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Effects of pathogens on sensory-mediated interactions between plants and insect vectors
Kerry E Mauck¹, Consuelo M De Moraes and Mark C Mescher

Vector-borne plant pathogens frequently alter host-plant quality and associated plant cues in ways that influence vector recruitment and pathogen acquisition. Furthermore, following acquisition by the vector, pathogens may influence subsequent vector behavior either directly or via effects on the host plant. Given that such effects have significant implications for pathogen acquisition and inoculation, selection might be expected to favor patterns of pathogen effects on host-vector interactions that are conducive to transmission. Consequently, we might also expect to observe broad similarity in the effects of pathogens sharing similar modes of transmission. Here we discuss some specific hypotheses arising from these expectations and the implications of recent empirical findings. On the whole, this evidence is consistent with the expectation that pathogen effects on host-vector interactions are often (though not always) adaptive with respect to transmission.

Pathogen effects on information-mediated interactions among hosts and vectors

The transmission of vector-borne pathogens requires that vectors interact with infected hosts in a manner conducive to pathogen acquisition and then subsequently interact with other, uninfected hosts in ways that lead to inoculation. Pathogens can potentially influence this process via effects on the plant or on the vector that modify the frequency and nature of interactions between them. Furthermore, because such interactions are mediated by sensory cues (Figure 1), pathogen effects on the transfer of information between hosts and vectors are likely to have important implications for transmission. Vectors of plant pathogens include pollinators [10] and herbivores (the focus of the current discussion), which rely on cues such as leaf odors to locate plants and to assess their resource value [11,12].

Pathogen infection can alter host-derived sensory cues, as well as the resource value of the host for vectors, either as part of an adaptive strategy of indirect (host-mediated) manipulation of vector behavior or as a by-product of pathology [6,8,12] (Figure 1). Some pathogens that colonize and persist in vector tissues following acquisition may also exert direct effects on vector responses to plant cues that influence the efficiency of transmission (e.g., [14,15]) (Figure 1). Given that effective transmission is critical to the fitness of vector-borne pathogens, we may assume that pathogens are frequently under selection to produce (or maintain) host phenotypes and effects on vectors that are conducive to transmission. Consequently, we might also predict some degree of convergence in the effects of vector-borne parasites that share similar modes of transmission, and hence are expected to benefit from...
Potential behavioral sequences of a vector in response to aspects of the host plant phenotype (stimuli) that can be altered by pathogen infection. In most cases, pathogen acquisition by the vector occurs during host plant feeding (persistently transmitted pathogens), but can also occur for some pathogens during the more superficial ‘assessment’ behavior (nonPersistently transmitted pathogens). Pathogens could potentially manipulate multiple stimuli in a single host in order to facilitate a specific sequence of vector behaviors that are conducive to transmission (green boxes). Following pathogen acquisition by the vector, there is also the potential for direct effects on vector behavior in relation to hosts, depending on the degree of intimacy between pathogen and vector (blue boxes). Additionally, dispersal of vectors that have established feeding on a host can also be stimulated by predators, competitors, or abiotic factors.

similar patterns of vector behavior with respect to hosts. As discussed below, empirical work on the ecology of plant pathogens that differ in their interactions with herbivorous vectors offers a promising opportunity to evaluate the validity of this prediction.

**Persistently and non-persistently transmitted plant pathogens**

A key factor influencing the ecology of vector-borne parasites is the duration and extent of their association with vectors. Some plant pathogens reside — and in some cases replicate — within the vector for extended periods, during which time the vector can repeatedly infect new hosts [15]. Owing to this prolonged relationship, the transmission of such pathogens is said to be ‘persistent.’ In contrast, other plant pathogens utilize ‘non-persistent’ transmission mechanisms whereby they form only transitory associations with the vector (namely, viruses that attach to aphid styles) [16]. This distinction between persistently transmitted pathogens (PTPs) and non-persistently transmitted pathogens (NPTPs) has potentially significant implications for the evolution of parasite effects on host–vector interactions. For instance, given their intimate association with vectors, PTPs can potentially exert direct effects on vector behavior [14,15]. Furthermore, we might also expect PTPs and NPTPs to exhibit different patterns of indirect, plant-mediated effects on vectors, since the efficient transmission of pathogens of each type is facilitated by divergent patterns of host–vector interactions. For example, PTPs typically benefit from sustained feeding by vectors in phloem, which leads to acquisition of more pathogen units that can persist within the vector [15,17]. In contrast, NPTP transmission is typically impeded by long-term phloem feeding because these pathogens are acquired and inoculated when vectors salivate into and sample contents of non-vascular plant cells [16,18,19]. As a result, NPTP transmission is most efficient if vectors disperse shortly after acquiring the pathogen and before phloem feeding (Figure 2).
On the basis of these considerations, we have previously explored the hypothesis that PTPs and NTPs exhibit divergent patterns of effects on host plant traits that mediate interactions with vectors [6,20,21]. While such divergence would be an expected outcome of adaptive manipulation by pathogens with differing modes of transmission, the predicted patterns do not depend on the assumption that vector-borne plant pathogens are always adapted to manipulate host–vector interactions. Rather, the minimal assumption is only that selection will tend to weed out pathogen effects on host phenotypes that have adverse effects on vector transmission, resulting in PTP and NPTP effects on host–vector interactions that are broadly conducive to their divergent modes of transmission. Mescher and Pearse (this volume) make a somewhat similar point regarding the interpretation of broader patterns of information-mediated interactions among plants and other organisms [22].

Specific predictions and evidence from early empirical studies

In keeping with these general expectations, it is possible to derive specific hypotheses regarding the potentially divergent effects of PTPs and NPTPs on host–vector
Box 1 Hypotheses regarding effects of PTPs versus NPTPs on aspects of host phenotype or vector behavior that influence transmission efficiency

**Hypothesis 1:** Insect vectors must interact with infected hosts in order to acquire a pathogen. Therefore, it follows that both PTPs and NPTPs should induce changes in host plant phenotype that increase (or at least do not reduce) vector contacts with infected hosts.

**Hypothesis 2:** Long-term feeding is required for PTP acquisition and inoculation, while short probes without long-term feeding are required for NPTP acquisition and inoculation. Therefore, it follows that PTPs should enhance (or at least not reduce) plant palatability for vectors. In contrast, NPTPs should generally reduce (or at least not enhance) plant palatability in order to discourage vector phloem feeding, and subsequent pathogen loss, following acquisition.

**Hypothesis 3:** Vectors must eventually disperse from infected plants in order to transmit. Therefore, it follows that PTPs should exert direct effects on vector behavior (following acquisition) that increase vector dispersal and contacts with healthy, susceptible hosts. NPTPs are presumably not capable of exerting direct effects, so any stimulation of vector dispersal following acquisition should occur only as a result of (indirect) pathogen effects on host-plant phenotype.

Pathogen effects on vector dispersal and inoculation behavior are likely to be important given that pathogen fitness depends on transmission. A reversal of vector preferences following pathogen acquisition (i.e., from favoring infected hosts to favoring healthy hosts) has been termed a conditional vector preference because the preference shift depends on the vector acquiring the pathogen [23**]. Conditional vector preferences have been observed (and modeled) in PTP systems, where pathogens residing in the vector can facilitate preference shifts via direct effects on vector behavior in response to host cues [23**]. Such direct effects can operate in combination with pathogen effects on host phenotype (indirect effects) that allow both pathogen-free and pathogen-infected vectors to differentially discriminate between infected and healthy hosts, leading to greater pathogen spread regardless of the relative proportions of healthy and infected hosts in the landscape (Figure 2) [23**]. Although NPTPs presumably cannot exert direct effects, vector dispersal following pathogen acquisition remains critical to transmission and can be influenced via pathogen-induced changes in host-plant cues (Figure 2). Such indirect effects can mediate host-dependent vector preferences that yield outcomes similar to those mediated by conditional vector preferences (Figure 2). Consistent with the transient association between NPTPs and vectors, host-dependent vector preferences are temporary (i.e., not leading to permanent avoidance of infected hosts), while conditional vector preferences may persist as long as the pathogen resides in the vector.

Early empirical reports of pathogen effects on host–vector interactions often documented effects on host quality and vector performance [6*,8*,13], while far fewer studies directly assessed vector orientation, feeding, or dispersal behavior. Despite this limitation, the findings of these studies, discussed in detail in recent reviews [6*,8*], were largely consistent with the first two hypotheses presented in Box 1. Overall, vectors frequently orient towards infected plants versus healthy plants regardless of the transmission mechanism of the infecting pathogen (Hypothesis 1). Meanwhile, studies reporting performance effects reveal a trend in which PTPs tend to enhance host palatability and encourage vector settling and feeding, while NPTPs (which were the focus of far fewer studies) often exhibit negative [24,25] or neutral [25–27] effects on the palatability of infected hosts for vectors (Hypothesis 2).

While these studies support Hypotheses 1 and 2, they generally did not account for the infectious state of the vector (Hypothesis 3). Yet theoretical considerations suggest that, in the absence of preference shifts, vector orientation and feeding preferences for infected hosts (or increases in infected host quality) could actually reduce pathogen spread, since vectors will presumably avoid visiting healthy, susceptible hosts [23**,28,29]. Despite this, early empirical work often took for granted that pathogens derive fitness benefits by enhancing vector arrestment and fecundity, even though there may be situations where this is not advantageous. For instance, vectors that arrest and reproduce on infected hosts may not disperse until much later in the season, creating a temporal separation of infectious vectors from susceptible hosts (which are often vulnerable only during early phenological stages) [30]. Furthermore, even if vectors disperse within an appropriate window for initiating new infections, they might still prefer infected hosts, which could disfavor transmission [23**]. Since most early studies did not examine vector preferences following pathogen acquisition (immediately or over the course of disease progression), these studies did not provide sufficient data to test the expectation that most effects have neutral to positive effects on transmission due to a failure to account...
for all of the relevant effects on host–vector interactions within a single system.

**Filling the gaps: evidence from recent empirical studies**

Vector dispersal from infected hosts has now been examined across diverse PTP and NPTP pathosystems and reveals that effects on plant quality, palatability, and attractiveness reported in early empirical work (supporting Hypotheses 1 and 2) likely do increase pathogen fitness in light of evidence that vectors prefer healthy plants following pathogen acquisition (supporting Hypothesis 3). For example, while virus-free aphids prefer to feed on wheat infected with the PTP *Barley yellow dwarf virus* (BYDV; Luteoviridae), aphids carrying BYDV either have no preference [31] or prefer to feed on healthy rather than infected wheat [14**]. This shift appears to be a direct effect of the virus, as it was observed regardless of whether aphids acquired BYDV from infected plants or from artificial diet [14**]. A similar reversal of feeding preferences was reported for aphids that acquired *Potato leafroll virus* (PLRV; Luteoviridae) from infected potatoes, along with a corresponding reversal of preferences for odors cues from healthy and infected plants [32**]. The olfactory preferences of (both infected and uninfected) aphids are mediated by virus-induced changes in infected plants, which emit volatile blends enriched in monoterpenes, aldehydes and sesquiterpenes compared to those of uninfected plants [32**]. These changes in volatile emissions also vary over the course of disease progression in ways that are conducive to transmission. Infected plants are only attractive to non-infectious vectors at 4–6 weeks post-inoculation, but not at 2, 8 or 10 weeks post-inoculation [33], or when inoculated at later phenological stages [34]. This should facilitate vector visits to infected hosts when infection prevalence is low but titer in infected hosts is high (4–6 weeks) and facilitate visits to healthy hosts once infected plants are more prevalent (8–10 weeks) [33,34]. Thus, for both BYDV and PLRV, conditional vector preferences seem to be influenced by both direct effects (confirmed for BYDV, putative for PLRV) and indirect effects mediated by changes to the host plant phenotype that permit discrimination between infected and healthy hosts.

New evidence supporting a combination of host-mediated indirect effects (Hypotheses 1 and 2) and direct effects (Hypothesis 3) has also been forthcoming for PTPs from other virus families. For instance, Begomoviruses in the family Geminiviridae generally have neutral to positive effects on plant quality for whitefly vectors [67,35]. Recent work suggests that vector settling preferences are congruent with observed virus effects on host–plant quality (Hypothesis 2) but also vary with the infectious condition of the vector (Hypothesis 3). Virus-free whiteflies exhibit a settling preference for tomatoes infected with *Tomato yellow leaf curl virus* (TYLCV; Geminiviridae) but prefer healthy plants once the virus is acquired [36,37**]. Whitefly perception of, and discrimination among, infected and healthy hosts is likely mediated by begomovirus suppression of terpene synthase genes that synthesize whitefly-deterrent volatile terpenoids (direct defenses) [38,39]. There is also some evidence that the virus may alter vector perception of host cues. For example, whiteflies carrying the TYLCV pathogen and feeding on healthy plants made more contacts with phloem and exhibited longer durations of salivation into phloem sieve elements relative to virus-free whiteflies — behaviors that enhance inoculation [40,41*]. Since phloem contact frequency and salivation are both mediated by plant cues that indicate tissue identity and suitability [42], the change in behavior following acquisition is likely due to pathogen effects on vector sensitivity to plant cues or the range of responses to these cues. A similar effect was observed for the persistent propagative pathogen *Tomato spotted wilt virus* (TSWV; Bunyaviridae), whose presence in male thrips increased inoculative behavior (non-ingestion probing, salivation) [43*]. The apparent convergence of direct effects on feeding behavior across distantly related viral PTPs revealed by these recent studies supports Hypothesis 3 and suggests that viral PTPs may be adapted for manipulating vector responses to cues associated with infected and healthy hosts.

Additional support for the expectation of transmission mechanism-based convergence in pathogen effects can be found in recent studies of non-viral PTPs. For example, the phytoplasma *Candidatus liberibacter asiaticus* (*Las*) enhances attraction of psyllid vectors to infected citrus trees (consistent with Hypotheses 1 and 2); however, over a period of several days, vectors subsequently disperse from infected to healthy plants (consistent with Hypothesis 3) [44**]. In this system, the initial attraction of the vector appears to be mediated by pathogen-induced emission of the volatile compound methyl salicylate from infected trees. The same compound is also emitted from uninfected plants in response to psyllid feeding and acts as an aggregation cue for the insect, perhaps by indicating potential mates [44**]. Furthermore, *Las*-carying psyllids also have a greater capacity and propensity for dispersal relative to *Las*-free psyllids [45**], again revealing direct effects of the pathogen on vector behavior and providing support for Hypothesis 3. The related PTP *Candidatus liberibacter solanacearum* induces a similar pattern of vector behavior towards infected and healthy potato plants: vectors orient towards, and settle on, infected plants, but subsequently disperse to healthy plants after acquiring the pathogen [46]. In both systems, the conditional vector preference is likely mediated by a combination of direct pathogen effects on vector physiology and pathogen-induced changes in host phenotype that permit discrimination between infected and healthy hosts.
The finding of conditional vector preferences across diverse PTP systems supports our expectation of convergence in pathogen effects depending on shared transmission mechanism. Analogous observations of host-dependent vector preferences consistent with expectations for NPTPs (Box 1 and Figure 2) have also now been shown for more systems. For example, multiple strains of Cucumber mosaic virus (CMV: Bromoviridae) enhance aphid attraction to squash plants by elevating volatile emissions of infected hosts (Hypothesis 1) [20] but also reduce plant palatability by altering nutrient cues, which encourages aphid dispersal after probing behavior conducive to acquisition (supporting Hypotheses 2 and 3) [20,21**]. Complementary findings have also been reported for CMV infecting Arabidopsis thaliana, where infection induces production of 4-methoxy-indol-3-yl-methylglucosinolate, a mild aphid feeding deterrent that stimulates dispersal after probing [48]. And in Cucumis sativus, aphids feeding on CMV-infected plants increase short superficial probes and reduce phloem feeding, effects that are conducive to acquisition and retention of CMV by aphid vectors [49]. A similar increase in short, superficial probes was also found for another, distantly related NPTP (Potato virus Y: Potyviridae) infecting tobacco [50].

The increasing number of studies addressing NPTPs has also produced findings which suggest that these pathogens may have more variable effects on host–plant phenotypes than PTPs [21**,51–53,54*,55]. This is not surprising given that many NPTPs are transmitted by a large number of vectors, including non-colonizing species that already exhibit host-dependent vector preferences conducive to NPTP transmission (rapid dispersal following assessment) [56] (Figure 2). As a result, selection may act more frequently or strongly on NPTP traits that enhance attractiveness of hosts to vectors (Hypothesis 1) and less frequently or strongly on pathogen traits that influence palatability (Hypotheses 2 and 3). This is consistent with findings showing that some isolates of Bean yellow mosaic virus, Zucchini yellow mosaic virus, and Turnip mosaic virus (all Potyviridae) induce changes in host color cues that are attractive to aphids generally, but also enhance settling and feeding of colonizing aphids [53,54*,55,56,57]. Given that each of these viruses can be transmitted, at varying efficiencies, by many non-colonizing aphids, the isolates examined may not have undergone strong selection pressure for effects on palatability. Apparently maladaptive effects of NPTPs on host–vector interactions may also reflect pathogen adaptation to local hosts. For example, CMV-KVPG2, a squash-adapted pathogen, changes squash phenotype to favor vector attraction followed by dispersal after probing, but has an opposite, mal-adaptive effect in pepper, a novel (but susceptible) host for CMV-KVPG2 [21**]. Meanwhile, an isolate adapted to pepper (CMV-PEP) has largely neutral effects on pepper phenotype (consistent with expectations in Box 1) that are more conducive to CMV transmission [21**]. This observation suggests that pathogens may have less adaptive effects on secondary hosts. It is also important to point out that neutral effects of pathogens on host phenotype, such as those observed for CMV-PEP, may be under-reported despite the fact that they could reflect selection against pathogen genotypes inducing maladaptive changes to host phenotype [21**]. More studies examining the effects of different pathogen

<p>| Table 1 |
|-------------------------|-------------------------|-------------------------|-------------------------|
| <strong>Research priorities for future studies of pathogen effects on host-vector communication and selected examples of recent progress</strong> |</p>
<table>
<thead>
<tr>
<th>Research goal</th>
<th>Key questions</th>
<th>Supporting literature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Examine pathogen effects on communication between multiple hosts and herbivorous vectors</td>
<td>Can multi-host pathogens manipulate the phenotype of multiple hosts? To what extent does the evolutionary history of a pathogen with a given host or suite of hosts influence such effects? Do direct effects of a PTP on a vector vary depending on pathogen, vector, or host genotype?</td>
<td>[21**,37**,58-60]</td>
</tr>
<tr>
<td>Determine mechanisms underlying pathogen-induced changes in host phenotypes and direct effects of pathogens on vector behavior</td>
<td>How does plant phenotype change throughout disease progression in the host? How does vector perception of, or response to stimuli change when carrying a PTP? What pathogen traits are responsible for inducing these changes?</td>
<td>[9**,33,34,39,41*,43*,47,48*,51,54*,55,61,62*]</td>
</tr>
<tr>
<td>Explore the ecological context of pathogen effects on host and vector physiology</td>
<td>How is communication between plants and vector natural enemies changed by pathogen effects on plant phenotypes? Do pathogen effects also influence plant interactions with non-vector herbivores? Do pathogen effects influence plant resistance to abiotic stress?</td>
<td>[63-71]</td>
</tr>
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</table>
The genotypes with vector of genetic alterations would enhance our understanding of the genetic basis for such effects and provide information on the frequency with which these effects are likely to occur.

Conclusions and future directions
The literature discussed above is broadly consistent with our expectation that pathogens should tend to have neutral to positive effects on aspects of host–vector communication that influence pathogen transmission. Furthermore, there appears to be evidence for convergence of pathogen effects based on transmission mechanism, particularly for phylogenetically divergent PTPs (Box 1 and Figure 2). Importantly, recent work focusing on vector dispersal provides evidence that the preferences of PTP-carrying vectors frequently change to favor movement from infected to healthy plants (Hypothesis 3). As a result, earlier reports that these same PTPs enhance vector attraction to and arrestment on infected hosts (Hypotheses 1 and 2) can now be taken as evidence of positive effects with respect to pathogen transmission. Furthermore, recent work in NPTP systems demonstrates the existence of host-dependent vector preferences that operate entirely via pathogen effects on host cues. More broadly, the literature reviewed here suggests a shift away from the historical focus on single phenotypic alterations and towards the sensory perspective of herbivorous vectors, which involves consideration of multiple interacting aspects of the host phenotype as well as direct effects of PTPs on vector perception [4]. Future work should continue to test expectations (Box 1 and Figure 2) by increasing the number of pathogen–host–vector combinations examined from the vector sensory perspective and by shifting the context of empirical studies beyond the laboratory (Table 1).

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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


6. Mauck K, Bosque-Pérez NA, Eigenbrode SD, De Moraes CM, Mescher MC: Transmission mechanisms shape pathogen effects on host-vector interactions: evidence from plant viruses. Funct Ecol 2012, 26:1162-1175. This review provides a comprehensive, semi-quantitative synthesis of early reports describing plant virus effects on host quality, palatability, or attractiveness for vectors. This synthesis highlights key differences between effects of PTPs versus NPTPs and provides a framework for future studies in this area.


8. Bosque-Pérez NA, Eigenbrode SD: The influence of virus-induced changes in plants on aphid vectors: insights from luteovirus pathosystems. Virus Res 2011, 159:201-205. This is a review of Luteovirus effects on host–vector communication that summarizes findings before the discovery of conditional vector preferences in Luteovirus systems.

9. Shapiro L, De Moraes CM, Stephenson AG, Mescher MC: Pathogen effects on vegetative and floral odors mediate vector attraction and host exposure in a complex pathosystem. Ecol Lett 2012, 15:1430-1438 http://dx.doi.org/10.1111/j.1461-0248.2012.02001.x. This study describes a host-dependent vector preference shift that occurs in a pathosystem consisting of a beetle vector, a bacterial pathogen, and cucurbits hosts. This pathogen simultaneously enhances the attractiveness (via odor cues) and palatability of host foliage to cucumber beetle vectors while diminishing the attractiveness of odor cues from flowers. This pattern is significant because beetles readily acquire the bacteria by feeding on infected foliage, while inoculation of healthy plants is thought to frequently occur via floral nectarines when beetles aggregate within the flowers and deposit frass.


14. Ingwell LL, Eigenbrode SD, Bosque-Pérez NA: Plant viruses alter insect behavior to enhance their spread. Sci Rep 2012, 2:578 http://dx.doi.org/10.1038/srep00578. This study is one of the first to test for conditional vector preferences in a PTP system. Notably, the authors used artificial feeding systems to allow vectors to acquire pathogens without exposure to host cues. In doing so, they demonstrate that shifts in vector preference are likely mediated by direct effects of the pathogen on the vector, rather than learning of cues or changes in host quality.


18. Martin B, Collar JL, Tjallingii WF, Fereres A: Intracellular ingestion and salivation by aphids may cause the acquisition and

This study demonstrates that host-dependent vector preference shifts for an NPTP depend on the identity of the infected host and the genotype of the pathogen. When infecting the host from which it was isolated, pathogen genotypes induced changes that were conducing to transmission, but in a novel host, a mal-adaptive host phenotype was induced. This comparative study suggests that pathogens may undergo local adaptation for effects on host phenotype.


This paper builds on prior modeling attempts that incorporate vector preferences for diseased hosts and extends these findings to include shifts in vector preferences following pathogen acquisition. The authors show that conditional vector preferences are essential for pathogen spread when vectors favor diseased hosts, validating much of the prior empirical literature reporting evidence of such preferences.


This paper provides evidence that the effects of PTPs on a pathogen might extend to effects on orientation behavior. Pathogen alteration of plant odors usually renders them more attractive to vectors, but when infectious, vectors favor odors of healthy plants. This study points out that pathogen effects on host-vector communication are complex and may include effects on the host phenotype and vector physiology.


This study demonstrates that conditional vector preferences for whiteflies depend on both the vector and host genotype. A conditional vector preference was found for one genotype of whitefly interacting with infec tive and healthy plants at a highly susceptible host genotype, but was not present when the vector genotype was different or the host genotype was a highly tolerant variety.


41. 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 http://dx.doi.org/10.1371/journal.pone.0061543.

This study demonstrates a putative direct effect of a viral pathogen on its whitefly vector by examining how the presence of the pathogen within the vector affects behaviors related to inoculation. Results suggest that carrying a PTP can alter the processing of information by vectors because vectors have different behaviors in response to healthy host cues.


This elegant study uses the electrical penetration graphing technique in a non-traditional way (on thrips vectors) to illustrate that a PTP that replicates in the vector can alter feeding behavior on healthy, susceptible hosts. This is one of the few reports of purely direct effects of a PTP on the probability of pathogen inoculation and further suggests that harboring a PTP changes vector perception of and responses to cues of healthy host plants.


This study is one of the first reports of a conditional vector preference in a non-viral PTP (the phytoplasma Candidatus liberibacter asiaticus). It also suggests that the pathogen is able to co-opt a signal (methyl salicylate) that is already attractive to the psyllid vector. Methyl salicylate is produced by healthy plants in response to vector feeding and is produced by psyllids to locate conspecics.
45. Martini X, Hoffmann M, Coy MR, Stalinski LL, Pelz-Stelinski KS: Selection of an insect vector with a bacterial plant pathogen increases its propensity for dispersal. PLOS ONE 2015, 10:e0129373 http://dx.doi.org/10.1371/journal.pone.0129373. This study demonstrates that a phytoplasma PTP can enhance dispersal behaviors following acquisition and colonization of psyllid vector tissues, which is beneficial for pathogen spread. Dispersal propensity and duration were both tightly linked to pathogen titer in the vector, strongly suggesting active direct manipulation by the pathogen. Vectors that had low titers or that did not successfully acquire the pathogen suffered lower dispersal capacity relative to uninfected vectors growing on healthy plants, but those that had high titers had the highest dispersal capacity.


48. Westwood JH, Groen SC, Du Z, Murphy AM, Anggoro DT, Tungadi T, Long V-N, Lewsey MG, Rossiter JT, Powell G, Smith AG: A trio of viral proteins tunes aphid–plant interactions in Arabidopsis thaliana. PLOS ONE 2013, 8:e83066 http://dx.doi.org/10.1371/journal.pone.0083066. In this paper, the authors manipulate expression of individual virus genes in a host plant in order to examine the effects of each virus component on host phenotype and vector behavior. They show that a combination of several genes is necessary to produce a phenotype that is conducive to transmission. This approach could be possible in other systems where model plants are potential hosts.


54. Casteel CL, Yang C, Nanduri AC, 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. This paper describes the genetic basis for effects of TuMV on aphid settling and performance. It also demonstrates that for this NPTP, transmission can occur even after long-term colonization by the vector. This suggests that studies into virus-induced changes in host phenotype can also shed new light on aspects of the virus–host–vector interaction (such as potential flexibility in transmission mechanism) that may have been previously overlooked.


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62. MacLean AM, Orlovskis Z, Kowitwanich K, Zdzierska AM, Angenent GC, Immink RG, Hogenhout SA: Phytoplasma effector SAP54 hijacks plant reproduction by degrading MADS-box proteins and promotes insect colonization in a RAD23-dependent manner. PLoS Biol 2014, 12:e1001835 http://dx.doi.org/10.1371/journal.pbio.1001835. This study uses a model plant–pathogen system to identify a specific pathogen-produced effector protein as the factor responsible for inducing a host plant phenotype which is more palatable to leafflower vectors. This system shows promise as a model for future work to understand the mechanisms underlying pathogen effects on host–vector communication.


