions, are difficult to analyse with the methods successfully applied to TMV and related viruses. For example, attempts to resolve the atomic structure of *Potato virus X* (PVX, genus *Potexvirus*) by X-ray fibre diffraction, while revealing some features of PVX virions, generally failed (Parker et al., 2002). Cryoelectron microscopy of PVX confirmed the previously determined helix characteristics and provided information on structural features of the virion surface, but could generate the structure only at a relatively low resolution of 14 Å (Kendall et al., 2008). In view of these technical issues, alternative indirect methods were used. Among those, 'tritium planigraphy' was employed to identify the CP regions exposed on the surface of PVX virions (Baratova et al., 1992, 2004). These data, combined with the data obtained with other indirect approaches, were used to develop a model for the PVX CP structural fold (Nemykh et al., 2008). Experimental data on the structure of potexvirus CP subunits and virions have been recently published. In 2012, X-ray analysis of the CP expressed in bacteria along with cryoelectron microscopy of virions revealed the *Papaya mosaic virus* (PapMV) CP tertiary structure, which appeared to be distant from known folds of other plant viruses (Yang et al., 2012). In 2015, the structure of two other potexviruses was resolved at high resolution by cryoelectron microscopy, which shed new light on the details of inter-subunit interactions in filamentous virions (Agirrezabala et al., 2015; DiMaio et al., 2015). Additionally, the structure of rod-shaped *Barley stripe mosaic virus* (BSMV, genus * Hordeiviruses*) has recently been obtained at near-atomic resolution, showing striking differences from TMV in inter-subunit contacts (Clare et al., 2015).

### Strong inter-subunit interactions in tobamovirus virions

Interactions between protein subunits in viral capsids and, in particular, helical capsids of plant viruses, involve electrostatic interactions, hydrophobic contacts and hydrogen bonds (Bloomer et al., 1978; Champness et al., 1976; Clare et al., 2010; Namba et al., 1989; Stubbs et al., 1977). In the TMV CP, most of the charged amino acid residues are grouped into four clusters involved in electrostatic interactions within and between CP molecules in the capsid (Namba et al., 1989). One charged cluster located at a low virion radius forms the main lateral inter-subunit contact. It includes the calcium-binding site formed by Glu95 from one CP subunit and Glu106 from its lateral neighbour, and two inter-subunit salt bridges, Arg113–Asp115 and Asp88–Arg122 (Fig. 1a). This region also includes sites for binding of RNA phosphate groups (Namba et al., 1989). Another charged cluster, which is located at a medium radius in the axial inter-subunit interface, includes a potential metal-binding site formed by Asp77 in one CP subunit and Glu50...
from a subunit in the underlying helix turn. Additionally, Glu50 is stabilized by Arg134 from the subunit diagonally opposite to it (Fig. 1a). Two other clusters of charged residues in the TMV virion are both located entirely within one CP subunit and do not contribute to the interaction between CP molecules (Namba et al., 1989).

Carboxylate interactions, which involve pairs of residues Glu95–Glu106 and Glu50–Asp77, take part in inter-subunit interactions and play an important role in assembly and disassembly of TMV virions (Caspar & Namba, 1990). These interactions are not found in the two-layer 20S disk serving as an intermediate in the assembly of...
tobamoviruses, and are believed to be formed during virion assembly, upon the transition of the 20S disk consisting of two flat rings of CP molecules into a 'lock washer' structure with a helical arrangement of CP subunits (Bloomer et al., 1978). Under virion-stabilizing conditions, negatively charged carboxylate groups bind calcium ions that can compensate their mutual electrostatic repulsion (Lu et al., 1996). On the other hand, the loss of calcium ions under other conditions results in the repulsion of interacting carboxylate groups, diminished inter-subunit contacts and destabilization of the virion. The role of carboxylate interactions in virion disassembly is confirmed by mutagenesis experiments, in which replacements of Glu and Asp residues in the interacting pairs by Gln and Asn, respectively, considerably inhibited the disassembly of TMV virions, with the most pronounced effect being observed for Glu50 involved in axial contacts (Lu et al., 1996). Therefore, the carboxylate interactions are thought to serve as a virion structural element involved in a structural transition required for virion translational activation and co-translational disassembly, as described below.

The positions of residues involved in carboxylate interactions differ considerably in tobamoviruses with available high-resolution structures, namely TMV, TMV strain U2, CGMMV, RMV and ORSV. Carboxylate interactions at a high virion radius are found for all these viruses except RMV; however, in TMV, TMV-U2 and ORSV this interaction involves the Asp50 and Asp77 residues, while in CGMMV it involves the Glu46 and Asp126 residues. Among these closely related viruses, the carboxylate interactions at a low virion radius can vary to a greater extent and be more complex. In particular, the lateral carboxylate interactions in RMV involve the Glu95, Glu98, Glu99 and Glu106 residues, while in CGMMV they involve the Glu95 and Asp98 residues (Wang et al., 1998). Therefore, the carboxylate interactions, due to their importance for virion assembly and disassembly, are generally preserved among tobamoviruses; however, their exact positions can vary. It has been suggested that the sites of electrostatic contacts could change their location within subunit interfaces in the evolution of CP (Wang et al., 1998). Presumably, the ability of TMV CP assemblies for the transition between CP disks and helical ‘lock washer’ structures, which depends on carboxylate interactions, dictates the mode of tobamovirus virion assembly and, to a great extent, determines the structure of these viruses.

Besides the strong electrostatic interactions making a major contribution to the inter-subunit interactions, the TMV capsid is stabilised by a so-called ‘hydrophobic girdle’ formed by lateral hydrophobic contacts between neighbouring subunits at a high virion radius (Bloomer et al., 1978) (Fig. 1a). Therefore, the hydrophobic girdle represents a continuous array of contacts that follows the protein helix in the virion. Contacts within the hydrophobic girdle involve aromatic amino acid residues, proline residues and hydrophobic side chains (Bloomer et al., 1978; Namba et al., 1989). The hydrophobic girdle contributes to virion stability, and isolates the CP regions at a lower radius from the outside environment (Bloomer et al., 1978).

Thus, the inter-subunit contacts in tobamovirus virions include strong electrostatic interactions, both lateral and axial, and the hydrophobic girdle. These interactions are believed to determine the stability of tobamovirus virions, which is exceptionally high among helical plant viruses (Makarov et al., 2013).

### Flexible inter-subunit contacts in potexviruses

Inter-subunit contacts in filamentous viruses, so far studied in detail only for potexviruses, drastically differ from those found in tobamoviruses. Unlike tobamovirus capsids, which are largely stabilized by multiple electrostatic interactions involving residues in the CP core domain, the formation of potexvirus capsids depends mostly on hydrophobic interactions involving the CP N- and C-terminal regions.

The lateral inter-subunit contact is made by the CP N-terminal region, a part of which represents an ‘interacting arm’ (the IA domain) extending to a neighbouring subunit and interacting with a hydrophobic groove, or pocket, formed by the neighbouring subunit on the outer virion surface (Agirrezabala et al., 2015; DiMaio et al., 2015; Yang et al., 2012) (Protein Data Bank accession numbers 4DOX, 5AZT and 5FN1) (Fig. 2a, b). Importantly, the IA domain is connected to the CP core domain by a flexible hinge. The interaction of the IA domain with the hydrophobic pocket appeared to be similar in three studied potexviruses. For example, in PapMV the IA domain residues involved in inter-subunit interactions include Ala12, Phe13, Ile16, Met21, Ile24 and Val26, with the Phe13 residue being buried in the hydrophobic pocket formed by Leu39, Val42, Met46, Val56, Ala60, Phe106, Tyr109 and Phe110 (Yang et al., 2012). In *Bamboo mosaic virus* (BaMV) virions, the IA domain forms a short α-helix located in the hydrophobic groove of the neighbouring subunit; additionally, Trp41 of one protein subunit forms a strong stacking interaction with Trp68 of another CP molecule (DiMaio et al., 2015). The IA domain of *Pepino mosaic virus* (PepMV) comprises Phe28, functionally analogous to PapMV Phe13 and BaMV Trp41 (Agirrezabala et al., 2015). The Phe28 residue is crucial for PapMV capsid assembly, since a point mutation affecting this residue results in a virus unable to form stable virions (Agirrezabala et al., 2015). Similarly, a deletion affecting the IA domain in PapMV CP, as well as a mutation in PapMV CP Phe13, completely abolishes homologous protein–protein interactions (Lecours et al., 2006). Therefore, the lateral inter-subunit interaction involving the IA domain is supposed to provide the mechanism of CP polymerization resulting in the formation of filamentous plant virus virions (Yang et al., 2012).

The C-terminal region of potexvirus CP is involved in axial inter-subunit contacts (Agirrezabala et al., 2015; DiMaio et al., 2015; Yang et al., 2012). The N-terminal region of potexvirus CP is involved in lateral inter-subunit contacts (Agirrezabala et al., 2015; DiMaio et al., 2015; Yang et al., 2012).
et al., 2015). The C-terminus is located on the inner virion surface and represents a long coil making multiple relatively weak contacts. In the case of BaMV, the polypeptide chain of C-terminal region goes along the axial channel and contacts CP subunits in the protein layer located closer to the encapsidated RNA 5′-terminus, turns around, and its extreme C-terminal residues contact a CP subunit in the layer closer to the RNA 3′-terminus (DiMaio et al., 2015) (Fig. 2b, c). Therefore, in potexvirus virions, lateral inter-subunit contacts on the outer virion surface and axial contacts on the inner surface lining the axial channel are formed independently by the N- and C-terminal CP regions, respectively.

It is notable that the absence of salt bridges between neighbouring subunits and the inter-subunit contacts, made predominantly by the terminal protein regions, can contribute to the structural lability of potexvirus virions, a feature clearly distinguishing these virions from rigid, structurally invariant tobamovirus virions with different types of virion CP interactions (DiMaio et al., 2015). This difference between potexviruses and TMV is clearly demonstrated by comparisons of the inter-subunit contact surface calculated per CP subunit. In the TMV virion, the surface of contacts made by the CP core domain is 4499 Å²; in BaMV virions the surface of contacts involving residues of the CP core is 1806 Å² and the overall contact surface including the N- and C-terminal regions is 5752 Å² (DiMaio et al., 2015). Thus, the CP molecules in potexvirus virions interact mostly by their terminal regions, which constitute more than two-thirds of all inter-subunit contacts.

Based on general considerations, the local bending of helical virus capsids can only be possible due to weak interactions between successive turns of the helix or, in other words, weak or less-specific axial interactions between protein subunits forming the helical array (Caspar & Klug, 1962). Therefore, the axial contacts between CP subunits in potexvirus virions made by the protein C-terminal region must be changeable and distensible enough to permit variations in the distance between turns of the helix. On the other hand, unlike TMV and other rod-shaped viruses, where all subunits have identical conformation, make the same bonds and are therefore equivalent, protein subunits in flexuous virions lacking a straight axis of symmetry cannot be exactly equivalent. These subunits make similar but not equivalent contacts, adopt similar but not equivalent conformations, and are therefore considered quasi-equivalently related (Caspar & Klug, 1962). In the capsids of potexviruses, the ability of CP to exist in different conformations necessary for the formation of a quasi-equivalent helical array is specified by the inter-subunit interaction mode, predominantly involving contacts made by the CP terminal regions flexibly connected to the CP core domain. Such an interaction provides a mechanism for local virion bending and formation of flexuous virus particles.

Fig. 2. Contacts between subunits in capsids of potexviruses. Images for BaMV are presented. (a) The lateral inter-subunit contact made by the CP N-terminal region (residues 39–60), which is shown in green. Two neighbouring CP subunits in one helix turn are shown. (b) An axial view of six CP subunits, three of which are located in one helix turn of the BaMV capsid and three others in the next helix turn. The C-terminal protein region (residues 191–242), which is involved in formation of a network of axial contacts on the inner virion surface, is shown in red. (c) A lateral view of a segment of the inner virion surface formed by the six CP subunits shown in (b). Molecular graphics were generated with the UCSF Chimera package (Pettersen et al., 2004) using data deposited in Protein Data Bank under the accession number 5a2t.
Intermediate mode of subunit interaction in hordeivirus capsids

The structure of all rod-shaped helical plant viruses was expected to be similar to that of TMV. In support of this view, the cryoelectron microscopy-based reconstruction of rod-shaped *Barley stripe mosaic virus* (BSMV, genus * Hordeiviruses*) at a resolution of 19 Å revealed no drastic differences from TMV in the virion structure (Kendall et al., 2013). However, structural studies of the BSMV CP and virions employing analyses of circular dichroism spectra, differential scanning calorimetry and tryptophan spectra have demonstrated that the BSMV virions are considerably more labile than the TMV virions and, in general, the physicochemical characteristics of BSMV virions resemble those of flexuous filamentous viruses rather than TMV (Makarov et al., 2013). The recently reported high-resolution structure of BSMV at 4.1 Å obtained by cryoelectron microscopy (Protein Data Bank accession numbers 5a79 and 5a7a) provides an explanation for this contradiction (Clare et al., 2015).

In contrast to other studied helical plant viruses, two structurally distinct forms of BSMV virions were identified, ‘wide’ and ‘narrow’ virions of diameters 224 Å and 216 Å, respectively. Wide virions have 111 subunits per helix period, while narrow ones have 106 subunits, corresponding to a difference of one CP subunit per helix turn (Clare et al., 2015). Accordingly, both the conformation of CP molecules and their contacts differ in the two virion forms, providing another example of quasi-equivalent interactions in plant virus helical capsids. The functional difference between the two forms of BSMV virions is not understood. Both forms co-exist in populations of BSMV virions, but it is not known whether the two virion forms are assembled simultaneously and independently from each other in one cell or tissue, or if only one virion form is assembled under the given conditions and later undergoes a structural transition resulting in the observed mixed virion populations. Structural data show that the narrow BSMV virions are more stable than wide virions due to stronger CP contacts with RNA and additional contacts between CP subunits (Clare et al., 2015). Hypothetically, the stable narrow virion form could be considered as a *bona fide* storage container for the viral genome, while the wide form could be an intermediate in virion uncoating. This hypothesis implies the possibility of structural transitions, either uni- or bidirectional, between the two virion forms. The details of inter-subunit contacts in BSMV virions do not contradict this possibility.

Inter-subunit interactions in the BSMV virions differ considerably from those described for TMV. The primary lateral contact is made by an internal loop of the CP polypeptide chain interacting with the adjacent subunit on the outer surface of the virion (Fig. 1b). This contact involves both charged and hydrophobic residues (Clare et al., 2015). The functional importance of this contact is confirmed by site-directed mutagenesis: mutation of Ile86 and Tyr91 in the interacting loop to Gly residues results in the inability of BSMV CP to form stable virions. Such a contact is not found for TMV, since the BSMV interacting loop represents an ‘insertion’, and is absent from the TMV CP sequence. Importantly, amino acid sequence alignments reveal that the CPs of all known rod-shaped viruses, except TMV and related tobamoviruses, contain such ‘insertions’ (Clare et al., 2015), suggesting that their CP subunits can interact in assembled virions similarly to the BSMV CP molecules. From this point of view, tobamovirus capsids stabilized solely by lateral electrostatic interactions can represent a peculiar exception to the general rule for rod-shaped viruses, which are presumably stabilized by the interacting loop on the surface of virions. It should be noted that the main BSMV lateral contact is mechanistically similar to the lateral contact made by the IA domain in potexvirus CP. The CPs of hordeiviruses and potexviruses are structurally and evolutionary unrelated, and the domain responsible for lateral inter-subunit contacts is formed by either the N-terminal protein region in potexviruses or the internal loop in hordeiviruses. Therefore, the similarity of lateral inter-subunit contacts made by CP structural elements on the outer surface of hordeivirus and potexvirus virions exists only at the functional level, suggesting their convergent evolution.

It should be noted that narrow BSMV virions are stabilized by two additional lateral contacts, which are not found in wide virions, and represent salt bridges at a low and high virion radius (Asp44–Arg69, Glu125–Arg128) (Clare et al., 2015) (Fig. 1b). Therefore, the narrow form of the BMSV virion involves lateral contacts similar to those found in tobamoviruses and potexviruses.

Another important feature of hordeivirus virions is the lack of substantial contacts between successive turns of the helix. No axial salt bridges have been identified between the BSMV subunits. Moreover, since the axial subunit distance is as large as 6 Å, axial contacts, if they exist, are very weak (Clare et al., 2015). This structural feature provides a possibility for proposed transitions between the wide and narrow forms of the BSMV virions. Such transitions, involving alterations in the number of CP subunits per helix turn, are impossible in the TMV virion because of strong electrostatic axial contacts, and also in the potexvirus virion because of axial interactions made by the C-terminal protein region. On the other hand, as the inter-subunit interactions in all rod-shaped viruses (except tobamoviruses) are thought to be similar to those of hordeiviruses, one can predict that virions of many rod-shaped plant viruses can exist in more than one structural conformation.

**CP folds in rod-shaped and filamentous viruses and their evolution**

A quarter of a century ago, detailed sequence analyses of CPs encoded by helical plant viruses established that the CPs forming rod-shaped and filamentous capsids
constituted two distinct monophyletic protein families, with a lack of significant overall sequence similarity between them (Dolja et al., 1991). These data indicate an independent evolutionary origin of the CPs encoded by rod-shaped and filamentous viruses and their structural unrelatedness. Indeed, recent structural studies reveal that the CPs in rod-shaped and filamentous plant viruses have dissimilar, unrelated folds. Among flexuous viruses, structural data are available only for potexviruses. The potexvirus CP structure consists of seven α-helices (H1–H7), with H1 being located in the N-terminal region protruding from the protein core formed by H2–H7 (Yang et al., 2012). Surprisingly, the tertiary structure of potexvirus CP is similar to that of the nucleoprotein (NP protein) encoded by viruses of the genus Phlebovirus (family Bunyaviridae) (Agirrezabala et al., 2015). The NP protein binds single-stranded phlebovirus RNA to form ribonucleoproteins (RNPs) representing sinuous loose helices with a variable pitch (Raymond et al., 2012; Zhou et al., 2013). Taking into account the distant evolutionary relationship of mammal-infecting phleboviruses and plant potexviruses, the evolution of these virus groups is suggested to involve an event of horizontal gene transfer of a CP–NP progenitor (Agirrezabala et al., 2015).

According to the data on physicochemical characteristics of virions (Makarov et al., 2001; Zayakina et al., 2008), similarly, virus-like particles are detected in protoplasts infected with the potexvirus White clover mosaic virus encoding the CP C-terminally truncated by 31 amino acid residues (Forster et al., 1992). Additionally, even the rigid rod-like hordeivirus capsid can be formed in the absence of any substantial axial inter-subunit contacts, as described above. These observations suggest that in flexible viruses, the shape of RNA–protein assemblies, instead of being determined by axial contacts, more probably depends on the degree of freedom exerted by the flexible hinge interconnecting the protein core and the arm interacting with a neighbouring protein subunit. Indeed, the interacting arm of monomeric phlebovirus NP protein can fold back and interact with the core domain of the same protein molecule, therefore showing a wide range of hinge bending (Raymond et al., 2010), whereas the potexvirus CP molecule lacking an interacting counterpart has a structured N-terminal arm extending outward from the protein core (Yang et al., 2012). Therefore, we assume that the filamentous shape of potexvirus virions is determined by a limited flexibility of the hinge between the IA and core domains of CP.

The CP of rod-shaped tobamoviruses consists mainly of four α-helices forming a structural domain termed the ‘four-helix bundle’ (Namba et al., 1989), and exhibits no structural similarity to potexvirus CPs. The CP of the rod-shaped hordeivirus BSMV has the same fold as tobamovirus CP (Clare et al., 2015). A similar type of polypeptide fold, though not found in other viruses, is seen in many non-viral proteins including globins, cytochrome C and somatotropin (Kamtekar & Hecht, 1995), indicating that the CP of rod-shaped helical viruses was acquired early in their evolution and did not contribute to the evolution of other virus lineages.

As the CP of rod-shaped viruses has the four-helix-bundle fold, while the CPs of filamentous viruses exemplified by potexviruses have another fold, it can be concluded that the CP fold determines the shape of virions. However, as noted above, potexviruses and phleboviruses sharing a CP fold form different RNA–protein assemblies, either flexuous virions or loose helical RNPs, due to differences in the interactions between protein subunits. We propose that the general appearance of helical virus capsids depends mostly on the flexibility of their subunit contacts. Indeed, extremely flexible lateral contacts of the NP molecules in phlebovirus RNPs can give rise to assemblies with the loose coil conformation, while filamentous virions of potexviruses have much less flexible lateral contacts involving the IA domain, and rigid rod-shaped hordeivirus virions are characterized by the firm lateral contacts made by the inflexible interacting loop. The axial contacts appear to make a lesser contribution to the determination of the appearance of the virion, since rigid hordeivirus virions mostly lack axial inter-subunit contacts, while filamentous potexvirus virions comprise an axial contact network formed by the C-terminal CP region.

According to the data on physicochemical characteristics of virions (Makarov et al., 2013), salt bridges and other electrostatic interactions in the TMV virion provide even more stable inter-subunit contacts than the firm lateral contacts in hordeivirus virions. The TMV-like lateral contacts, based mainly on electrostatic interactions and devoid of any structural element for inter-subunit contacts on the outer surface of virions, appear to be unique for tobamoviruses. As noted above, the CPs of all rod-shaped plant viruses except tobamoviruses have sequence elements potentially forming an inter-subunit interaction loop (Clare et al., 2015). Therefore, we propose that during evolution, tobamoviruses lost this structural element, and instead gained other means for stabilization of the virion helix, resulting in the formation of exceptionally stable virions. Such an evolutionary scenario can be imaginatively recapitulated by a row of known rod-shaped virus structures: the lateral inter-subunit
contacts are represented mostly by the interacting loop in the wide BSMV virion, by both the interacting loop and salt bridges in the narrow BSMV virion, and by electrostatic contacts only in the TMV virion. Additional stabilization of TMV virions is provided by the axial electrostatic interactions and the hydrophobic girdle. Thus, with the exception of tobamoviruses, helical virions of plant viruses are labile to different extents, and this may be important during different stages of the viral life cycle, such as a virion translational activation and translocation through plasmodesmata (see below).

**Intrinsically disordered N-terminal regions in the CPs of filamentous viruses**

Structural data available for the CPs of potexviruses BaMV, PapMV and PepMV reveal that their IA domain is preceded by the extreme N-terminal regions of 38, 10 and 20 amino acid residues in length, respectively, which are not resolved in structure reconstructions, indicating that this protein region is disordered (Agirrezabala et al., 2015; DiMaio et al., 2015; Yang et al., 2012). Accordingly, secondary structure predictions employing several algorithms of analysis, such as those implemented in the web-based services FoldIndex (Prilusky et al., 2005) and DISOPRED3 (Ward et al., 2004), reveal that the extreme N-terminal sequences in the CPs of BaMV, PapMV and PepMV are predicted with a high probability as being intrinsically disordered (Fig. 3). Moreover, similar analyses carried out for other potexviruses demonstrate that in all cases the extreme CP N-termini preceding the IA domain are predicted to be intrinsically disordered (Fig. 3 and data not shown). This region is hereafter called the N-terminal intrinsically disordered (NID) domain. Thus, the N-terminal region of potexviral CPs consists of the NID and IA domains, with the NID domain being quite variable both in its length and amino acid sequence (Fig. 3).

Intrinsically disordered proteins and protein domains represent a recently recognized class of polypeptides, which lack a specific three-dimensional structure and are often involved in molecular recognition, particularly in interactions with proteins and nucleic acids (Fink, 2005; Uversky, 2011; van der Lee et al., 2014). In the CPs of icosahedral viruses, intrinsically disordered domains were shown to take part in stabilization of the capsid structure, control of the capsid assembly and disassembly and interaction with nucleic acids (Helgstrand et al., 2004).

Mutagenesis analyses reveal possible functions of the NID domain in potexvirus CPs. Deletion of the N-terminal 21 amino acid residues of the PVX CP, most of the 31-residue NID domain, as well as the deletion of residues 7–31, results in a functional virus able to produce virions (Chapman et al., 1992; Lico et al., 2006). BaMV with the deletion of N-terminal 35 CP residues is capable of virion formation and systemic transport (Lan et al., 2010). The N-terminal 14 amino acid residues of the *Plantago asiatica* mosaic virus (PlaMV) CP are dispensable for the formation of virions (Ozeki et al., 2009). These data clearly demonstrate that the NID domain is not essential for potexvirus assembly. However, deletions and mutations of the NID domain often result in altered characteristics of viral infection. First, deletions in the NID domain of PVX CP resulted in formation of bundles of virions twisted around each other, while another alteration in this domain resulted in the formation of large stacked virion arrays (Betti et al., 2012; Chapman et al., 1992). Secondly, the NID domain is involved in virus–host interactions. Deletion of the region encompassing residues 7–31 of PVX CP results in a delayed systemic infection and milder symptoms (Chapman et al., 1992). In BaMV, the NID domain is required for necrotic local lesion induction in *Chenopodium quinoa* and symptom expression leading to systemic infection in *C. quinoa* (Chapman et al., 1992, 1993).

in Nicotiana benthamiana (Lan et al., 2010). Amino acid residues 11–26 of the PepMV CP residing within the NID domain contain a virus pathogenicity determinant influencing the infection phenotype in N. benthamiana (Duff-Farrier et al., 2015). A point mutation in the PlaMV CP (Leu to Ala mutation at position 3) results in blocked virus cell-to-cell movement, suggesting that this protein region is involved in interactions with cell-to-cell movement factors other than the viral movement protein TGB1, binding to which is not affected by this particular mutation (Ozeki et al., 2009). Thus, one can conclude that the NID domain of potexvirus CP can be involved at least in (i) interactions with host factors affecting virus movement and symptom induction, a function consistent with the general view on intrinsically disordered domains as protein modules involved in multiple interactions (Uversky, 2011); and (ii) prevention of virion aggregation.

Systematic analyses of the CP sequences in other filamentous plant viruses reveal that the presence of the NID domain is not unique to potexviruses. Intrinsically disordered regions are confidently predicted for all four families of flexuous plant viruses, Alphaflexiviridae, Betaflexiviridae, Closteroviridae and Potyviridae in their CP sequences preceding the core domain (Fig. 4). The exceptions are four genera of the family Betaflexiviridae (Capillovirus, Trichovirus, Vitivirus and Tepovirus), which encode the CPs constituting a separate evolutionary branch among the CPs of Alphaflexiviridae and Betaflexiviridae (Martelli et al., 2007). On the other hand, disordered N-terminal CP regions are not found in TMV or BSMV, and are not predicted for the CPs of other rod-shaped viruses. These observations suggest that most flexuous filamentous virions require functions provided by the N-terminal intrinsically disordered CP regions. Interestingly, similarly to multiple functional interactions found for the potexvirus NID domain, mutational analyses of the potyvirus CP demonstrate that the N-terminal intrinsically disordered region, which is dispensable for virion formation, can influence viral cell-to-cell movement and is essential for systemic transport as well as for transmission by an insect vector (Atreya et al., 1991; Dolja et al., 1994).

**Translational activation of helical virions**

Rod-shaped and filamentous viruses differ in their mechanisms of translational activation of virions or, in other words, the process of virion structural changes giving access of the translational machinery to the encapsidated RNA. In rod-shaped tobamoviruses, translational activation is believed to take place stochastically, as a result of structural micro-fluctuations leading to dissociation of a number of CP molecules from the virion end encapsidating the 5' -terminus of genomic RNA; uncoating of the RNA 5'-terminus can permit translation initiation and further co-translational virion disassembly (Mundry et al., 1991; Wilson & Shaw, 1987). Such relative instability of one virion end is thought to be due to the properties of the TMV RNA 5'-untranslated region devoid of G residues and therefore interacting weakly with CP subunits (Mundry et al., 1991).

In flexuous potexviruses, the encapsidated RNA cannot be translated; however, some external stimuli can cause virion translational activation, not necessarily accompanied by partial or full virion disassembly in this case (Atabekov et al., 2000, 2001; Kiseleva et al., 2003). First, the PVX virions can be translationally activated by the phosphorylation of in-virion CP, presumably its N-terminal disordered region, by serine/threonine protein kinases (Atabekov et al., 2001; Karpova et al., 2006a). As one possible explanation of this effect, one can propose that a charge gained by the N-terminal CP region due to phosphorylation can weaken the lateral contacts formed by the N-terminal IA domain, and changes in the inter-subunit contacts can lead to a structural alteration in the virion and its translational activation. Secondly, the PVX virions are translationally activated upon their interaction with virus-encoded protein TGB1, several molecules of which can bind in vitro to one virion end containing the 5'-end of PVX genomic RNA (Atabekov et al., 2000; Karpova et al., 2006b). Deletion mutagenesis data show that 10–18 C-terminal residues of the PVX CP are involved in the interaction with TGB1 (Zayakina et al., 2008). According to the available data on the structure of BaMV and PepMV, the C-terminal regions of potexvirus CP form an axial interaction network on the inner virion surface, and only the C-termini of CP molecules in the first turn of the virion capsid helix are available for interaction with the TGB1 protein. We assume that such an interaction can initiate a disruption of the axial hydrophobic contacts at one end of the PVX virion that further spreads as a domino effect along the virion axis, finally resulting in a structural virion switching into an altered or remodelled conformation accompanied by translational activation. The mechanisms of translational activation induced by phosphorylation and TGB1 binding are seemingly different; however, the N-terminal domain of PVX CP can influence the conformational state of the C-terminal region of the CP in the virion. Indeed, in particles with the 19–21 N-terminal CP residues removed by proteolysis, the C-terminal region, which normally faces the inner virion channel, is now located at the outer virion surface (Baratova et al., 1992). In the context of this review, the ability of the N-terminal CP region to determine structural characteristics of virion CP molecules and mediate, upon phosphorylation or deletion, their conformational switch (changes in inter-subunit contacts and/or conformational alteration of a peptide backbone) is most significant, demonstrating that external stimuli can influence the structure of filamentous virions and thereby emphasizing their structural lability.

**Role of virions in cell-to-cell transport of filamentous viruses**

Cell-to-cell movement of plant viruses occurs through plasmodesmata interconnecting cells in plant tissues and requires dedicated virus-encoded movement proteins...
Fig. 4. Intrinsically disordered regions in the CPs of viruses belonging to four families of filamentous plant viruses, predicted by the DISOPRED3 algorithm. The disorder probability is plotted against the residue number in the protein amino acid sequence. For each family, the prediction is shown for type viruses of three genera. Amino acid residues are considered disordered when the plotted value is above the confidence score of 0.5 (the horizontal dashed line). Light grey areas show the positions of the CP core domain in protein sequences. The dark grey area in the prediction plot for CLV indicates the position of the conserved sequence domain characteristic of the genus *Carlavirus* (pfam08358). PVX, Potato virus X; ShVX, Shallot virus X; LoLV, Lolium latent virus; CLV, Carnation latent virus; CLBV, Citrus leaf blotch virus; ASPV, Apple stem pitting virus; BYV, Beet yellows virus; LIYV, Lettuce infectious yellows virus; GLRaV-3, Grapevine leafroll-associated virus 3; PVY, Potato virus Y; BaYMV, Barley yellow mosaic virus; SPMMV, Sweet potato mild mottle virus.
(MPs), which are essential for the intracellular delivery of the viral genome to plasmodesmata and its translocation through plasmodesmata micro-channels to neighbouring cells (Lucas, 2006). Viral RNA genomes are thought to be transported cell-to-cell in the form of either non-virion RNP s composed of viral RNA and MP, or virions (Heinlein, 2015; Lucas, 2006; Tilsner et al., 2014). The latter alternative is considered for many flexuous filamentous plant viruses, in which the CP is essential for cell-to-cell movement. Here we discuss the role of virions in cell-to-cell movement of helical viruses, not considering virus long-distance transport via the phloem, a less studied process with specific requirements for transported entities.

For potyviruses, the hypothesis of virion transport is supported by an elegant experiment employing PVX virion-specific antibodies, which interact with virus particles but not with dissociated CP subunits. Using such an approach, PVX virions have been detected within plasmodesmata interconnecting virus-infected cells (Santa Cruz et al., 1998). An alternative hypothesis of a non-virion complex transport has been suggested on the basis of indirect evidence (Lough et al., 2000) and seemed less probable until newer supporting data were published in recent years. For example, a PepMV mutant carrying a point mutation of the Tyr residue in the IA domain blocking inter-subunit interactions is unable to form virions, but moves from cell to cell (Agirrezabala et al., 2015). On the other hand, point mutations in the PepMV CP RNA-binding site prevent both virion assembly and virus movement (Agirrezabala et al., 2015), demonstrating that the binding of RNA by viral CP is necessary for cell-to-cell transport. Taking into account that the inability of the IA domain mutant to form virions was inferred from the inability to isolate intact virus particles from infected tissues using a standard purification protocol, we presume that in infected tissues this mutant might form a virion-like complex serving as the viral transport form, but insufficiently stable to be isolated. This hypothesis is in line with the known propensity of potyvirus virions to undergo structural transitions, or, in other words, to exist in alternative structural states presumably including a less stable virion-like structure, which can serve as a virus transport form.

Similarly, the available data show that potyviruses move from cell to cell as virions or complexes similar to virions in their structure. Mutations introduced into the core domain of the potyvirus *Tobacco etch virus* CP result in the inability of the protein to form virions and virus incompetence for cell-to-cell movement, whereas deletions in either the N- or C-terminal CP regions, which are dispensable for virion formation, do not abolish viral cell-to-cell movement, although they do make it slower compared with the wild-type virus (Dolja et al., 1994, 1995). In a similar manner, point mutations in the CP C-terminal region of *Soybean mosaic virus*, another potyvirus, block both virion formation and viral cell-to-cell movement (Seo et al., 2013), indicating a direct link between them. Furthermore, immuno-gold labelling and electron microscopy studies have revealed the potyvirus CP in plasmodesmal cavities (Roberts et al., 1998; Rodriguez-Cerezo et al., 1997), and its localization within plasmodesmata often correlates with the presence of CP-containing fibrillar material, with the dimensions of fibrils similar to the virion diameter (Roberts et al., 1998). Therefore, potyviruses move from cell to cell in the form of either virions, or virion-like complexes composed of the same components as true virions, but differing in their fine structure. For phloem-limited viruses of the family Closteroviridae, virions are detected in plasmodesmata interconnecting sieve element and phloem parenchyma cells (Esau et al., 1967; Hoefert et al., 1988). In view of these data, and taking into account that all structural proteins of closterovirus virions are essential for virus cell-to-cell movement, the virion is considered the transport form of the closterovirus genome (Dolja, 2003).

In contrast to filamentous viruses translocated through plasmodesmata as virions or virion-like structures, the CP of rod-shaped viruses is not required for viral cell-to-cell movement (Herzog et al., 1998; Petty & Jackson, 1990; Saïto et al., 1990; Savenko et al., 2003; Schmitt et al., 1992; Tamada et al., 1996; Ziegler-Graff et al., 1991). In these viruses, the transport form of the virus genome is assumed to be a non-virion RNP, as has been reported for well-studied TMV and BSMV (Brakke et al., 1988; Citovsky et al., 1990, 1992). Therefore, the rod-shaped helical virus particles seem to be incompatible with the virion translocation through plasmodesmata, and viruses with such type of virions necessarily encode an MP capable of forming transport RNPs, either the MP of TMV type or the N-terminally-extended TGB1 protein (for details, see Morozov & Solovyev, 2003). Conversely, transport forms of filamentous viruses (virions, structurally modified virions or virion-like assemblies) have characteristics that permit their translocation through plasmodesmata micro-channels. The 10–13 nm diameter of flexuous virions is smaller compared with that of rod-shaped virions (18–22 nm) but considerably bigger than the effective diameter of plasmodesmata micro-channels, estimated to range from 2.4–3.1 nm for MP-gated plasmodesmata (Wolf et al., 1989). However, the presence of virions, or virion-like transport entities similar in their diameter to virions within plasmodesmata, demonstrates that the true dimensions of plasmodesmata micro-channels may be higher than estimated. It can be proposed that (i) the cell-to-cell transport of filamentous virions induces more severe changes in the internal structure of plasmodesmata compared with those induced by individual viral MPs, and (ii) the virion-like transport form could be structurally modified upon translocation through plasmodesmata. Conceivably, the structure of the filamentous virion can be globally remodelled or destabilized, as shown for PVX virions with bound TGB1 protein, and can additionally undergo local chaperoning, which affects virion regions passing through plasmodesmata. Energy for the structural transitions can be provided by other viral proteins involved in cell-to-cell transport. Interestingly, in the case of phloem-limited closteroviruses, such structural changes may appear to be unnecessary, since phloem plasmodesmata are different in structure and have a higher size.
exclusion limit compared with other tissues (Oparka & Santa Cruz, 2000; Van Bel & Kempers, 1998).

The available data suggest that the labile structure of filamentous viroins and their capability of structural conversion permit their transport, either in the non-altered or a structurally remodelled form, through plasmodesmata. However, it should be noted that a number of filamentous viruses of the family Betaflexiviridae (genera Capillovirus, Citrovirus, Tepovirus, Trichovirus and Vitivirus) encoding the TMV-type MPs (Martelli et al., 2007) most probably move from cell to cell in a non-virion form.

Virion terminal structures and the mechanism of cell-to-cell transport

Cell-to-cell movement of the transport forms of filamentous viruses (virion, modified virion or a virion-like assembly) requires specialized structures made of non-CP viral protein(s) and located at the virion end containing the 5'-terminus of genomic RNA.

Closteroviruses, which have unusually large RNA genomes (Dolja et al., 2006; Kiss et al., 2013), in addition to the major CP, encode a minor capsid protein (CPm) considered to be a diverged duplicated copy of the CP. The main virion part (approximately 95 % of its length) is made of the CP, while the remaining segment, which is located at the virion end encapsidating the 5'-terminus of genomic RNA, is formed by the CPm (Agranovsky et al., 1995; Peremyshlov et al., 2004; Satyanarayana et al., 2004). Additionally, the CPm-formed region of BYV virions contains three other viral proteins, namely an Hsp70 homologue (Hsp70h), a p64 protein with a CP-like structural domain in its C-terminal region and p20 (Kiss et al., 2013; Napuli et al., 2000, 2003; Tian et al., 1999). The latter is dispensable for virion formation and virus cell-to-cell movement, but is essential for the viral systemic transport through the plant vascular system (Prokhnevsky et al., 2002). Atomic force microscopy studies demonstrate that the closterovirus virion terminal structure has a complex morphology and consists of three distinct segments, of which the tip segment is likely to be formed by the p20 protein (Peremyshlov et al., 2004). Since the CPm, Hsp70h and p64 proteins are required for BYV cell-to-cell movement (Alzhanova et al., 2000; Peremyshlov et al., 1999; Prokhnevsky et al., 2002), it has been concluded that the terminal structure represents the specialized device evolved to facilitate translocation of closterovirus viroins to and through plasmodesmata (Dolja et al., 2003). Of these proteins, Hsp70h may be the key element of BYV transport machinery, since this protein, when expressed in the absence of other viral products, is able to localize to motile granules, which are transported to the cell periphery in an actin-dependent manner, and can associate with plasmodesmata (Prokhnevsky et al., 2005). Moreover, the ATPase activity of Hsp70h is required for cell-to-cell BYV transport (Alzhanova et al., 2001).

Potyviruses are characterized by the genome-linked protein VPg covalently attached to the 5'-terminus of viral genomic RNA (Revers & García, 2015). VPg can interact with a virus-encoded protein HC-Pro; this interaction is believed to result in the formation of virion terminal structures, which are distinguishable by electron and atomic force microscopy and can be labelled with HC-Pro antibodies (Torrance et al., 2006). Moreover, a sub-population of particles isolated from plants infected with Potato virus A has been shown to contain the viral CI (cylindrical inclusion) protein localized to one end of the virion (Gabrenaite-Verkhovskaya et al., 2008), presumably due to the reported ability of CI to interact with HC-Pro (Revers & García, 2015). The CI protein is targeted to plasmodesmata-associated sites by the recently discovered CI-interacting potyvirus protein P3N-PIPO, which is essential for viral transport and is capable of directed intracellular trafficking to plasmodesmata and translocation through plasmodesmata to neighbouring cells (Chung et al., 2008; Vijayapalani et al., 2012).

Virions of potexviruses have no morphologically distinguishable terminal structures. On the other hand, TGB1 molecules can bind to the PVX virion end containing the 5'-terminus of genomic RNA (see above). In addition to converting virions into a translatable form, TGB1 binding can be pertinent to virus transport in plants, since TGB1 can be directed to plasmodesmata by the TGB2 and TGB3 proteins (discussed in Morozov & Solovyev, 2003; Solovyev et al., 2012; Verchot-Lubicz et al., 2010). This plausible hypothesis is consistent with the observations that deletions in the potexvirus CP C-terminal region involved in interactions with the TGB1 protein, while having little effect on virion formation (see above), block virus cell-to-cell movement (Fedorkin et al., 2001; Forster et al., 1992). According to the current view, attachment of TGB1 molecules to virions serves for segregation of a progeny virion sub-population, which is both destined for cell-to-cell transport and capable, due to structural destabilization upon interaction with TGB1, of co-translational uncoating (discussed above) after translocation to a new cell (Atabekov et al., 2001). As a result, by the TGB1-dependent coupling of virion structural conversion and cell-to-cell movement, the uncoating of virions is avoided in cells, where they are assembled. Another likely result of the TGB1-induced virion structural transition might be virion conversion to a movement-competent form. One can speculate that after TGB1-induced structural transition this protein region might also appear to be located on the outer surface of transport-competent TGB1-bound virions similar to the localization of C-terminal PVX CP region to the outer virion surface upon protease treatment (Baratova et al., 1992), and to directly participate in interactions required for viral movement. It should be kept in mind that potexvirus cell-to-cell transport is coupled with the replication of viral RNA (Tilsner et al., 2013). Since an Ala residue in the C-terminal region of BaMV CP has been found to be involved in the CP interaction with viral replicase, and point mutations of this residue in both
BaMV and *Foxtail mosaic virus* CPs result in the virus being unable to move from cell to cell (Lee et al., 2011), it is conceivable that the viral replicase binds to the CP C-termini exposed on one end of the virion, competing therefore with the TGB1 protein. Alternatively, the replicase may bind to CP molecules with the C-terminal region presumably exposed on the virion surface upon interaction with TGB1.

In addition to other functions, the protein components of terminal structures can participate in transport form remodelling, which may involve their enzymatic activities, such as the NTPase activity of the Hsp70 protein in closteroviruses and the NTPase/helicase functions of the potexvirus TGB1 protein and the potyvirus CI protein. In fact, terminal structures can participate in transport form TGB1. Bind to CP molecules with the C-terminal region presumed to be involved in the interactions within and between subunits (Jankowsky et al., 2001). Therefore, the terminal structures found in filamentous viruses are essential for the delivery of viral transport form to plasmodesmata and its transfer to adjoining cells in a polar manner, with the genome 5′-terminus being transferred first.

Conclusions

Recent data on the structures of helical plant viruses have considerably advanced our knowledge of the field. The structures of helical plant viruses appear to be much more labile than expected previously, often allowing structural transitions, remodelling and the existence of alternative structural forms of virions. Therefore, the rigid invariant structure of TMV virions, previously considered a paradigm for helical plant viruses, can now be considered as an exception. The virion shape, as well as its internal lability, is found to be determined mainly by contacts between CP molecules forming the helical array. Available data suggest that the lability of helical virions can be of functional importance at different stages of the virus life cycle, such as co-translational disassembly and viral cell-to-cell transport. We expect that further research on helical plant viruses will elucidate both the molecular mechanisms underlying virion structural transitions and their exact functions.

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