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Effects of pathogens on sensory-mediated interactions between plants and insect vectors

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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.

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Introduction

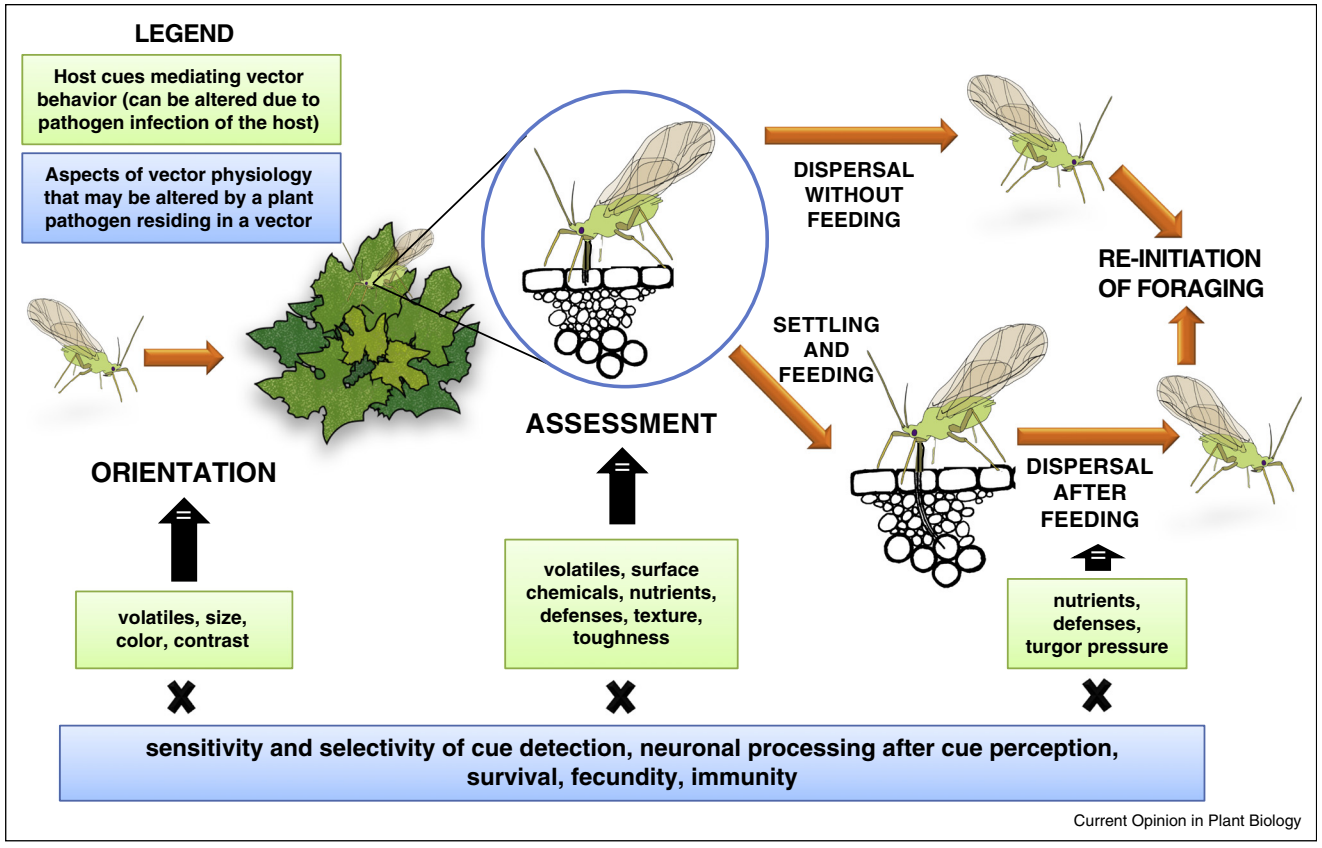
Parasites frequently alter the phenotypes of their hosts in ways that enhance their own transmission and fitness, and such effects can have profound implications not only for parasite transmission but also for the structure and dynamics of ecological communities [1]. Work exploring the ecological implications of host manipulation has focused primarily on animal parasites [2–4], but, given the importance of plants in terrestrial ecosystems, the manipulative effects of plant parasites on host phenotypes might have equal or greater significance for ecology [1]. A key way in which parasites influence the interactions of their hosts with other organisms, and thus transmission, is by influencing potential sensory cues produced by infected hosts. For example, some trophically transmitted animal parasites elicit or alter the production of cues that make intermediate hosts more conspicuous to predators that are primary hosts [3]. Similarly, cues from infected hosts can also make them more conspicuous to vectors [5,6•,7], including cues from plants infected with pathogens that are transmitted by insect herbivores [6•,8•,9••]. Consequently, selection may be expected to favor plant pathogens that influence plant cues and vector behavior in ways that are conducive to pathogen acquisition and inoculation by vectors. Here we discuss recent findings suggesting that vector behavior is influenced both by pathogen effects on plant cues that facilitate discrimination between infected and healthy hosts and by direct pathogen effects on vector physiology and cue perception.

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•,13] (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

Figure 1



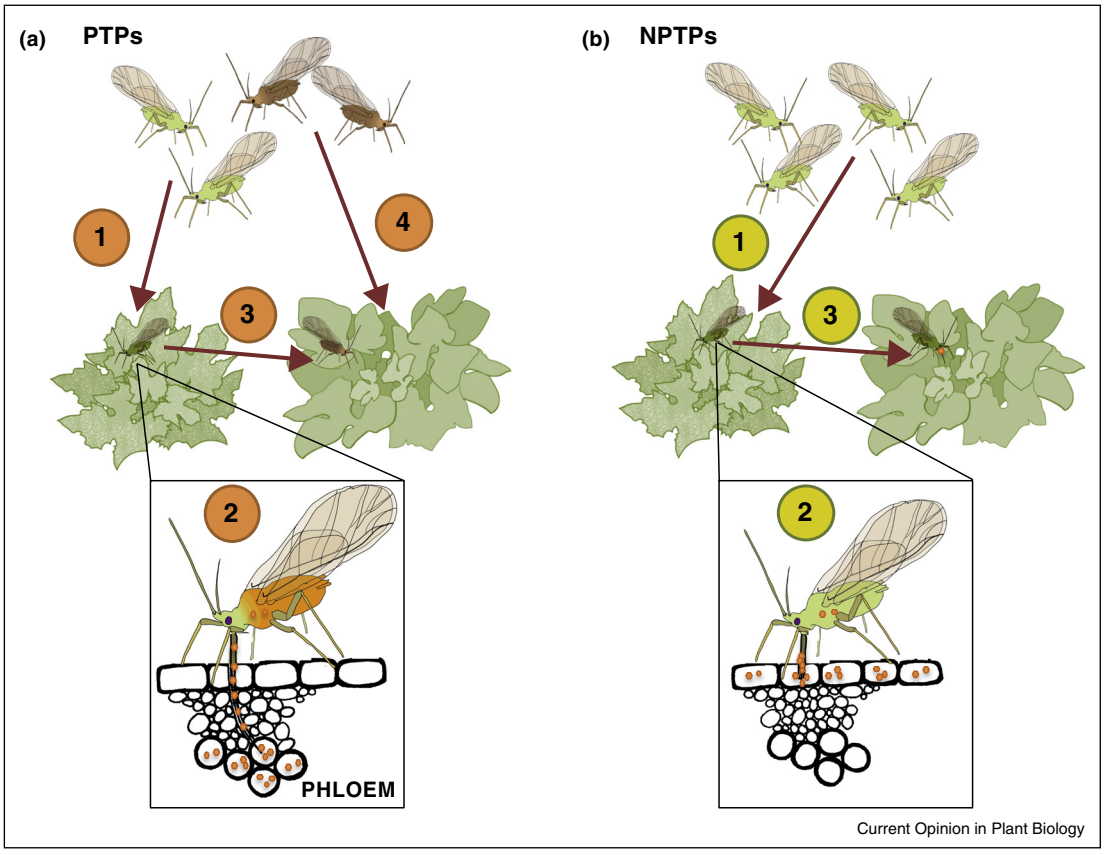
Potential behavioral sequences of a vector in response to aspects of the host plant phenotype (stimuli) that can be altered by pathogen infection. In most cases, pathogen acquisition by the vector occurs during host plant feeding (persistently transmitted pathogens), but can also occur for some pathogens during the more superficial 'assessment' behavior (non-persistently 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 stylets) [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).

Figure 2



Expectations for plant pathogen effects on herbivorous vector behavior for **(a)** persistently transmitted pathogens (PTPs), and **(b)** non-persistently transmitted pathogens (NPTPs). PTPs are generally acquired during long-term feeding, usually in the phloem. They exit the gut into the hemocoel to colonize other tissues (e.g., salivary glands) from which they can reside or replicate to be inoculated to multiple plants. In contrast, non-persistently transmitted pathogens (NPTPs) are acquired and inoculated during brief tastes of outer plant cells. NPTPs bind to specific regions of the mouthparts, but are only retained for a few hours and can only be inoculated to one or two plants following acquisition. If the vector proceeds to long-term feeding on the infected host after acquiring a NPTP, then the NPTP particles are lost from the mouthparts. On the basis of these requirements for transmission, it is expected that should induce changes in the host phenotype, or in vector perception/response to host cues, that favor (i) orientation of non-infectious vectors (green) to infected hosts (point A1), (ii) settling and feeding of non-infectious vectors on the infected host for a period sufficient to acquire the PTP (usually from the phloem) (point A2), and (iii) eventual dispersal of infectious vectors (orange) from infected hosts *to* healthy hosts (point A3). For PTPs, infectious vectors (orange) can remain in the general vector pool for long periods of time (point A4), and can continue to inoculate healthy plants after a single acquisition. Therefore, we also expect to see differences in vector preferences and/or feeding behaviors depending on infectious condition (conditional vector preferences). In contrast, NPTPs do not persist in the vector pool for more than a few hours and do not circulate within the vector. It is therefore expected that NPTPs should induce changes in the host phenotype that favor (i) orientation of vectors towards the infected plant (point B1), (ii) probing of the outer plant cells and uptake of cell contents without proceeding to phloem feeding (so that the NPTP is retained on the mouthparts) (point B2), and (iii) dispersal from the infected plant to a healthy plant within a short time period (hours) (point B3 — host dependent vector preferences).

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.

interactions (stated in Box 1 and elaborated in Figure 2). These hypotheses function as a set of criteria for determining whether a given pathogen has effects that are conducive to transmission, including effects on vector orientation in response to host cues (Hypothesis 1), pathogen acquisition during assessment of host quality (Hypothesis 2), and vector dispersal following assessment and/or pathogen acquisition (Hypothesis 3). These hypotheses are informed by both early studies focused on pathogen effects on plant cues mediating acquisition (Hypotheses 1 and 2) (reviewed in [6•,8•,13]) and by more recent studies exploring vector dispersal from infected plants to healthy hosts after assessment or feeding (Hypothesis 3).

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 [6•,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*-carrying 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••,47]. 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

Table 1

Research priorities for future studies of pathogen effects on host–vector communication and selected examples of recent progress

Research goal	Key questions	Supporting literature
Examine pathogen effects on communication between multiple hosts and herbivorous vectors	Can multi-host pathogens manipulate the phenotype of multiple hosts?	[21••,37••,58–60]
	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?	
Determine mechanisms underlying pathogen-induced changes in host phenotypes and direct effects of pathogens on vector behavior	How does plant phenotype change throughout disease progression in the host?	[9••,33,34,39,41•,43•,47,48•,51,54•,55,61,62•]
	How does vector perception of, or response to stimuli change when carrying a PTP?	
	What pathogen traits are responsible for inducing these changes?	
Explore the ecological context of pathogen effects on host and vector physiology	How is communication between plants and vector natural enemies changed by pathogen effects on plant phenotypes?	[63–71]
	Do pathogen effects also influence plant interactions with non-vector herbivores?	
	Do pathogen effects influence plant resistance to abiotic stress?	

genotypes across multiple host–vector combinations 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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- • of special interest
- •• of outstanding interest

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This study describes a host-dependent vector preference shift that occurs in a pathosystem consisting of a beetle vector, a bacterial pathogen, and cucurbit 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 nectaries when beetles aggregate within the flowers and deposit frass.

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