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Manipulation of hosts and vectors by plant viruses and impact of the environment

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The effect of environmental factors on the efficiency of plant virus transmission is extremely difficult to predict, because they obviously impact concomitantly multiple steps of the complex three-way plant–virus–vector interaction. This review summarizes the diversity of the relationship between plants, viruses and insect vectors, and highlights the numerous phases of this process that can be altered by the virus in ways that can potentially enhance its transmission success. Many of the reported cases are often considered to be possible viral manipulations acting through modifications of the physiology of the host plant, indirectly reaching to the insect vector. Plants are extremely responsive to environmental fluctuations and so interferences with these putative viral manipulations are highly expected. The role of environmental factors in plant virus transmission can thus be envisaged solely in the context of this complexity. It is only briefly evoked here because this field of research is in its infancy and currently suffers from an impressive lack of experimental data.

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Introduction

As opposed to animals, the immense majority of plants are incapable to move away from any sudden or gradual change of their environment, nor from attacks by herbivores and pathogens. Consequently, plants have developed a large panel of constitutive or inducible protections, defenses, or more generally phenotypic plasticity, to confront and accommodate such changes. A corollary of this fact is that plants have also evolved a very sophisticated arsenal of sensory/perception systems in order to monitor all environmental periodic fluctuations and unpredictable 'anomalies' including abiotic and biotic stresses. This sophistication in plant sensory potential allows timely, diverse and specifically adapted physiological responses and, in some instances, their communication to neighbor plants via volatile emission or information transfer through soil microorganisms [1–3].

In the three-way interaction between a plant, a virus and its insect vector, one must bear in mind that both viral infection and vector feeding will rapidly induce dramatic changes in plant physiology, defensive or not, and that changes induced by viruses and vectors can either be independent, synergistic or antagonistic. The possibility for a virus to antagonize or assist the feeding, settling and development of its vector on their common host is opening the way for possible manipulations, which can ultimately potentiate transmission [4••,5,6•].

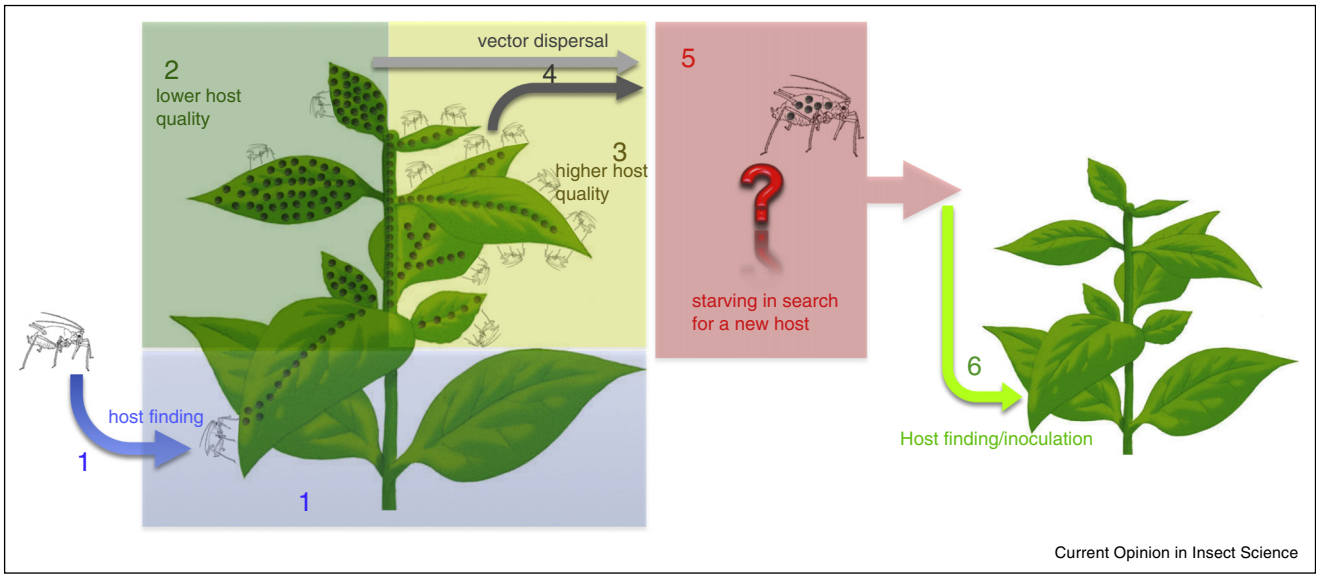
Many plant responses to biotic and abiotic stresses are interconnected [7]. Hence, when environmental factors impact on plant physiology they concomitantly interfere with plant–virus and/or plant–vector relationships and thus with the potential mechanisms by which a virus could manipulate both its host and its vector. The primary scope of this review is to summarize the different modes of vector-transmission of plant viruses [8,9], with a dedicated attention to virus-induced changes in plants and vectors that potentially increase transmission (Figure 1). Though of obvious importance, how environmental factors can modulate these changes and interfere with virus transmission is only briefly evoked because of the paucity of data in the current literature.

Different modes of insect-transmission by plant viruses

Plant viruses are transmitted by fungi, nematodes, mites, and insects [10], but insects are the only vectors for which sufficient knowledge is available on the complex interactions reviewed in this chapter.

There are few but important distinctions in the mechanisms of interaction between insect vectors and viruses of animals versus plants [11,12]. The first one is that the majority of animal arboviruses actually infect (and thus replicate in) their vectors, whereas most plant viruses do not. In fact, a minority of plant viruses, designated 'circulative propagative' and belonging to families whose members may infect either plants or animals (*Rhabdoviridae*, *Reoviridae*, *Bunyaviridae*), replicate in their insect vectors (Figure 2). These are thought to derive from insect viruses that have secondarily acquired the capacity

Figure 1



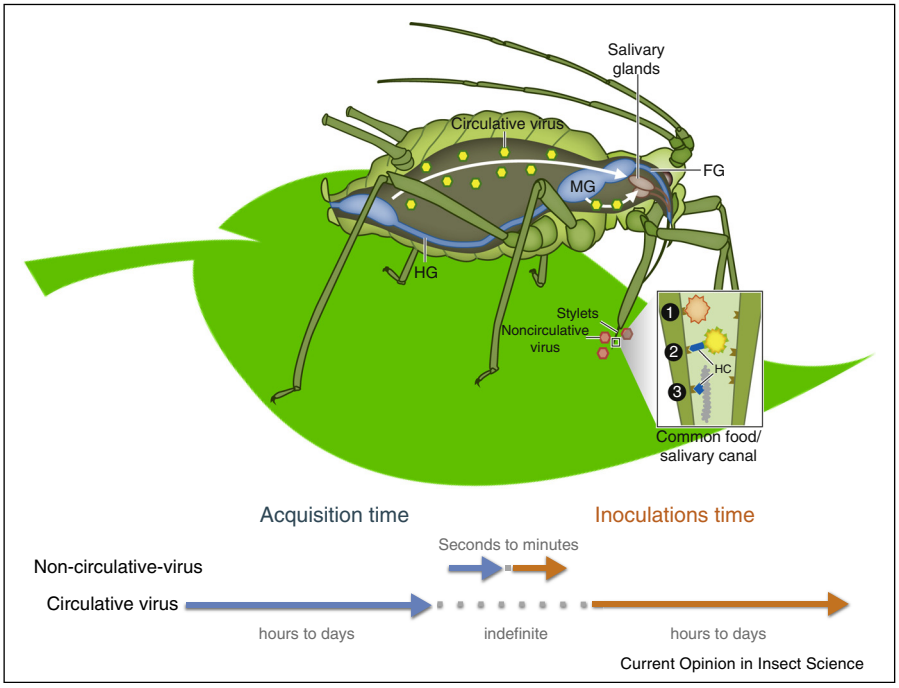
Steps in the virus transmission process affected by virus and/or the environment. This figure depicts all steps of a plant virus life cycle where the virus can affect both the host plant and the insect vectors in ways that can potentially increase transmission. 1 (lower blue arrow and rectangle): Insect vectors, here aphids, are attracted to infected plants by visual and olfactory cues. 2 (left upper green rectangle): For non-circulative viruses, the quality of the host plant can be decreased by the infection. The insect vectors rapidly acquire the virus from superficial tissues and soon leave in search for a healthy plant (light gray arrow 4). 3 (yellow upper rectangle): For circulative viruses, the quality of the host plant can be improved by the infection. The insect vectors settle, feed from deep tissues, and ingest the virus. The vector population growth is accelerated, leading to overcrowding and increased emigration in search for new host plants (dark gray arrow 4). 5 (red rectangle): This step is the journey of the viruliferous insect vectors away from any host plant. If the vector fails to find a new host, it will die together with the viruses it carries. Viruses could manipulate the motility or survival time of the insect vectors when away from any host. 6 (green arrow): contrary to the preference of virus-free vectors for infected plants (1), virus-loaded vectors are sometimes better attracted by healthy plants. Environmental factors could modify this scheme at any steps in unpredictable ways.

to replicate in and infect plants [11,13,14]. In these cases, insect vectors can be aphids, hoppers, and thrips, and their vector capacity and/or competence is conceptually identical to that amply studied for arboviruses of animals, discussed in [11,14,15].

A second marked distinction is that the 'circulative non-propagative' transmission is frequent and well characterized in plant but not reported in animal viruses [12]. All member species of the families *Luteoviridae*, *Nanoviridae* and *Geminiviridae* traverse the gut of their respective vectors to reach the hemolymph and diffuse to the salivary glands, with no detectable replication (Figure 2), except perhaps for one geminivirus species discussed in [16]. Circulative non-propagative transmission has been reported for vectors such as aphids, whiteflies, and hoppers where viruses accumulate exclusively in gut and salivary gland cells and appear excluded from any other organs [9,10,14]. It seems reasonable to assume that the vector capacity/competence, despite the absence of viral replication, is affected by environmental factors, such as temperature for instance, but limited data are available. Some studies unequivocally demonstrate that the efficiency of transmission of nanoviruses, luteoviruses and geminiviruses by aphids and whiteflies is intimately linked to temperature but the underlying mechanisms have rarely been investigated ([17,18] and references within). The influence of environmental factors on the circulative non-propagative transmission is an emerging research area further discussed in the last section.

Finally, a third distinctive category of virus–vector relationship is the so-called non-circulative transmission. This might be compared to the 'mechanical' transmission of animal arboviruses (believed to result from non-specific contamination of biting-insect mouthparts) [12], but in all well studied cases of plant non-circulative viruses, a very specific molecular interaction between unidentified receptors located in the anterior alimentary tract of the vectors and viral ligands has been evidenced. Despite these specific molecular interactions, the contact between non-circulative viruses and their insect vectors is external, limited to the cuticle lining the vectors' mouthparts or foregut [9,19] (Figure 2), and thus it may be anticipated that the virus has little opportunities to significantly directly modify vector behavior or life history traits. A legitimate question is thus whether it is relevant to

Figure 2



Different modes of vector-transmission of plant viruses. Emblematic vectors of plant viruses are aphids (represented here). Other sap-feeding insects have similar relationships with the virus they transmit and the different transmission modes depicted here apply to all insect vectors. Gut and salivary glands are represented in blue and brown, respectively. The green hexagons represent circulative viruses following a route from mid- or hind-gut to salivary glands through the hemolymph (white arrows). During this cycle in the vector body, non-propagative viruses accumulate solely in gut and salivary gland cells and do not replicate, whereas propagative viruses replicate and may also colonize other organs. Depending on the virus and vector species, the retention sites of non-circulative viruses (red hexagons) are located either at the tip of the stylets (non-persistent viruses) or further up on the cuticle lining the lumen of the foregut (semi-persistent viruses). The common food and salivary canal is enlarged in the inset at the tip of the aphid maxillary stylets. Non-circulative viruses (detailed as icosahedral or filamentous particles) are retained on putative receptors at the surface of the cuticle, either directly or with the intermediate of a virus-encoded 'helper component' (blue molecular link). The acquisition (blue arrows), retention (dotted gray lines) and inoculation (orange arrows) time are short (few minutes to hours) for non-circulative viruses and long (hours to days) for circulative viruses. Adapted from [9]. FG: foregut, MG: midgut, HG: hindgut.

investigate the role of environmental factors on the efficiency of such rapidly reversible external interactions. The answer is clearly yes. Indeed, just as for circulative viruses, non-circulative ones can induce changes in the plant, which in turn modify the plant–insect interaction in ways that could potentially affect transmission, and environmental factors could alter this process.

Virus-induced changes in plants and insects altering the efficiency of transmission

All virus-induced changes in the behavior and life history traits of insect vectors can potentially facilitate or impede transmission (Figure 1). Because transmission is a key component of virus fitness, any heritable viral phenotype affecting the success of transmission is submitted to selection. There is a variety of ways whereby virus vector-transmission can be optimized, some of which have often been interpreted as manipulative 'behaviors' [4••,6•,20–22] and are described below, with emphasis on most recent contributions and remaining major gaps in this field of investigation.

Increased attractiveness of infected host plants to vectors

The very first requirement for virus transmission by an insect vector is that the two actually come into contact. Hence, attracting insect vectors on infected plants would benefit the virus. It has long been evidenced that visual cues such as shape and color affect the choice of plant hosts by sap-feeding insects [23]. Interestingly, a large proportion of virus-related symptoms include a change of leaf color, mostly to light green or yellow colors [24] that appear to be preferred by aphids [25,26] and whiteflies [27].

A growing body of evidence is demonstrating that, beyond visual cues, volatiles emitted by infected plants are of major importance in attracting insect vectors. This has been clearly demonstrated in a number of cases reviewed

in [23]. An emblematic illustration is Cucumber mosaic virus (CMV)-infected squash plants that emit a similar volatile blend as healthy plants but in greater amount [28•]. Another series of very interesting studies suggested that viruses can further modify the odor-related host-choice of their insect vectors, not only by changing the plant odor, but also by changing the perception and response of the insect vector [21,29••]. Aphids of the species *Rhopalosiphum padi* reared on healthy wheat plants prefer to settle on new plants infected by the luteovirus Barley yellow dwarf virus (a BYDV-PAV isolate). In contrast, when individuals of the same aphid species are reared on BYDV-infected plants, their choice is reversed and they preferentially settle on healthy ones. Remarkably, the same change of aphid choice for infected to healthy wheat plants has been reported when they are previously fed on artificial diet added with purified BYDV particles [29••]. This result indicates that the virus circulating within the aphid body directly alters its preference for healthy or infected host plants, although BYDV is a luteovirus and does not replicate within aphids. While the preference of aphids for infected or non-infected plants is based on their different volatile emission [30–32], the mechanisms underlying the reversion of this choice in viruliferous aphids are not yet elucidated. The obvious consequence appears profitable to virus transmission since non-viruliferous aphids tend to get the virus on infected plants, whereas viruliferous ones tend to disseminate it to healthy ones [33]. Recent publications reported similar observations not only in other plant–aphid–luteovirus systems [32], but also in the case of Tomato yellow leaf curl virus (TYLCV, *Geminiviridae*) transmitted by whiteflies [34].

Altered feeding behavior of insect vectors favoring viral transmission

Animal arboviruses have been reported in several instances to modify the feeding behavior of their arthropod vectors. In the majority of the described cases, viral infection compromises the blood-engorgement process and thus induces repeated biting, a phenomenon that increases contacts between vectors and hosts and supposedly increases viral transmission (reviewed in [4••]). The feeding process of sap- or more generally plant-feeding insect has been described in much detail, thanks to the electrical penetration graph (EPG) technology. The successive feeding phases and their specific association with both the acquisition and inoculation of circulative and non-circulative plant viruses are thoroughly characterized [35]. It is surprising to note, however, that modification of EPG-monitored feeding behaviors on infected plants in ways that would possibly increase the acquisition/inoculation of the virus have seldom been reported [36•,37,38]. A compelling case is that of the thrips *Frankliniella occidentalis* infected with the circulative–propagative Tomato spotted wilt virus (TSWV, family *Bunyaviridae*). In this system, TSWV is acquired by larvae and inoculated by adults. Through unknown mechanisms, the virus within the adult male thrips directly induces numerous non-destructive probing punctures, during which there is no ingestion of plant material but intense salivation, which can efficiently inoculate the virus into newly colonized healthy plants and thus potentially increase transmission [39].

Changing plants into a better host for insect vectors

Once insect vectors have alighted on an infected plant they probe, evaluate the quality of the host, and then chose to stay or leave. Depending on the type of virus–vector relationship, virus transmission could benefit from either of these vector choices. Circulative viruses (whether propagative or not) require a long feeding period for both acquisition and inoculation (Figure 2), most often from deep phloem tissues [14], and may thus be better acquired and transmitted if their vectors decide to stay on the infected plant. On the opposite, non-circulative viruses are often acquired and inoculated within a few minutes from or in superficial plant tissues, and retained infectious in their vector's mouthparts for a very limited time (Figure 2) [9]. Their transmission may thus be better ensured by vectors rapidly aborting their feeding on infected plants and deciding to leave immediately in search for healthy ones.

There is a general trend for plant viruses to increase the quality of the host plants for their respective vectors. Consistent with the above considerations, however, some nuances have been demonstrated and discussed [6•]. Conferring increased fecundity, longevity and shorter development time to insect vectors has been repeatedly reported for tospoviruses, luteoviruses and geminiviruses, respectively transmitted by thrips, aphids and whiteflies (reviewed in [23]). These viruses are all circulative viruses and their positive impact on the vector fitness can operate either directly inside the insect or indirectly through changes in the plant physiology: down regulation of defense pathways, reduced callose deposition, altered amino acid content of the sap [40–43]. The faster growth of vector populations on individual infected plants can potentially increase vector density in the environment, enhance transmission opportunities for all viruses transmitted by the corresponding vector species, and thus have strong ecological impacts on the connected species community [40,44,45]. In contrast, the effect on vector growth related to infection of host plants by non-circulative viruses is much less clear. Under the assumption that viruses can manipulate the interaction between plant hosts and insect vectors, plants infected by non-circulative viruses should rapidly deter vectors and force emigration onto neighbor healthy plants. This has been very well described for CMV (isolate KCPG2)-infected squash, deterring aphid vectors of the species *Myzus persicae* and *Aphis gossypii* at early stages of the feeding process, favoring rapid dispersion of viruliferous aphids

and thus virus transmission [28•,46]. However, counter examples are also described: CMV (P1 isolate)-infected pepper has a neutral effect on the same aphid species [47], highlighting the importance of plant genotype × virus isolate × aphid species or biotypes interactions. More surprising, Turnip mosaic potyvirus (TuMV) is favoring the settlement and increasing the fecundity of the aphid vector *M. persicae* on infected host plants [43,48]. In this latter case, despite a possible long-term effect on the increase of the density of the population of aphid vectors in the field, it is hard to conclude on an immediate benefit for non-circulative TuMV transmission (but see discussion in [48]).

Increased emigration of viruliferous vectors from infected plants

It has long been established that overcrowding of aphid and other hemipteran insect populations on host plants increases the production of winged individuals. Viruses infecting plants often increase the proportion of winged aphids either 'spontaneously' or indirectly because of the overcrowding resulting from enhanced growth discussed in the previous section [49]. These observations may be interpreted as a virus manipulation of the vector phenotype that increases emigration from the infected plants and thus long distance viral dispersal ([50], and discussion and reference cited in [29••,35]).

It is conceivable that viruses, once fully installed in their insect vectors, may induce a migratory behavior, depending or not on the density of the population on a given host plant [51]. Circulative viruses may do it through direct interaction with the insect vector while accumulating in its body or through the alteration of plant quality, whereas non-circulative viruses could only do it by acting on plant quality. As far as we are aware, the emigration rate of vectors from healthy plants and from plants infected with a circulative virus have never been directly quantified and formally compared. Likewise, whether virus-induced enhanced emigration of these vectors could occur independently of overcrowding is unknown. In contrast, for non-circulative viruses, the case of CMV already mentioned in previous sections appears as an excellent illustration of this phenomenon. In what has recently been named a pull–push strategy [37], CMV-infected squash plants attract aphid vectors through enhanced volatile emission [28•], and deter and force them away from the infected plant as soon as they have acquired the virus and distasted some chemical compounds produced preferentially in infected plants [46].

Conferring 'super power' to vectors when away from any host plant

The previous sections comment on the investigated and/or predicted targets for possible manipulation by viruses in order to increase their transmission success: vector attraction, feeding behavior, growth, deterring, and emigration. The available literature, however, reveals an important gap that is indicated by the red rectangle and interrogation mark in Figure 1. One crucial but overlooked consideration is that, in order to efficiently transmit, viruliferous insect vectors must travel for variable length of time and distances away from any host plant. There are several ecological configurations where this situation can be critical: (i) when plants are rare and scattered in large areas, (ii) when host plants are intimately mixed with a very dense population of non-host plant species, or (iii) when insect vectors are blown away, in high atmospheric streams that have been reported to promote viral dispersion over huge distances (long range dispersal is discussed in [35,52]). In all these cases, the exploratory behavior and motility of the insect vectors, but most of all their capacity to survive away from host plants, are traits of prime importance for the virus: if the insect vector dies before finding the next host, the virus also dies. Whatever the environmental conditions and the structure of the landscape, the capacity of a vector to efficiently find the next host is crucial and might be 'improved' when a virus is present. This yet ignored aspect deserves more attention as a possible target for virus manipulation, particularly for circulative viruses.

Concluding remarks

To summarize, the multiple physiological changes induced by viruses, either in host plants and/or in insect vectors, modify the interactions in ways that often have the potential to increase transmission. Nevertheless, whether these changes are adaptive viral traits and represent true cases of host and vector manipulation is hard to confirm for two reasons. The first is the frequent report of counter examples where viral infection induces no changes in vector life history traits ([47] and compare [34,38,41,53]), or changes with less evident potential to increase transmission (compare [28•,48,54]). On the one hand, these counter examples could indicate that some virus-induced changes are unrelated to manipulation, but rather incidentally benefit transmission or not. On the other hand, a different interpretation could be that true manipulation requires tight virus–host–vector co-evolution, and would thus be effective solely in very specific plant–virus–vector species associations [47]. It is highly likely that the literature contains all possible situations: true manipulation of host plants and insect vectors by the virus, cases of maladapted plant–virus–vector biological systems, and also cases of virus-induced changes unrelated to the manipulation of transmission but eventually and incidentally impacting it. On top of this blurred picture, there is another layer of possible confusion, related to the fact that some manipulations could align the interests of both viruses and vectors (i.e. increasing survival of viruliferous vectors when traveling away from host plants), whereas other may oppose them (virus attracting aphids to lower quality host plants, or reducing vector fitness in any way). The latter situation may induce partial resistance to manipulation (PRM) or bypass to manipulation

(BPM). These concepts have recently been developed in the context of host manipulation by parasites [22]. They can lead to host responses that render manipulation by parasites even more elusive and they should be carefully considered when investigating vector manipulation.

In any case, whether adaptive or not, virus-induced changes with a high potential to facilitate transmission and to impact the epidemiology of the corresponding diseases do exist. Because they are related both to plant and vector physiology, multiple and very diverse environmental factors can interfere with these processes. Such factors can be biotic factors as for example unrelated pathogens also eliciting plant defenses and altering plant quality [55,56], or insect predators and parasites affecting the dispersal of insect vectors [57,58]. Most importantly, however, abiotic stresses are perfectly known to elicit sensing, transduction and effectors of defense pathways in ways at least partially redundant with those elicited by viruses and insect vectors [7]. It is clear that water deprivation, temperature and CO_2 increase will impact the plant–virus–vector relationship and so the epidemiology of related diseases, but this field of research is in its infancy and is thus far illustrated by very few examples. Wheat plants are 'protected' from extreme drought when infected by BYDV-PAV, and the fitness of the aphid vector *R. padi* is further favored in these conditions, evoking a possible increased transmission (which was not tested in these studies) under conditions of strong water deprivation [59,60•]. In situations of elevated CO_2, the green biomass of pepper plants was increased, but the population growth of the aphid vector *M. persicae* was hampered. Surprisingly enough, limited changes were noted in the feeding behavior of the aphids in these conditions and the transmission of CMV was reduced for unidentified reasons [36•]. These two examples illustrate and confirm the evident impact of the environment on the plant–insect–virus three-way interactions. They also demonstrate the unpredictability of this impact, which thus stands as a major challenge and research horizon for the epidemiology of vector-borne diseases in the context of the forthcoming global changes.

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References and recommended reading

Papers of particular interest, published within the period of review, have been highlighted as:

- • of special interest
- •• of outstanding interest

1. Mescher MC, De Moraes CM: **Role of plant sensory perception in plant–animal interactions**. *J Exp Bot* 2015, **66**:425-433.

2. Babikova Z, Gilbert L, Bruce TJ, Birkett M, Caulfield JC, Woodcock C, Pickett JA, Johnson D: **Underground signals carried through common mycelial networks warn neighbouring plants of aphid attack**. *Ecol Lett* 2013, **16**:835-843.

3. Dong F, Fu X, Watanabe N, Su X, Yang Z: **Recent advances in the emission and functions of plant vegetative volatiles**. *Molecules* 2016, **21**.

4. •• Lefevre T, Thomas F: **Behind the scene, something else is pulling the strings: emphasizing parasitic manipulation in vector-borne diseases**. *Infect Genet Evol* 2008, **8**:504-519.
This paper is a seminal review on the possible manipulation by parasites in vector-borne diseases.

5. Lefevre T, Lebarbenchon C, Gauthier-Clerc M, Misse D, Poulin R, Thomas F: **The ecological significance of manipulative parasites**. *Trends Ecol Evol* 2009, **24**:41-48.

6. • Mauck K, Bosque-Pérez NA, Eigenbrode SD, De Moraes C, Mescher M: **Transmission mechanisms shape pathogen effects on host–vector interactions: evidence from plant viruses**. *Funct Ecol* 2012, **26**:1162-1175.
This perusal of the bibliography is one rare paper actually providing evidence of manipulation of vectors by viruses by demonstrating the existence of convergent evolution in distantly related plant–virus–vector systems.

7. Pandey P, Ramegowda V, Senthil-Kumar M: **Shared and unique responses of plants to multiple individual stresses and stress combinations: physiological and molecular mechanisms**. *Front Plant Sci* 2015, **6**:723.

8. Whitfield AE, Falk BW, Rotenberg D: **Insect vector-mediated transmission of plant viruses**. *Virology* 2015.

9. Blanc S, Drucker M, Uzest M: **Localizing viruses in their insect vectors**. *Annu Rev Phytopathol* 2014, **52**:403-425.

10. Bragard C, Caciagli P, Lemaire O, Lopez-Moya JJ, MacFarlane S, Peters D, Susi P, Torrance L: **Status and prospects of plant virus control through interference with vector transmission**. *Annu Rev Phytopathol* 2013, **51**:177-201.

11. Gray SM, Banerjee N: **Mechanisms of arthropod transmission of plant and animal viruses**. *Microbiol Mol Biol Rev* 1999, **63**:128-148.

12. Blanc S, Gutierrez S: **The specifics of vector transmission of arboviruses of vertebrates and plants**. *Curr Opin Virol* 2015, **15**:27-33.

13. Nault LR: **Arthropod transmission of plant viruses: a new synthesis**. *Ann Entomol Soc Am* 1997, **90**:521-541.

14. Hogenhout SA, Ammar el D, Whitfield AE, Redinbaugh MG: **Insect vector interactions with persistently transmitted viruses**. *Annu Rev Phytopathol* 2008, **46**:327-359.

15. Rotenberg D, Jacobson AL, Schneweis DJ, Whitfield AE: **Thrips transmission of tospoviruses**. *Curr Opin Virol* 2015, **15**:80-89.

16. Rosen R, Kanakala S, Kliot A, Cathrin Pakkianathan B, Farich BA, Santana-Magal N, Elimelech M, Kontsedalov S, Lebedev G, Cilia M *et al.*: **Persistent, circulative transmission of begomoviruses by whitefly vectors**. *Curr Opin Virol* 2015, **15**:1-8.

17. Anhalt MD, Almeida RP: **Effect of temperature, vector life stage, and plant access period on transmission of banana bunchy top virus to banana**. *Phytopathology* 2008, **98**:743-748.

18. Pusag JCA, Jahan SMH, Lee KS, Lee S, Lee KY: **Upregulation of temperature susceptibility in *Bemisia tabaci* upon acquisition of Tomato yellow leaf curl virus (TYLCV)**. *J Insect Physiol* 2012, **58**:1343-1348.

19. Ng JC, Zhou JS: **Insect vector–plant virus interactions associated with non-circulative, semi-persistent transmission: current perspectives and future challenges**. *Curr Opin Virol* 2015, **15**:48-55.

20. Casteel CL, Jander G: **New synthesis: investigating mutualisms in virus–vector interactions**. *J Chem Ecol* 2013, **39**:809.

21. Bosque-Perez NA, Eigenbrode SD: **The influence of virus-induced changes in plants on aphid vectors: insights from luteovirus pathosystems**. *Virus Res* 2011, **159**:201-205.

22. Daoust SP, King KC, Brodeur J, Roitberg BD, Roche B, Thomas F: **Making the best of a bad situation: host partial resistance and bypass of behavioral manipulation by parasites?** *Trends Parasitol* 2015, **31**:413-418.

23. Fereres A, Moreno A: **Behavioural aspects influencing plant virus transmission by homopteran insects**. *Virus Res* 2009, **141**:158-168.

24. Li Y, Cui H, Cui X, Wang A: **The altered photosynthetic machinery during compatible virus infection**. *Curr Opin Virol* 2015, **17**:19-24.

25. Hodge S, Powell G: **Do plant viruses facilitate their aphid vectors by inducing symptoms that alter behavior and performance?** *Environ Entomol* 2008, **37**:1573-1581.

26. Fereres A, Kampmeier GE, Irwin ME: **Aphid attraction and preference for soybean and pepper plants infected with potyviridae**. *Ann Entomol Soc Am* 1999, **92**:542-548.

27. Isaacs R, Willis MA, Byrne DN: **Modulation of whitefly take-off and flight orientation by wind speed and visual cues**. *Physiol Entomol* 1999, **24**:311-318.

28. • Mauck KE, De Moraes CM, Mescher MC: **Deceptive chemical signals induced by a plant virus attract insect vectors to inferior hosts**. *Proc Natl Acad Sci U S A* 2010, **107**:3600-3605.
This paper is an experimental demonstration of a complex virus-induced modification of the vector behavior that elegantly matches the criterion ''purposiveness of design'' discussed in reference No. 4.

29. •• Ingwell LL, Eigenbrode SD, Bosque-Perez NA: **Plant viruses alter insect behavior to enhance their spread**. *Sci Rep* 2012, **2**:578.
This is thus far the only study demonstrating that a plant virus can directly modify the behavior of an insect vector by observing the effect after artificially feeding aphids with solutions containing purified virus particles.

30. Medina-Ortega KJ, Bosque-Perez NA, Ngumbi E, Jimenez-Martinez ES, Eigenbrode SD: ***Rhopalosiphum padi* (Hemiptera: Aphididae) responses to volatile cues from Barley yellow dwarf virus-infected wheat**. *Environ Entomol* 2009, **38**:836-845.

31. Ngumbi E, Eigenbrode SD, Bosque-Perez NA, Ding H, Rodriguez A: ***Myzus persicae* is arrested more by blends than by individual compounds elevated in headspace of PLRV-infected potato**. *J Chem Ecol* 2007, **33**:1733-1747.

32. Rajabaskar D, Bosque-Perez NA, Eigenbrode SD: **Preference by a virus vector for infected plants is reversed after virus acquisition**. *Virus Res* 2014, **186**:32-37.

33. Roosien BK, Gomulkiewicz R, Ingwell LL, Bosque-Perez NA, Rajabaskar D, Eigenbrode SD: **Conditional vector preference aids the spread of plant pathogens: results from a model**. *Environ Entomol* 2013, **42**:1299-1308.

34. Legarrea S, Barman A, Marchant W, Diffie S, Srinivasan R: **Temporal effects of a Begomovirus infection and host plant resistance on the preference and development of an insect vector, *Bemisia tabaci*, and implications for epidemics**. *PLOS ONE* 2015, **10**:e0142114.

35. Stafford CA, Walker GP, Ullman DE: **Hitching a ride: vector feeding and virus transmission**. *Commun Integr Biol* 2012, **5**:43-49.

36. • Dader B, Fereres A, Moreno A, Trebicki P: **Elevated CO_2 impacts bell pepper growth with consequences to *Myzus persicae* life history, feeding behaviour and virus transmission ability**. *Sci Rep* 2016, **6**.
These two studies represent rare examples that considers the impact of environmental factors on the outcome of the complex interaction between a virus, its host plant and insect vector.

37. Carmo-Sousa M, Moreno A, Garzo E, Fereres A: **A non-persistently transmitted-virus induces a pull–push strategy in its aphid vector to optimize transmission and spread**. *Virus Res* 2014, **186**:38-46.

38. Moreno-Delafuente A, Garzo E, Moreno A, Fereres A: **A plant virus manipulates the behavior of its whitefly vector to enhance its transmission efficiency and spread**. *PLOS ONE* 2013, **8**:e61543.

39. Stafford CA, Walker GP, Ullman DE: **Infection with a plant virus modifies vector feeding behavior**. *Proc Natl Acad Sci U S A* 2011, **108**:9350-9355.

40. Su Q, Mescher MC, Wang S, Chen G, Xie W, Wu Q, Wang W, Zhang Y: **Tomato yellow leaf curl virus differentially influences plant defence responses to a vector and a non-vector herbivore**. *Plant Cell Environ* 2016, **39**:597-607.

41. Su Q, Preisser EL, Zhou XM, Xie W, Liu BM, Wang SL, Wu QJ, Zhang YJ: **Manipulation of host quality and defense by a plant virus improves performance of whitefly vectors**. *J Econ Entomol* 2015, **108**:11-19.

42. Ziebell H, Murphy AM, Groen SC, Tungadi T, Westwood JH, Lewsey MG, Moulin M, Kleczkowski A, Smith AG, Stevens M *et al.*: **Cucumber mosaic virus and its 2b RNA silencing suppressor modify plant–aphid interactions in tobacco**. *Sci Rep* 2012, **1**:187.

43. Casteel CL, De Alwis M, Bak A, Dong H, Whitham SA, Jander G: **Disruption of ethylene responses by turnip mosaic virus mediates suppression of plant defense against the green peach aphid vector**. *Plant Physiol* 2015, **169**:209-218.

44. Malmstrom CM, Melcher U, Bosque-Perez NA: **The expanding field of plant virus ecology: historical foundations, knowledge gaps, and research directions**. *Virus Res* 2011, **159**:84-94.

45. Mauck K, De Moraes C, Mescher M: **Cucumber mosaic virus-induced changes in volatile production and plant quality: implications for disease transmission and multitrophic interactions**. *Phytopathology* 2014, **104** 151–151.

46. Mauck KE, De Moraes CM, Mescher MC: **Biochemical and physiological mechanisms underlying effects of Cucumber mosaic virus on host-plant traits that mediate transmission by aphid vectors**. *Plant Cell Environ* 2014, **37**:1427-1439.

47. Mauck KE, De Moraes CM, Mescher MC: **Evidence of local adaptation in plant virus effects on host–vector interactions**. *Integr Comp Biol* 2014, **54**:193-209.

48. Casteel CL, Yang C, Nanduri AC, De Jong HN, Whitham SA, Jander G: **The NIa-Pro protein of Turnip mosaic virus improves growth and reproduction of the aphid vector, *Myzus persicae* (green peach aphid)**. *Plant J* 2014, **77**:653-663.

49. Müller CB, Williams IS, Hardie J: **The role of nutrition, crowding and interspecific interactions in the development of winged aphids**. *Ecol Entomol* 2001, **26**:330-340.

50. Higashi CHV, Bressan A: **Influence of a propagative plant virus on the fitness and wing dimorphism of infected and exposed insect vectors**. *Oecologia* 2013, **172**:847-856.

51. Gutiérrez S, Michalakis Y, Van Munster M, Blanc S: **Plant feeding by insect vectors can affect life cycle, population genetics and evolution of plant viruses**. *Funct Ecol* 2013, **27**:610-622.

52. Canto T, Aranda MA, Fereres A: **Climate change effects on physiology and population processes of hosts and vectors that influence the spread of hemipteran-borne plant viruses**. *Glob Change Biol* 2009, **15**:1884-1894.

53. Rubinstein G, Czosnek H: **Long-term association of tomato yellow leaf curl virus with its whitefly vector *Bemisia tabaci*: effect on the insect transmission capacity, longevity and fecundity**. *J Gen Virol* 1997, **78**:2683-2689.

54. Blua MJ, Perring TM, Madore MA: **Plant virus-induced changes in aphid population development and temporal fluctuations in plant nutrients**. *J Chem Ecol* 1994, **20**:691-707.

55. Goyal RK, Mattoo AK: **Multitasking antimicrobial peptides in plant development and host defense against biotic/abiotic stress**. *Plant Sci* 2014, **228**:135-149.

56. Ma KW, Ma W: **Phytohormone pathways as targets of pathogens to facilitate infection**. *Plant Mol Biol* 2016.

57. Mauck KE, De Moraes CM, Mescher MC: **Infection of host plants by Cucumber mosaic virus increases the susceptibility of *Myzus persicae* aphids to the parasitoid *Aphidius colemani***. *Sci Rep* 2015, **5**:10963.

58. Ryabov EV, Keane G, Naish N, Evered C, Winstanley D: **Densovirus induces winged morphs in asexual clones of the rosy apple aphid, *Dysaphis plantaginea***. *Proc Natl Acad Sci U S A* 2009, **106**:8465-8470.

59. Davis TS, Bosque-Pérez NA, Popova I, Eigenbrode SD: **Evidence for additive effects of virus infection and water availability on phytohormone induction in a staple crop**. *Front Ecol Evolut* 2015, **3**:114.

60. • Davis TS, Bosque-Perez NA, Foote NE, Magney T, Eigenbrode SD: **Environmentally dependent host–pathogen and vector–pathogen interactions in the Barley yellow dwarf virus pathosystem**. *J Appl Ecol* 2015, **52**:1392-1401.

These two studies represent rare examples that considers the impact of environmental factors on the outcome of the complex interaction between a virus, its host plant and insect vector.