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REVIEW

Insect transmission of plant viruses: multilayered interactions optimize viral propagation

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Abstract By serving as vectors of transmission, insects play a key role in the infection cycle of many plant viruses. Viruses use sophisticated transmission strategies to overcome the spatial barrier separating plants and the impediment imposed by the plant cell wall. Interactions among insect vectors, viruses and host plants mediate transmission by integrating all organizational levels, from

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molecules to populations. Best-examined on the molecular scale are two basic transmission modes wherein virus-vector interactions have been well characterized. Whereas association of virus particles with specific sites in the vector's mouthparts or in alimentary tract regions immediately posterior to them is required for non-circulative transmission, the cycle of particles through the vector body is necessary for circulative transmission. Virus transmission is also determined by interactions that are associated with changes in vector feeding behaviors and with alterations in plant host's morphology and/or metabolism that favor the attraction or deterrence of vectors.

A recent concept in virus-host-vector interactions proposes that when vectors land on infected plants, vector elicitors and effectors 'inform' the plants of the confluence of interacting entities and trigger signaling pathways and plant defenses. Simultaneously, the plant responses may also influence virus acquisition and inoculation by vectors. Overall, a picture is emerging where transmission depends on multi-layered virus-vector-host interactions that define the route of a virus through the vector, and on the manipulation of the host and the vector. These interactions guarantee virus propagation until one or more of the interactants undergo changes through evolution or are halted by environmental interventions.

Key words arbovirus; defense; host plant; insect vector; interactions; plant virus; transmission

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Introduction

Viruses are obligate parasites that rely on their prokaryotic or eukaryotic hosts for all steps of their replication cycle. The hosts provide the energy and infrastructures necessary for the multiplication, encapsidation, and where applicable, spread of the virus within the host. However, once replication is completed and the host's resources are depleted, viruses must overcome the hurdle of having to cross physical and spatial barriers to reach and invade another host. This process, transmission, is achieved by using different but specific strategies. Transmission can be vertical (Gray & Banerjee, 1999), meaning that the pathogen passes from an infected progenitor directly to the next generation. Transmission can also be horizontal, wherein the virus is transmitted from one individual host to another of the same species or even one that is potentially unrelated. In the simplest case, viruses lyse host cells and then diffuse freely by Brownian movement through the medium (or extracellular matrix) to reach new host cells. This vector-less and support-less transmission is found, for example, among microbe-infecting viruses such as virophages and bacteriophages. Transmission by direct or indirect contact, for example through open wounds, as reported for animal, human, fungal and plant viruses, also belongs to this category of transmission. In airborne transmission, viruses use inert supports to reach and infect new hosts. For example, human rhinoviruses and other viruses are contained within aerosols produced by sneezing and are distributed in the air to be inhaled by new hosts; while other viruses are dispersed by hitching a ride on dust and other solid particles. All other modes of horizontal transmission rely on vectors, biological entities that help viruses to bridge the distance between two hosts. The reasons for the success of vector transmission are quite obvious: contrary to passive distribution by wind or water, vectors are capable of actively and directionally seeking and moving towards new hosts. Moreover, many insect vectors are sap or blood feeding parasites, and can deliver viruses directly into cells and vessels of a new host. In fact,

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the needle-like mouthparts of piercing-sucking insects—unlike those of chewing ones—inflict minimal damage on their hosts, leaving the punctured cells and tissues mostly intact and thus compatible for virus infection and propagation. The term ‘flying syringes’ has been coined to illustrate this fact. Vectors are found among different phyla. However, other than the less frequently encountered vectors like fungal spores and nematodes, most vectors transmitting animal, human or plant viruses are arthropods. Of the different classes of arthropods, insects and arachnids play especially important roles as plant, invertebrate and vertebrate virus vectors, although parasitic crustaceans may also vector viruses (Overstreet *et al.*, 2009). Recent reviews have provided thorough coverage of discrete biological and molecular aspects of insect vector-mediated plant virus transmission (Blanc & Gutiérrez, 2015; Drucker & Then, 2015; Ng & Zhou, 2015; Rosen *et al.*, 2015; Whitfield *et al.*, 2015; Mauck *et al.*, 2016). In a departure from these archetypal reviews, we present here instead a general conspectus of the biology of insect transmission of plant viruses, from molecular vector-virus interactions (how viruses are transported by insects) to the ecology of virus transmission (how viruses modify vector and host performance to promote transmission).

Molecular virus-vector determinants of transmission

Viruses need to interact physically with vectors during all steps of transmission. During the transmission process the requirements change drastically. After acquisition during vector feeding, the virus must be retained in the vector’s body in a transmissible form meaning it must protect itself from the potentially hostile environment in the vector. Then, the virus must be inoculated at the right time into a new host. This necessitates opposing actions: acquisition is only possible by physical binding to the vector and inoculation only by physical release from the latter, and the intermediate retention phase must neither interfere with acquisition nor inoculation. In this chapter we discuss

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the different molecular virus-insect interactions that have evolved to accomplish the three steps (acquisition, retention and inoculation) of transmission.

Most insect vectors of plant viruses belong to the order Hemiptera and have piercing-sucking mouthparts, called stylets, designed for feeding in the phloem. Consequently, the stylets are the first zone of contact between viruses and vectors when the latter feed on infected plants. Differences in the feeding behaviors of different phytophagous hemipteran insects (Ng & Zhou, 2015; Ng & Walker, 2016) notwithstanding, the initial exploratory stages of feeding all involve some form of probing activities in which vectors insert their stylets briefly into plant cells to assess the suitability of the plant as a feeding host. When a suitable plant is identified, the stylets ultimately penetrate into the phloem for sustained feeding. In both cases, some saliva is first injected into the target and then a mixture of saliva and cell or sieve tube contents is ingested and passes through the stylets, the foregut and eventually into the intestine. Virus particles (virions) contained in the ingested fluid are thus temporarily (cells) or continuously (phloem sap) flushed through the digestive system. Viruses must find a way in this set-up to bind to the vector for retention and later for inoculation. Two virion binding locations have been identified, each with its own uniqueness but both are superbly geared toward the common goal of optimizing virus delivery. One location is the stylets or regions farther up in the alimentary tract referred to as the foregut. The advantage of binding to the stylets is that the retention site can also be used for inoculation when the insect switches from ingestion to salivation or egestion/regurgitation. The disadvantage is that there is only an extremely small window of time during which virions flushing through can bind, and that virions are retained in the vector only for a short time (Table 1). Nonetheless, this transmission mode, named non-persistent transmission (Watson & Roberts, 1939) or, to reflect the fact that virions do not cycle through the vector, non-circulative transmission (Kennedy *et al.*, 1962), is used by hundreds of viruses. Virus-

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binding to vector mouthparts is often considered as being similar to contact transmission, requiring no particular prerequisites (from either the virus or the vector). Yet there is a conspicuous degree of vector specificity in that a non-circulative virus can be transmitted by several species of one insect family but not by members of another insect family. For example, *Cucumber mosaic virus* (CMV) is transmitted by at least 80 aphid species (Zitter & Murphy, 2009), but by no whitefly species. This suggests that non-circulative transmission is not the result of the non-specific contamination of vector mouthparts. Many non-circulative viruses use their capsid for binding to the vector mouthparts. The reason for this is certainly that the capsid protects the viral genome from potential dangers that exist throughout the transmission process including unfavorable environments in the mouthparts and that epitopes unique to coat proteins can serve as specific vector binding sites. For example, specific regions in the coat protein of CMV are determinants of capsid stability and/or vector interaction (Perry *et al.*, 1998; Bricault & Perry, 2013). Capsid determinants of transmission have also been identified for other non-circulative viruses (Table 2). While many viruses bind like CMV directly, others bind indirectly, relying for this on viral encoded non-structural proteins, so-called Helper Components (HCs). HCs contain vector-binding and virion-binding regions that function as molecular bridges to mediate virion binding to vector ligands (Pirone & Blanc, 1996). The HCs identified in various potyviruses and the caulimovirus *Cauliflower mosaic virus* (CaMV) have been best studied (Table 2). A remarkable feature of helper-mediated transmission is that virions may exist in two forms, either complexed to the HC or not. Furthermore, it has been shown that transmissible complexes can form in infected plant cells, before or at the time of acquisition, or in the vector. Since HCs bind to vectors independently of virions, sequential acquisition of HC and virions is possible. This enables the phenomenon called HC-transcomplementation (Froissart *et al.*, 2002), where a HC acquired initially by the vector assists in the transmission of virions located in the

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same cell, in other cells, or even in other host plants probed by the vector. HC-transcomplementation has been speculated to increase diversity of the transmitted virus population because it favors assembly of transmission units from different plant sites (Froissart *et al.*, 2002). However, it might just as well serve to economize cell resources by forming transmission units only when they are needed, i.e. when vectors probe on plants, leaving the virions and HCs available for accomplishing other steps in the replication cycle the rest of the time.

After binding to still unknown ligands and retaining in the stylets or foreguts following acquisition feeding, non-circulative viruses must be inoculated into a new host to complete the transmission cycle. The conceptual problem is that the retention sites are also the sites from which virions are inoculated, and must be compatible with binding and release. The current hypothesis is that different biochemical conditions (pH, redox potential, fluid composition) in ingested saliva/plant sap and secreted saliva favor virus binding and release, respectively. This theory provides a credible framework for explaining the inoculation of virions bound to the common canal (located at the stylet tips) formed by the convergence of the food and the salivary canals that run parallel to each other (Uzest *et al.*, 2007). This common region is constantly exposed to fluids whose constituents include inward streaming (ingested) virus-containing plant sap or outward streaming saliva. The situation is less well understood for foregut-borne viruses like whitefly-transmitted criniviruses (Chen *et al.*, 2011; Ng, unpublished). It would seem less dependable for alternating ingestion/salivation processes to mediate the transmission of such viruses since the virions are localized way posterior to the junction of the food and the salivary canals. For a more in-depth treatise on this subject, readers are encouraged to refer to the work by Zhou *et al.* (2017) in this special issue.

The second binding sites for acquired virions are located deeper in the vector's digestive system, in the mid- and the hindgut (Brault *et al.*, 2010). Here, available immobilization times can be much

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longer, but the virions must resist the lytic conditions in the gut lumen. Furthermore, the digestive tube is a one-way flow system towards the anus and the retention sites cannot function as launch pads for virus inoculation. Instead, virions must traverse the gut epithelia into the body cavity (hemocoel) and invade the salivary glands. From there, virions are inoculated together with saliva into a new host. This transmission mode is named persistent transmission (Watson & Roberts, 1939) or circulative transmission (Kennedy *et al.*, 1962), because the viruses cycle through the vector and vectors remain transmitters for long periods of time (Table 1).

Clearly, circulative transmission requires highly specific virus-insect interactions. The viral capsid is a part of the virion that is the most exposed to the environment and also to the vector. Therefore, as with non-circulative transmission, the capsid not only protects the viral genome, but it must also facilitate virion passage through the vector. Indeed, motifs on capsid and capsid-associated proteins have been implicated in circulative transmission (Table 2). Insects use like animals besides paracellular transport receptor-mediated and receptor-independent endocytosis for the uptake of specific ligands present in the gut lumen (Schelhaas, 2010), and also for the internalization of hemocoel components by organs including the salivary glands. Electron microscopy studies have indicated that viruses hijack one or both endocytosis systems for gut-hemocoel and hemocoel-salivary glands transfer (Brault *et al.*, 2007; Ammar *et al.*, 2009). For this to happen, virions must interact specifically with endocytosis components, for example natural ligands, endocytosis receptors, adapter proteins and others. The coat proteins of specific viruses have been implicated to be involved in such interactions, but their insect targets are largely unknown (Table 2). An exception is membrane alanine aminopeptidase N, a putative gut receptor protein of the *Pea enation mosaic virus* (PEMV) (Linz *et al.*, 2015). Interestingly, this protein is also involved in endocytosis of animal enteroviruses (i.e. Delmas *et al.*, 1992), suggesting that viruses infecting phyla as different as plants,

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insects, and animals evolved independently similar strategies for internalization. A problem that endocytosed virions must avoid is degradation in lysosomes, the usual destination of endocytic vesicles. How circulative plant viruses achieve this is unknown.

A unique form of circulative transmission involves the propagation of viruses, referred to as propagative transmission. Viruses exhibiting this mode of transmission replicate in plants as well as in various vector organs. It is proposed that circulative propagative viruses are insect viruses that have acquired the ability to invade plant tissues (Nault, 1997). Thus, there are a few insect viruses such as *Nilaparvata lugens virus* (NLRV) that can be insect-to-insect-transmitted through plant tissue, but do not infect the plant (Nakashima & Noda, 1995).

Taken together, an overarching theme concerning the molecular virus-vector determinants of transmission appears to be one in which the virus capsid or capsid-associated proteins are involved in controlling acquisition, retention and inoculation for both non-circulative and circulative virus transmission, while corresponding contributions from the vector constitute different, and still very poorly understood, insect components. One fundamental distinction between non-circulative and circulative transmission is that extracellular cuticular factors in the mouthparts or the foregut are used for non-circulative acquisition/retention, while intracellular components are used for circulative acquisition/retention. Saliva, on the other hand, is involved in the inoculation of all circulative and most non-circulative viruses, with the situation for foregut-borne viruses being less clear. Why do two very different transmission modes exist? The answers may fall beyond our complete understanding. However, we are clear about some of the benefits that the viruses derive from these systems. For example, the short time that non-circulative viruses spend retaining in their vectors might be counteracted by the benefit of their immediate transmissibility. In addition, unlike for circulative viruses, which are dependent on closely related vector species for transmission, the

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much lower vector specificity of non-circulative viruses enhances their chances to encounter a suitable vector. Thus, non-circulative and circulative transmission might represent generalist and specialist strategies, respectively.

Viruses modify vector performance and behavior to enhance transmission

The continuum of processes occurring during insect-mediated plant virus transmission (Ng & Zhou, 2015) extends beyond the mere molecular contacts between the virus, vector, and plant host. They consider as well how virus infection in plants alter insect vector behaviors associated with host plant selection. Host plant selection by insects is a complex process involving different stimuli and responses (Fereres & Moreno, 2009). This process is best-described for aphids, but it basically applies to other species of the orders *Hemiptera* or *Thysanoptera* as well. The process comprises four steps: (1) visual and chemical attraction to a host plant, (2) alighting and initial assessment of the leaf surface, (3) probing on epidermis and mesophyll tissues, and (4) sustained feeding of phloem sap. Each of these steps can be influenced by the physiological status of the plant, and since viral infection changes plant physiology, it can, in principle, also change host selection by insects. Indeed, many studies have provided evidence that viruses themselves might alter specific aspects of host plant phenotypes in ways that enhance virus transmission. This has been conceptualized in the Vector Manipulation Hypothesis (VMH) proposed by Ingwell *et al.* (2012). Plant viruses influence vector-host interactions in two different manners: (1) direct effects due to the presence of virions in the vector's body, and (2) indirect effects mediated by viral interference with the host plant. This review will focus on both, beginning with the latter.

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Indirect vector manipulation

Plant color and volatile organic compounds (VOC) can both be modified by virus infection and, in turn, alter the initial attractiveness of the infected plants to vectors (Ferreles & Moreno, 2009). Then, after alighting, insects probe the infected plants for quality as hosts and either commit to staying and colonizing, or leave. Theoretical considerations suggest that both non-circulative and circulative viruses would enhance vector attraction to infected plant hosts as a strategy to improve virus acquisition efficiency. However, once attracted, it is reasonable to surmise that non-circulative and circulative viruses would require different feeding arrestment periods and behaviors in their insect vectors to mediate optimal pathogen spread (Mauck *et al.*, 2012). Rapid degradation and low nutrition quality of plants would discourage long-term settlement and induce quick vector dispersal from the deteriorating plant. For non-circulative viruses, this would increase transmission efficiency because one feature of non-circulative transmission is that vectors acquire viruses quickly, but retain the transmission ability only for brief periods after probing on infected plants (Hodge & Powell, 2008). In contrast, this would clearly be a disadvantage for circulative viruses where acquisition periods are considerably longer. Therefore, the decline in plant quality should be slower for circulative virus-infected plants to favor sustained feeding on the infected host (Mauck *et al.*, 2012).

What evidence exists that supports or refutes the models of non-circulative virus-induced changes (in plants) on vector manipulation? In free-choice assays, *Acyrtosiphon pisum* Harris aphids settled preferentially on leaf disks infected with *Bean yellow mosaic virus* (BYMV) compared to healthy disks (Hodge & Powell, 2008). *Aphis gossypii* Glover alates preferred plants infected with CMV over mock-inoculated plants up to 30 minutes after insect release, but later aphids changed their behavior and settled on healthy plants. This behavior can be correlated with the initial acquisition of virions from infected plants by aphids, followed by the migration of these viruliferous aphids and their

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inoculation of virions to healthy plants (Carmo-Sousa *et al.*, 2014). The soybean aphid *Aphis glycines* Matsumura was attracted to plants infected with *Soybean mosaic virus* (SMV) for up to 60 minutes (Peñaflor *et al.*, 2016). Within this period, aphids initiated sustained feeding on SMV-infected plants more so than on mock-inoculated plants, a behavior disadvantageous to transmission of non-circulative viruses like SMV (Hodge & Powell, 2008). By contrast, SMV-infected soybean plants did not attract aphids *Myzus persicae* Sulzer and *Rhopalosiphum maidis* Fitch, but the latter remained for a shorter period on the infected plants before taking off. This potentially increases the probability of probing on another healthy host before the vector loses transmissibility of the virus (Ferrerres *et al.*, 1999). These and other examples (Table 3A) might seem controversial, but they clearly reflect on the complexity of specific plant-virus-vector interactions and their impacts on virus transmission.

Evidence of aphid attraction to circulative virus-infected plants was mostly obtained from studies on viruses from the family Luteoviridae (Table 3B). The emerging picture gathered from these studies appears to be more cohesive than that of non-circulative viruses. Thus, *M. persicae* settling on potato leaves was significantly more marked on plants infected by the circulative *Potato leafroll virus* (PLRV) than on plants infected by the non-circulative *Potato virus Y* (PVY), the non-vectorized *Potato virus X* (PVX), or on virus-free plants (Castle *et al.*, 1998). In separate studies, olfactometer tests and emigration bioassays showed again the preference of *M. persicae* for PLRV-infected potato leaves over non-infected ones. The attraction increased over time for up to 60 minutes after aphid release, and was correlated to VOCs released by the plants (Eigenbrode *et al.*, 2002; Alvarez *et al.*, 2007; Werner *et al.*, 2009; Rajabaskar *et al.*, 2013). Attraction to infected plants, assisted by VOC, was also found for other *Luteoviridae*-aphid pathosystems (Table 3B), strongly suggesting that this

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phenomenon is characteristic for circulative viruses, or, at minimum, for members of the *Luteoviridae*.

Considering vector fitness, the model outlined above predicts that non-circulative viruses reduce the quality of the host and this, in turn, imposes adverse effects on aphid fitness. In support of this, infection by *Turnip mosaic virus* (TuMV) of Chinese cabbage shortened the life of *Brevicoryne brassicae* L. colonies, prolonged the nymphal period, and reduced the live weight of adults (Hodgson, 1981). Furthermore, Mauck *et al.* (2014) showed that CMV infection decreased the nutritional value of infected plants, but concurrently made the plants more attractive for vectors by changing VOC and ethylene production, thereby encouraging an attraction/deterrence behavior to favor transmission.

However, beneficial effects of non-circulative virus infection were also reported (Table 3A). Nymphs of *M. persicae* reached maturity faster, and adults were heavier and deposited larvae more frequently on TuMV-infected Chinese cabbage (Hodgson, 1981). Increased fitness of *M. persicae* was also observed on TuMV-infected tobacco and *Arabidopsis* (Casteel *et al.*, 2014; 2015), and of *A. gossypii* on *Zucchini yellow mosaic virus* (ZYMV)-infected squash plants (Blua & Perring, 1992). Interestingly, in this case aphid fitness changed with infection stage. Fitness was higher on recently infected plants than on older infected plants, concomitant with rejection of the older plants and conducive with transmission. Thus, as with the attractiveness studies, apparently contradictory results have been reported from studies on the effect of non-circulative virus infection on vector fitness.

In contrast with non-circulative viruses, it seems beneficial for circulative viruses to augment aphid fitness by increasing the plant host's quality as a strategy to prolong feeding arrestment and optimize virus acquisition. Indeed, *M. persicae* showed significantly increased growth rates and

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intrinsic rates of natural increase on PLRV-infected potato plants than on uninfected ones (Castle & Berger, 1993). The same response, i.e. better vector fitness, has been reported for other viruses (Table 3B). Contrasting results have been reported as well. Fiebig *et al.* (2004) found that BYDV negatively affected *Sitobion avenae* Fabricius development. However, that study also showed that more alate insects developed on the infected plants. Since alates potentially spread BYDV better than wingless forms, this possibly neutralizes the negative impact. Thus, most results support the general notion that circulative viruses increase vector fitness, at least in the case of aphid vectors. Less data on the influences of virus infection (in plants) on vector fitness are available for other hemipteran vectors. The results provided in the few available reports (Table 3B) are all in line with those of aphid vectors, which is not surprising given the similarities in feeding behaviors among phytophagous hemipterans. It should be noted that better performance of vectors on infected plants might not always be specific for a pathosystem, since increased fitness has also been reported for the chewing-biting Mexican bean beetle (*Epilachna varivestis* Mulsant) feeding on bean plants infected with *Southern bean mosaic virus* (SBMV, which it vectors) or with BPMV (which it does not transmit) (Musser *et al.*, 2003).

Taken together, it seems that circulative viruses show more consistent effects on attraction and fitness of vectors than do non-circulative viruses. This could be attributed to the intimate and more specific/sustained relationships between circulative viruses and their vectors, which might not allow large variations of the parameters controlling transmission. Non-circulative virus-vector interactions are transient and less specific, and other interaction parameters that are usually not considered by the general models might greatly impact non-circulative transmission. Plant longevity is an example of one such parameter that can play a role on vector fitness. Hily *et al.* (2014) showed that CMV-infected *Arabidopsis* plants with a short-lived genotype supported larger *M. persicae* populations

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and were more susceptible to infection than plants with a long-lived genotype, with consequences for their role as pathogen reservoirs.

Another parameter impacting not only transmission but also vector fitness, is insect feeding behavior. Using Electrical Penetration Graph (EPG) technique, which analyzes tissue penetration and sap ingestion by vector mouthparts in real time, several authors have identified modified probing behavior of aphids that are potentially associated with the enhanced transmission of non-circulative viruses. *Myzus persicae* probed more frequently on PVY-infected pepper plants, and transmission of PVY was associated with shorter than average probes during acquisition feeding (Collar *et al.*, 1997). Carmo-Sousa *et al.* (2014) observed that *A. gossypii* made more intracellular probing punctures on CMV-infected cucumbers. At the same time, the aphids showed increased phloem feeding deterrence. An implicit explanation for these observations is that non-circulative viruses are lost when aphids are allowed to probe, after virus acquisition, the phloem on the same plant (Ferreles & Moreno, 2009).

The modification of vector feeding behavior by circulative viruses has also been observed. *Schizaphis graminum* Rondani aphids on BYDV-infected oats displayed increased feeding activity, characterized by a shorter time before phloem ingestion and an increase in ingestion duration, whereas *R. padi* did not (Montllor & Gildow, 1986). EPG results obtained for *M. persicae* on PLRV-infected or healthy potato plants suggested a reduction in plant resistance to stylet penetration on infected plants, with fewer numbers of short duration probes before the first phloem activity (Alvarez *et al.*, 2007). Similar results were obtained in other pathosystems (Table 3B). Most studies support the view that virus infection incites phloem feeding, which would speed up virus acquisition of the often phloem-restricted circulative viruses.

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Collectedly, this short review section of the literature shows that there is reasonable evidence that infection in plants by circulative and non-circulative viruses tends toward the specific alterations of insect feeding behaviors that impact the transmission of specific viruses.

Direct modification of the behavior of viruliferous vectors

Besides plant-mediated indirect modification of insect behaviors and performance, studies have also documented that the sole presence of viruses in insect vectors is associated with changes in the vectors' behaviors that are amenable to transmission. For example, BYDV-viruliferous *R. padi* preferred to feed on non-infected wheat plants, while non-viruliferous aphids preferred BYDV-infected plants (Medina-Ortega *et al.*, 2009; Ingwell *et al.*, 2012). Similar observations were made for the *M. persicae*/PLRV (Rajabaskar *et al.*, 2014) and *A. gossypii*/CABYV pathosystems (Carmo-Sousa *et al.*, 2016). This differential behavior, depending on the virus-carrying status of the aphid, may promote pathogen spread since the non-viruliferous vectors' preference for infected plants and the viruliferous vectors' preference for non-infected plants tend towards the promotion of virus acquisition and inoculation, respectively. At least in some cases the altered preferences might be mediated by volatiles (Ngumbi *et al.*, 2007; Medina-Ortega *et al.*, 2009). Studies on the direct modification of behavior in other viruliferous vector species yielded similar results as for aphids (Table 3C).

Thus, many examples show that circulative viruses modify vector behavior and it is easily imaginable that viruses do so by interfering/interacting with insect components during their passage through the vector. Although the mechanisms underpinning these associations are unknown, possible vector targets could be localized in the gut, the salivary glands, and even the nervous system. It is not assumed that the presence of non-circulative viruses in insects changes behaviors,

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but it is principally imaginable that they, too, interact with ligands lining the mouthparts or the intestine and thereby alter feeding behavior. Evidence for this might come from the work by Collar *et al.* (1997) who reported that viruliferous aphids transmitting PVY from infected pepper plants had different feeding behavior than those that did not transmit the virus from the same plants. Since not all cells are infected in PVY-infected leaves, one could speculate that the feeding behavior changed when aphids probed infected cells, possibly due to a viral component or a virus-induced plant factor. Another example are *Bemisia tabaci* Gennadius whiteflies viruliferous with the non-circulative, semi-persistent *Cucurbit chlorotic yellows virus* that showed different behaviors than virus-free whiteflies (Lu *et al.*, 2017).

Overall, substantial data on viruses' manipulation of insects and plants have been obtained. They show clearly that viruses may interfere at all stages of insect-plant interactions to enhance transmission. These 'manipulations' are extremely different from one virus-plant-insect pathosystem to another. However, it seems that circulative viruses modify different plant-insect pathosystems more similarly than non-circulative viruses do. A challenge for the future will be to reveal the mechanisms behind these, especially in regards to how the vector's preferences are modulated in the vector itself.

Plant defense-related pathways involved in virus transmission

Plant defense responses against herbivores such as callose deposition, phloem clogging, and VOC synthesis, render plants unattractive or inaccessible for insects, and deter them from feeding. This is not advantageous for plant viruses that depend on insect vectors for transmission. However, viruses

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and insect vectors have the capacity to engage each other in synergism and beneficial mutualism to manipulate plant defense responses to their benefit.

Typical plant defense against herbivorous insects follows the two-staged 'Zigzag' model that was initially proposed for the defense against microbes (Jones & Dangl, 2006). These plant defenses rely on recognition by the plant of broad-specificity pathogen-associated molecular patterns (PAMPs) in the first step, pathogen-triggered immunity (PTI), and on that of highly specific pathogen molecules in the second step, effector-triggered immunity (ETI). They drive fast defense responses like reactive oxygen bursts and callose deposition and slower defense responses that by mediation of phytohormones propagate throughout the plant and install systemically acquired resistance.

Viruses can interfere with these defense systems and decrease plant defenses against insects, and, in so doing, increase plant quality for the vectors and vector fitness. The rationale is that better insect survival will increase the number of vectors loaded with virus, thus promoting virus transmission. One example of a pathogen effector is CMV protein 2b that tampers with jasmonic acid signaling (Lewsey *et al.*, 2010) to possibly promote aphid infestation and transmission (Ziebell *et al.*, 2011; Wu *et al.*, 2017). In another example, Casteel *et al.* (2014) demonstrated that TuMV infection suppresses callose deposition and increases aphid fecundity on tobacco and Arabidopsis plants. A single conserved TuMV protein, NIa-Pro, was found to suppress the resistance against aphids. Concomitantly, the NIa-Pro-associated interference of the plant defense system was accompanied by modified ethylene signaling (Casteel *et al.*, 2015). The underlying mechanisms remain unknown. More is known about *Tomato yellow leaf curl China virus* (TYLCCV)–*B. tabaci* interaction. Here the viral satellite-coded β C1 protein interacts with the host protein AS1 to suppress a set of jasmonic acid responses. This correlates with an increase in fitness of whiteflies (Zhang *et al.*, 2012). β C1 interacts

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also with the host transcription factor MYC2 and reduces the synthesis of toxic terpenes, again boosting the survival of whiteflies (Li *et al.*, 2014).

There is yet another perspective when considering the interactions associated with virus transmission, and that is the insect vectors themselves. Their interactions with plant defenses might pave the way for viral infection and transmission. For example, some insect- or tick-transmitted animal viruses are more efficiently transmitted by ticks or by artificial inoculation with saliva-coated needles than by inoculation with uncoated needles. This phenomenon, termed 'saliva-activated transmission' (Nuttall *et al.*, 2000), is due to the tampering of saliva proteins with the immune system (i.e. Thangamani *et al.*, 2010; Lieskovská *et al.*, 2015). For the transmission of plant viruses, the interference of plant defenses by effector proteins in the saliva of insect vectors may be thought of as an equivalent of saliva-activated transmission used to facilitate virus inoculation. Several studies have alluded to this possibility. In a study by Will *et al.* (2007), an unknown factor in aphid saliva was found to prevent sieve tube clogging; it is tempting to speculate that this or other factors might also participate in the inoculation of viruses. Saliva components are also assumed to modify PTI, ETI and other defense responses, but hardly any candidates are known (reviewed by Kaloshian & Walling, 2016; van Bel & Will, 2016) and their possible role in facilitating virus transmission is even less explored. On a promising note, there are ongoing efforts to analyze the roles of aphid saliva in plant defense mitigation (i.e. Jaouannet *et al.*, 2014; Kettles & Kaloshian, 2016; Mugford *et al.*, 2016; Rodriguez *et al.*, 2017) and we are hopeful that specific gaps in knowledge might soon be filled. Taken together, plant defenses may play a prominent role in virus transmission. This role is mostly indirect and involves changing plant and insect performances to favor transmission. However, there is recent evidence on a possible direct role of plant defenses in transmission. The non-circulative CaMV responds to aphid probing by inducing within seconds transmissible virus forms that are

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acquired by the insects (Bak *et al.*, 2013; Martinière *et al.*, 2013). Likewise, the potyviral NIa-Pro protein relocates transiently to vacuoles when aphids probe on TuMV-infected leaves and this correlates with inhibition of plant defenses (Bak *et al.*, 2017). It has been suggested that early steps of plant defense against aphids are somehow deviated by these viruses to enable these reactions.

Outlook

Transmission is the result of multi-layered interactions at each step of the transmission process (Fig. 1). In the beginning, virus-modified plant parameters (volatiles, color, nutrients, etc) may attract/deter vectors, then viral transmission-specific proteins and, in some cases, vector-induced formation of transmission morphs may control the acquisition step. Subsequently, again with the influences of virus-modified plant parameters (phloem composition, volatiles, etc), vector fitness and behaviors may be altered to determine the duration of virus acquisition and to encourage the vector to seek out a new host. Finally, viruses may modify vector performance directly, by interacting with vector factors, to guide transmission. And, to complete the cycle (Fig. 1), insects could interact with plant defenses during plant probing and infestation to facilitate viral infection.

Despite the large body of evidence documenting changes in infected plants and vectors that are related to transmission, the molecular determinants, especially on the plant and vector sides are only beginning to be revealed. It is a challenge for the future to identify these pathways and mechanisms, for pure scientific pleasures but also because their knowledge will help to develop new virus and insect control strategies.

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Disclosure

The authors have declared that no competing interests exist.

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Table 1 Characteristics of the different viral transmission strategies by vectors (with data from Gray & Banerjee, 1999; Brault *et al.*, 2010; Whitfield *et al.*, 2015).

Transmission mode	Non-circulative (non-persistent, semi-persistent)		Circulative (persistent)	
Transmission strategy	Capsid	Helper	Non-propagative	Propagative
Acquisition time	Seconds to minutes		Hours to days	
Latency period	None		Hours to days	Days to months
Inoculation time	Seconds to minutes		Minutes to hours	
Retention time	Minutes to hours or days		Days to lifetime	Lifetime
Retention place for inoculation	Stylets and foregut		(Accessory) salivary glands	
Virus retained after molt	No		Yes	
Multiplication in the vector	No		No	Yes
Transmission to the progeny	No		Sometimes	
Acquisition from	Epidermis, mesophyll, phloem		Phloem	
Inoculation into	Epidermis, mesophyll, sometimes xylem		Epidermis, mesophyll, phloem	
Particularity		Allows transcomplementa		Insects and plants are virus hosts

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		tion		
Examples (virus)	<i>Cucumovirus</i> , <i>Alfamovirus</i> , <i>Crinivirus</i>	<i>Potyvirus</i> , <i>Caulimovirus</i> , <i>Waikavirus</i>	<i>Luteovirus</i> , <i>Polerovirus</i> , <i>Begomovirus</i>	<i>Phytoreovirus</i> , <i>Fijivirus</i> , <i>Oryzavirus</i>
Examples (insects)	Aphids, whiteflies, planthoppers	Aphids, whiteflies, leafhoppers	Aphids, whiteflies, beetles	Aphids, planthoppers, thrips

Table 2 Examples of viral proteins involved in vector interaction. Only proteins for which virus-vector interaction domains have been characterized are listed.

Virus	Family	Vector	Helper	Vector Interaction	Reference
Non-circulative viruses					
<i>Cucumber mosaic virus</i>	<i>Bromoviridae</i>	Aphids	No	Coat protein	Chen & Francki, 1990; Perry <i>et al.</i> , 1998; Ng <i>et al.</i> , 2000
<i>Lettuce infectious yellows virus</i>	<i>Closteroviridae</i>	Whiteflies	No	Minor coat protein	Tian <i>et al.</i> , 1999; Chen <i>et al.</i> , 2011
<i>Tobacco vein mottling virus</i> , <i>Potato virus Y</i> , <i>Tobacco etch virus</i>	<i>Potyviridae</i>	Aphids	Yes	HC-Pro	Atreya <i>et al.</i> , 1992; Wang <i>et al.</i> , 1996; Blanc <i>et al.</i> , 1998; Peng <i>et al.</i> , 1998
<i>Cauliflower mosaic virus</i>	<i>Caulimoviridae</i>	Aphids	Yes	P2	Woolston <i>et al.</i> , 1987; Moreno <i>et al.</i> , 2005; Leh <i>et al.</i> , 1999
Circulative viruses					
<i>Barley yellow dwarf virus</i>	<i>Poleroviridae</i>	Aphids	No	Coat protein, Readthrough protein	Chay <i>et al.</i> , 1996
<i>Beet western yellow virus</i> , <i>Potato leaf roll virus</i>	<i>Poleroviridae</i>	Aphids	No	Coat protein, Readthrough protein	Reinbold <i>et al.</i> , 2001; Peter <i>et al.</i> , 2008

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Table 2 Examples of viral proteins involved in vector interaction. Only proteins for which virus-vector interaction domains have been characterized are listed.

Virus	Family	Vector	Helper	Vector Interaction	Reference
<i>Abutilon mosaic virus</i>	<i>Geminiviridae</i>	Whiteflies	No	Coat protein	Höhnle <i>et al.</i> , 2001
<i>Potato yellow dwarf virus</i>	<i>Rhabdoviridae</i>	Leafhopper	Yes	Coat protein G	Gaedigk <i>et al.</i> , 1986
<i>Rice dwarf virus, Wound tumor virus</i>	<i>Reoviridae</i>	Leafhopper	Yes	Coat proteins P2 and P8, non-structural protein Psn4	Omura & Yan, 1999; Chen <i>et al.</i> , 2015
<i>Tomato spotted wilt virus</i>	<i>Bunyaviridae</i>	Thrips	Yes	Coat protein G _N , Non-structural protein NSs	Whitfield <i>et al.</i> , 2008; Margaria <i>et al.</i> , 2014

Table 3 Indirect and direct effects of viruses on behavior and performance of insect vectors.

Virus	Family	Vector	Host	Vector response	Reference
(A) Indirect effects of non-circulative viruses					
<u>Vector settlement</u>					
<i>Bean yellow mosaic virus</i>	<i>Potyviridae</i>	<i>A. pisum</i>	<i>V. faba</i>	+	Hodge & Powell, 2008
<i>Cucumber mosaic virus</i>	<i>Bromoviridae</i>	<i>A. gossypii</i>	<i>C. sativus</i>	+, up to 30 min	Carmo-Sousa <i>et al.</i> , 2014
<i>Turnip mosaic virus</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>N. benthamiana</i>	+	Casteel <i>et al.</i> , 2015
<i>Soybean mosaic virus</i>	<i>Potyviridae</i>	<i>A. glycines</i>	<i>G. max</i>	+, up to 60 min	Peñaflor <i>et al.</i> , 2016
<u>Vector arrestment</u>					
<i>Soybean mosaic virus</i>	<i>Potyviridae</i>	<i>R. maidis</i>	<i>G. max</i>	–	Fereres <i>et al.</i> , 1999
<i>Cucumber</i>	<i>Bromoviridae</i>	<i>A. gossypii</i>	<i>C. sativus</i>	–	Carmo-Sousa <i>et al.</i> , 2014

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<i>mosaic virus</i> <i>Soybean mosaic virus</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>G. max</i>	0	Fereres <i>et al.</i> , 1999
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Vector fitness

<i>Turnip mosaic virus</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>B. rapa</i>	+	Hodgson, 1981
<i>Zucchini yellow mosaic virus</i>	<i>Potyviridae</i>	<i>A. gossypii</i>	<i>C. pepo</i>	+	Blua & Perring, 1992
<i>Turnip mosaic virus</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>A. thaliana</i>	+	Casteel <i>et al.</i> , 2014, 2015
<i>Turnip mosaic virus</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>N. benthamiana</i>	+	Casteel <i>et al.</i> , 2015
<i>Turnip mosaic virus</i>	<i>Potyviridae</i>	<i>B. brassicae</i>	<i>B. rapa</i>	-	Hodgson, 1981
<i>Wheat streak mosaic virus</i>	<i>Potyviridae</i>	<i>S. graminum</i>	<i>T. aestivum</i>	-	Michels <i>et al.</i> , 1994
<i>Alfalfa mosaic virus</i>	<i>Bromoviridae</i>	<i>A. glycines</i>	<i>G. max</i>	-	Donaldson & Gratton, 2007
<i>Bean pod mottle virus</i>	<i>Secoviridae</i>	<i>A. glycines</i> (non-vector)	<i>G. max</i>	-	Donaldson & Gratton, 2007
<i>Soybean mosaic virus</i>	<i>Potyviridae</i>	<i>A. glycines</i>	<i>G. max</i>	-	Peñaflor <i>et al.</i> , 2016
<i>Bean pod mottle virus</i>	<i>Secoviridae</i>	<i>E. varivestis</i>	<i>P. vulgaris</i>	+	Musser <i>et al.</i> , 2003
<i>Southern bean mosaic virus</i>	<i>Sobemovirus</i>	<i>E. varivestis</i>	<i>P. vulgaris</i>	+	Musser <i>et al.</i> , 2003

Vector feeding behavior

<i>Potato Virus Y</i>	<i>Potyviridae</i>	<i>M. persicae</i>	<i>C. annuum</i>	+	Collar <i>et al.</i> , 1997
<i>Cucumber mosaic virus</i>	<i>Bromoviridae</i>	<i>A. gossypii</i>	<i>C. sativus</i>	+	Carmo-Sousa <i>et al.</i> , 2014

(B) Indirect effects of circulative virusesVector settlement

<i>Potato leaf roll virus</i>	<i>Poleroviridae</i>	<i>M. persicae</i>	<i>S. tuberosum</i>	+	Castle <i>et al.</i> , 1998; Eigenbrode <i>et al.</i> , 2002; Alvarez <i>et al.</i> , 2007; Werner <i>et al.</i> , 2009; Rajabaskar <i>et al.</i> , 2013
<i>Barley yellow dwarf virus</i>	<i>Poleroviridae</i>	<i>R. padi</i>	<i>T. aestivum</i>	+	Jiménez-Martínez <i>et al.</i> , 2004b

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Tomato spotted wilt virus	Bunyaviridae	<i>F. occidentalis</i>	<i>C. annuum</i>	+	Maris <i>et al.</i> , 2004
Pea enation mosaic virus	Poleroviridae	<i>A. pisum</i>	<i>V. faba</i>	+	Hodge & Powell, 2008
Bean leaf roll virus	Poleroviridae	<i>A. pisum</i>	<i>P. sativum</i>	+	Wu <i>et al.</i> , 2014
Pea enation mosaic virus	Poleroviridae	<i>A. pisum</i>	<i>P. sativum</i>	+	Wu <i>et al.</i> , 2014

Vector fitness

Potato leaf roll virus	Poleroviridae	<i>M. persicae</i>	<i>S. tuberosum</i>	+	Castle & Berger, 1993
Barley yellow dwarf virus	Poleroviridae	<i>S. avenae</i>	<i>T. aestivum</i>	+	Fereres <i>et al.</i> , 1989
Barley yellow dwarf virus	Poleroviridae	<i>S. graminum</i>	<i>A. sativa</i>	+	Montllor & Gildow, 1986
Barley yellow dwarf virus	Poleroviridae	<i>R. padi</i>	<i>T. aestivum</i>	+	Jiménez-Martínez <i>et al.</i> , 2004a
Tomato spotted wilt virus	Bunyaviridae	<i>F. occidentalis</i>	<i>C. annuum</i>	+	Maris <i>et al.</i> , 2004
Bean leaf roll virus	Poleroviridae	<i>A. pisum</i>	<i>P. sativum</i>	+	Wu <i>et al.</i> , 2014
Tomato yellow leaf curl virus	Begomovirus	<i>B. tabaci</i>	<i>S. lycopersicum</i>	+	Maluta <i>et al.</i> , 2014
Barley yellow dwarf virus	Poleroviridae	<i>S. avenae</i>	<i>T. aestivum</i>	-	Fiebig <i>et al.</i> , 2004

Vector feeding behavior

Barley yellow dwarf virus	Poleroviridae	<i>S. graminum</i>	<i>A. sativa</i>	+	Montllor & Gildow, 1986
Potato leaf roll virus	Poleroviridae	<i>M. persicae</i>	<i>S. tuberosum</i>	+	Alvarez <i>et al.</i> , 2007
Cucurbit aphid-borne yellows virus	Poleroviridae	<i>A. gossypii</i>	<i>C. sativus</i>	+	Carmo-Sousa <i>et al.</i> , 2016
Barley yellow dwarf virus	Poleroviridae	<i>R. padi</i>	<i>A. sativa</i>	0	Montllor & Gildow, 1986

(C) Direct effects of circulative viruses

Vector settlement

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Barley yellow dwarf virus	Poleroviridae	<i>R. padi</i>	<i>T. aestivum</i>	+	Medina-Ortega <i>et al.</i> , 2009; Ingwell <i>et al.</i> , 2012; Rajabaskar <i>et al.</i> , 2014
Potato leaf roll virus	Poleroviridae	<i>M. persicae</i>	<i>S. tuberosum</i>	+	Rajabaskar <i>et al.</i> , 2014
Cucurbit aphid-borne yellows virus	Poleroviridae	<i>A. gossypii</i>	<i>C. sativus</i>	+	Carmo-Sousa <i>et al.</i> , 2016
Soybean vein necrosis virus		<i>N. variabilis</i>	<i>G. max</i>	+	Keough <i>et al.</i> , 2016
Southern rice black-streaked dwarf virus	Reoviridae	<i>Sogatella furcifera</i>	<i>Oryza sativa</i>		Lu <i>et al.</i> , 2016

Vector arrestment

Tomato yellow leaf curl virus	Geminiviridae	<i>B. tabaci</i>	<i>S. lycopersicum</i>	+	Moreno-Delafuente <i>et al.</i> , 2013
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Vector fitness

Tomato yellow leaf curl virus	Geminiviridae	<i>B. tabaci</i>	<i>S. lycopersicum</i>	+	Maluta <i>et al.</i> , 2014
Soybean vein necrosis virus	Bunyaviridae	<i>N. variabilis</i>	<i>G. max</i>	+	Keough <i>et al.</i> , 2016

Vector feeding behavior

Tomato spotted wilt virus	Bunyaviridae	<i>F. occidentalis</i>	<i>D. stramonium</i>	+	Stafford <i>et al.</i> , 2011
Tomato yellow leaf curl virus	Geminiviridae	<i>B. tabaci</i>	<i>S. lycopersicum</i>	+	Moreno-Delafuente <i>et al.</i> , 2013

+: positive, -: negative, 0: neutral effect.

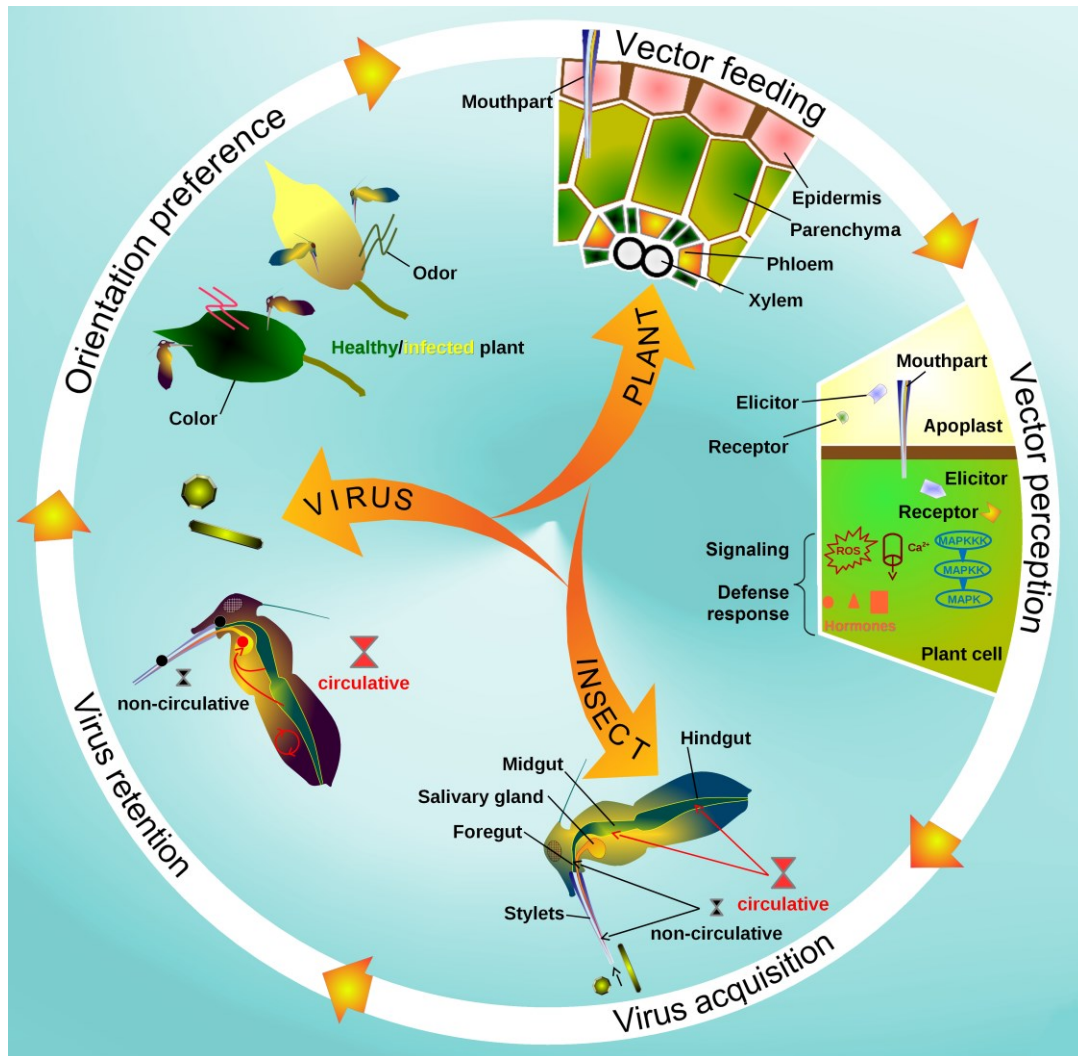
Legend to figure

Fig. 1 The transmission cycle. Virus-free insect vectors are more attracted to infected plants than to healthy ones. This is due to plant color, odor, metabolism etc. altered by viruses, which provide positive or negative cues for subsequent virus acquisition during insect feeding. Again, acquisition is

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often facilitated by viral modifications of the plants promoting 'acquisition-friendly' feeding behavior, *e.g.* longer feeding for circulative and deterrence for non-circulative viruses. For this, viruses, working on altering plant defense responses and other pathways, may interfere with the perception of insects by plants, with plant defense responses and with other pathways. At least one virus (CaMV) responds instantly to the vector's presence by forming transmission morphs. Viruses are retained in the insects after acquisition for short (non-circulative viruses) or longer (circulative viruses) periods. During retention, insects may be manipulated by viruses to promote transmission, *e.g.* encouraging viruliferous vectors to prefer healthy over infected plants or enhancing their fitness to allow for longer-lasting transmissibility. After virus inoculation in a new host, which could be assisted by vector factors, the cycle restarts.

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