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Evolutionary Determinants of Host and Vector Manipulation by Plant Viruses

Kerry E. Mauck*,[1], Quentin Chesnais*, Lori R. Shapiro†
*Department of Entomology, University of California, Riverside, Riverside, CA, United States
†Department of Applied Ecology, North Carolina State University, Raleigh, NC, United States
[1]Corresponding author: e-mail address: kerry.mauck@ucr.edu

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Abstract

Plant viruses possess adaptations for facilitating acquisition, retention, and inoculation by vectors. Until recently, it was hypothesized that these adaptations are limited to virus proteins that enable virions to bind to vector mouthparts or invade their internal tissues. However, increasing evidence suggests that viruses can also manipulate host plant

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phenotypes and vector behaviors in ways that enhance their own transmission. Manipulation of vector–host interactions occurs through virus effects on host cues that mediate vector orientation, feeding, and dispersal behaviors, and thereby, the probability of virus transmission. Effects on host phenotypes vary by pathosystem but show a remarkable degree of convergence among unrelated viruses whose transmission is favored by the same vector behaviors. Convergence based on transmission mechanism, rather than phylogeny, supports the hypothesis that virus effects are adaptive and not just by-products of infection. Based on this, it has been proposed that viruses manipulate hosts through multifunctional proteins that facilitate exploitation of host resources and elicitation of specific changes in host phenotypes. But this proposition is rarely discussed in the context of the numerous constraints on virus evolution imposed by molecular and environmental factors, which figure prominently in research on virus–host interactions not dealing with host manipulation. To explore the implications of this oversight, we synthesized available literature to identify patterns in virus effects among pathogens with shared transmission mechanisms and discussed the results of this synthesis in the context of molecular and environmental constraints on virus evolution, limitations of existing studies, and prospects for future research.

1. INTRODUCTION

Vector-borne plant viruses are obligate intracellular parasites that can fundamentally change the physiology of their host plants. The outcomes of these changes, such as reductions in crop yield or quality, have driven much of the research on virus–host interactions. But virus effects on host plants extend well beyond agronomically relevant metrics. There is now increasing evidence that viruses can alter aspects of the host plant phenotype (cues) that mediate interactions with other organisms, including the mobile insect vectors responsible for much of virus transmission (Casteel and Falk, 2016; Eigenbrode and Bosque-Perez, 2016; Mauck, 2016; Mauck et al., 2012, 2016). These cues include visual and tactile characteristics, odors, induced defenses, secondary metabolites, sugars, free amino acids, and likely other undescribed factors (Bosque-Pérez and Eigenbrode, 2011; Casteel et al., 2014; Mauck et al., 2014a,b). Insect vectors make their initial foraging decisions by integrating visual and odor cues, which convey information about plant presence, identity, and quality. After contacting a host plant, vectors assess additional cues from leaf or stem surfaces, parenchyma, and vascular tissues through olfactory and gustatory sensory systems. The insects' probing, feeding, and dispersal behaviors in response to plant cues directly determine the probability that virions will be acquired, retained, and transported (Fereres, 2016; Fereres and Collar, 2001; Hogenhout et al., 2008;

Madden et al., 2000; Moreno et al., 2012; Ng and Falk, 2006). Thus, if a plant virus alters aspects of the host plant that provide cues for its herbivorous vectors, these changes have potential to influence rates of host–vector contact and vector-feeding behaviors that determine virus transmission. Given that transmission is critical to the fitness of vector-borne plant viruses, it has been proposed that viruses evolve traits that induce (or at least maintain) host phenotypes and effects on vectors that encourage virus spread.

Consistent with this hypothesis, there are now more than 100 published reports of plant viruses purportedly "manipulating" host plant phenotypes to increase vector attraction to infected plants, or elicit transmission-conducive feeding behaviors (reviewed in Casteel and Falk, 2016; Eigenbrode and Bosque-Perez, 2016; Fereres and Moreno, 2009; Heil, 2016; Mauck, 2016; Mauck et al., 2012, 2016). More recently, there is evidence that some plant viruses can also manipulate vector behaviors to favor virus transmission to new hosts by interacting with the vector's tissues following acquisition from infected hosts or artificial substrates (Ingwell et al., 2012; Moreno-Delafuente et al., 2013; Rajabaskar et al., 2014; Stafford et al., 2011). The idea that plant viruses can manipulate hosts and vectors to enhance transmission is not unique. Manipulation of host phenotypes by parasites is well documented across a wide range of taxonomic groups (Heil, 2016). The fitness advantages of manipulation as a strategy are captured by the "adaptive host manipulation hypothesis," which proposes that parasites can evolve to control elements of their host's phenotype that help maintain or enhance rates of transmission (Poulin, 2010). Thus, "manipulated" hosts exhibit additional hallmarks of infection beyond those associated with the basic need for a parasite to attenuate host immunity and use host resources for reproduction (Lefèvre et al., 2009). Although there are hundreds of reports of putative host manipulation by parasites (Lafferty and Shaw, 2013; Lefèvre et al., 2009; Mauck et al., 2012, 2016; Poulin, 2010), only a handful of these studies have made progress in pinpointing the *parasite* as the "manipulator"—that is, the organism having genetic control over the altered host phenotype. An equally likely explanation is that the phenotype of the infected host is due to an immune response under genetic control of the host. Alternatively, observed phenotypes could represent by-products of pathology, or even the residual influence of inherited ancestral traits that were adaptive in one host–parasite context but have become maladaptive in another context (Heil, 2016). Parsing these explanations has proven difficult for eukaryotic parasites that provide the most charismatic examples of host manipulation, but prove to be intractable laboratory models. The growing evidence of host and vector

manipulation by plant viruses provides new opportunities to explore the adaptive significance of parasite manipulation in the context of environmental variation using pathosystems that are more amenable to experimental methods involving functional genomics.

Although we do not yet have a thorough mechanistic understanding of plant virus genes that confer manipulative traits, there is still evidence to support the hypothesis that host and vector manipulation is adaptive for plant viruses. Theoretical studies demonstrate that "manipulative" viruses inducing transmission-enhancing effects in hosts (or vectors) will spread more rapidly, and from lower starting frequencies, relative to viruses that have neutral effects, or viruses that elicit changes that deter virus acquisition by vectors (Jeger et al., 2004; McElhany et al., 1995; Roosien et al., 2013; Shaw et al., 2017; Sisterson, 2008). Thus, under ideal conditions, manipulative virus genotypes are expected to enjoy higher fitness than nonmanipulative virus genotypes. Additionally, the nature of virus influence on vectors generally corresponds with the virus transmission mechanism regardless of virus phylogeny (Mauck et al., 2012). In other words, virus-induced changes in host cues and quality for vectors are not uniform, but differ depending on the requirements for virion uptake and transmission that are inherent to a given virus (Mauck et al., 2012, 2016). Each insect-borne plant virus is classified into one of the four transmission mechanism groups depending on requirements for acquisition, retention, and inoculation (Table 1). These requirements are based on virus localization within hosts and the nature of associations with vectors, which range from transient binding to cuticular surfaces of vector mouthparts (noncirculative, nonpersistent viruses), to invasion of the hemocoel (circulation) and retention (persistence) of ingested virions in salivary glands (circulative-persistent viruses). Within the circulative-persistent category, some viruses undergo active replication in salivary glands and other internal vector tissues (propagative), while others localize to salivary glands but do not replicate (nonpropagative) (Table 1). These associations determine what vector behavioral sequences most favor efficient virus acquisition, retention, and eventual inoculation. Thus, transmission mechanism groups can serve as a basis for generating predictions about how any given virus might be expected to alter host phenotypes (or vector physiology) to influence probing, feeding, and dispersal behaviors in ways that are conducive to its own transmission. The adaptive significance of virus effects on hosts and vectors can then be explored by evaluating evidence for convergence in virus effects across phylogenetically diverse viruses that share a transmission mechanism group.

Table 1 Characteristics of Plant Virus Transmission Mechanism Groups

	Circulative		Noncirculative	
Transmission Modes	**Persistent Propagative**	**Persistent Nonpropagative**	**Semipersistent**	**Nonpersistent**
Acquisition time[a]	Minutes to hours		Seconds to hours	Seconds to minutes
Retention time[b]	Days to months		Minutes to hours	Seconds to minutes
Inoculation time[c]	Minutes to hours		Seconds to hours	Seconds to minutes
Association with vectors[d]	Internal		External	
Replication in vectors	Yes	No	No	
Association with plants	Restricted to phloem		Some restricted to phloem, others not	Not restricted to phloem
Abbreviation	C-P-Prop	C-P-NProp	NC-SPer	NC-NPer

[a]Time required for a vector to efficiently acquire virus particles following initiation of probing or feeding.
[b]Time during which the virus remains infectious within its vector following acquisition.
[c]Time required for a vector to efficiently inoculate infectious virus particles to a new healthy plant.
[d]Internal means that the virus enters the inner body of its vector, passing through cellular barriers. External means that the virus binds to cuticular surfaces (stylet or foregut) and never passes through cellular barriers. Adapted from Brault, V., Uzest, M., Monsion, B., Jacquot, E., Blanc, S., 2010. Aphids as transport devices for plant viruses. C. R. Biol. 333 (6–7), 524–538. https://doi.org/10.1016/j.crvi.2010.04.001.

We took this approach for our earlier quantitative synthesis of 55 papers reporting putative instances of virus manipulation (Mauck et al., 2012), which was the first to show that phylogenetically divergent plant viruses transmitted via the same sequences of vector behavior induce similar phenotypes in their host plants. By demonstrating convergent effects based on transmission mechanism group, this synthesis provided support for the hypothesis that virus effects are the result of adaptations and not just by-products of infection. Since the publication of this synthesis, the number of empirical reports of putative plant virus manipulation has more than doubled. Despite the popularity of this topic and its clear relevance for understanding virus epidemiology, there has not been any subsequent attempt to comprehensively reevaluate virus effects with regard to transmission mechanism groups, or to place this body of work within the context of constraints

on virus evolution imposed by molecular and environmental factors, which should not be lightly dismissed. Plant viruses have small genomes that often encode less than 10 functional proteins, sometimes through overlapping open reading frames. Virus proteins perform multiple functions in the host plant, interact with each other extensively, and may play a dual role in facilitating interactions with both plant and vector tissues. These features enable rapid replication and maintain vector transmissibility but impose major limitations on virus evolution because most mutations are likely to be deleterious and will be rapidly purged. Molecular constraints will further interact with environmental factors to shape virus evolution. In a field context, plant viruses are subject to heterogeneous host environments at intraspecific and interspecific levels, as well as variation in the frequency of transmission-conducive contacts with vectors (Elena et al., 2014; Gutiérrez et al., 2013; Pagán et al., 2012; Rodelo-Urrego et al., 2013; Roossinck and García-Arenal, 2015). These constraints will influence the evolution and maintenance of manipulative traits in plant viruses, and thus all reports of putative manipulation of hosts and vectors must be considered within the context of these constraints.

To explore the extent to which the existing literature considers molecular and environmental axes of virus evolution, and to revisit the question of whether viruses exhibit convergence in effects within each transmission mechanism group, we performed a comprehensive review and quantitative synthesis of all studies reporting putative instances of virus manipulation of hosts and vectors following the guidelines used in Mauck et al. (2012). Here, we discuss this synthesis in the context of the methodologies employed and molecular and environmental factors that may facilitate, or hinder, the evolution of manipulative functions. Our results provide evidence of convergence in virus effects within transmission mechanism groups while revealing a number of inadequacies in the current literature that provide a roadmap for future research directions.

2. VIRUS EFFECTS ON HOST PHENOTYPES AND VECTOR BEHAVIOR

Virus manipulation of vector behavior can occur via two mechanisms that are not mutually exclusive. The most reported mechanism, and the first discussed in our synthesis, involves changes in aspects of the host phenotype that influence vector orientation to, and feeding behaviors on, virus-infected plants (Eigenbrode and Bosque-Perez, 2016; Fereres and Moreno, 2009;

Mauck et al., 2012, 2016). This pathway is *indirect* because it is mediated by the host resource being shared by both the virus and its vector(s). A second, more recently described behavioral modification occurs when the virus is acquired by the vector and *directly* interacts with vector tissues (Ingwell et al., 2012; Stafford et al., 2011). These more intimate associations create opportunities for viruses to alter vector physiology in ways that affect behaviors related to virus transmission (e.g., relative preferences for host cues mediating orientation to, and feeding on, infected or healthy hosts). The question of whether viruses can evolve these functions in natural or agricultural settings is central to our understanding of the ecological and epidemiological importance of host and vector manipulation. To begin to address this question, we provide a conceptual outline of predictions for adaptive virus effects that is based on the transmission requirements outlined in Table 1 and Fig. 1, and then present a quantitative synthesis of all experiments on plant virus manipulation of vector behavior performed to date. Our goal is to create a framework for discussing the mechanisms underlying the evolution of manipulative functions, molecular and environmental constraints on these mechanisms, limitations of the existing studies, and possible avenues for future research.

2.1 Transmission Mechanisms and Predicted Virus Effects

Much emphasis is placed on the host plant as a selective agent driving virus evolution. This supposition is based on the idea that the host is the fundamental environment in which the virus resides (Elena et al., 2014). While this is true, plant virus fitness ultimately depends on the virus capacity to infect more than one host, which is often achieved via the feeding activities of mobile insect vectors (Whitfield et al., 2015). As discussed briefly in the Introduction, plant viruses are classified into different transmission mechanism groups according to their associations with hosts and vectors (Table 1) and the corresponding vector–host interactions required for transmission. Arthropod-borne circulative-persistent viruses (designated here as C-P) can be either propagative (C-P-Prop viruses) or nonpropagative (C-P-NProp viruses) and are transmitted by insects and arachnids with variations on the piercing–sucking mode of feeding (aphids, whiteflies, leafhoppers, planthoppers, and mites) (Table 1; Fig. 1). Propagative viruses replicate within their vectors, while nonpropagative viruses circulate and reside in specific tissues but do not engage in replication (Fig. 1). Both are generally phloem restricted and acquired during long-term phloem sap ingestion (Table 1).

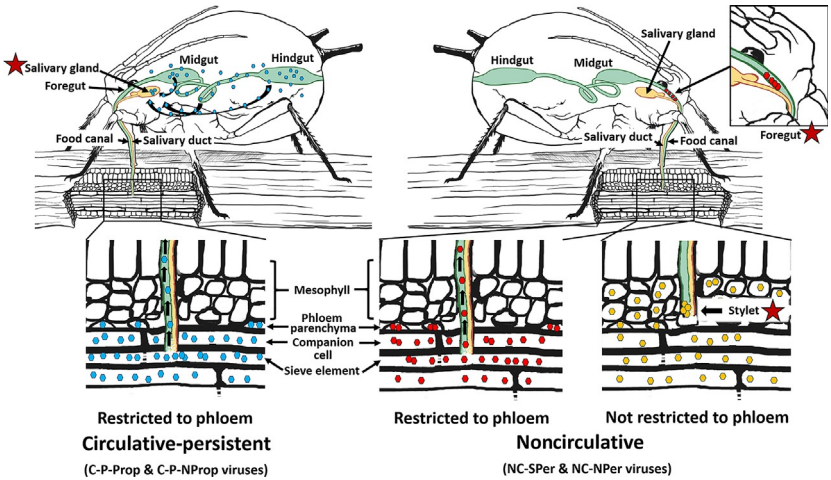

Fig. 1 Schematic representations of plant virus transmission mechanisms. Retention sites for each virus are indicated by a *red star* adjacent to the labeled area. *Left*: Schematic representation of associations between hosts, vectors, and circulative-persistent propagative (C-P-Prop) viruses or circulative-persistent nonpropagative (C-P-NProp) viruses. Nearly all circulative-persistent viruses are restricted to the phloem, and so acquisition requires phloem sap ingestion by the vector. Virions are retained in salivary glands after acquisition and, for propagative viruses, within several other vector tissues. *Right*: Schematic representation of associations between hosts, vectors, and noncirculative viruses. Depending on where the virus is localized within the plant, virions of noncirculative viruses will be acquired during long-term phloem sap ingestion (like phloem-restricted circulative-persistent viruses) or during brief probes of epidermal or mesophyll tissue prior to phloem contact. Noncirculative semipersistent (NC-SPer) viruses exhibit both types of association with plants, whereas noncirculative nonpersistent (NC-NPer) viruses are only acquired from epidermal and mesophyll cells and are lost if the vector feeds in the phloem.

Noncirculative viruses (designated as NC) can be either semipersistent (NC-SPer viruses) or nonpersistent (NC-NPer viruses). NC-SPer viruses are transmitted by aphids, whiteflies, mealybugs, beetles, and leafhoppers (Fereres and Raccah, 2009) and are usually retained in the foregut—a chitinous anterior region of the alimentary canal that immediately precedes the gut (Fig. 1). Virion retention is typically measured in hours (Table 1) and virions can be inoculated to multiple hosts following a single acquisition event. Some NC-SPer viruses are phloem-limited (Ng and Zhou, 2015), so the vector behaviors that favor efficient acquisition (long-term phloem ingestion) are similar to those required for circulative-persistent viruses (Fig. 1). Other NC-SPer viruses are not restricted to the phloem and can also occupy mesophyll tissue (Fig. 1). These viruses are acquired during brief ingestion events

from nonvascular tissues (similar to NC-NPer viruses) (Fig. 1). NC-NPer viruses are exclusively transmitted by aphids and are acquired rapidly during brief probes in the nonvascular tissues of the plant *before* phloem ingestion occurs. The virus binds to target sites on the aphid stylet (Hogenhout et al., 2008; Martin et al., 1997; Ng and Falk, 2006) and virions can be lost if the vectors proceed to initiate sustained phloem sap ingestion on the infected host plant (Ng and Falk, 2006) (Fig. 1). Thus, in contrast to phloem-restricted C-P viruses, transmission of NC viruses is favored by dispersal of a vector from an infected plant shortly after initial probing to assess host suitability. Following NC-NPer virion acquisition, vectors typically remain viruliferous (i.e., carry virus) for a brief period (minutes) and are capable of inoculating only a few plants before losing viruliferous status.

Each virus transmission mechanism is characterized by different host and vector associations, and requirements for acquisition and inoculation. But among taxa that share a transmission mechanism group, the same patterns of vector behavior are required for virus spread. We used these shared requirements for transmission as a basis for generating predictions about how phylogenetically diverse viruses within a group might alter host phenotypes and/or vector physiology to enhance specific probing, feeding, and dispersal behaviors in ways that are generally conducive to transmission. These predictions are described in Fig. 2 and tested using our quantitative synthesis in the ensuing sections.

2.2 Host-Mediated Effects of Plant Viruses on Vector Behavior

Viruses can alter multiple biochemical pathways in plants, each of which might disrupt cues that are important for vector foraging. Identifying each of the cues mediating a putative manipulation, and the virus genes responsible, would be extremely challenging. Instead of taking this approach, we used previously published methods (Mauck et al., 2012) to find evidence of convergence in virus effects on host phenotypes and vector behavior across distantly related taxa that share a transmission mechanism group. Here, we present the results of this expanded quantitative synthesis, which includes all literature from 1960 to 2017. In brief, we searched Google Scholar and the ISI Web of Science for publications related to virus–host–vector interactions following the criteria for manuscript identification and selection described by Mauck et al. (2012). Our first review summarized 55 papers published between 1960 and 2012. Here, we consider an additional 67 newer papers (for a total of 122). We parsed each study into individual experiments, each

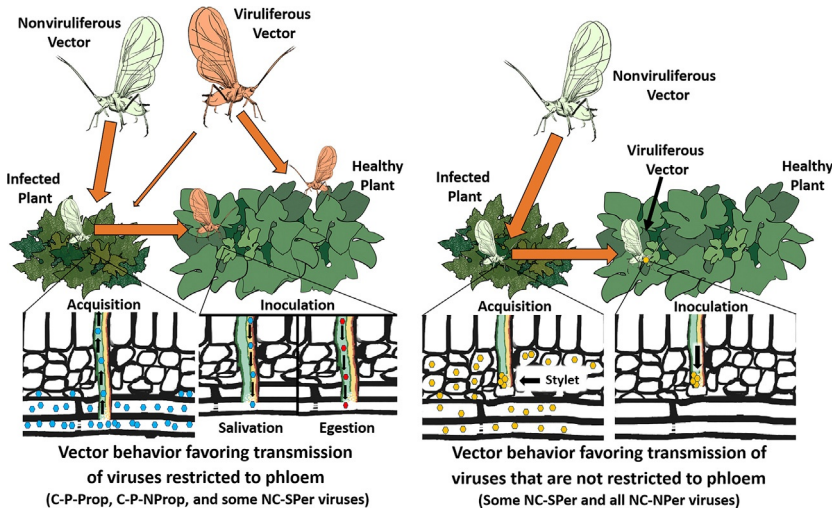

Fig. 2 Graphical representations of predictions for transmission-conducive effects of viruses on the behavior of their insect vectors. We expect to see convergence of virus effects across phylogenetically distant virus groups that share the same transmission mechanism, and, therefore, are transmitted via similar sequences of vector probing, feeding, and dispersal behavior (Fig. 1; Table 1). *Prediction 1*: Consistent with the need to maintain or increase vector contact rates with infected hosts, we expect both viruses that are restricted to phloem (*left*) and viruses that are not phloem restricted (*right*) will have neutral to positive effects on plant cues mediating vector orientation preference. *Prediction 2*: Viruses that are restricted to phloem diverge from viruses that are not restricted to phloem in requirements for vector probing/feeding frequencies and durations (Fig. 1; Table 1). We expect phloem-restricted viruses (C-P-Prop, C-P-NProp, and some NC-SPer viruses) to have neutral to positive effects on plant palatability cues that encourage vector settling and feeding prior to dispersal (to facilitate virion uptake from the phloem) (*left*). In contrast, we expect viruses that are not restricted to phloem (*right*) to reduce plant palatability in ways that discourage sustained feeding to ensure vectors disperse after probing and acquiring virions, and before virions are lost during subsequent salivation events (Ng and Falk, 2006). This category includes all NC-NPer viruses and some NC-SPer viruses. *Prediction 3*: We expect to see plant quality effects that are consistent with virus-induced changes in host palatability. For phloem-restricted viruses (C-P-Prop, C-P-NProp, and some NC-SPer viruses), enhanced host quality may lead to higher reproductive rates, crowding, accelerated use of host resources, and eventual dispersal of viruliferous vectors (*left*). For viruses that are not restricted to the phloem (all NC-NPer viruses and some NC-SPer viruses), we expect to see reductions in plant quality, which will increase vector restlessness and opportunities for virus acquisition (*right*). *Predictions regarding direct effects*: For C-P-Prop and C-P-NProp viruses that are phloem-restricted (*left*), dispersal is beneficial for the virus only if it occurs after sufficient virions have been acquired to establish a viruliferous state (either by retention or by replication). Therefore, we expect that these viruses may evolve mechanisms to encourage dispersal following several hours to days of feeding on infected hosts, and to discourage viruliferous vectors from visiting virus-infected hosts (indicated by a *smaller*

addressing a single virus strain or isolate by host species (or cultivar) by vector interaction (440 experiments total). Each experiment was categorized according to the virus transmission mechanism group (Table 1; Fig. 1) and the vector response component (orientation preference, settling/feeding preference, or performance) (Fig. 2). For experiments measuring preferences (orientation or settling/feeding), results were classified as preference for virus-infected plants, preference for healthy plants or no preference. For measurements of vector performance, results were classified as a positive, neutral, or negative effect of virus infection in a host plant on the vector(s). Compiled results were evaluated for departure from an expected even distribution of effects (1:1:1 for *infected/no preference/healthy* within behavioral effect categories, and *positive/neutral/negative* within the performance effect categories) across the three interaction types using chi-square tests followed by pairwise comparisons (*P*-value adjustment method: "fdr," R package "RVAideMemoire"). To test our hypothesis that neutral effects should also be considered adaptive for the virus, we then evaluated results using two-way chi-square tests by grouping the "no preference" or "neutral" categories with the appropriate "transmission-enhancing" category for each transmission mechanism group. Individual groupings for each transmission mechanism group are described within the predictions below.

Prediction 1: Both circulative-persistent viruses and noncirculative viruses will have neutral to positive effects on plant cues mediating vector orientation.

Our analysis of the 52 available experiments related to this first prediction indicates that C-P-NProp viruses often induce changes in host phenotype that result in enhanced attraction of vectors to virus-infected plants when presented in a choice scenario vs healthy plants (Tables 2 and 3). An important caveat of this result is that almost all the experiments studying this effect for C-P-NProp viruses focused on Luteoviridae, with the exception of one study on a Begomovirus showing that the whitefly vector, *Bemisia tabaci*, exhibited no orientation preference between *Tomato severe rugose virus*-infected (*ToSRV*, Geminiviridae) and healthy tomatoes (Fereres et al., 2016). For the NC-NPer viruses, while the number of available studies is more limited, our

orientation arrow toward infected hosts, and a *larger orientation arrow* to healthy hosts). These mechanisms could include direct effects of retained/replicating viruses on vector behavior (e.g., perception or processing of host cues), indirect effects on host plants (e.g., changes in palatability or quality that only occur following vector feeding), or a combination of both of these mechanisms.

Table 2 Quantitative Synthesis of Overall Host-mediated Effects of Plant Viruses on Vector Behavior and Performance

			Effect on Orientation Preference			Effect on Settling/Feeding Preference			Effect on Performance		
Mode of Transmission	**Virus taxon**	**Genome**	**Infected**	**No Pref.**	**Healthy**	**Infected**	**No Pref.**	**Healthy**	**Positive**	**Neutral**	**Negative**
C-P-Prop viruses	Tospoviridae	(−) seg. RNA	0	0	0	21	3	1	8	8	3
	Reoviridae	ds RNA	4	1	0	1	1	3	3	2	2
		Sum	4	1	0	22(a)	4(b)	4(b)	11	10	5
			Chi-square = not tested			*Chi-square = 21.6, P < 0.001*			*Chi-square = NS*		
C-P-NProp viruses	Luteoviridae	(+) ss RNA	20	8	2	24	13	3	37	10	6
	Geminiviridae	ss circ. DNA	0	1	0	10	2	2	37	20	14
	Nanoviridae	ss circ. DNA	0	0	0	1	0	0	1	0	0
		Sum	20(a)	9(b)	2(b)	35 (a)	15(b)	5(c)	75(a)	30 (b)	20(b)
			Chi-square = 15.94, P < 0.001			*Chi-square = 25.45, P < 0.001*			*Chi-square = 41.20, P < 0.001*		

			Effect on Orientation Preference			Effect on Settling/Feeding Preference			Effect on Performance		
Mode of Transmission	**Virus taxon**	**Genome**	**Infected**	**No Pref.**	**Healthy**	**Infected**	**No Pref.**	**Healthy**	**Positive**	**Neutral**	**Negative**
NC-SPer viruses	Closteroviridae	(+) ss RNA	0	1	2	8	2	1	6	3	1
	Sobemovirus	(+) ss RNA	0	0	0	1	0	0	1	0	0
	Caulimoviridae	ds DNA	0	0	0	0	1	1	0	0	2
	Secoviridae	ss RNA	1	0	0	4	0	0	1	1	1
		Sum	1	1	2	13(a)	3(b)	2(b)	8	4	4
			Chi-square = not tested			*Chi-square = 12.33, P = 0.002*			*Chi-square = NS*		
NC-NPer viruses	Bromoviridae	(+) ss RNA	4	2	0	3	5	7	3	5	15
	Potyviridae	(+) ss RNA	3	3	0	14	12	12	17	12	13
		Sum	7	5	0	17	17	19	20	17	28
			Chi-square = 6.5, P = 0.039			*Chi-square = NS*			*Chi-square = NS*		

Transmission group abbreviations: C-P-NProp, circulative-persistent nonpropagative viruses; *C-P-Prop*, circulative-persistent propagative viruses; *NC-NPer*, noncirculative nonpersistent viruses; *NC-SPer*, noncirculative semipersistent viruses. Genome abbreviations: *dsRNA*, double-stranded RNA viruses; *(−) seg. RNA*, negative sense, segmented, single-stranded RNA viruses; *ss circ. DNA* = single-stranded circular DNA viruses; *(+) ssRNA* = positive sense, single-stranded RNA viruses; *ssRNA*, single-stranded RNA viruses, sense unknown. Different lower case letters indicate statistically significant differences between effects (pairwise comparisons using chi-squared tests, $P < 0.05$).

Table 3 Quantitative Synthesis of Adaptive vs Nonadaptive Host-mediated Effects of Plant Viruses on Vector Behavior and Performance

			Effect on Orientation Preference		Effect on Settling/ Feeding Preference		Effect on Performance	
Mode of transmission	**Virus Taxon**	**Genome**	**Adaptive**	**Maladaptive**	**Adaptive**	**Maladaptive**	**Adaptive**	**Maladaptive**
C-P-Prop viruses	Tospoviridae	(−) seg. RNA	0	0	24	1	16	3
	Reoviridae	ds RNA	5	0	2	3	5	2
		Sum	5	0	26	4	21	5
			Chi-square = not tested		*Chi-square = 16.13, $P < 0.001$*		*Chi-square = 9.85, $P = 0.002$*	
C-P-NProp viruses	Luteoviridae	(+) ss RNA	28	2	37	3	47	6
	Geminiviridae	ss circ. DNA	1	0	12	2	57	14
	Nanoviridae	ss circ. DNA	0	0	1	0	1	0
		Sum	29	2	50	5	105	20
			Chi-square = 23.52, $P < 0.001$		*Chi-square = 36.81, $P < 0.001$*		*Chi-square = 57.79, $P < 0.001$*	

			Effect on Orientation Preference		Effect on Settling/ Feeding Preference		Effect on Performance	
Mode of transmission	**Virus Taxon**	**Genome**	**Adaptive**	**Maladaptive**	**Adaptive**	**Maladaptive**	**Adaptive**	**Maladaptive**
NC-SPer viruses	Closteroviridae	(+) ss RNA	1	2	10	1	9	1
	Sobemovirus	(+) ss RNA	0	0	1	0	1	0
	Caulimoviridae	ds DNA	0	0	1	1	0	2
	Secoviridae	ss RNA	1	0	4	0	2	1
		Sum	2	2	16	2	12	4
			Chi-square = not tested		*Chi-square = 10.89, P = 0.001*		*Chi-square = 4.00, P = 0.045*	
NC-NPer viruses	Bromoviridae	(+) ss RNA	6	0	12	3	20	3
	Potyviridae	(+) ss RNA	6	0	24	14	25	17
		Sum	12	0	36	17	45	20
			Chi-square = 12.00, P < 0.001		*Chi-square = 6.81, P = 0.009*		*Chi-square = 9.61, P = 0.019*	

The "adaptive" category for each transmission mechanism group includes experiments demonstrating neutral effects plus the effect predicted to enhance transmission for viruses within that group (according to group characteristics in Table 1 and predictions in Fig. 2). Transmission group abbreviations: *C-P-NProp*, circulative-persistent nonpropagative viruses; *C-P-Prop*, circulative-persistent propagative viruses; *NC-NPer*, noncirculative nonpersistent viruses; *NC-SPer*, noncirculative semipersistent viruses. Genome abbreviations: *dsRNA*, double-stranded RNA viruses; *(−) seg. RNA*, negative sense, segmented, single-stranded RNA viruses; *ss circ. DNA*, single-stranded circular DNA viruses; *(+) ssRNA*, positive sense, single-stranded RNA viruses; *ssRNA*, single-stranded RNA viruses, sense unknown.

synthesis suggests that virus infections in host plants either enhanced or had no effect on orientation preference by vectors (Table 2). If we combine the two categories that would be considered "adaptive" for the virus (orientation preference toward infected plants + no preference) (Table 3), there is evidence of selection *against* virus genotypes that reduce host attractiveness to vectors. This pattern is unlikely to be the result of publication bias, since an adverse effect of a virus on its own transmission is still of ecological interest (Chen et al., 2015a,b). Concerning the two other transmission mechanism groups (C-P-Prop viruses and NC-SPer viruses), too few studies have been conducted to perform a statistical analysis; however for C-P-Prop viruses, it is again notable that no experiments reported orientation preferences for healthy plants (Tables 2 and 3). Thus, the pattern for C-P-Prop viruses is consistent with the other transmission classes, and the prediction described in Fig. 2; both circulative-persistent and noncirculative viruses have neutral to positive effects on plant cues mediating vector orientation.

Prediction 2: Nonviruliferous vectors prefer to settle and feed on plants infected with viruses acquired during phloem sap ingestion. We identified 156 experiments exploring vector settling preferences for virus-infected or healthy host plants. Consistent with our prediction (Fig. 2), our analysis suggests that nonviruliferous vectors preferred to settle and feed on plants infected with C-P-Prop viruses, C-P-NProp viruses, and NC-SPer viruses more than on healthy plants (Tables 2 and 3). Within the NC-SPer viruses, the pattern is driven largely by phloem-restricted Closteroviridae, which require sustained feeding for acquisition just like C-P-Prop and C-P-NProp viruses (Table 2). Experiments with Closteroviridae largely report settling preferences for infected plants (eight experiments) or no preference (two experiments), with just one experiment showing a preference for healthy plants. However, the intriguing patterns observed within the NC-SPer viruses must be considered with caution as there are only a limited number of studies (18 experiments) for this transmission mechanism group. For NC-NPer viruses, effects were evenly distributed among all three choices (Table 2). This pattern suggests that viruses within the NC-NPer transmission mechanism group may be less likely to manipulate hosts in ways that *enhance* vector dispersal following virion acquisition (Fig. 2). However, in the two-way comparison of adaptive vs maladaptive outcomes, there is evidence of selection *against* NC-NPer viruses that increase host palatability and thus inhibit vector dispersal and virus transmission (Table 3). Furthermore, the two virus families that make up the NC-NPer group differ in their effects. The Bromoviridae tend to induce

adaptive changes in host palatability that enhance or at least do not reduce the probability of vectors dispersing after acquiring virions (*chi-square* = 4.26, $P = 0.039$), while effects of Potyviridae are more evenly distributed (*chi-square* = 2.14, $P = 0.144$). This result is notable because we did not observe the same degree of divergence in effects according to virus family within the other transmission mechanism groups.

Prediction 3: Vectors perform better on plants infected by viruses restricted to phloem, and poorly on plants infected by viruses that are not restricted to phloem.

Among the 232 experiments addressing this prediction, our analyses suggest that C-P-Prop viruses and C-P-NProp viruses have mostly neutral to positive effects on vector performance (i.e., survival, fecundity, or both). This pattern is congruent with effects of these transmission classes on palatability (Tables 2 and 3) and supports our prediction (Fig. 2). Positive effects of virus infection are most evident for C-P-NProp viruses (Tables 2 and 3). For C-P-Prop viruses and NC-SPer viruses the analysis did not show significant differences from the expected even distribution (Table 2). But if neutral to positive effects are considered together (both of these being adaptive for the virus) (Table 3), there is evidence of selection against viruses that reduce vector performance (Table 3). It is particularly notable that C-P-Prop viruses have 21/26 experiments in the neutral or positive categories considering that vectors feeding on C-P-Prop virus-infected plants will also serve as hosts for virus replication (Barandoc-Alviar et al., 2016; Hogenhout et al., 2008). This is evidence of selection for genotypes that maintain or enhance host quality, and against genotypes that are overly pathogenic within vectors feeding on these infected hosts. For NC-SPer viruses, effects on performance seem to track with effects on host palatability, but this pattern must be considered with caution as the overall sample size is low (16 experiments). Phloem-restricted Closteroviridae represent more than half of the available experiments (10/16) and show strong evidence of adaptive effects on host plant quality for vectors (9/10 studies). This is consistent with the expectation that phloem-limited NC-SPer viruses acquired during long-term feeding should induce host phenotypes that are similar to those generated by circulative-persistent virus infection (Fig. 2). While the number of available studies is limited for nonphloem-restricted NC-SPer viruses (such as *Cauliflower mosaic virus* [CaMV, Caulimoviridae]), it is notable that the trend for this virus, which is acquired much more rapidly, seems to be the opposite.

For NC-NPer viruses, effects on host plant quality for vectors are evenly distributed across the three performance categories (Table 2) but fall largely

in the "adaptive" category in the two-way comparison of neutral + negative effects vs positive effects (Table 3). Notably, as was evident for settling preferences, the two virus families in the NC-NPer group diverge with respect to the distribution of experiments into adaptive vs maladaptive categories (Table 3). Viruses in the family Bromoviridae have mostly neutral + negative effects on plant quality (*chi-square* = 12.56, $P < 0.001$), while effects were evenly distributed between neutral + negative effects and positive effects for Potyviridae (*chi-square* = 1.52, $P = 0.217$) (Table 3). The divergence of effects on both plant palatability and plant quality between these two families within the NC-NPer group may reflect other biological characteristics of viruses within each taxon. For example, the Bromoviridae studied thus far are multipartite viruses that depend on vectors acquiring and retaining each encapsidated portion of the genome. Requirements for dispersal following probing may be more stringent for these multipartite viruses relative to monopartite Potyviridae, and this may favor more extreme reductions in plant quality to encourage vector restlessness and probing behavior. In fact, there is evidence from one Potyviridae system (*Turnip mosaic virus*, TuMV) that vectors are still capable of transmitting even after successfully colonizing the infected host plant (Casteel et al., 2014). This is unusual for an NC-NPer virus because virions are acquired during brief probes in nonvascular tissue and are generally lost from the stylet if the vector proceeds to colonize the plant and initiate phloem ingestion (Fig. 1; Table 1). Thus, while transmission mechanisms provide a good metric for exploring potentially adaptive virus effects on host phenotypes, our synthesis reveals interesting patterns *within* transmission mechanisms that warrant further exploration.

2.3 Mechanisms Underlying Host-Mediated Effects

If transmission-conducive effects on host phenotype are the product of virus adaptations, then the targets of manipulation should frequently include plant cues known to mediate interactions with vectors. However, unlike clear cases of parasite manipulation involving protozoan or metazoan parasites of animals, it may be difficult in individual plant virus pathosystems to distinguish adaptive indirect (host-mediated) effects from by-products of pathology because plant viruses can induce subtle changes in suites of cues that are already produced by healthy hosts. A fraction of the studies in our analysis profiled plant cues and/or transcriptional responses of hosts to viruses, and an even smaller subset used functional genomics approaches to identify virus proteins involved in the induction of specific host phenotypes. Here, we review these

studies to identify points of convergence in the mechanisms underlying virus effects on host phenotypes and track overall progress toward the goal of demonstrating that such effects are the product of virus adaptations rather than generalized host responses to pathogen infection.

2.3.1 Virus Effects on Long-Range Cues

Many sucking insects that transmit plant viruses (i.e., aphids, whiteflies, thrips, planthoppers, leafhoppers), as well as chewing vectors (i.e., beetles), use visual and olfactory cues to orient toward potential host plants. Aphids are the most well-represented vectors in the studies included in our synthesis (55.7% of virus–host–vector combinations), and both winged and wingless aphid morphs make use of volatile cues while walking and during initial host plant assessments (Eigenbrode et al., 2002; Mauck et al., 2010; Webster, 2012). Thus, we expect that these cues will frequently be targets of manipulation by plant viruses of all transmission classes (Figs. 1 and 2). Out of nine studies in our analysis that profiled volatile metabolites of infected and healthy plants and odor-based vector preferences, seven found that vectors preferred the odor source that emitted a greater quantity of volatiles per unit of leaf tissue—usually the infected host (Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004b; Lu et al., 2016; Mauck et al., 2010, 2014a; Rajabaskar et al., 2014; Werner et al., 2009). Volatile blends of infected hosts were characterized by enhanced emissions of volatiles produced constitutively as a result of normal physiological activity, rather than induction of novel compounds or major changes in blend component ratios. In one case, virus infection suppressed volatile emissions (BPMV infecting soybean), which corresponded with reduced attraction of vectors (Peñaflor et al., 2016). While not adaptive for the virus, this result is still consistent with the pattern of higher emitting plants being more attractive. In two cases out of the nine total studies, virus infection caused variable changes in the quantities of each volatile emitted rather than overall enhanced emissions of all compounds, with divergent outcomes for vector attraction. Wu et al. (2014) reported that infection of peas by either of two different viruses in the Luteoviridae (*Bean leafroll virus* and *Pea enation mosaic virus*) increased the ratio of green leaf volatiles (*trans*-3 hexen-1-ol, *cis*-3-hexen-1-ol, 1-hexanol, and *cis*-3-hexenyl acetate) to monoterpenes (β-pinene and β-ocimene). This change was associated with increased attractiveness of infected plants to the vector (*Acyrthosiphon pisum*) and is consistent with the possibility that the viruses manipulate the lipoxygenase pathway, in which multiple green leaf volatiles are synthesized from hydroperoxy intermediates derived from

linolenic acid (Dudareva et al., 2013). In Mauck et al. (2014a), a squash-adapted isolate of CMV infecting a novel host (pepper) induced changes in the percent composition of seven compounds within the volatile blend and had no overall effect on total volatile emissions. Aphids were not more attracted to this volatile blend but were attracted to the overall enhanced volatile blend emitted by squash (the primary host) infected with this same CMV genotype. Several studies also report virus effects on visual cues (spectral reflectance or light polarization) that may enhance vector attraction to infected hosts (Ajayi and Dewar, 1983; Maxwell et al., 2017), and synergism between enhanced volatile cues and altered visual cues (Fereres et al., 2016).

Although not numerous, these studies suggest that induction of a "supernormal stimulus" (i.e., exaggeration of existing cues) might be a common mechanism by which viruses enhance attractiveness of hosts to vectors (Dawkins and Krebs, 1979). This makes sense given that viruses are often transmitted by more than one vector species, and certainly by different genotypes within a species, making manipulation of the "host present" signal a better strategy than changes in blend composition that might compromise attractiveness to a subset of vectors. The range of potential manipulations could also be limited by molecular constraints on multifunctional proteins (discussed in Section 3). Unfortunately, there is little information about how the viruses studied thus far might induce overall volatile increases. We can speculate that manipulation of green leaf volatiles might arise as a derivation of virus effects on plastid membranes that create protected sites of replication. Membranes contain the precursors for the lipoxygenase pathway (linolenic and linoleic acids) and virus proteins are already adapted for interacting with these molecules. Manipulation of terpenoid compounds might involve virus effects on stomatal apertures, which are known release sites for sesquiterpenes (Seidl-Adams et al., 2015), and possibly other volatiles. For example, the CMV 2b protein, a virus suppressor of RNA silencing, has been shown to interfere with abscisic acid signaling to increase stomatal permeability in *Arabidopsis thaliana* (Westwood et al., 2013b), and it also induces an attractive odor phenotype in this same host (Wu et al., 2017). Uncovering the mechanisms by which viruses alter host volatile emissions will require a functional genomics approach that explores the role of individual proteins alone, and in combination, across different host environments.

2.3.2 Virus Effects on Contact and Palatability Cues

Following contact with a potential host, insect vectors detect leaf surface cues and ingest small quantities of plant material to rapidly discriminate

between suitable and unsuitable host plants (Margaritopoulos et al., 2005; Powell et al., 2006). The outcome of these assessments is initiation of dispersal behavior (from unsuitable hosts) or initiation of subsequent feeding activities (on suitable hosts). A variety of plant characteristics and cues are thought to mediate host plant acceptance, although a comprehensive understanding of host assessment is lacking for most piercing–sucking vector insects (Powell et al., 2006). Leaf toughness, thickness, trichome density, and phloem accessibility will influence the number and duration of probing events. Specialists may respond to just a few plant cues (e.g., a particular class of secondary metabolites) that enable identification of a limited range of suitable hosts (Gabrys and Tjallingii, 2002), while generalists use a variety of hosts that presumably vary widely in secondary chemistry. Nutritional cues, such as free amino acids and sugars, are expected to play a larger role in host discrimination by generalists (Douglas, 2003; Tosh et al., 2003). These cues may interact with plant defense status. Stylets distort the cell walls during penetration, which leads to induction of rapid changes in ion permeability of the plasma membrane, ion exchange (Ca^{2+} and H^+ in, K^+ and Cl^- out), and production of reactive oxygen species as a first line of defense (Mai et al., 2013; Powell et al., 2006). Vectors, and especially those that sample intracellular contents of parenchyma, might be sensitive to these rapidly induced defenses and could be using them as additional measures of host quality.

Experiments on vector settling/feeding preferences are numerous in our quantitative synthesis, but only a handful of these studies quantified plant cues. Most do not separate the relative influence of nutritional vs defense-related cues because it is logistically difficult to do so, and pathway mutants are not always available for susceptible hosts. In general, vector preferences for infected plants are correlated with increased quantities of free amino acids in leaf tissue (Casteel et al., 2014; Cui et al., 2016; Fiebig et al., 2004; McMenemy et al., 2012), but strong relationships are not always apparent. Costa et al. (1991) reported that whiteflies preferred several species of host plants infected with Geminiviridae or Closteroviridae, but found no relationship between whitefly oviposition preferences and total quantities of free amino acids in parenchymal tissues. Blua et al. (1994) showed that squash plants infected with the NC-NPer viruses, *Zucchini yellow mosaic virus* (ZYMV, Potyviridae), had higher levels of free amino acids in leaf tissue at all stages of disease progression, but aphids had difficulty feeding on this tissue only when plants had been infected for 4 weeks. Using microscopy, metabolomics, and techniques for monitoring stylet activities, it was subsequently shown that infected plants in this late stage of disease progression had

higher trichome densities and a reduction in the ratio of sucrose to total free amino acids (Blua and Perring, 1992b; Blua et al., 1994). This nutrient ratio is known to be a key mediator of hemipteran host preferences (Abisgold et al., 1994), and it may be the reason why some studies focusing only on absolute amino acid concentrations did not find a clear relationship with vector preference.

Nutritional changes can also interact with virus-induced alterations of plant defenses. *Tomato yellow leaf curl virus* (TYLCV, Geminiviridae) increases both free amino acids and sugars in phloem sap of infected tomatoes and attenuates induction of defenses against the vector, *B. tabaci* (Su et al., 2015). Whiteflies prefer to settle on TYLCV-infected tomatoes (Legarrea et al., 2015) and acquisition-associated feeding behaviors are enhanced by TYLCV infection (Liu et al., 2013). Attenuation of defenses also occurs in tobacco infected by the related *Tomato yellow leaf curl china virus* (TYLCCNV, Geminiviridae) (Luan and Yao, 2013; Zhang et al., 2012), which improves whitefly performance, but the same enhancement of amino acids in the phloem sap of TYLCCNV-infected tobacco was not detected (Wang et al., 2012). Studies across both geminivirus pathosystems illustrate the complexity of virus-induced changes in host phenotypes, as well as the difficulties associated with identifying whether changes in suites of metabolites are under the genetic control of the pathogen or represent immune responses on the part of the host plant.

Among the NC-NPer viruses *Cucumber mosaic virus* (CMV, Bromoviridae) is one of the best-studied systems with regard to mechanisms underlying virus effects on host phenotypes because it is tractable in the laboratory and amenable to functional genomics studies. As with the ZYMV pathosystem (Blua et al., 1994), Mauck et al. (2014b) found that the ratio of sugars to amino acids in leaf tissue of squash plants infected with the Fny strain of CMV played a role in mediating the rapid dispersal of aphid vectors from infected hosts following probing (Mauck et al., 2010). Using this same strain of CMV in *A. thaliana*, Westwood et al. (2013a) showed that, in this host, rapid dispersal behavior is instead mediated by a secondary metabolite (the glucosinolate compound 4-methoxy-indol-3-yl-methylglucosinolate), which is a mild aphid feeding deterrent. A reverse genetics approach identified the CMV 2a protein (RNA-dependent-RNA-polymerase) as the virus factor responsible for production of the glucosinolate compound by activating defensive signaling in the host (Westwood et al., 2013a). The 2a protein works in concert with the 2b protein, which enhances attractiveness of *Arabidopsis* plants to aphid vectors via effects on volatile cues (Westwood et al., 2013a; Wu et al., 2017),

to produce a host phenotype that deceives vectors into visiting plants that they will ultimately find unsuitable after acquiring virions (Mauck et al., 2010; Westwood et al., 2013a). However, on tobacco, the Fny strain of CMV neither renders plants more attractive to aphid vectors (Tungadi et al., 2017) nor induces an unpalatable phenotype (Ziebell et al., 2011).

A reverse genetics approach was also employed to characterize virus proteins mediating effects of TuMV (Potyviridae) on host phenotypes and aphid vector behavior (Bak et al., 2017; Casteel et al., 2014, 2015). TuMV is an NC-NPer virus that apparently has relaxed restrictions on vector behaviors favoring transmission relative to other NC-NPer viruses. While CMV transmission is clearly reduced when aphid vectors engage in phloem feeding (Fereres and Collar, 2001; Martin et al., 1997), transmission of TuMV can occur following aphid colonization of the host plant (Casteel et al., 2014). Correspondingly, Casteel et al. (2014) reported that expression of the NIa-Pro virus protease protein in *Nicotiana benthamiana* enhanced palatability of host plants for *Myzus persicae* by increasing free amino acid content in leaf tissue and decreasing deposition of feeding-deterrent callose tissue in response to aphid feeding. In an elegant study, Bak et al. (2017) further showed that long-term aphid vector colonization and performance are due to rapid relocalization of NIa-Pro to the vacuole after perception of local signals associated with aphid feeding. Relocalization was not as strongly induced when the attacking insect was a nonvector, and it was also host specific, occurring in *N. benthamiana* but not *Nicotiana tabacum*. Together with work on CMV, these studies highlight the complexity of virus effects on hosts, and the difficulty in demonstrating that such effects are the product of virus adaptations rather than host responses to pathogen infection. Changes in host phenotype can result from the effects of single or multiple virus proteins and can vary depending on the host plant. These findings suggest that molecular and environmental constraints on virus evolution may limit the emergence of manipulative functions (discussed in Sections 3 and 4). Future mechanistic studies should combine functional genomics approaches with artificial selection experiments to elucidate factors favoring the evolution of host plant manipulation.

2.4 Changes in Vector Behavior Following Virus Acquisition

The best-documented cases of putative host manipulation involve protozoan and metazoan parasites. Some of these parasites directly influence the behavior of intermediate animal hosts in ways that enhance the probability that the

parasite will subsequently be ingested by primary hosts, where it can complete its life cycle and, if possible, reproduce sexually (Moore, 2013; Poulin, 2010; Thomas et al., 2005). Although the mechanisms underlying these effects are still poorly characterized, the prevailing hypothesis is that parasites change the behaviors of animal hosts by *directly* targeting the host's neural, endocrine, neuromodulatory, and immunomodulatory systems during infections (Lafferty and Shaw, 2013). Plant viruses with circulative-persistent transmission mechanisms (C-P-Prop viruses and C-P-NProp viruses) may also be capable of targeting these systems in their insect vectors during interactions with vector tissues following acquisition (Fig. 1; Hogenhout et al., 2008). C-P-NProp viruses encounter and traverse a diversity of membrane barriers in different tissue systems during the journey from the midgut to the salivary glands, while C-P-Prop viruses use vector resources directly by replicating in gut epithelium, muscle tissue, salivary glands, fat bodies, and the nervous system. Consistent with these intimate associations, a growing number of studies provide evidence of *direct* manipulation of insect vector behavior by C-P-Prop and C-P-NProp viruses.

As demonstrated in Section 2, viruses frequently induce changes in host plants that render them more attractive to insect vectors (Tables 2 and 3), which is hypothesized to increase virus spread (Roosien et al., 2013; Sisterson, 2008). However, once vectors acquire virions and become viruliferous, there are no additional benefits (for the virus) if vectors continue visiting infected hosts (Table 1; Fig. 1) (Hogenhout et al., 2008; McElhany et al., 1995). Changes in vector preferences following virion acquisition have been named *conditional vector preferences* because the relative attractiveness of plant cues associated with infected and healthy plants is conditional on the viruliferous status of the vector. There are several published reports of conditional vector preferences for C-P-Prop and C-P-NProp pathosystems. For example, the white-backed planthopper (*Sogatella furcifera*), which transmits the C-P-Prop *Southern rice black-streaked dwarf virus* (SRBSDV, Reoviridae), and the brown planthopper (*Nilaparvata lugens*), vector of the C-P-Prop, *Rice ragged stunt virus* (RRSV, Reoviridae), are both differentially attracted to healthy or infected plants depending on their viruliferous status, the infection status of the host, and the stage of disease progression relative to a same-age healthy host (Lu et al., 2016). Preference changes are also evident in C-P-NProp virus pathosystems. Aphids carrying *Potato leafroll virus* (PLRV, Luteoviridae) preferred volatiles from healthy plants, whereas nonviruliferous aphids preferred odor cues from infected plants (Rajabaskar et al., 2014). The same effect was observed for a whitefly-transmitted C-P-NProp virus

(*Tomato severe rugose virus* [ToSRV, Geminiviridae]), where viruliferous whiteflies preferred the odors of mock-inoculated healthy plants over those of ToSRV-infected plants (Fereres et al., 2016). Theoretical explorations of conditional vector preferences demonstrated a substantial positive influence on virus spread (Roosien et al., 2013; Shaw et al., 2017). Indeed, while a constant preference for infected hosts is expected to increase virus spread only when infected plants are rare, a conditional preference eliminates this trade-off and increases virus spread at all stages of epidemic progression (Roosien et al., 2013; Shaw et al., 2017).

Conditional vector preferences based on plant palatability cues are also evident for several C—P virus pathosystems, with the most well-studied group being aphid-transmitted viruses in the Luteoviridae. Carmo-Sousa et al. (2016) demonstrated that *Aphis gossypii* carrying *Cucurbit aphid-borne yellows virus* (CABYV, Luteoviridae) preferred to settle on healthy cucumber plants over CABYV-infected plants, while no settling preference was observed for nonviruliferous aphids. In the PLRV system described earlier, Rajabaskar et al. (2014) found that viruliferous aphids have a clear settling preference for healthy potato plants, whereas nonviruliferous aphids preferred settling on infected plants. And using the well-studied *Barley yellow dwarf virus* (BYDV)-wheat pathosystem, Ingwell et al. (2012) observed the same preference reversal for viruliferous aphids as seen in the PLRV system. As a further step, this study demonstrated that the reversal occurred even when aphids acquired BYDV virions by feeding on an artificial medium containing virions without contact with the infected plants. This provides additional evidence that the observed shifts in behavior are the result of a direct effect of the plant virus on the aphid vector and not mediated by proteins or chemical compounds associated with infected plants. A whitefly-transmitted virus (TYLCV (Geminiviridae) also induces palatability-based conditional vector preferences following acquisition (Legarrea et al., 2015; Moreno-Delafuente et al., 2013), although these effects appear to be specific to the virus genotype by vector combination under study (Fang et al., 2013). Beyond plant viruses, similar conditional vector preferences have been documented for several psyllid-transmitted phytoplasmas in the *Candidatus liberibacter* group (Mann et al., 2012; Martini et al., 2015; Mas et al., 2014). Evidence of convergence among viruses and phytoplasmas that both reside and replicate within their vectors lends further support to the hypothesis that these effects may be the product of pathogen adaptations.

The studies above propose that transmission-conducive direct effects are under genetic control of the virus because some of the outcomes appear to

be detrimental for the vector (e.g., discouraging visitation to, and feeding on, nutritionally superior virus-infected hosts). While there has been no attempt to dissect the roles of individual virus proteins in induction of direct effects, other mechanistic studies provide some insight into specific behavioral processes that change in response to virus acquisition and retention. The electrical penetration graphing (EPG) technique is a powerful tool for monitoring the probing and feeding behaviors of piercing–sucking vectors during interactions with plant hosts (Tjallingii, 1988). EPG systems translate stylet position in the plant (mesophyll, xylem, or phloem tissues) and associated activities (intercellular stylet progression, intracellular penetration, salivation, or ingestion) into distinct waveforms that can be analyzed quantitatively, making this tool particularly valuable for dissecting transmission-relevant behavioral differences between viruliferous and nonviruliferous vectors. For example, in the SRBSDV system, where Lu et al. (2016) observed a preference of viruliferous white-backed planthoppers for healthy plants, Lei et al. (2016) showed that viruliferous white-backed planthoppers spent more time in salivation and phloem sap ingestion on healthy plants than nonviruliferous insects. These behaviors are expected to increase SRBSDV inoculation. In another C-P-Prop virus pathosystem (*Tomato spotted wilt virus* [TSWV, Tospoviridae]), viruliferous males of *Frankliniella occidentalis* performed a greater number of probes into plant tissue, and specifically more noningestion probes that consist of salivation without extensive cellular damage, which is expected to increase the probability of virus establishment in living cells (Stafford et al., 2011). Among C-P-NProp virus pathosystems, Moreno-Delafuente et al. (2013) showed that *B. tabaci* carrying TYLCV had a larger number of phloem contacts and longer salivation phases in phloem sieve elements than nonviruliferous whiteflies. Viruliferous whiteflies also moved half as quickly as nonviruliferous whiteflies and tended to settle and feed more readily. This arrestment behavior is likely related to the enhanced probing and feeding of viruliferous whiteflies on healthy plants. Minor effects on prephloem activities (mean durations of nonprobing, intercellular pathway, and intracellular punctures) were also observed in the CABYV-cucumber pathosystem discussed earlier (Carmo-Sousa et al., 2016). There is even evidence of one foregut-borne NC-SPer virus *Cucurbit chlorotic yellows virus* (CCYV, Closteroviridae) inducing earlier and longer salivation phases in viruliferous male whiteflies relative to nonviruliferous whiteflies (Lu et al., 2017).

While examples are not numerous, the studies discussed earlier suggest that salivation and feeding behaviors are targets for direct manipulations by C-P plant viruses. Virus proteins from both C-P-Prop viruses and C-P-NProp

viruses interact with a variety of host proteins while residing or replicating in the salivary glands. Mar et al. (2014) demonstrated that SRBSDV coimmunoprecipitates with at least 18 proteins from its insect vector, *S. furcifera*, including proteins in the nervous system and the ubiquitin proteasome system, which regulates numerous cellular processes. A differential proteomics analysis of *F. occidentalis* infected with TSWV showed abundant expression of proteins involved in signaling, stress and defense responses, translation, and lipid metabolism (Ogada et al., 2017). Similarly, Luteovirids and Geminivirids (C-P-NProp viruses) both perturb hundreds of biochemical pathways during retention in their respective vectors (Gray et al., 2014; Hasegawa et al., 2018; Luan et al., 2011). Many of these perturbations involve suppression of vector immune responses that might limit virus invasion of vector tissues, providing evidence that C-P-NProp viruses can evolve to manipulate core pathways in their insect vectors. Therefore, it may not be such an evolutionary leap for C-P viruses to evolve adaptations for manipulating vector-feeding behavior. One potential mechanism is via virus effects on the expression levels of salivary effector proteins that modify the defense status and suitability of plants for vector feeding (Elzinga and Jander, 2013). Mutti et al. (2008) demonstrated that this route is feasible by showing that reduced expression of a putative salivary effector protein in pea aphids resulted in a lowered frequency and duration of phloem feeding on a suitable host, as measured by EPG recordings. There are dozens of other known salivary protein effectors in aphids (Elzinga and Jander, 2013) and hundreds of putative secretory proteins have been identified in whiteflies (Su et al., 2012) and planthoppers (Ji et al., 2013). As these proteins are characterized and more functional genomics tools become available for important herbivorous vectors, it will be interesting to explore how C—P virus retention influences salivary effector delivery and efficacy.

3. MOLECULAR CONSTRAINTS ON THE EVOLUTION OF MANIPULATIVE FUNCTIONS

Section 2 provides evidence that viruses can evolve to manipulate vector behavior indirectly, via effects on host plant cues (Tables 2 and 3) (Sections 2.1–2.3), and directly, via effects on vectors (Section 2.4). The handful of studies identifying putative virus effectors of host phenotypes provide additional evidence that virus proteins can evolve manipulative functions. But these same studies also suggest that there are constraints on the

evolution of these functions. For example, manipulation of host phenotype is species specific (Bak et al., 2017; Mauck et al., 2014a; Westwood et al., 2013a,b; Ziebell et al., 2011) and can even be limited to certain *genotypes* within a species (Rajabaskar et al., 2013a,b). This suggests that plant viruses cannot evolve manipulative functions that induce a transmission-conducive phenotype in all possible hosts. Limitations on the evolution of manipulative functions are likely governed by the same molecular constraints that limit virus evolution and adaptation generally. There are numerous reviews on the mechanisms underlying the generation and fixation of mutations in virus genomes, particularly for RNA viruses, which are thought to have relatively high mutation rates and extreme limitations on genome size (Belshaw et al., 2007; Duffy et al., 2008; Gilbertson et al., 2003). Here, we provide a short overview of several key constraints on virus evolution (genome size, pleiotropy and epistasis, and maintenance of vector transmission) as a framework for understanding barriers to the evolution of secondary functions in virus proteins that may confer manipulative traits.

3.1 Genome Size and Secondary Structure

Plant viruses have some of the smallest genomes of any organism (4–20 kb). Restrictions on plant virus genome size are imposed by several factors. Rigid plant cell walls force most plant viruses to use plasmodesmata as a means of establishing systemic infections (Lucas, 2006). Both DNA and RNA viruses accomplish this by actively increasing the size-exclusion limits of plasmodesmata and exploiting the endogenous RNA trafficking mechanisms of the host using movement proteins (Lucas, 2006). But the size-exclusion limit typically cannot be increased beyond a certain point (large enough for passage of a 10 kDa protein) (Hanley-Bowdoin et al., 2000; Lucas, 2006). This constraint limits the size of the virus genome, which must pass through the modified plasmodesmata with the help of viral proteins (Lucas, 2006). A second factor that limits the size of plant virus genomes is mutation. If the mutation rate is too high, increasingly large genomes will experience progressive decay due to rapid accumulation of lethal mutations. This is one mechanism to explain the finding that RNA plant viruses tend to have smaller genomes than DNA plant viruses (Belshaw et al., 2008). RNA-dependent RNA polymerases encoded by RNA viruses lack 3′ to 5′ exonuclease activity and therefore do not proofread during synthesis of new RNA molecules (Drake et al., 1998). The existence of secondary RNA structure also poses challenges for polymerase fidelity and may lead to

deletions during template slippage (Duffy et al., 2008). The realities of RNA genome structure and replication have led to the hypothesis that RNA plant viruses experience higher overall mutation rates relative to DNA plant viruses (Drake et al., 1998). However, recent studies with plant viruses having single-stranded DNA (ssDNA) genomes suggest that these pathogens experience similar mutation rates to those observed in RNA plant viruses even though they are using host plant DNA polymerases for replication. For example, the monopartite whitefly-transmitted begomovirus, TYLCV, and the bipartite begomovirus, *East African cassava mosaic virus*, both exhibit nucleotide substitution rates within the range of $\sim 1.56 \times 10^{-3}$ to 4.63×10^{-4} substitutions per site, per year, which are in line with estimates of nucleotide substitution rates for RNA viruses (Duffy and Holmes, 2008, 2009).

Regardless of the mechanisms underlying restrictions on genome size in plant viruses, the outcome of such restrictions is the tight packing of coding sequences within the available space for information storage. Many plant viruses with RNA genomes exhibit some level of gene overlap, where there are simultaneously two open reading frames (ORFs) coded by the same set of nucleotides. In an analysis of 701 reference RNA virus genomes, Belshaw et al. (2007) found that 56% of the sequences exhibited some gene overlap. Of this 56%, approximately 29% (116 of 392 reference genomes) belonged to plant viruses. A subsequent analysis of the Potyviridae found that this group (represented by 54 sequences in Belshaw et al., 2007) also has gene overlap (Chung et al., 2008). Across all virus families, viruses with smaller genomes tended to have proportionally more gene overlap than viruses with genomes larger than the median length (Belshaw et al., 2007). It is hypothesized that gene overlap benefits the virus because it allows more information to be encoded in a smaller size molecule (e.g., as terminal overlaps of the 3′ end of one gene with the 5′ end of a second gene). It also accommodates the creation of new genes (as internal overlaps) without a concurrent increase in genome size. Maintenance of a smaller genome via gene overlap may facilitate higher replication rates and lower mutation rates, but is also hypothesized to impose additional constraints on virus evolution (Sanjuán and Elena, 2006; Sanjuán et al., 2004, 2005). Even though mutation rates of plant viruses may be high enough to generate the necessary diversity for the evolution of alternative protein functions (including manipulative functions), mutations that occur in overlapping regions are more likely to be detrimental and may subsequently be rapidly purged from the population. Plant viruses also experience extreme reductions in population sizes (bottlenecks) during the initiation of systemic infections (Gutiérrez et al., 2010; Li and Roossinck, 2004; Monsion et al., 2008)

and during inoculation by vectors (Ali et al., 2006). Bottlenecks introduce an additional level of stochasticity in the selection process by reducing the chances that any given mutation will proliferate within the population (Ali et al., 2006; Power, 2000). The frequency with which plant viruses experience population bottlenecks suggests that even beneficial mutations, including those that confer manipulative functions, may be regularly purged by chance events.

3.2 Pleiotropy and Epistasis

Most plant viruses are capable of infecting multiple host species and certainly multiple host genotypes within a species. Therefore, mutations that do go to fixation during infection of one host (because they are either beneficial or, more rarely, neutral) might be detrimental during infection of a subsequent host if the mutation abolish infectivity (host-selective lethality) or limit within-host fitness. This phenomenon is categorized as a type of antagonistic pleiotropy because the same gene controls virus fitness outcomes in two different hosts. Thus, pleiotropic effects impose constraints on virus evolution by limiting the number of host environments available for infection (potentially selecting for specialist viruses), or by imposing costs on viruses that have evolved a generalist lifestyle (Agudelo-Romero et al., 2008; García-Arenal and Fraile, 2013; Malpica et al., 2006). Plant viruses serve as good models for basic research on host trade-offs because many of them can be mechanically passaged through homogeneous or heterogeneous host environments in a logistically simple, replicated design. Studies of this nature repeatedly show the evolution of species-specific mutations, many of which confer reductions in fitness in novel hosts relative to the primary host (Bedhomme et al., 2012; Elena, 2016; Miyashita et al., 2016). In an alternative approach, the artificial introduction of point mutations into infectious clones of plant viruses demonstrates how mutational effects interact with host identity (Elena and Lalić, 2013; Lalić et al., 2011). This approach was used to explore the effects of 20 single point mutations on the fitness of the Solanaceae-specialist RNA virus, *Tobacco etch potyvirus* (TEV), in its primary host environment (*N. tabacum*), other related Solanaceae, and phylogenetically distant hosts in the Asteraceae and Amaranthaceae (Lalić et al., 2011). Effects of mutations on virus fitness varied depending on the host environment, providing clear evidence of pleiotropic effects. Looking across the axis of host relatedness (*N. tabacum*), the fraction of beneficial mutations was slightly higher in non-Solanaceae hosts. Thus, the negative outcomes of antagonistic pleiotropy depend on the phylogenetic distance between two possible host

species (Lalić et al., 2011). In the context of the evolution of secondary, manipulative functions in virus proteins, the widespread occurrence of antagonistic pleiotropy suggests that any mutations that confer manipulative ability to a virus in one host have the potential to confer maladaptive effects in a second host, with such effects depending on the phylogenetic distance between host species.

Plant viruses with tightly packed genomes with overlapping ORFs encoding multifunctional proteins will also experience significant evolutionary constraints due to nonmultiplicative interactions among mutations, a phenomenon known as epistasis (Lalić and Elena, 2013; Sanjuán et al., 2004; Torres-Barceló et al., 2010). The importance of epistasis as a factor limiting the evolutionary pathways available to viruses is evident from numerous experimental evolution and double-mutant interaction studies, most of which employ RNA viruses (Bedhomme et al., 2015). In a follow-up study to the one described earlier, Lalić and Elena (2013) explored epistasis effects using 10 novel TEV genotypes each having a different combination of 2 mutations whose effects in single occurrence were known (Lalić et al., 2011). They found that the magnitude (fitness value) and sign (positive or negative) of epistatic interactions among mutations varied depending on the host background. In hosts of the same family as the origin host (Solanaceae), TEV mutants experienced fewer independent fitness effects of epistasis relative to TEV infections in distantly related hosts (Asteraceae or Amaranthaceae) (Lalić and Elena, 2013). Epistatic effects included both within-host fitness reductions and host-dependent lethality, and mutant pairs in different proteins led to similar epistatic effects in several cases. These examples suggest that epistatic interactions among virus genes are expected to further limit pathways available for the evolution of manipulative functions, particularly for multihost viruses that infect phylogenetically divergent host species.

3.3 Maintenance of Vector Transmission

Most studies exploring constraints on plant virus evolution do not include insect vectors. This is primarily due to logistical constraints (mechanical inoculations are more consistent), not lack of interest in the interactive effects of selection pressures imposed by host plants and vectors. Nonetheless, we can hypothesize that the need to maintain vector transmissibility will further constrain plant virus evolution. Consistent with the multifunctional nature of most virus proteins, there is evidence that mutations in protein

domains mediating virion interactions with vectors can have effects on within-host replication and systemic spread. In the well-studied *Barley yellow dwarf virus* pathogen (BYDV, Luteoviridae), a nonstructural protein encoded by ORF 4 is required for both aphid transmission and systemic movement in plants. Mutation of the read-through domain of this protein abolished the capacity for virions to invade aphid salivary glands and also reduced the accumulation of virus in infected plants (Chay et al., 1996). In a similar scenario, a single amino acid change in the coat protein of CMV reduced aphid transmissibility and induced necrosis in tobacco plants (Ng et al., 2005). The consequences of this mutation will have a twofold effect on virus fitness: the probability of transmission will be reduced *directly* as a result of inefficiencies in virion binding to aphid mouthparts, and *indirectly* as a result of lethal necrotic symptoms that limit the duration of time that the host can serve as a source of inoculum. In both examples, effects are pleiotropic because each respective mutation influences multiple seemingly unrelated traits (host infection and vector transmissibility). But unlike the examples earlier (Bedhomme et al., 2012; Lalić et al., 2011), the signs of pleiotropic effects do not change when the consequences of the mutations are explored in host and vector environments. The cumulative negative effects of mutations that simultaneously disrupt both host–virus and vector–virus interactions will impose additional limits on pathways available for evolution of novel protein functions, including functions that result in the induction of specific host phenotypes.

Host phenotype and vector transmissibility can also be modified by the presence of subviral RNAs—molecular parasites of viruses that reproduce and proliferate using virus-encoded proteins (Simon et al., 2004). Plant viruses are associated with a particular type of subviral RNA (satellites), which are unique in that their sequences are mostly or entirely unrelated to those of the helper virus (Simon et al., 2004). For example, various satellite RNAs of CMV (Bromoviridae) interact with the primary CMV genotype to modify virus accumulation and symptom expression in the host. Satellite RNAs that induce a necrotic phenotype in the host plant will only persist at high vector densities (when there are more opportunities for transmission) because aphid vectors are less likely to perform transmission-conducive behaviors on plants infected with CMV + a necrogenic satellite RNA (Escriu et al., 2000, 2003). Association with a satellite RNA can therefore mask any manipulative effects of the "host" virus genotype on plant phenotype, effectively eliminating selection for, or against, mutations conferring manipulative functions.

4. ENVIRONMENTAL CONSTRAINTS ON THE EVOLUTION OF MANIPULATIVE FUNCTIONS

Molecular constraints on virus evolution operate in the context of environmental factors that have both spatial and temporal dimensions, and that are more or less stochastic depending on anthropogenic influences and abiotic variability. Virus variants that manipulate host phenotype and vector behavior may be selected against if the mutations responsible reduce virus fitness in some environments. Environmental features such as host diversity and vector population size will impose genetic structure on plant virus populations by increasing or decreasing the stringency of molecular constraints, and by augmenting the number of bottlenecks (reductions in genetic diversity) and founder events (chance survival of one particular genotype) (Ali et al., 2006; Betancourt et al., 2008; Geoghegan et al., 2016; Gutiérrez et al., 2012; Roossinck and García-Arenal, 2015). Furthermore, abiotic stressors, nonvector herbivores, and competing pathogens will impact host (and by extension, virus) survival. Thus, transmission-conducive effects of plant viruses will be favored only if they do not increase plant vulnerability to abiotic and biotic threats because plants that succumb to these stressors will no longer serve as sources of inoculum. Here, we review key environmental factors shaping virus evolution in a community context in order to generate predictions about how these factors might interact with molecular constraints to favor, or hinder, the evolution of secondary manipulative functions.

4.1 Host Community Composition

Fitness trade-offs among hosts have been repeatedly documented using laboratory experiments that explore virus evolution during serial transmission events, or the distribution of mutational fitness effects across host environments following artificial introductions of point mutations (discussed in Section 3). These studies demonstrate that adaptation to a primary host can result in reduced fitness in novel hosts due to antagonistic pleiotropy, although there are also documented cases of no apparent costs to virus fitness in novel hosts (Bedhomme et al., 2012). When trade-offs are evident, reductions in virus fitness tend to be more severe with increased phylogenetic distance between primary and novel hosts (Lalić and Elena, 2013; Lalić et al., 2011). Physiological aspects of host life history that determine reservoir potential (life span and investment in immune defenses) will also determine

whether a host is "permissive" or "restrictive" for virus infection and replication (Cronin et al., 2010, 2014; Hily et al., 2014). Based on this experimental evidence, it is logical to expect that the diversity of hosts in the landscape functions as one major axis along which the evolution of manipulative functions will be directed and possibly constrained depending on pathogen and host characteristics.

This expectation has not been explored empirically, either through artificial evolution experiments in the laboratory or through characterization of the phenotypic effects of virus isolates from different host communities. But it is still possible to generate predictions regarding the mechanisms by which host composition might influence evolution of manipulative functions. One scenario under which manipulation is expected to enhance parasite fitness is when opportunities for transmission are constrained to a small-time window (Heil, 2016). In regard to plant viruses, this scenario is characteristic of agricultural monocultures that have an abundance of genetically uniform hosts, some of which may only be susceptible for a brief period during development due to age-related resistance (Panter and Jones, 2002; Sigvald, 1985). Use of chemical insecticides that target vectors, or targeted removal of infected hosts, could impose additional limitations on opportunities for virus acquisition. Additionally, annual crops with rapid life cycles and poor immune defenses are considered "permissive" host environments for virus replication (Cronin et al., 2010, 2014; Hily et al., 2014), which will lead to higher virus replication rates and more opportunities for mutations that could confer manipulative functions. Annual crops are also dead-end hosts for the virus due to harvesting or natural senescence. Based on these features, it is expected that annual monocultures will favor the evolution of manipulative functions that increase the probability of vectors acquiring, retaining, and transporting viruses from crops to alternative reservoirs before crop destruction.

Within these uniform host environments, we might expect to see more frequent evolution of manipulative functions for viruses with circulative-persistent transmission mechanisms (Table 1; Figs. 1 and 2). This is because most circulative-persistent viruses are exclusively transmitted by a limited suite of colonizing vectors that must engage in long-term feeding to acquire and retain the pathogen. The window of opportunity for circulative-persistent virus acquisition and inoculation is therefore constrained not only by the availability of susceptible hosts but also by the presence of colonizing vectors (Fereres and Moreno, 2009; Hogenhout et al., 2008). Selection will tend to favor virus genotypes that enhance the number of contacts with these vectors and encourage feeding behaviors conducive to transmission (Fig. 2).

In contrast, noncirculative viruses, particularly NC-NPer viruses, are often transmitted by a large number of species, many of which may be transient visitors but nonetheless efficient vectors (Perring et al., 1999; Pirone and Perry, 2002; Radcliffe and Ragsdale, 2002). For example, there are more than 50 known aphid vectors of the NC-NPer viruses, *Potato virus Y* (PVY), but only a few of these species actually colonize the solanaceous crop hosts that are susceptible to PVY (Radcliffe and Ragsdale, 2002). Spread of PVY in field environments is not driven by the abundance of colonizing vectors, but rather by the number of those that are transient, noncolonizing visitors engaging in rapid probing and dispersal behaviors conducive to NC-NPer virus transmission (Mondal et al., 2016; Radcliffe and Ragsdale, 2002) (Fig. 2). Spread of two other NC-NPer viruses, *Watermelon mosaic virus* and *Papaya ringspot virus*, in cucurbit crops was also linked to abundance of several noncolonizers, but not of colonizing aphids (Angelella et al., 2015). These less-specific vector–host relationships create more opportunities for successful transmission events and might "dilute" the features of annual monoculture environments that favor the evolution of manipulative functions.

Mixed host plant communities are expected to be less conducive to the evolution of manipulative functions relative to communities with low host diversity because the potential for antagonistic pleiotropy is greater (Bedhomme et al., 2015; Elena, 2016). Multihost viruses that induce transmission-conducive effects in one host might induce transmission-limiting effects in a second host, which will favor the spread of nonmanipulative genotypes that have neutral effects on host–vector interactions. Furthermore, direct effects of viruses on vector behavioral responses are unlikely to be robust across host plants with vastly different chemistries (Carrasco et al., 2015). Even among hosts in the same species, phenotypic effects may vary with genotype, competitive status, or other heterogeneous factors in diverse, unmanaged plant communities. These constraints suggest that the evolution of manipulative functions in mixed-host environments should be limited to specialist viruses that have narrow host ranges and tightly coevolved vector relationships. If susceptible hosts are few or patchily distributed, opportunities for transmission will be rare—a condition that is hypothesized to favor the evolution of host and vector manipulation (Heil, 2016). Specialist viruses may evolve manipulative functions in such environments if the cues responsible for mediating host–vector contacts are very specific and consistent (e.g., secondary metabolites associated with a specific plant family) and if the host range of the vectors is also similarly specialized. But the majority of characterized plant

viruses naturally infect hosts across two or more plant families. Thus, heterogeneous host environments are expected to constrain, rather than favor, the evolution of manipulative functions.

4.2 Prevalence of Competent Vectors

If competent vectors are prevalent, these vectors are expected to contact and colonize host plants frequently regardless of plant infection status or phenotype. Agricultural monocultures are particularly likely to have large vector populations. Vectors with high reproductive rates, such as aphids and whiteflies, will go through successive generations on one crop host and emigrate in large numbers to other, more appealing crops following declines in initial host quality or harvesting (Carrière et al., 2017; Mondal et al., 2016; Thomas et al., 1993). Vector amplification and emigration are such a reliable phenomenon in agriculture that continent-wide, suction trap networks are in place in many locations to monitor vector emigrants and predict optimal planting dates (Harrington et al., 2004). This can be an effective strategy for mitigating virus impacts because there is ample evidence that rates of pathogen spread are strongly tied to vector abundance (Jeger et al., 2011; Madden et al., 2000; McElhany et al., 1995; Shaw et al., 2017; Sisterson, 2008). If planting dates align with high vector numbers, there are more vectors per host niche and almost every plant is contacted or colonized, creating opportunities for transmission. This will favor the spread of both manipulative and nonmanipulative virus genotypes and reduce the relative fitness benefits of host and vector manipulation. If planting dates do not align with high vector numbers, there are fewer vectors per host niche, and manipulated hosts should serve as sources of inoculum more often than nonmanipulated hosts.

Frequency of virus transmission is not only influenced by vector prevalence. Potential vectors must also be *competent* (capable of transmitting the virus) and *efficient* (transmit at high enough rates to contribute significantly to virus spread). Vector competence refers to the ability of an organism to acquire, retain, and inoculate a virus (or other microbial entity). Transmission efficiency refers to the probability of a competent vector transmitting a virus from one host to the next and is determined by characteristics of the virus, host, and vector as well as external factors, such as temperature, that modify relationships among these players (Anhalt and Almeida, 2008; Bosquee et al., 2016; Chatzivassiliou et al., 2002). Vector behavior is one major determinant of transmission efficiency (Fereres, 2016; Fereres and

Collar, 2001; Madden et al., 2000; Martin et al., 1997; Roosien et al., 2013; Shaw et al., 2017; Sisterson, 2008; Wang and Ghabrial, 2002). If the most abundant vectors have poor efficiency because they do not perform behaviors conducive to acquisition or inoculation, then transmission events will be infrequent (Madden et al., 2000; Shaw et al., 2017). Evolution of manipulative functions may be favorable under these conditions if such functions selectively increase transmission-conducive interactions with efficient vectors. Alternatively, if efficient vectors are abundant and most plants experience vector contacts independent of infection status, selection for manipulative functions will be weaker because there are more opportunities for transmission of all virus genotypes.

4.3 Off-Target Effects of Virus-Induced Host Phenotypes

As discussed in Section 2, plant viruses can alter plant nutritional quality, defense responses, and plant-derived sensory cues, either as part of an adaptive strategy of indirect manipulation of vector behavior or as a by-product of pathology. These often-significant impacts on host plant phenotypes are expected to strongly influence broader community interactions, with implications for the longevity, survival, and fitness of the infected host and the virus that depends on its resources (Alexander et al., 2013; Mauck et al., 2015). Nonvector arthropods are sensitive to many of the same cues and plant quality changes that mediate host plant interactions with insect vectors. And virus effects on conserved phytohormone signaling pathways will alter how hosts respond to both biotic and abiotic stressors (Aguilar et al., 2017). Therefore, evolution of manipulative functions in plant viruses will potentially be limited to effects on plant phenotype that maintain or enhance transmission-conducive vector behaviors without significantly increasing host vulnerability to nonvector organisms (off-target effects).

Limited explorations of the off-target effects of putative manipulations have been reported for several of the pathosystems covered in our quantitative synthesis (Tables 2 and 3). In the well-studied *Tomato spotted wilt virus*-pepper-thrips pathosystem (Tospoviridae), Belliure et al. (2010) reported that virus infection increased host susceptibility to a nonvector herbivore (*Tetranychus urticae*) that feeds in a similar manner as the thrips vector. Kersch-Becker and Thaler (2013) also demonstrated increased susceptibility of tomato plants infected with *Potato virus Y* (Potyviridae) to two chewing herbivores, including a Solanaceae specialist (*Leptinotarsa decemlineata*). More recently, Ángeles-López et al. (2016, 2017) showed that infection of chili

plants by *Pepper golden mosaic virus* (Geminiviridae) induces a phenotype that is expected to increase transmission by its whitefly vector (*B. tabaci*), but which also increases host susceptibility to, and quality for, a nonvector whitefly (*Trialeurodes vaporariorum*). The authors additionally report that *T. vaporariorum* feeding significantly reduces virus titers in infected host plants and attenuates aspects of the (putatively) transmission-conducive phenotype induced by the virus (Ángeles-López et al., 2017). Coinfections with other pathogens may similarly disrupt transmission-conducive phenotypes (Peñaflor et al., 2016), although one report suggests that when coinfecting viruses are transmitted by the same vector, selection may favor genotypes of the "nonmanipulating" virus species that have minimal effects on the transmission-enhancing functions of the manipulative virus species (Salvaudon et al., 2013). Together, this small set of studies reveals at least three scenarios under which selection will disfavor manipulative virus genotypes in a community context: when virus-induced susceptibility to nonvectors (i) reduces host tissue volume or survival, (ii) abolishes the transmission-conducive phenotype, or (iii) directly reduces within-host virus fitness.

Manipulative genotypes are more likely to emerge and spread if the virus-induced host phenotype confers additional benefits to the host plant or at least has neutral effects on host resistance to other stressors. Recent studies highlight a few pathosystems where this appears to be the case. CMV (Bromoviridae) induces a transmission-enhancing phenotype in several host plants (*Cucurbita pepo*, *Cucumis sativus*, *A. thaliana*, *N. tabacum*) (Carmo-Sousa et al., 2014; Mauck et al., 2010, 2014a,b; Shi et al., 2016; Westwood et al., 2013a; Wu et al., 2017). In at least some of these hosts, the transmission-enhancing phenotype also confers drought tolerance (Westwood et al., 2013a; Xu et al., 2008) and reduces susceptibility to generalist and specialist nonvector herbivores (Mauck et al., 2015; Saad et al., 2017), both of which will increase the length of time infected hosts can persist as sources of inoculum. The putative host manipulation and the beneficial effects of virus infection on host resistance to drought and nonvector herbivores have been partially attributed to the functions of the 2b silencing suppressor protein (Westwood et al., 2013a,b). Several other reports demonstrate selectivity in the effects of virus-induced host phenotypes, with benefits only being realized for the vector, and not for common, co-occurring nonvectors (Cassone et al., 2014; Peñaflor et al., 2016; Sadeghi et al., 2016). But for the majority of pathosystems summarized in Tables 2 and 3, we have no knowledge of how putative virus manipulations of host phenotypes are likely to influence resistance to biotic and abiotic stressors.

5. LIMITATIONS OF EXISTING STUDIES IN THE CONTEXT OF MOLECULAR AND ENVIRONMENTAL CONSTRAINTS

Our quantitative synthesis of the literature (Tables 2 and 3) supports the predictions about convergent effects of plant viruses on host phenotypes and vector behavior outlined in Fig. 2, and first proposed in Mauck et al. (2012). Regardless of transmission mechanism, vectors are generally attracted to virus-infected plants over noninfected plants (Tables 2 and 3). Viruses that require sustained feeding for acquisition (C-P-Prop, C-P-NProp, and most NC-SPer viruses) generally enhance palatability and quality, whereas viruses that require rapid dispersal following acquisition (NC-NPer viruses) have more mixed effects, with a greater proportion of experiments demonstrating no change or a reduction in host palatability and quality (Tables 2 and 3). Convergence in the effects of viruses from multiple lineages that share a common transmission mechanism suggests that such effects are adaptive, or at least that viruses are subject to selection *against* the evolution of traits that have transmission-limiting effects (Poulin, 2010; Thomas et al., 2005). The few studies that have explored putative virus effectors of host phenotypes indicate that just one or two virus proteins can influence whole suites of host cues in ways that influence vector behavior. Despite this evidence, the broader literature on the molecular and environmental factors shaping virus evolution (Sections 3 and 4) suggests that there are only a few scenarios that favor the emergence and maintenance of manipulative traits, and many scenarios that do not. In light of these constraints, we performed a second quantitative synthesis of the same set of literature (Fig. 3) to determine how many studies considered the molecular and environmental factors discussed in Sections 3 and 4, and to identify target areas for future research to resolve multiple unknowns regarding virus manipulation of hosts and vectors in environments outside of the laboratory.

5.1 Pathogen Provenance

Nearly every study in our quantitative synthesis proposes that transmission-conducive effects are evidence of adaptive manipulation on the part of the virus. Presumably, this means that manipulative functions evolved in, and were selected for, an environment where they conferred fitness benefits to the pathogen. But more than 60% of the studies used virus strains that have been in laboratory culture for many years, or even decades (Fig. 3A). A significant proportion of studies with laboratory strains report culture

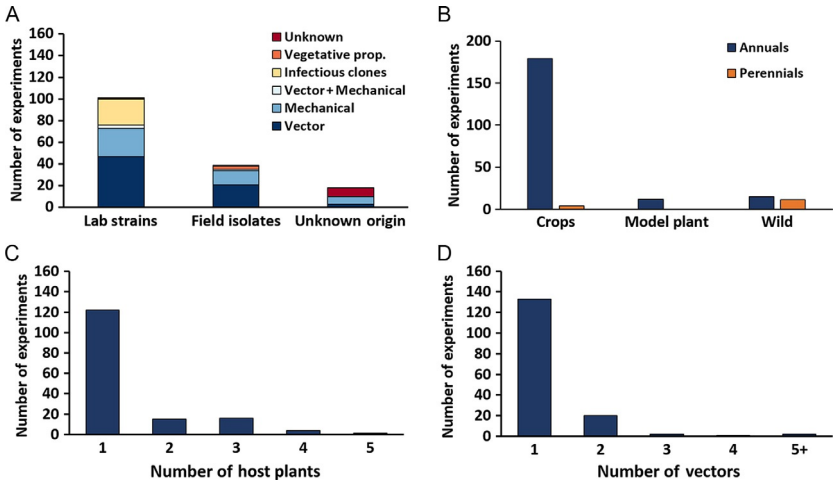

Fig. 3 Pathosystem features of the studies included in the quantitative synthesis (Tables 2 and 3). (A) Virus provenance; (B) host physiological phenotype and domestication status; (C) number of plant hosts examined for a single focal virus strain or isolate; (D) number of vector species or biotypes studied for each virus-host combination examined. For (A), (C), and (D), we parsed each article (122) into individual experiments, each addressing a single virus strain or isolate (158 experiments total). For (B), individual experiments represent a single virus (strain or isolate) studied on a single host (species or cultivar) (221 experiments total).

maintenance via serial transfer using vectors (44.9% of studies with lab strains) or mechanical transmission (29.1% of studies with lab strains). There is little to no methodological detail provided regarding these culture techniques (e.g., host age at inoculation, age of the inoculum source, number of vectors used to transmit, etc.). But we know from the experimental evolution studies discussed in Section 3 that repeated passage in a single host can drive virus evolution, including the fixation of mutations that influence host phenotype and constrain pathways for evolving novel functions (Bedhomme et al., 2012, 2013; Elena, 2016; Miyashita et al., 2016). Drastic changes in host phenotype can be the result of a single amino acid change in just one virus protein (Lewsey et al., 2009). Mutations that occur in overlapping ORFs may have proportionally larger effects on multiple aspects of the virus–host interaction (Belshaw et al., 2007), while epistatic interactions among virus proteins will further shape the impacts of any one mutation (Lalić and Elena, 2013; Sanjuán et al., 2004; Sanjuán and Elena, 2006). In the absence of selection pressures imposed by environmental factors, bottlenecks during vector transmission and systemic invasion of the host plant are expected to lead to genetic

drift and, potentially, to the random loss of manipulative functions that might have evolved in the original environment from which a virus was isolated (Ali et al., 2006; Li and Roossinck, 2004). By the same mechanism, maladaptive mutations might emerge and become fixed because among-host virus fitness is under the watchful eye of the researcher, and not dependent on vector behavior.

Under these experimental conditions, it is difficult to assert that transmission-conducive effects are the result of selection for manipulative functions. Serial passage in a single culture host is far more likely to select for mutations that enhance exploitation of that host environment regardless of effects on host phenotype (e.g., higher replication rates, higher virus titer, more rapid systemic colonization). Indeed, higher virus titers have been positively associated with transmission-enhancing virus effects in several studies with *Cucumber mosaic virus* (Bromoviridae) (Mauck et al., 2014a; Shi et al., 2016), *Barley yellow dwarf virus* (Luteoviridae) (Fereres et al., 1989; Jiménez-Martínez et al., 2004a,b; Medina-Ortega et al., 2009), and *Tomato yellow leaf curl virus* (Geminiviridae) (Legarrea et al., 2015). But the majority of studies in our synthesis did not consider virus titer or other metrics of infection (virulence, symptom expression), and very few have taken a comparative approach among virus genotypes that differ in levels of host exploitation and degree of phenotypic manipulation. To fully explore alternative explanations for putative manipulations, future studies should compare closely related viruses that induce transmission-enhancing and transmission-limiting phenotypes and collect data on changes in host phenotype that are apparently unrelated to vector manipulation, but important for virus fitness.

5.2 Host Physiological Phenotype

The most permissive hosts for plant viruses are short-lived species that have poor immune defenses, high nutrient levels (especially phosphorus), low leaf mass per area, and fast metabolisms—all of which are features that make them susceptible to both pathogen exploitation and vector feeding (Cronin et al., 2010; Elser et al., 2010; Hily et al., 2014; Lind et al., 2013; Reich, 2014). Domestication has exacerbated these features for many annual crops by breeding out functional pathways for production of secondary metabolites and other defenses as a means of increasing productivity, edibility or palatability for human consumption (Chen et al., 2015a; Gaillard et al., 2018). Almost all studies exploring putative instances of plant virus manipulation used

domesticated annual plants or laboratory models (*N. benthamiana* and *A. thaliana*) (Fig. 3B). This is likely because fast-growing annual crops and model hosts are logistically feasible for laboratory work, and funding is available to support research on mitigating virus impacts on annual crops. As discussed in Section 4, annual monocultures are expected to favor the evolution of manipulative functions in plant viruses, so use of annuals as model hosts is logical given this expectation. However, it also means that we cannot rule out the possibility that the host physiological phenotype is playing a role in determining some of the patterns observed in our quantitative synthesis.

With the current data set, there are not enough studies using wild or perennial hosts to test predictions regarding variation in virus effects along a continuum of host physiological phenotypes (e.g., fast vs slow lifestyles). But within studies using annual hosts, the few that have explored changes in virus effects over the course of disease progression provide evidence that host physiological phenotype does play a role. Plants have age-related resistance to virus infection (Panter and Jones, 2002). Young plants are typically more susceptible to viruses and other pathogens, and this susceptibility decreases as the plant progresses through different phenological stages. We identified 12 studies that explicitly examined virus effects at different stages of disease progression and host phenology (Blua and Perring, 1992a,b; Blua et al., 1994; Higashi and Bressan, 2013; Legarrea et al., 2015; Lu et al., 2016; Mann et al., 2008; Rajabaskar et al., 2013b; Shi et al., 2016; Wang et al., 2012; Werner et al., 2009; Williams, 1995). Several studies with NC-NPer viruses showed that palatability and quality for vectors were enhanced early in disease progression but reduced at later stages (Blua and Perring, 1992a,b; Blua et al., 1994; Shi et al., 2016). Surprisingly, this same pattern was observed in studies with C-P-NProp viruses. Werner et al. (2009) demonstrated that volatiles from potato plants infected with PLRV (Luteoviridae) were more attractive to vectors at 4 weeks postinoculation, but not at 8 or 10 weeks postinoculation. Rajabaskar et al. (2013a,b) further demonstrated that attraction to PLRV-infected plants at 4 weeks postinoculation depends on infection occurring between 1 and 3 weeks posttransplanting and is not induced when older plants are inoculated. Legarrea et al. (2015) found that whiteflies preferred TYLCV-infected tomato plants at 6 weeks postinfection, but not at 9 weeks postinfection. According to this limited suite of reports, virus effects on host attractiveness or palatability may be transient. Early stages of infection in younger plants are associated with greater palatability or attractiveness, while later stages are associated with reduced palatability or attractiveness, regardless of the virus transmission mechanism. This pattern clearly requires further

study, but it does suggest that host physiological phenotype may determine *whether* a virus can manipulate a host, as well as how long transmission-enhancing phenotypic changes are expressed by the host. It will be interesting to explore how other determinants of plant traits and host physiological phenotypes, such as nutrient supplies and ratios, influence virus-induced changes in host plant cues and quality (Borer et al., 2010; Cebrian et al., 2009). This approach would be particularly useful if integrated with comparisons of wild and cultivated congeners, or perennial and annual hosts, which also differ widely in plant traits that influence interactions with vectors.

5.3 Pathosystem Complexity

As discussed in Section 3, antagonistic pleiotropy limits the extent to which plant viruses can equally exploit multiple host plants (Agudelo-Romero et al., 2008; García-Arenal and Fraile, 2013; Malpica et al., 2006). The widespread occurrence of antagonistic pleiotropy suggests that any mutations that confer manipulative ability to a virus in one host have the potential to confer maladaptive effects in a second host, with such effects potentially depending on the phylogenetic distance between host species (Lalić et al., 2011). Epistatic interactions among virus proteins may also vary from one host environment to the next, resulting in different outcomes for virus effects on host phenotype and vector behavior (Lalić and Elena, 2013). Many of the pathogens included in our quantitative synthesis can infect multiple hosts, sometimes across several families. Yet only about 23% of the studies employed more than one plant species or genotype (Fig. 3C). Of those that did explore virus infections across multiple hosts, over 57% report that virus-induced changes in host phenotype are species specific. Some studies even report variation in effects among different genotypes within the same host species (Araya and Foster, 1987; Fereres et al., 1990; Hily et al., 2014; Liu et al., 2014; Rajabaskar et al., 2013a,b). In general, when effects diverge depending on host environment, the shift is from a transmission-enhancing phenotype to a phenotype that has neutral effects on transmission probability, but shifts to maladaptive phenotypes have also been reported (Mauck et al., 2014a). It is also possible that some of the potentially maladaptive phenotypes revealed in our quantitative synthesis represent instances of a lack of virus adaptation to a particular host (Tables 2 and 3). But the insufficient information on virus provenance and use of laboratory strains in most studies limit our ability to interpret these maladaptive effects within an evolutionary context. Although few, these studies support our expectation that

heterogeneous host environments are likely to disfavor the evolution of manipulative functions because mutations conferring these functions may reduce transmission from a subset of susceptible hosts (Section 4). Moving forward, it will be particularly important to incorporate landscape heterogeneity and temporal limitations on virus effects (discussed in Section 4) into models that describe the epidemiological outcomes of virus manipulations (Roosien et al., 2013; Shaw et al., 2017).

The number of vectors studied per host combination is also quite limited (Fig. 3D) even though many plant viruses are transmitted by multiple vector species, especially NC-NPer viruses (e.g., CMV and PVY are each transmitted by more than 50 species of aphid), but also a number of the most well-studied C-P-NProp viruses (e.g., B/CYDVs, PLRV, TSWV each have more than five vector species). Only about 15.8% of studies tested the behavioral or performance-related responses of multiple vector species, biotypes, or genotypes. Of these 15.8%, the majority (68%) show at least one instance of vector specificity (different responses of two vectors to the same host phenotype). In some cases, this tracks with vector efficiency. For instance, Chesnais et al. (2017) demonstrated that infection of *Camelina sativa* by *Turnip yellows virus* (a C-P-NProp virus in the Luteoviridae) enhanced palatability for *M. persicae*, an efficient vector, but did not enhance palatability for *Brevicoryne brassicae*, an inefficient vector. In other cases, there is no apparent relationship with vector efficiency. *M. persicae* and *B. brassicae* are both efficient vectors of the NC-NPer viruses, TuMV (Potyviridae), but TuMV infection in the same cultivar of turnips enhanced host quality for *M. persicae* and reduced host quality for *B. brassicae* (Hodgson, 1981). As discussed in Section 4, the robustness and consistency of vector responses to virus-induced changes in host phenotypes are expected to determine the frequency with which viruses having manipulative functions will be transmitted to new hosts. Viruses vectored by only one or two species may be more likely to evolve manipulative functions targeting multiple aspects of the transmission process (orientation, settling/feeding, and performance). But viruses vectored by many species (e.g., many NC-NPer viruses) may be less likely to evolve manipulative functions or may only evolve functions that target broad-spectrum cues (e.g., color or host odor enhancements that increase attraction of many vector species). Our quantitative synthesis shows that effects of NC-NPer viruses are more variable than those of C-P-Prop viruses or C-P-NProp viruses, which supports these expectations (Tables 2 and 3), but additional studies are needed to identify convergence or divergence in responses of multiple vectors to putative virus manipulations of host phenotypes.

6. CONCLUSIONS

Parasites shape the ecology of all life on earth and pose major threats to food security and human health. Understanding the selection pressures driving parasite evolution and emergence—including the evolution of manipulative traits—is essential from both basic and applied perspectives. There are now hundreds of reports of insect-vectored plant viruses inducing transmission-enhancing phenotypes in their host plants, and theoretical work elucidates the importance of these putative manipulations for disease spread in agricultural systems. Our quantitative synthesis of these reports, along with the limitations of the present body of work in the context of constraints on virus evolution, highlights numerous areas for future research to understand the relevance of manipulative plant viruses in real-world scenarios. Reverse genetics approaches coupled with phenotype profiling (metabolomics, transcriptomics) are beginning to reveal how virus proteins interact with host components to elicit changes in chemical cues or host quality for vectors. Artificial selection experiments with model pathogens will test the hypothesis that the evolution of secondary functions in virus proteins can be directed when selection is performed based on detectable differences in host phenotypes. Incorporation of multiple hosts, including wild-type and pathway mutants, will reveal trade-offs associated with manipulative functions and host factors involved in phenotype induction. And state-of-the-art phenotypic profiling techniques will provide a wealth of information about host cues and defenses mediating vector-feeding activities (most of which are presently unknown), and how virus infection augments these factors. Results of these studies will enrich our understanding of the evolution of parasite manipulation and facilitate development of new epidemiological models for predicting and mitigating virus threats to agriculture.

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