DESK ENCYCLOPEDIA OF Plant and Fungal Virology

Edited by BRIAN W. J. MAHY
MARC H. V. VAN REGENMORTEL

Desk Encyclopedia of PLANT AND FUNGAL VIROLOGY

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and
Dr MARC H V VAN REGENMORTEL

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Amsterdam • Boston • Heidelberg • London • New York • Oxford
Paris • San Diego • San Francisco • Singapore • Sydney • Tokyo
Academic Press is an imprint of Elsevier

ACADEMIC
PRESS

Academic Press is an imprint of Elsevier
Linacre House, Jordan Hill, Oxford, OX2 8DP, UK
525 B Street, Suite 1900, San Diego, CA 92101-4495, USA

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloguing in Publication Data
A catalogue record for this book is available from the Library of Congress

ISBN: 978-0-12-375148-5

For information on all Elsevier publications
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PRINTED AND BOUND IN SPAIN
08 09 10 11 10 9 8 7 6 5 4 3 2 1

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PREFACE

The *Desk Encyclopedia of Plant and Fungal Virology* is the fourth in a series of four volumes that reproduces many entries that appeared in the third edition of the *Encyclopedia of Virology*, edited by Brian W J Mahy and Marc H V van Regenmortel, published by Academic Press/Elsevier in 2008.

It consists of 85 chapters that highlight recent advances in our knowledge of the viruses that infect plants and fungi. The first section of the book, comprising 10 chapters, discusses general topics in plant virology such as the movement of viruses in plants, the transmission of plant viruses by vectors, antiviral defense mechanisms in plants, and the development of virus-resistant transgenic plants. A chapter is devoted to viroids.

The second section of 48 chapters presents an overview of the properties of a selection of 20 well-studied plant viruses, 23 plant virus genera and a few larger groups of plant viruses.

The third section of 12 chapters describes the most economically important virus diseases of cereals, legumes, vegetable crops, fruit trees, and ornamentals. This section is abundantly illustrated and should be very useful to plant pathologists and all those interested in viral infections in plants. The last section of 15 chapters describes the major groups of viruses that infect fungi.

As all the chapters initially appeared in an encyclopedia, little prior specialized knowledge is required to follow the material that is presented. When used in conjunction with the first volume of the series, which is devoted to *General Virology* and describes the structure, replication, molecular biology, and general properties of viruses, this volume could form the basis of an introductory course on virology, suitable for students of plant sciences.

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GENERAL TOPICS

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Movement of Viruses in Plants

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Glossary

Ancillary viral proteins Virus-encoded proteins that do not meet the definition of a movement protein, but are required for virus movement.
Intercellular movement Movement between two cells.
Intracellular movement Movement within a single cell.
Microfilaments A component of the cytoskeleton formed from polymerized actin monomers.
Microtubules A component of the cytoskeleton composed of hollow tubes formed from α–β tubulin dimers.
Molecular chaperones A family of cellular proteins that mediate the correct assembly or disassembly of other polypeptides.
Movement protein Virus-encoded proteins that can transport themselves cell to cell, bind RNA, and increase the size exclusion limits of plasmodesmata.
Phloem Vascular tissue that transports dissolved nutrients (e.g., sugars) from the photosynthetically active leaves to the other parts of the plant. In most plants there is only one phloem class, but for some plant families this tissue is divided into two classes: (1) internal phloem (internal or adaxial to xylem) and (2) external phloem (external or abaxial to xylem).
Systemic movement Movement through vascular tissue to all parts of the plant.
Viroid A plant pathogen containing nucleic acid that encodes no proteins.
Xylem Vascular tissue that transports water and minerals through the plant.

Introduction

In order for a plant virus to infect its host systemically, it must be capable of hijacking the host's cellular machinery to replicate and move from the initially infected cell. Plant viruses require wounding, usually by insect or fungal vectors or mechanical abrasion, for an infection to begin. Once inside a cell, the virus initiates transcription (DNA viruses) and translation and replication (DNA and RNA viruses) activities. Some of these viral products are required for virus movement and often interact with host factors (proteins or membranes) to carry out this function.

Virus movement in plants can be broken down into three distinct steps: (1) intracellular movement, (2) intercellular movement, and (3) systemic movement. Intracellular movement refers to virus movement to the periphery of a cell and includes all metabolic activities necessary to recycle the host and viral constituents required for the continued transport of the intracellular complex. Intercellular movement refers to virus movement between cells. In order for a plant virus infection to spread between cells, viruses must move through specific channels in the cell wall, called plasmodesmata (PD), that connect neighboring cells. Once intracellular and intercellular movement is established, the virus can invade the vascular cells of the plant and then spread systemically through the open pores of modified PD within the sugar-transporting phloem sieve elements. Upon delivery by the phloem to a tissue distant from the original infection site, virus exits the vasculature and resumes cell-to-cell movement via PD in the new tissue. Although it will not be discussed further in this article, it is important to know that a few viruses utilize the water-transporting xylem vessels for systemic transport.

When contemplating plant virus movement it is critical to understand that each virus movement complex varies in viral and host factor composition over time as it travels within and between cell types. In addition, individual viruses often utilize unique host factors to support their movement. The diverse and dynamic nature of virus movement complexes makes it difficult to summarize plant virus movement in a simple unified model. However, there is evidence that some stages of virus movement, although carried out by apparently unrelated host or virus proteins, do have functional convergence.

Virus movement in plants has been studied with a wide range of virus genera, including, but not limited to, tobamoviruses, potexviruses, hordeiviruses, comoviruses, nepoviruses, potyviruses, tombusviruses, tospoviruses, and geminiviruses. In this article we do not review virus movement by all plant viruses, but rather focus on model viruses within genera that provide the most information on the subject. We review what is currently known about the three steps of virus movement in plants and attempt to convey the complexity of movement mechanisms utilized by members of different virus genera. However, we also highlight recent findings indicating that irrespective of the presence of seemingly unrelated host or viral factors, functional similarities exist for some aspects of movement displayed by viruses from different genera.

Intracellular Movement

Intracellular movement is necessary to deliver the virus genome to PD for cell-to-cell transmission. This has been an understudied area, as researchers have only recently had the ability to label and observe the movement of viral proteins and RNA in near-real-time conditions. Early studies relied on static images of immunolabeled viral proteins from light and transmission electron microscopes to determine their intracellular location. While a few of these studies related the intracellular location of the viral protein to the stage of infection, most did not and thus the importance of the intracellular location for virus movement was not understood. Other early studies of virus movement relied on the mutation of specific viral genes in virus genomic clones and the assessment of the intercellular movement of the resulting mutant virus, through the presence of local (representing intracellular and intercellular movement) or systemic (representing intracellular, intercellular and vascular movement) disease. Although these genetic experiments often determined which viral proteins were important for virus intercellular or systemic movement, they could not determine whether the mutation prevented intracellular or intercellular movement, both outcomes being visually identical. In more recent studies, fusion of viral proteins with fluorescent reporter genes such as the green fluorescent protein (GFP) have given researchers a powerful method to observe both the intracellular movement and final subcellular destination of many viral proteins in near-real-time conditions. However, it is important not to over-interpret movement studies using GFP since GFP maturation for fluorescence emission takes hours and thus the visible movement and position of the GFP or GFP:viral protein fusion may not reflect early movement activity. Additionally, the level of GFP within the movement form of the virus may be too low to detect during critical phases of movement.

Although intracellular virus movement in plants is just beginning to be elucidated, it is clear that specific viral proteins regulate this activity. Chief among these are the virally encoded movement proteins (MPs), named to indicate their genetically determined requirement for intercellular virus movement. MPs are defined based upon three functional characteristics: their (1) association with, or ability to increase, the size exclusion limit (SEL) of PD; (2) ability to bind to single-stranded RNA (ssRNA); and (3) ability to transport themselves or viral RNA cell to cell. Based upon these defining characteristics, a number of proteins have been classified as MPs (**Table 1**). Many viral MPs have similar sequences indicating a shared evolutionary history. However, a considerable number have no obvious sequence similarity between them. The absence of a shared sequence for these MPs suggests convergent evolution for movement function by unrelated predecessor proteins. MPs often interact with host proteins that modify their amino acid backbone (e.g., through phosphorylation) or host proteins associated with intracellular trafficking (e.g., cytoskeletal or vesicle-associated proteins) (**Table 1**). However, the role of MPs in intracellular movement remains largely unknown because technical limitations have prevented visualizing movement of individual viral RNA or DNA associated with MPs in real time. In addition, it is becoming clear that ancillary viral proteins (**Table 1**), which do not fulfill the classical definition of an MP, are essential for virus movement. These proteins are often associated with membranes or cytoskeletal elements and thus likely function primarily for intracellular virus movement. The interaction of MPs with host factors and the impact of the ancillary viral proteins on intracellular virus movement are discussed in detail in the following section. Models for intracellular virus movement of particular genera of viruses are presented based on some of this information (**Figure 1**).

Host factors and intracellular virus movement

Host proteins shown to interact with viral MPs include kinases, chaperones, nuclear-localized proteins (often transcription co-activators), and proteins that are associated with or are core components of the cytoskeletal or vesicle trafficking systems (**Table 1**). In addition, some MPs have been shown to associate with host membranes.

For geminiviruses, whose DNA genomes replicate in the nucleus, it is not surprising that nuclear factors may be necessary to transport viral genetic components required for virus replication into or out of the nucleus. For RNA viruses, however, there must be other reasons for an interaction between a nuclear protein and viral MP since these viruses are replicated in the cytoplasm. Some of the nuclear host proteins are non-cell-autonomous factors (e.g., HiF22) and thus it has been suggested that their interaction with MPs may inadvertently aid in virus intracellular and intercellular movement. It is also possible that MP and nuclear protein interactions occur to prevent transcription of host defense proteins or enhance transcription of host proteins necessary for virus movement, either within the infected cell or after transport to uninfected cells at the infection front.

The discovery over 10 years ago that tobacco mosaic virus (TMV) MP associates with microtubules (MTs) and microfilaments (MFs) was the first evidence that the host cytoskeleton might be involved in virus movement in plants. Although results from early studies indicated that disruption of MT arrays or their association with TMV MP could inhibit TMV movement, later studies suggested this was not so. Disruption of MT arrays with pharmacological agents or by tubulin transcript knockdown using virus-induced gene silencing had no effect on TMV movement or MP localization. Other work showed that the association of the MP with MTs happened late in

Table 1 Proteins necessary for the cell-to-cell movement of plant viruses

Virus	*MP*[a]	*Ancillary viral proteins*[b]	*Host protein interactors with MP*
Tobacco mosaic virus, Tomato mosaic virus	**30 kDa**	126 kDa	Actin, tubulin, MPB2C, PME, KELP, MBF1, calreticulin
Red clover necrotic mosaic virus	**35 kDa**		
Groundnut rosette virus	ORF4		
Cowpea chlorotic mottle virus	3a		
Brome mosaic virus	**3a**	CP	
Cucumber mosaic virus	**3a**	CP	NtTLP1
Bean dwarf mosaic virus	BC1	BV1	
Tobacco etch virus	CP	CI, HC-Pro, VPg	
Barley stripe mosaic virus	TGBp1	TGBp2, TGBp3	
Potato virus X	**TGBp1**	TGBp2, TGBp3 + CP	TIPs
Cowpea mosaic virus	**48 kDa**	CP	
Cauliflower mosaic virus	**38 kDa**	CP	MPI7, PME
Turnip crinkle virus	**p8** + p9		Atp8
Tomato bushy stunt virus	**P22**		HFi22, REF
Potato leaf roll virus	17 kDa		
Tomato spotted wilt virus	**NS_m**		DnaJ-like, At4/1
Beet necrotic yellow vein virus	TGBp1	TGBp2, TGBp3, p14	
Grapevine fanleaf virus	2B	CP	Knolle, actin, tubulin
Rice yellow stunt virus	P3		
Rice dwarf virus	Pns6		
Southern bean mosaic virus	ORF1		
Turnip yellow mosaic virus	69 kDa		
Alfalfa mosaic virus	**P3**	CP	
Prunus necrotic ringspot virus	3a		
Tobacco rattle virus	**29 kDa**		
Soil-borne wheat mosaic virus	37 kDa		
Peanut clump virus	P51	P14, P17	
Potato mop top virus	**TGBp1**	TGBp2, TGBp3	
Commelina yellow mottle virus	N-term 216 kDa		
Beet yellows virus	p6, Hsp70h, p64	CPm, CP	
Rice stripe virus	Pc4		
Apple stem grooving virus	36 kDa		
Raspberry bushy dwarf virus	39 kDa		

[a]Regular (i.e., no bold) font indicates marginal classification as MP because the protein either has not been fully tested or has some but not all of the functions classically associated with MP (see text for definition).
[b]Necessary for viral cell-to-cell movement.

infection, probably after virus movement had occurred. Also, during time-course studies it was determined that the MP disappeared during late stages of infection. This finding, in combination with the discovery that a mutant virus expressing a functionally enhanced MP with limited affinity for MTs moved cell to cell better than the parental virus, led to the idea that the association of MP with MTs is critical for MP degradation rather than to aid virus cell-to-cell movement. Further support for this idea came from the finding that the *Nicotiana tabacum* host protein, MPB2C, binds to MP and promotes its accumulation at MTs, yet acts as a negative effector of MP cell-to-cell transport. The role of MTs during TMV movement remains to be fully understood, but at this time it appears that they are more involved with MP degradation or compartmentalization than with virus movement (**Figure 1(a)**).

In contrast to the large body of work focusing on the role of the MT–MP interaction in TMV movement, studies on the role of the MFs in the movement of TMV and other viruses have only recently been published. It was demonstrated that intracellular movement of TMV viral replication complexes (VRCs; large multiprotein complexes comprised of host and viral factors) and cell-to-cell spread of the virus were blocked by MF inhibitors (pharmacological and transcript silencing agents). VRCs were later determined to physically traffic along MFs (**Figure 1(a)**). The interaction of TMV VRCs with MFs may be mediated by the TMV 126 kDa protein (a protein containing helicase, methyltransferase, and RNA silencing suppressor domains), since expression of a 126 kDa protein:GFP fusion in the absence of the virus results in fluorescent protein bodies that, like VRCs, traffic along MFs. VRC association with MFs may be mediated through a direct interaction of the 126 kDa protein with MFs or through an intermediary cell membrane. MFs are known to associate with membranes in plant cells and

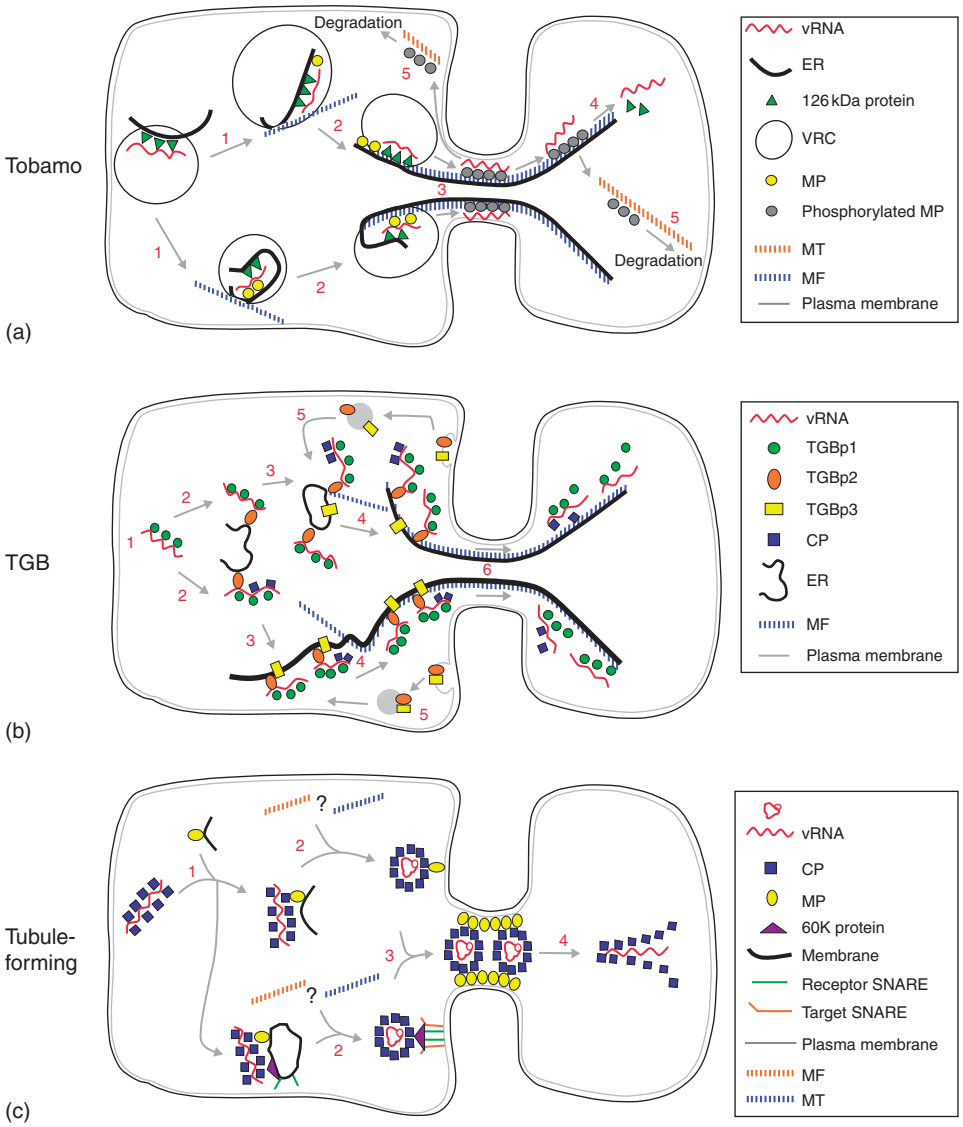

Figure 1 Models for cell-to-cell movement of plant viruses using tobamovirus triple gene blocks (TGBs), or tubule-forming strategies. (a) Viral 126 kDa protein binds both viral RNA (vRNA) and endoplasmic reticulum (ER) forming a cytoplasmic body in the cell termed a VRC. MP associates with the ER and possibly the vRNA within the VRC (step 1). VRCs associated with microfilaments (MFs) traffic toward plasmodesmata (PD; step 2). Here we show an indirect association of the VRC with actin mediated by the ER, but it is also possible that this interaction is mediated directly by the viral 126 kDa protein or MP. At the PD, vRNA is released from its association with the 126 kDa protein and is transported through the PD in association with MP (step 3). Phosphorylation of the MP occurs either within the cytoplasm, the cell wall, or both, and likely regulates transport to and through PD and subsequent translation of the vRNA in the new cell (steps 3 and 4). MP is degraded in the later stages of infection, likely via association with MTs and delivery to specific cellular sites of degradation (step 5). (b) Progeny vRNA binds to TGB protein 1 (TGBp1; step 1). The TGBp1/vRNA complex, either in the presence or absence of coat protein (CP, depending on virus genus), then binds TGBp2 attached to the ER to form a movement-competent ribonucleoprotein complex (RNP, step 2). The RNP then interacts with TGBp3, either directly or indirectly, to be positioned near the PD (steps 3 and 4). RNPs associate with actin MFs likely through an interaction with TGBp2, which may be responsible for transport to the PD. Following delivery of vRNA to the PD, TGBp2 and TGBp3 are likely recycled via an endocytic pathway (step 5). vRNA is actively transported through the PD via an unknown mechanism (step 6), although TGBp1 or CP may be involved, and is released from associated proteins in the next cell to allow replication to initiate. (c) CP-bound vRNA associates with the MP (itself associated with a membrane of unknown origin, step 1). The complex then moves, either as a vesicle directed to the PD through targeting proteins such as those from the SNARE family, or through other unknown targeting signals to the cell periphery (step 2). Interaction between SNAREs and virus may be mediated by a viral 60K protein for cowpea mosaic virus. The requirement for the cytoskeleton in transport of MP–vRNA complex is unclear since the nepovirus, grapevine fanleaf virus, requires cytoskeletal elements for proper delivery of its MP to the cell wall while cowpea mosaic virus does not. At or near the PD, the vesicular or nonvesicular membranes fuse with the plasma membrane and the attached MP directs the CP-associated vRNA through the PD (step 3). The vRNA is then released into the next cell to initiate virus replication and movement (step 4). Reproduced from Nelson RS (2005) Movement of viruses to and through plasmodesmata. In: Oparka KJ (eds.) *Plasmodesmata*, 1st edn., pp. 188–211. Oxford: Blackwell, with permission from Blackwell.

the 126 kDa protein binds to an integral membrane host protein, TOM1. However, the MP of TMV is long known to bind actin and associate with membranes, so the relative importance of the TMV 126 kDa protein or MP for directing intracellular VRC movement is unclear (**Figure 1(a)**).

Recently, MFs were demonstrated to co-localize with ancillary proteins required for movement of the hordeivirus, potato mop-top virus (PMTV). PMTV encodes a conserved group of proteins termed the triple gene block (TGB) that are required for cell-to-cell virus movement (see **Table 1**). Two of these TGB proteins (TGBp2 and TGBp3) co-localize with motile granules that are dependent upon the endoplasmic reticulum (ER)–actin network for intracellular movement. In addition, the TGBp2 protein from the potexvirus, potato virus X (PVX), localizes to MFs in what are likely ER-derived vesicles (**Figure 1(b)**). The association of TGBp2 from potexviruses and the 126 kDa protein from TMV with MFs and their requirement for successful virus movement provide an elegant example of convergent evolution since TGBp2 and 126 kDa protein have no sequence identity.

The role of the cytoskeleton in the intracellular transport of some plant viruses is unclear. Cowpea mosaic virus (CPMV), for example, does not require the host cytoskeleton for the formation of tubular structures containing MP on the surface of protoplasts. These tubular structures are similar to the tubules formed in modified PD (that likely do not contain cytoskeleton) which are necessary for intercellular movement of this virus (**Figure 1(c)**). The role of tubules in intercellular transport of CPMV is discussed in next section. Work with grapevine fanleaf virus (GFLV), another tubule-forming virus, has revealed the possibility that this virus may be targeted to the PD by membrane vesicle SNARE (v-SNARE)-mediated trafficking. The MP of GFLV co-immunoprecipitates with KNOLLE, a target SNARE (t-SNARE). The 60 kDa protein of CPMV has been shown to bind a SNARE-like protein. t-SNAREs such as KNOLLE act as specific receptors for targeted delivery of Golgi-derived vesicles to sites where fusion with the plasma membrane will occur. Thus, it is possible that the SNARE trafficking machinery delivers viral proteins (and possibly associated viral RNA) to the plasma membrane near PD (**Figure 1(c)**).

There is evidence that, following movement of the viral RNA to the PD, some viral factors involved in this movement may be recycled for further use. The TGBp2 and TGBp3 proteins from PMTV localize to endocytic vesicles as evidenced by labeling with FM4-64 dye, a marker for internalized plasma membrane (**Figure 1(b)**). Additionally, TGBp2 co-localizes with Ara7, a marker for early endosomes. The functional significance of this endocytic association of viral proteins remains to be determined and it is not known whether proteins from other viruses may also traffic in the host cell's endocytic pathway.

Intercellular Movement

Following intracellular movement to the cell periphery, the virus must then move through PD in order to spread into neighboring cells. PDs are plasma membrane-lined aqueous tunnels connecting the cytoplasm of adjacent cells. An inner membrane, termed the desmotubule, is a tubular form of the ER and is an extension of the cortical ER. PDs can be subdivided structurally into simple (containing a single channel) or branched (containing multiple channels) forms. The SELs of PD are increased by the disruption of MFs indicating a role for actin in PD gating and indeed both actin and myosin have been observed in PD. Thus, it is possible that cytoskeletal-mediated transport of viral components results in direct delivery of virus to and passage through PD.

Protein movement through PD is dependent on the developmental stage of the PD. For example, free GFP (27 kDa) moves through simple but not branched PDs. Branched PDs generally have smaller SELs than simple PDs and the presence of more branched PDs in mature photosynthate-exporting (source) versus immature photosynthate-importing (sink) leaves represents a developmental change that limits transport of macromolecules through PD. This developmental change also affects the localization pattern for some viral MPs. For example, both cucumber mosaic virus and TMV MPs are observed predominantly or solely within branched PD in source leaves and not simple PD in sink leaves. The TMV MP expressed in transgenic plants, however, is sufficient to complement the movement of an MP-deficient TMV mutant in both source and sink leaves. Thus, the presence of MP in the central cavity of branched PD in source leaves may not represent a site of function for the MP, but rather the final deposition of inactive MP. Although it is possible that the level of MP binding in simple PD is below the detection limits of the current technology, questions remain about where and how the MP functions in virus movement.

A clue to TMV MP function during virus movement comes from findings showing that a TMV MP–viral RNA complex could not establish an infection in protoplasts, but could do so when introduced into plants. It was suggested that a change in the phosphorylation state of the MP at the cell wall was necessary to weaken the binding between the MP and viral RNA, thereby allowing translation of the viral genome and initiation of infection in the next cell. Indeed, a PD-associated protein kinase has been identified that phosphorylates TMV MP. Thus, the protein kinase in the cell wall may be necessary to end the involvement of MPs in virus movement and release the viral RNA for translation in the new cell (**Figure 1(a)**). Also, considering that there are additional phosphorylation sites on the TMV MP besides those

targeted by the PD-associated protein kinase, it is likely that proper sequential phosphorylation of this protein is necessary to allow it to function in both intracellular and intercellular virus movement. For potyviruses, the eukaryotic elongation factor, eIF4E, appears to modulate both virus accumulation, likely by affecting translation, and cell-to-cell movement. Thus, as for TMV, virus accumulation and movement may be linked activities.

Chaperones of host or viral origin may be required for PD translocation of some MPs. Host-encoded calreticulin modulates TMV intercellular movement and co-localizes with TMV MP in PD. The virus-encoded virion-associated protein (VAP) of cauliflower mosaic virus (CaMV) binds MP through coiled-coil domains and co-localizes with MP on CaMV particles within PD. The mechanism by which a molecular chaperone can support intercellular virus movement is illustrated by the virally encoded Hsp70 chaperone homolog (Hsp70h) of beet yellows virus. Hsp70h requires MFs to target it to the PD. The Hsp70h is a component of the filamentous capsid and its ATPase activity is required for virus cell-to-cell movement. These findings led to a model where Hsp70h mediates virion assembly and, once localized to the PD, actively translocates the virion from cell to cell via an ATP-dependent process. The idea that viral proteins may actively participate in plasmodesmal translocation of virus is further supported by the finding that the NTPase activity of the TGBp1 helicase from PMTV is necessary for its translocation to neighboring cells and that the coat protein (CP) of PVX, necessary for virus cell-to-cell movement, has ATPase activity (**Figure 1(b)**). It has also been found that the helicase domain of the TMV 126 kDa protein is required for cell-to-cell movement. In these cases it seems likely that the helicase activity is necessary to remodel viral RNA, thereby easing passage of the virus through PD.

Tubule-forming viruses have adopted another strategy for intercellular movement whereby virus-induced tubules span modified PD that lack a desmotubule in order to transmit capsids from cell to cell (**Figure 1(c)**). Such capsid-containing tubules are known to be composed, at least in part, of MP and have been identified for a number of viruses, including commelina yellow mottle virus, CaMV, CPMV, and tomato ringspot virus.

Although the tubule-forming viruses modify PD differently than those utilizing classical MPs, it was recently determined that the tubule-forming MP from tomato spotted wilt virus can functionally substitute for the non-tubule-forming TMV MP to support TMV movement. This is likely another example where two proteins with no sequence identity and therefore no apparent evolutionary relationship have independently evolved to functionally support movement of viruses.

Systemic Movement

Some viruses are limited to the phloem of plants (i.e., phloem-limited viruses) and require inoculation, often by aphids, directly to vascular cells for infection. Systemic movement of a non-phloem-limited virus through vascular tissue, however, requires that the virus moves from nonvascular cells into veins. Veins are defined as major or minor based on their structure, location, branching pattern, and function (**Figure 2**). Whether major or minor, each vein contains many different cell types with greatly differing structures. Within *N. tabacum*, minor veins include phloem parenchyma, xylem parenchyma, and companion cells, along with sieve elements and xylem vessels (**Figure 2**). All of these cells have distinct structures and locations within the vein which present unique regulatory sites for virus entry. Between plant species, companion cell morphologies vary greatly with an obvious difference being the number of PDs between these cells and other vascular cells. This difference is functionally related to the type of photosynthate transport system exhibited by the plant (i.e., apoplastic versus symplastic). In addition, bundle sheath cells, which have their own unique position and structure surrounding the minor veins, must be considered as potential regulators of virus movement. These complex cell types are difficult to study because it is problematic to directly access or isolate them.

Recently, studies have been conducted that conclusively indicate which veins, minor or major, can serve as entry sites for rapid systemic infection. Using surgical procedures to isolate specific veins and TMV or CPMV modified to express GFP as a reporter, it was determined that either major or minor veins in leaves of *Nicotiana benthamiana* and *Vigna unguiculata* can be invaded directly and serve as inoculum sources for systemic infection. In addition, for TMV, direct infection of cells in transport veins in stems yielded a systemic infection. Considering that major and transport veins do not have terminal endings bounded by nonvascular tissue, it is likely that virus entered these veins by passing through bundle sheath cells and interior vein cells.

Virus transport and accumulation are regulated within vascular tissue. In plants that have internal and external phloem, potyviruses and tobamoviruses accumulate in specific tissue depending on the tissue's position relative to the inoculated leaf. In the inoculated leaf and the stem below, virus accumulates in the external phloem, whereas in the stem and leaf veins above the inoculated leaves, virus accumulates in the internal phloem.

Exit of PVX, TMV, and CPMV from vascular cells in sink tissues only occurs from major veins. For a growing number of viruses, however, exit occurs from both major and minor veins indicating that there is not a uniform exit strategy for all viruses.

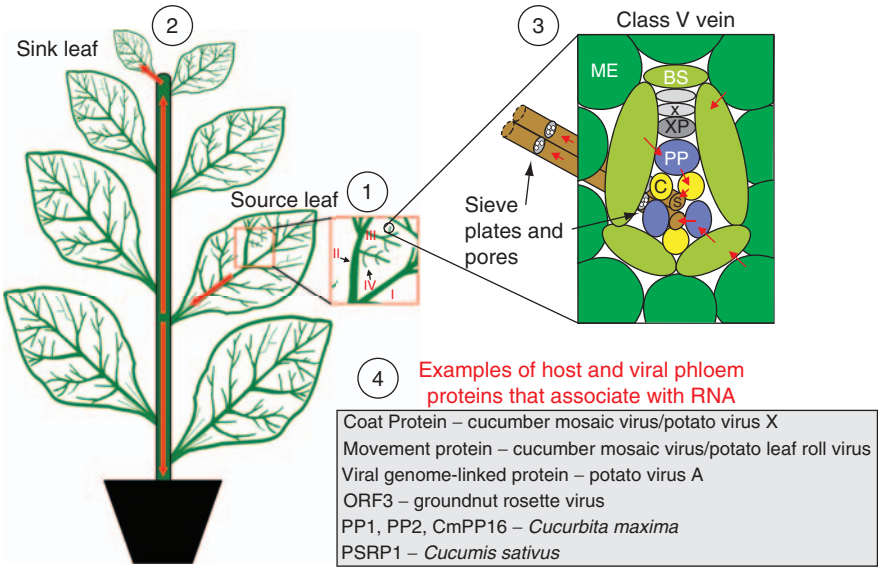

Figure 2 (1) Viral infection of a source leaf occurs by intercellular movement of the virus into the vasculature (class I–V veins indicated). (2) Virus travels through the phloem from the source leaf throughout the plant (red arrows) and exits vascular tissue to resume cell-to-cell movement in sink tissue. (3) In order to enter the phloem of a class V vein, a non-phloem-limited virus must travel through mesophyll cells (ME), bundle sheath cells (BS), and possibly phloem parenchyma cells (PP) before entering the companion cells (C) and finally the sieve elements (S). Movement through SEs requires passage of virus through pores within the sieve plates. A minority of viruses move through the xylem (X). (4) Examples of host and viral proteins that have been identified in phloem and that associate with RNA are indicated.

The virus and host factors that control systemic virus accumulation are becoming better understood, mostly through genetic studies. Virus factors include CP, some MPs, and some nonstructural proteins such as the 126 kDa protein of TMV. Although CPs are often necessary for systemic infection, it is clear that for some viruses, such as groundnut rosette virus, a CP is not present and the virus still produces a systemic infection in the host. Also, for viruses that normally require the CP for systemic infection, the loss of the CP through mutation or deletion may still allow systemic movement of the virus in specific hosts. Lastly, viroids, which do not encode any proteins, can systemically infect plants. These results indicate that although a capsid may be required to protect viral RNA for systemic transport in some hosts, other viral or host proteins can functionally mimic the CP and allow systemic infection.

MP function during systemic infection has, in one case, been uncoupled from its role during intra- and intercellular transport. Some point mutations in the red clover necrotic mosaic virus MP still allow intercellular movement, but prevent systemic movement. Additional support that MPs function to allow systemic movement comes from studies with the 17 kDa MP of potato leafroll virus, a phloem-limited virus. This MP, when expressed from within an infectious virus sequence in transgenic plants, is uniquely localized to PD connecting the companion cells with sieve elements, even though virus accumulated in both vascular and nonvascular cells. Thus, the PD between companion cells and sieve elements may be uniquely recognized by this MP to allow the virus to only invade vascular tissue. More recently, it has been shown that a host factor, CmPP16, that is thought to function by forming ribonucleoprotein complexes with phloem transcripts has sequence similarity with viral MPs. Thus, some MPs may function to protect RNA while in transit through the phloem.

Other viral proteins such as the 2b protein of CMV, p19 of TBSV, and the 126 kDa protein of TMV have been linked to supporting systemic movement of their respective viruses. Considering that all of these proteins are suppressors of gene silencing, it is possible that this activity is related to their function in supporting systemic movement. It is known that a member of the plant silencing pathway, specifically, the RNA-dependent RNA polymerase, RDR6, functions in sink tissue (e.g., the shoot apex) by responding to incoming signals for RNA silencing. RDR6 has also been shown to control virus accumulation in systemic, but not inoculated, leaves. Thus, it is possible that viral suppressor activity could function to specifically allow systemic accumulation of viruses.

Host factors that modulate virus systemic spread either support or restrict this activity. A protein methylesterase (PME) is involved in both intercellular and systemic movement of TMV. For systemic movement, PME is essential for virus to exit into nonvascular tissue of the uninoculated leaves. A phloem protein from cucumber, p48, was found to interact with CMV capsids and may function to protect the capsid during transport.

Host factors that restrict virus systemic movement include the restricted TEV movement (RTM) proteins, which are expressed only in phloem-associated cells and accumulate in sieve elements. RTM1 is related to the lectin, jacalin, while RTM2 has a heat shock protein motif. RTM1 may function in a plant defense pathway within the veins, although the jacalin-like proteins have not been previously linked to virus defense. RTM2 may function as a chaperone to prevent unfolding of a transport form of the virus within the sieve elements. A third protein that serves as a negative regulator is a cadmium-induced glycine-rich protein, cdiGRP. This protein does not act directly to restrict systemic movement. Instead, it induces callose deposits which are thought to restrict intercellular transport of the virus. This could prevent exit of virus from the vascular tissue. Interestingly, cadmium treatment inhibits the systemic spread of RNA silencing, lending support to the idea that spread of specific viruses affected by cadmium treatment (i.e., TMV and turnip vein clearing virus) is functionally similar to that of a host silencing signal.

See also: Bromoviruses; Carmovirus; Citrus Tristeza Virus; Cucumber Mosaic Virus; Furovirus; Luteoviruses; Nepovirus; Plant Resistance to Viruses: Engineered Resistance; Plant Resistance to Viruses: Geminiviruses; Potexvirus; Tobacco Mosaic Virus; Tobamovirus; Tobravirus; Tombusviruses; Tospovirus; Umbravirus; Viroids; Virus Induced Gene Silencing (VIGS).

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Vector Transmission of Plant Viruses

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Glossary

Fitness The relative ability of an individual (or population) to survive and reproduce in a given environment.

Helper component (HC)-transcomplementation An HC encoded by a viral genome X mediates the vector transmission of a virus particle containing a viral genome Y.

Horizontal transmission The transmission of a virus, parasite, or other pathogen from one individual to another within the same generation, as opposed to vertical transmission.

Pierce-sucking insects Insects adapted to sap or blood feeding, with the mouthparts transformed into long chitin needles that can pierce and penetrate tissues and allow pumping up their content.

Quasispecies Ensemble of mutant viral genomes constituting a viral population.
Vector Organism acquiring a pathogen on an infected host and inoculating it in a new healthy one.
Vertical transmission The transmission of a pathogen from the parent(s) to the offspring, usually through the germline.

Introduction

Viruses are intracellular parasites diverting the host cellular machinery for their own replication and offspring particles production. As such, they most often negatively affect the hosting cells, sometimes even killing them, and hence repeatedly and unavoidably face the problem of moving on and colonizing new healthy and potent 'territories'. Within a single host, viruses can both diffuse from cell to cell and be transported on longer distances by the vascular system. While animal viruses use membrane fusions (if enveloped) or membrane receptors to penetrate healthy cells, plant virus entry during the host invasion is always resulting from a passage through 'tunnels' traversing the cell wall, called plasmodesmata, and ensuring a cytoplasm continuity between adjacent cells. Any viral population can grow this way only until the physical limits of the host are reached. Then, a critical passage in the 'outside world' separating two compatible hosts has to be successfully achieved. Because animals are motile and often come in contact, some associated viruses can directly access either blood or permissive tissues of a healthy host and operate a cell entry resembling that involved during invasion of single hosts. However, a most frequently adopted strategy relies on additional organisms, capable of sampling the virus population within an infected host, transporting, disseminating, and efficiently inoculating infectious forms of this virus within host population. Such organisms are designated vectors, giving rise to the term vector transmission. Vector transmission is found frequently in animal viruses and, presumably due to stable hosts and to the need of covering considerable distances between them, has been adopted by the vast majority of plant viruses. Each virus species is submitted to different ecological conditions; hence, an impressive complexity of host–virus–vector interactions has been unraveled over a century of research efforts. The object of this chapter is to synthesize the knowledge available at present in the field of vector transmission of viruses, with a special emphasis on plant viruses, where a great diversity of strategies have been discovered and documented. Indeed, the numerous patterns of vector transmission described for plant viruses include all those reported in animal viruses and many more.

Plant Virus Vectors

Any organism that is creating a break into the cell wall, either for penetrating a plant or simply for feeding on it, and that is capable of covering the distance between two separated plants, can possibly be used as a vector by viruses, for traveling through space and time. Vectors have been described in groups of organisms as diverse as parasitic fungi, nematodes, mites, and most importantly insects (**Table 1**). The pattern of virus uptake, preservation, transport, dissemination, and inoculation can be very different, due to the specific biology of all three (plant, virus, and vector) partners. However, viruses transmitted by 'pierce-sucking' insects are quantitatively predominant, and the classification established for their various modes of transmission is widely used as a reference for comparison with others. For this reason, hemipteran insect transmission will be described first in details and succinctly compared later on with that by other types of vectors.

Transmission of Plant Viruses by Insects

History of the Classification of the Different Modalities of Transmission

The transmission of plant viruses has been investigated for over a century with the most common vectors being sap-feeding insects with pierce-sucking mouth parts, particularly aphids, and also whiteflies, leafhoppers, planthoppers, and mealybugs. Pioneer studies have demonstrated the complexity and diversity of the interactions between plant viruses and their insect vectors. Even as late as the 1950s, scientists, using the tools at hand, were merely measuring quantitative traits such as the time required for virus acquisition on infected plants and the time during which the virus remained infectious within the vector. Three categories were then defined: (1) the nonpersistent viruses, acquired within seconds and retained only a few minutes by their vectors; (2) the semipersistent viruses, acquired within minutes to hours and retained during several hours; and (3) the persistent viruses that require minutes to hours for acquisition, and can be retained for very long periods, often until the death of the vector. It is important to note that, though the classification and terminology have changed in the last decades, these categories are still used by a number of authors, and thus often encountered in the literature.

In an early study on 'nonpersistent' viruses, the transmission of potato virus Y was abolished by chemical (formaldehyde) or ultraviolet (UV) treatments of the extremity of the stylets of live viruliferous aphids, demonstrating that infectious virus particles were retained there. It was first believed that the transmission of 'nonpersistent' viruses could be assimilated to mechanical transmission, stemming from nonspecific contamination of the stylets, the

Table 1 Vectors and mode of transmission in families of plant viruses

Family[a]	*Vector*	*Mode of vector-transmission*[b]
Bromoviridae genus *Alfamovirus*	Aphids	Noncirculative capsid strategy
Bromoviridae genus *Cucumovirus*	Aphids	Noncirculative capsid strategy
Bromoviridae genus *Ilarvirus*	Thrips	?
Bromoviridae genus *Oleávirus*	?	?
Bromoviridae genus *Bromovirus*	Beetle	?
Bunyaviridae	Thrips, planthopper	Circulative propagative
Caulimoviridae	Aphid, mealybug, leafhopper	Noncirculative helper strategy
Circoviridae	Aphid	Circulative nonpropagative
Closteroviridae	Aphid, whitefly, mealybug	Noncirculative
Comoviridae genus *Comovirus*	Beetle	?
Comoviridae genus *Fabavirus*	Aphid	Noncirculative
Comoviridae genus *Nepovirus*	Nematode	Noncirculative capsid strategy
Geminiviridae	Leafhopper, whitefly	Circulative nonpropagative[c]
Luteoviridae	Aphid	Circulative nonpropagative
Partiviridae	?	?
Potyviridae genus *Potyvirus*	Aphid	Noncirculative helper strategy
Potyviridae genus *Ipomovirus*	Whitefly	Noncirculative
Potyviridae genus *Macluravirus*	Aphid	Noncirculative
Potyviridae genus *Rymovirus*	Mite	Noncirculative
Potyviridae genus *Tritimovirus*	Mite	Noncirculative
Potyviridae genus *Bymovirus*	Fungus	Circulative
Reoviridae	Planthopper, leafhopper	Circulative propagative
Rhabdoviridae	Leafhopper, aphid	Circulative propagative
Sequiviridae	Aphid, leafhopper	Noncirculative helper strategy
Tombusviridae	Fungus	Noncirculative

[a]The families are broken down to the genus level when they contain genera with totally different vectors and mode of transmission.
[b]The helper or capsid strategies (see **Table 2**) are mentioned when experimentally demonstrated for at least one of the member species. When no complement is added to either circulative or noncirculative, it reflects the lack of further information.
[c]For at least one member species (*Tomato yellow leaf curl virus*, TYLCV), replication within the vector is still being debated.
The noncirculative viruses, or assimilated as discussed in the text, are in blue. The circulative viruses, or assimilated as described in the text, are in green.

vector acting simply as a 'flying needle'. Consistent with this was the repeated demonstration that 'nonpersistent' viruses are lost upon moulting of the viruliferous vectors. Later on, the hypothesis of virus uptake during sap ingestion and inoculation during putative regurgitation led to a change from vectors as flying needles to vectors as 'flying syringes', the virus–vector relationship still being considered as nonspecific (**Figure 1**). It is interesting that, while in plant viruses recent data unequivocally convinced the scientific community that the situation is much more complex, likely involving specific receptors in vectors for specific virus species (see the next section), in animal viruses very few studies are available at present and this mode of transmission is still referred to as 'mechanical vector transmission'. The prime conclusion from these experiments is that the virus–vector association occurs externally, on the cuticle lining the food or salivary canal in the insect stylets. Because semipersistent viruses are also lost upon vector moulting, their association with the vector was also proposed to be external, likely in the stylets, though a possible location 'upstream', on the cuticle lining the anterior gut of the insect, was also proposed in some cases.

In sharp contrast, many persistent viruses were observed within the vector body by electron microscopy, in various organs and tissues, indicating an internal association with the vectors. Such viruses were shown to pass through the gut epithelium into the hemolymph and join the salivary glands to be ejected together with saliva (**Figure 1**). A latent period of hours to days after acquisition, during which the virus cannot be efficiently inoculated, is consistent with the time needed for completing this cycle within the vector body. Moreover, microinjection of purified persistent viruses within the insect hemolymph subsequently resulted in efficient transmission to new healthy plants, proving that virus within the vector body can get out and be inoculated to host plants.

Altogether, these results prompted a revision of the classification of the modes of transmission in the late 1970s, based on qualitative criteria and still valid today (**Table 2**). The non- and semipersistent viruses were grouped in a new category designated 'noncirculative viruses', and the persistent viruses were named 'circulative viruses'. While circulative animal viruses (arboviruses) in fact infect their vectors where they efficiently replicate, some circulative plant viruses can seemingly operate their cycle in the vector body without any cell infection and replication. Hence, the category 'circulative' has been broken down into the two subcategories: 'propagative'

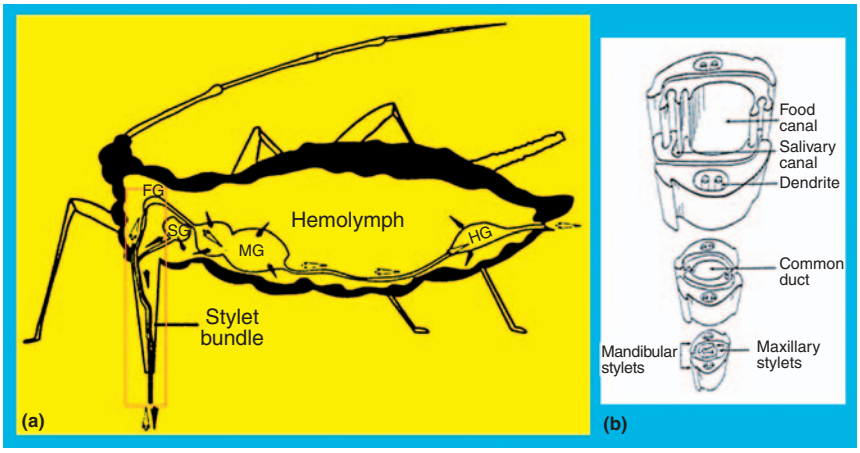

Figure 1 Different routes of plant viruses in their aphid vectors. (a) The white arrows represent the ingestion of circulative viruses, whereas the black arrows materialize their cycle within the aphid body, and inoculation in a new host plant. The red square area indicates the region of the anterior feeding system, where noncirculative viruses are retained in their vectors. (b) Cross sections of the stylet bundle illustrating the inner architecture of maxillary stylets which defines interlocking structures, food canal and salivary canal, fused at the distal extremity into a single common duct, where most noncirculative viruses are thought to be retained (see text). Adapted from Taylor CE and Robertson WM (1974) Electron microscopy evidence for the association of tobacco severe etch virus with the Maxillae in Myzuspersical (Sulz.). *Journal of Phytopathology* 80: 257–266.

Table 2 Different modes of plant virus transmission by insects with pierce-sucking mouth parts

	Circulative		*Noncirculative*	
Transmission modes[a]	*Propagative*	*Nonpropagative*	*Capsid strategy*	*Helper strategy*
Acquisition time[b]	Minutes to hours	Minutes to hours	Seconds to hours	Seconds to hours
Retention time[c]	Days to months	Days to months	Minutes to hours	Minutes to hours
Inoculation time[d]	Minutes to hours	Minutes to hours	Seconds to minutes	Seconds to minutes
Association with vectors[e]	Internal	Internal	External	External
Replication in vectors	Yes	No	No	No
Requirement of an HC[f]	No	No	No	Yes

[a]These modes of transmission were established and are widely accepted for virus transmission by pierce-sucking insects. As discussed in the text, they sometimes also apply to other types of vector.
[b]The length of time required for a vector to efficiently acquire virus particles upon feeding on an infected plant.
[c]The length of time during which the virus remains infectious within its vector, after acquisition.
[d]The length of time required for a vector to efficiently inoculate infectious virus particles to a new healthy plant.
[e]Internal means that the virus enters the inner body of its vector, passing through cellular barriers. External means that the virus binds the cuticle of the vector and never passes through cellular barriers.
[f]A helper component (HC) is involved in cases where the virus particles do not directly recognize vectors, acting as a molecular bridge between the two.

and 'nonpropagative'. The various families and/or genera of plant viruses and their associated vectors and modes of transmission are listed in **Table 1**.

During the last decades, the implementation of molecular and cellular biology has provided invaluable tools for studying the molecular mechanisms of virus–vector interaction. The data currently available for each category are summarized in the following subsections.

Circulative Transmission

Logically, circulative viruses are ingested by vectors, while feeding on infected plants. Some viruses are limited to phloem tissues, which the insect vector can reach within minutes to hours depending on the species, which explains the long feeding period required for their acquisition. As schematized in **Figure 1(a)**, the viruses cross the mid- or hindgut epithelium, are released into the hemolymph, and can then adopt various pathways to traverse the salivary glands, and be released in their lumen, wherefrom they will be inoculated upon salivation into healthy hosts. During this basic cycle , the virus encounters and must overcome diverse cellular barriers, where the existence of specific virus–vector interaction has long been established experimentally, though specific receptors have not been identified so far.