UC Riverside

UC Riverside Previously Published Works

Title

A synthesis of virus-vector associations reveals important deficiencies in studies on host and vector manipulation by plant viruses

Permalink

https://escholarship.org/uc/item/7fw8154c

Authors

Mauck, Kerry E
Chesnais, Quentin

Publication Date

2020-08-01

DOI

10.1016/j.virusres.2020.197957

Peer reviewed

Review

A synthesis of virus-vector associations reveals important deficiencies in studies on host and vector manipulation by plant viruses

Kerry E. Mauck[a],* kerry.mauck@ucr.edu, Quentin Chesnais[a,b]

[a]Department of Entomology, University of California, Riverside, Riverside, CA 92521, USA

[b]Université de Strasbourg, INRAE, SVQV UMR-A 1131, F-68000 Colmar, France

*Corresponding author.

Abstract

Plant viruses face many challenges in agricultural environments. Although crop fields appear to be abundant resources for these pathogens, it may be difficult for viruses to "escape" from crop environments prior to host senescence or harvesting. One way for viruses to increase the odds of persisting outside of agricultural fields across seasons is by evolving traits that increase transmission opportunities between crops and wild plant communities. There is accumulating evidence that some viruses can achieve this by manipulating crop plant phenotypes in ways that enhance transmission by vectors. Putative manipulations occur through alteration of plant cues (color, size, texture, foliar volatiles, in-leaf metabolites, defenses, and leaf cuticles) that mediate vector orientation, feeding, and dispersal behaviors. Virus effects on host phenotypes are not uniform but appear to exhibit convergence depending on virus traits underlying transmission, particularly the duration of probing and feeding required to acquire and inoculate distinct types of plant viruses. This shared congruence in manipulation strategies and mechanisms across divergent virus lineages suggests that such effects may be adaptive. To discern if this is the case, researchers must consider molecular and environmental constraints on virus evolution, including those imposed by insect vectors from organismal to landscape scales. In this review, we synthesize applied research on vector-borne virus transmission in laboratory and field settings to identify the main factors determining transmission opportunities for plant viruses, and thus, selection pressure to evolve manipulative traits. We then examine these outputs in the context of studies reporting putative instances of plant virus manipulation. Our synthesis reveals important disconnects between virus manipulation studies and actual selection pressures imposed by vectors in real-world contexts.

Introduction

Domestication of wild plants for agriculture fundamentally changed the evolution of plant viruses. Agriculture brings homogeneity in the form of fixed host genotypes planted over large areas, as well as instability, in the form of sudden and synchronized harvesting, crop rotations, and fallow periods. As a result, the pace and trajectory of virus-host coevolution has been forever altered by the presence of agriculture at interfaces with unmanaged habitats (Gibbs et al., 2010, 2008; Nguyen et al., 2013; Smith et al., 2014). Crop fields present huge swaths of potentially dead-end hosts, most of which are susceptible to infection during early phenological stages (Panter and Jones, 2002). These fields are magnets for arthropod vectors because crop hosts present visual and olfactory cues that are highly arresting (Döring, 2014; Powell et al., 2006; Webster, 2012). Plant viruses that rely on arthropod vectors for transmission have had to adapt to survive, and persist across seasons, under circumstances where their vectors prefer transient and unstable host environments.

How plant viruses are able to cope with agricultural landscapes characterized by frequent host turnover is still an open question. One strategy might be to increase the number of transmission-conducive vector contacts, thereby upping the odds of exploiting and "escaping" an infected crop host prior to harvest or senescence. Based on this, it has been proposed that plant viruses infecting annual crops have evolved to manipulate host phenotypes and vector behavior in ways that enhance transmission opportunities (Blua and Perring, 1992; Castle et al., 1998; Eigenbrode et al., 2002; Kennedy, 1951; Mauck et al., 2012, 2019, 2018, 2010). Virus infections in plants, and especially crops, will typically result in drastic phenotypic changes that alter physical and physiological characteristics, such as color, size, texture, volatile profiles, primary and secondary metabolite levels, metabolite partitioning among tissues, defenses, and cuticular waxes (reviewed in Culver and Padmanabhan 2007; Mauck et al. 2019). These phenotypic aspects determine how vectors perceive and interact with the infected host, and thereby, the probability of vectors engaging in transmission-conducive feeding and dispersal behaviors. If viruses can evolve to elicit specific changes in host phenotypes that encourage vectors to visit infected crop hosts and engage in behaviors optimal for virion acquisition, this may improve the odds of establishing infections in perennial vegetation that can support over-wintering. Thus, we might predict that manipulative viruses (i.e., those that alter host plants to increase vector contacts and transmission-conducive feeding behavior) will more effectively persist across seasons and disproportionately contribute to new infections over time, while viruses that are not able to manipulate their hosts and vectors will fail to do so.

Consistent with this prediction, there is now substantial evidence that numerous viruses of economic importance induce changes in host phenotypes and vector behavior in ways that are expected to increase transmission opportunities (reviewed in Mauck et al., 2018). Experiments demonstrating such effects typically combine ethological approaches with chemical analytical and molecular techniques to describe virus effects on plant physiology and quantify the impact of these changes on vector attraction, settling and feeding, and performance (reviewed in Mauck et al., 2019). In some cases, virus proteins underlying these effects have been linked to specific elements of the infected host phenotype that influence vectors (Ziebell et al. 2011; Zhang et al. 2012; Li et al. 2014; Wu et al. 2017; reviewed in Mauck et al. 2019). For example, an elegant series of studies on the species *T-urnip mosaic virus* (genus *Potyvirus*, family *Potyviridae*) revealed that a multifunctional protease, NIa-Pro, modifies plant defenses to increase attraction of aphids to infected hosts, and also relocalizes in the cell during aphid probing to selectively inhibit plant defenses against the vector (Casteel et al. 2014; Bak et al. 2017). In other cases, phenotypic changes are caused by multiple virus proteins. This can occur in both an additive (Patton et al. 2020) and synergistic manner (Westwood et al. 2013). Additionally, there is mounting evidence that infected host phenotypes and vector behaviors change over disease progression, with manipulative effects only

and Bressan 2013; Shi et al. 2016; Legarrea et al. 2015; Mann et al. 2008; Rajabaskar et al. 2013; Williams 1995; Lu et al. 2016; Shrestha et al. 2019).

Despite recent progress on molecular mechanisms of manipulation for single virus-host-vector combinations (reviewed in Mauck et al. 2019; Carr et al. 2020), logistical constraints have limited the number of vector species that can be explored for behavioral responses to any given pathosystem. This may not be problematic when the virus under study is transmitted by a limited suite of vectors in the field. But it may fail to represent real-world scenarios for numerous viruses that are transmitted by many vector species or biotypes having a range of relationships with commonly infected crop hosts. In these cases, extrapolating conclusions about the evolution of manipulative traits is not warranted because the experimental design replicates a selective environment that is not realistic. To explore whether there are systematic methodological issues limiting the study of plant virus manipulation, we provide a review of approaches to studying transmission in laboratory and field environments. We then use this information as a framework for exploring whether virus manipulation studies are congruent with selection pressures imposed by arthropod vectors. To achieve this, we structured both the review and synthesis portions of this paper around well-established transmission mechanism categories (Table 1). Transmission mechanisms are an excellent framework for studying the adaptive significance of virus manipulation because we expect phylogenetically divergent pathogens that share transmission mechanism traits to exhibit convergence in effects on host phenotypes and vector behavior. This has been demonstrated in prior quantitative syntheses of virus manipulation literature (Mauck et al., 2012, 2018). Here, we leverage these syntheses and combine their outputs with compiled information about virus transmission traits to reveal important gaps in our understanding of virus manipulation and identify research priorities for future work.

2 Identifying competent vectors of plant viruses and measuring transmission characteristics

Much of our knowledge about virus transmission by arthropod vectors comes from experiments performed in the laboratory or greenhouse using cultured virus isolates and vector colonies. These experiments provide crucial information about the transmission process and the nature of virus-vector relationships. For example, knowing the breadth of competent vector species, at least among those that are most commonly present, facilitates improved vector monitoring efforts, selection of appropriate insecticides or biological control agents, and identification of resistant plant material (Steinger et al., 2015). Knowledge of virus-vector associations is equally critical for understanding ecological aspects of virus movement across the agroecological interface, including possible alternative host ranges and prevalence in wild communities (Alexander et al., 2014; Wintermantel et al., 2009). Moreover, virus evolution is strongly constrained by the identity and abundance of competent vectors in the environment. Viruses need to maintain genetic components that enable associations with their vectors, and adaptive strategies to accomplish this vary considerably among viruses depending on selection pressures imposed by vector interactions with shared host plants (e.g., vector host range and preferences) (Chay et al., 1996; Ng et al., 2005). It has even been hypothesized that adaptation to transmission by a new vector species is more difficult, evolutionarily, than adaptation to new host genotypes or species (Lefeuvre et al., 2019). This hypothesis is supported by abundant evidence of rapid plant virus adaptation to infecting resistant crop germplasm (Djidjou-Demasse et al., 2017; Gómez et al., 2009; Rousseau et al. 2019) as well as strong congruence between virus coat protein phylogenies and vector phylogenies (Lefeuvre et al., 2019). Thus, despite logistical constraints, laboratory experiments to identify competent vectors are necessary as a starting point for applied, ecological, and molecular-evolutionary virus studies.

especially the minimum acquisition access period (AAP), latent period in the vector before transmission is possible, and minimum inoculation access period (IAP). These experiments are done in the laboratory using the most common crop hosts of the virus and vector, and occasionally non-crop hosts that are common in agricultural landscapes. Protocols typically involve labor-intensive factorial assays requiring tedious transfers of hundreds of individual vectors, maintaining large numbers of plants for sufficient durations to observe infections, and use of specialized equipment, such as electrical penetration graphing instrumentation, to identify stylet activities associated with virion acquisition and inoculation (Boquel et al., 2011; Fereres and Collar, 2001; Tjallingii, 1978). As a result, for many viruses, AAP and IAP information is only available for a fraction of the vector and host genotypes and species these pathogens may encounter, even in agricultural landscapes.

Despite limitations, accumulated knowledge of pathogen-vector interactions gleaned from laboratory studies is vital for improving our understanding of virus evolution and lifestyles. For example, establishment of the modern nomenclature for transmission mechanisms (Table 1) emerged in the 1930s following rigorous and meticulous vector transmission studies that only yielded useful information because researchers took steps to exclude myriad confounding factors typical of observational studies (Rand and Pierce, 1920; Watson and Roberts, 1939). *Beet curly top virus* (genus *Curtovirus*, family *Geminiviridae*) was the first insect-transmitted plant virus species to be described in detail by a logical series of transmission experiments identifying the one competent vector (beet leafhopper, *Circulifer tenellus* [Baker] [Hemiptera: Cicadellidae]), minimum AAPs, minimum latent periods, and susceptible alternative over-wintering hosts for both the virus and the vector (Bennett and Wallace, 1938; Rand and Pierce, 1920; Severin, 1931). The progression of studies on beet curly top virus established a methodological framework for future work, which rapidly expanded to include many new virus pathogens.

By 1940, clear distinctions in transmission requirements were evident, and researchers began classifying viruses into the categories of "persistent" and "non-persistent" based on differences in the durations of retention on, or in, the vector (Table 1). These categories were succinctly defined by Watson and Roberts (1939) following a series of studies on transmission of several non-persistently transmitted viruses (hereafter, non-persistent viruses), which also provided definitive proof that aphid transmission efficiency for viruses with this lifestyle declines with increasing time feeding on the infected host (Watson, 1936; Watson and Roberts, 1939; Watson and Russell, 1938). A more comprehensive review of non-persistent virus transmission followed in 1954 (Sylvester, 1954). Additional studies eventually led to differentiation of retention categories into "non-circulative" and "circulative" based on whether a virus is able to cross the gut barrier and invade the hemolymph and salivary glands of their vectors, thus being retained through molting (Kennedy et al., 1962). A further distinction, "propagative" vs. "non-propagative", was introduced to separate circulative viruses that replicate in vector tissues from those that do not (Kennedy et al., 1962).

The work of Watson and colleagues also clarified an important discrepancy in how researchers approach the study of persistent viruses, which are often obligately vector-transmitted, and non-persistent viruses, which can be mechanically sap-transmitted or via insect vectors. Watson and Roberts note in their 1939 paper that "When viruses are difficult to transmit mechanically, understanding of the insect-virus relationships is essential to easy propagation of the viruses, and no progress can be made without it. When the viruses are readily sap-transmissible their properties can more easily be determined without the use of their vectors. Also the specific and obligate nature of the insect transmission observed for the persistent viruses has led workers to consider it as of particular complexity and interest whereas the superficial nature of the studies which have so far been made on the non-persistent type has led some workers to the conclusion that they are of minor interest." It is interesting

research on non-persistent virus transmission largely focused on narrow suites of vectors known to colonize the most commonly infected crop hosts. Even to this day, papers still use the words "simple" and "mechanical" to describe the non-persistent transmission mechanism, despite obvious evidence to the contrary (Deshoux et al., 2018).

3 Quantifying vector contributions in the field

The approaches and research described in Section 2 pertain to virus-vector interactions under a narrow suite of laboratory conditions at single time points in disease progression and host phenology. For all but the most well-studied pathosystems, complementary field studies have not been performed to validate links between vector presence and patterns of virus spread. However, for those systems where such studies are available, we sometimes find important contrasts with laboratory results that further refine our understanding of selection pressures that certain groups of plant viruses face in real-world environments.

Non-persistent viruses provide excellent examples of disconnects between laboratory and field studies. These disconnects are important to explore because they have implications for how we design and interpret research on virus evolution and ecology. As noted in Table 1, unlike persistent viruses, non-persistent viruses are exclusively aphid-borne and have very brief AAPs and IAPs, typically only a few seconds. For the AAP, longer-term aphid feeding in vascular tissues is detrimental for virus retention, with the one exception being cCauliflower mosaic virus (CaMV), (species *Cauliflower mosaic virus*, genus *Caulimovirus* family *Caulimoviridae*), which has a bimodal non-persistent/semi-persistent transmission mechanism depending on the vector. Acquisition takes place during intracellular probes in epidermal and mesophyll tissues and virions are only retained for a few hours. If aphids feed from the phloem of the infected plant following virion acquisition, they often lose viruliferous status (Martin et al., 1997; Wang and Ghabrial, 2002; Watson and Roberts, 1939). Thus, unlike persistent and semi-persistent viruses, transmission of non-persistent viruses largely depends on vectors dispersing rapidly from infected hosts following probing, and before initiating phloem sap ingestion. Empirical studies demonstrating this (Fereres et al., 1992; Garzo et al., 2004; Martin et al., 1997; Wang and Ghabrial, 2002; Watson and Roberts, 1939; Watson and Russell, 1938) are supported by theoretical work modeling virus spread and vector movement; for non-persistently transmitted viruses to spread, it is critical that vectors move to new susceptible hosts over short time frames following virus acquisition (Madden et al., 2000).

Despite these stringent requirements for vector feeding and dispersal behaviors, laboratory experiments exploring basic aspects of non-persistent virus transmission often employ only colonizing vectors, which are more likely to settle and feed on the infected hosts (e.g., Boiteau et al., 1998; Srinivasan et al., 2012; Srinivasan and Alvarez, 2007, but see Boquel et al., 2015, 2012). This is probably because colonizing vectors were the insects seen in the field feeding on the crop when virus was also present, and so the presumption was that they are primary drivers of virus spread. For example, in the laboratory, pPotato virus Y (fPVY)] (species *Potato virus Y*, genus *Potyvirus*, family *Potyviridae*) transmission and ecology are most commonly explored using the aphid vector *Myzus persicae* (Sulzer) (Hemiptera: Aphididae), and occasionally *Macrosiphum euphorbiae* (Thomas) (Hemiptera: Aphididae) and *Aphis nasturtii* (Kaltenbach) (Hemiptera: Aphididae) (e.g., Bradley, 1963; Bradley and Rideout, 1953; Gibson et al., 1982; Govier and Kassanis, 1974; Powell, 1992; Watson and Roberts, 1939 among many others). These aphids are colonizers of the most economically important crop host (potatoes) and can colonize related indicator hosts (e.g., tobacco). Under laboratory conditions where the researcher controls the duration of probing behaviors to suit AAP and IAP requirements, both of these species are highly efficient vectors of PVY (Boquel et al., 2011). However, in the field, multiple studies have documented minimal

vectors that do not colonize solanaceous hosts as being responsible for the bulk of virus transmission events.

Because they do not feed on potato (i.e. no or reduced phloem sap ingestion), non-potato-colonizing aphids will automatically engage in the behaviors most conducive to PVY acquisition and inoculation, namely probing and rapid dispersal (Fereres and Moreno, 2009; Kennedy et al., 1961, 1959). They are also more likely to be present in high numbers during the most vulnerable early stages of the potato crop, which has not been in the field long enough to generate successive generations of potato-colonizing species or appreciable numbers of mobile alates. For example, an extensive study in England performed in the 1980's trapped 6769 individual aphids using a vertical net downwind of a PVY-infected potato plot and tested them, individually, for viruliferous status using susceptible hosts (Harrington et al., 1986). This massive undertaking yielded 165 transmission events by 20 vector species. A single non-colonizing aphid species, *Brachycaudus helichrysi* (Kaltenbach) (Hemiptera: Aphididae), was responsible for 52% of transmissions, while *M. persicae* and *M. euphorbiae* accounted for just 11.56% and 2.72% of transmissions (Harrington et al., 1986). The importance of the non-potato-colonizing *B. helichrysi* as a main driver of PVY spread was further reinforced by a long-range study that compiled over 20 years of country-wide virus incidence and aphid flight data from the Swiss seed certification program (Steinger et al., 2015). While *B. helichrysi* abundance was highly predictive of outbreaks, there was no relationship between *M. persicae* abundance and PVY incidence. A similar finding was reported the following year from a multi-year study in U.S. potato-growing regions (Mondal et al., 2016). The study documented marked increases in PVY prevalence in potato fields following peaks in trap catches of non-potato-colonizing aphid species (especially *Rhopalosiphum padi* L. [Linneaus] and *Metopolophium dirhodum* [Walker] [Hemiptera: Aphididae]), but weak connections between *M. persicae* and PVY spread.

The implication of non-potato-colonizing aphids as drivers of PVY spread in multiple geographic locations suggests that surrounding landscape composition may play more of a role in predicting infection rates in crops than any management efforts that target colonizing vectors (Carrière et al., 2014; Claflin et al., 2017). This speculation has been elegantly demonstrated by a series of studies on infection rates by non-persistent viruses infecting cultivated pumpkins. Like most cucurbits, pumpkins are subject to infection by a suite of non-persistently transmitted viruses in the families *Potyviridae* and *Bromoviridae*. Through a two year survey in the midwestern U.S., Angelella et al. (2015) determined that three non-colonizing aphids were associated with increased prevalence of several potyviruses and a cucumovirus in pumpkin fields. In subsequent work using the same data set, the authors constructed partial least-squares path models to explore the influence of within-field (i.e., weed cover) vs. extra-field land cover on virus spread (Angelella et al., 2016). Contrary to the prevailing narrative that weeds in the local vicinity of crop fields are sources of vectors, the path models revealed that extra-field cover (4-5 km) was more strongly predictive of total non-colonizing aphid numbers and virus prevalence in pumpkin fields (Angelella et al., 2016). In fact, weed cover deterred landing by non-colonizing aphids, possibly by disrupting the very attractive visual stimulus of green crop host plant tissue against a soil background (Döring and Chittka, 2007; Saucke and Döring, 2004; Schröder et al., 2017).

Knowing that local landscape features are strongly associated with vector abundance and virus spread enabled the use of molecular methods to identify the origins of one abundant non-pumpkin-colonizing aphid species, *Aphis craccivora* (Koch) (Hemiptera: Aphididae). Using single nucleotide polymorphism genotyping to identify host-associated differentiation, it was determined that early season *A. craccivora* originate from locust trees, while late-season *A. craccivora* originate largely from alfalfa (Angelella et al., 2019). This progression of work perfectly illustrates how knowing the identity of virus vectors in the field can inform studies that precisely

stages of growth in the crop. It also underscores the complexity of virus transmission at field scales (and especially non-persistent virus transmission) and reinforces how little it resembles the laboratory and greenhouse conditions under which most aspects of transmission have historically been studied. Other reports documenting prominent roles for non-colonizing aphids in non-persistent virus spread have been published for cucurbits growing in Europe (Katis et al., 2006), and for garlic (Perotto et al., 2014), papaya (Kalleshwaraswamy et al., 2005), soybeans (Halbert et al., 1981), passion fruit (Garcêz et al., 2015), stone fruits (Knoll et al., 2004) and many other crops. These studies are only snapshots of a few selected pathosystems, each using different approaches for quantifying spatial and temporal variations in vector richness and abundance in relation to virus incidence. We are now learning that aphid abundance and species richness can vary significantly over short (intra-annual) timescales, with the nature of this variation being strongly influenced by regional cropping structures (Claflin et al. 2019). Studies employing large sampling intervals, single time point collections, or limited spatial variation may not capture the influence of all crop-colonizing and non-colonizing vectors. But even with these limitations, the studies cited above still provide strong evidence that the actual insects transmitting non-persistent viruses in the field are quite often not those we are focusing on for management or studying in the laboratory.

4 Implications for the evolution of manipulative traits

Knowing the breadth of competent vectors and minimum AAPs and IAPs for at least a subset of these vectors allows researchers to make predictions about the range of transmission opportunities available to different viruses under real-world scenarios. Likewise, determining which vectors in the landscape provide the most transmission opportunities for any given virus is essential for ranking the relative contributions of vectors as drivers of virus spread in contexts that include spatial and temporal components. If we consider virus fitness across hosts as the number of new infections originating from a single inoculum source (the basic reproductive number, R_0), those viruses experiencing more transmission opportunities will enjoy greater fitness. Thus, the vector species that transmit in the field are ultimately the ones imposing selection pressure on a virus to take advantage of their presence and the transmission opportunities this provides.

It is within this context that manipulative traits enabling viruses to alter host phenotypes and vector behavior are purported to evolve. To explore congruence between studies reporting putative instances of virus manipulation and virus traits underlying transmission opportunities, we summarized transmission traits (number of reported competent vector species, minimum AAP, and whether extended feeding enhances acquisition) for viruses having at least one published report describing effects consistent with adaptive host and vector manipulation. These studies have been previously reviewed in Mauck et al. (2018) (see list of the studies as supplementary material). To compile transmission trait information, we considered online databases such as Plant Virus Online (Brunt et al., 1997), The Universal Virus Database (ICTVdB) (Buchen-Osmond, 2006) and DPVweb (Adams and Antoniw, 2006), all of which have the shared purpose of compiling and summarizing basic research generated for individual virus species over the last ~100 years. When different numbers of competent vectors were indicated within these databases, we kept the higher value. To complete our compilation, we also surveyed Edwardson and Christie (1991), which reviewed characteristics of viruses associated with legume crops, as well as specific articles for viruses characterized more recently and not reported in databases (indicated directly in Tables 2&3). To locate these articles, we searched Google Scholar using the following terms: "[virus name]" transmission-" and "[virus name]" acquisition access period". We then evaluated how transmission metrics have, or have not, informed the design and interpretation of empirical and theoretical explorations of plant virus

(families *Bromoviridae*, *Potyviridae* and some *Caulimoviridae*) can be transmitted by a greater number of vector species than most persistent and semi-persistent viruses (all other families, Tables 2&3). The average number of competent vector species for a non-persistent virus in the *Bromoviridae*, *Potyviridae*, or *Caulimoviridae* families is 34.5 +/- 9.3 vector species (range: 4 to 89 vectors), while the same metric for viruses from any other family is 3.8 +/- 0.84 vector species (range: 1 to 22). The compilation further shows that viruses having higher numbers of competent vector species tend to be those for which extended feeding in vascular tissue (more precisely, phloem sap ingestion) does not enhance virus acquisition rates. This is strongly congruent with the studies cited in Section 3 reporting major contributions of non-colonizing vectors to the spread of viruses in the families *Bromoviridae* and *Potyviridae* in field studies of potato and cucurbit crops.

In terms of virus evolution, the compilation suggests that non-persistent viruses generate transmission opportunities through adaptations facilitating interactions with multiple, potentially distantly related vectors, possibly by capitalizing on shared features of the aphids stylet anatomy (Uzest et al., 2010). As described in Section 3, outbreaks of non-persistent viruses occur when there are increases in abundance of one or more vectors that do not colonize the main crop hosts of the virus (Angelella et al., 2015; Steinger et al., 2015). The identity of these vectors may or may not be the same from year to year depending on landscape features that support growth of key vector populations (e.g., adjacent crops, which may rotate).

How might this reality shape the evolution of manipulative traits in non-persistent viruses? Clearly, most competent vector species (Tables 2&3) will engage in behaviors required for efficient non-persistent virus transmission (e.g., rapid probing and dispersal) due to host plant incompatibility, and regardless of infection status. Thus, for these viruses, there may be little gained, if anything, from maintaining mutations that manipulate host phenotype to encourage colonizing vectors to engage in transmission-conducive behaviors. Mutations may even be detrimental given that virus coding capacity is already severely limited and pleiotropic effects of mutations are common (Bedhomme et al., 2012; García-Arenal and Fraile, 2013; Sanjuán et al., 2005).

Surely, any study proposing to explore adaptive host and vector manipulation by non-persistent viruses must consider the selection pressures described above and in previous sections. Unfortunately, this is not the case: nearly all studies to date testing for host and vector manipulation by non-persistent viruses have focused exclusively on vectors that are the main colonizers of the hosts under study (reviewed in Mauck et al., 2019, 2018) (Fig. 1). Additionally, a single colonizing vector species is a common feature of every theoretical model exploring the implications of host and vector manipulation for virus spread. Not surprisingly, the results of both empirical and theoretical studies are unclear and sometimes contradictory. In one of the most recent reviews on this topic (Mauck et al., 2018), we performed a quantitative synthesis of the effects of non-persistent virus infections in host plants on vector orientation preferences, settling/feeding preferences, and performance. If non-persistent viruses are manipulating colonizing vectors, we would expect to see induction of host phenotypes that favor rapid probing and dispersal behaviors that are required for virus retention (Ng and Falk, 2006). But our quantitative synthesis of empirical work demonstrated that non-persistent viruses have no clear effect on host palatability for vectors, with nearly even numbers of experiments documenting increases, decreases, and neutral effects (Fig. 2) (Mauck et al., 2018).

alt-text: Fig. 1

Fig. 1
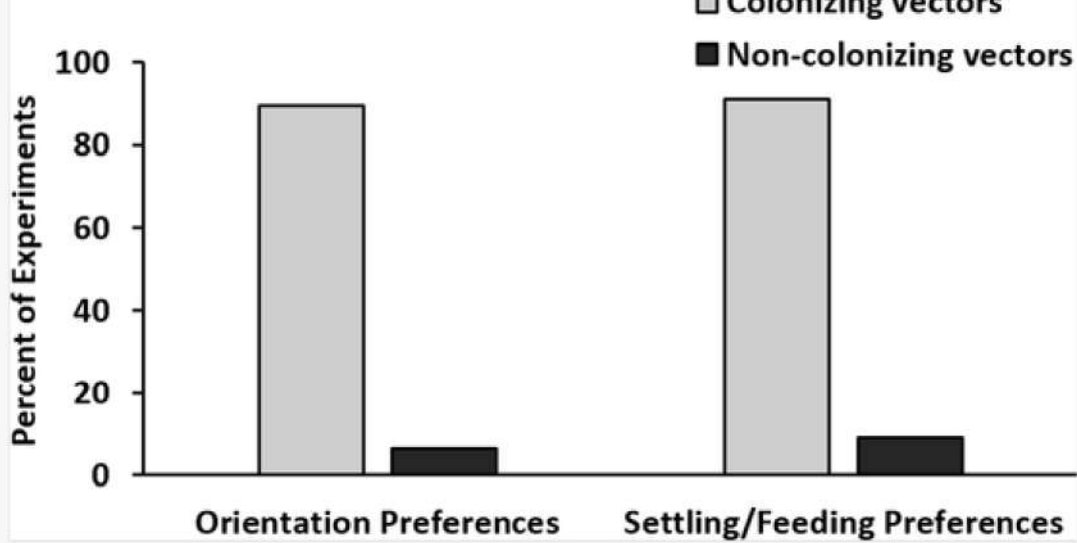
Summary of vector-host relationships examined in studies reporting putative instances of virus manipulation by non-persistently transmitted plant viruses. Calculations are based on all experiments on this pathogen group included in our prior quantitative synthesis (Mauck et al. (2018), supplementary material), and several recently published studies (orientation preferences category [n = 19] additionally includes Mwando et al. (2018) and Safari et al. (2019); settling/feeding preferences category [n = 90] additionally includes Angelella et al. (2018); Murphy et al. (2018); Gadhave et al. (2019); Guo et al. (2019)).
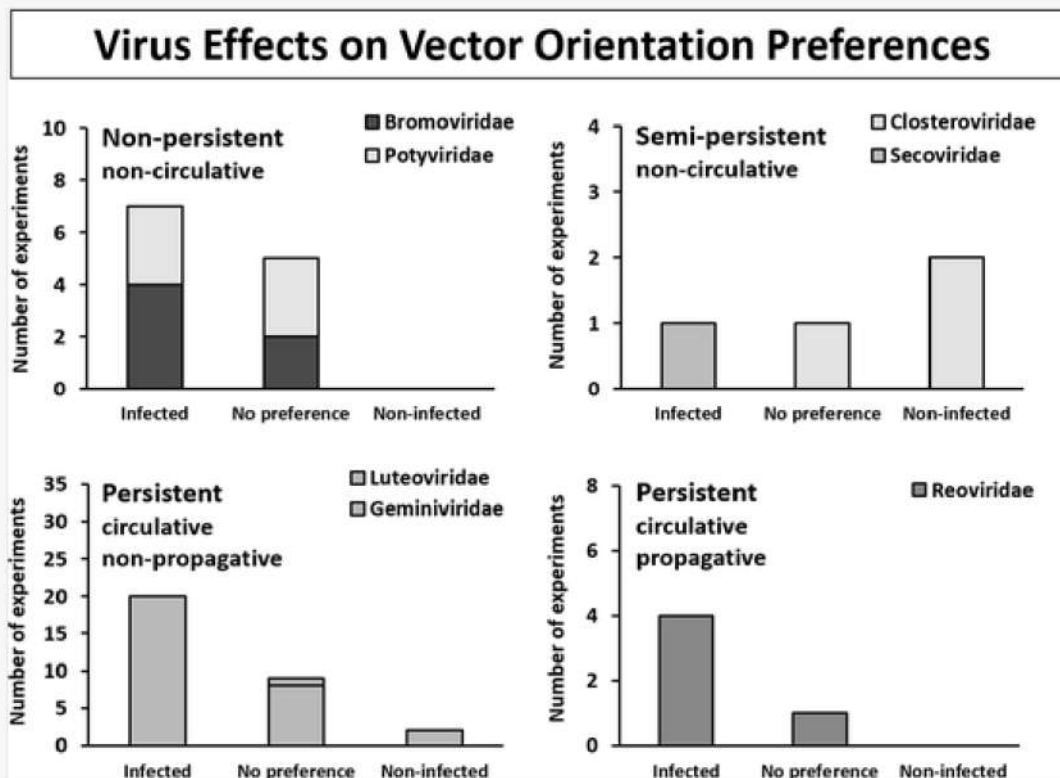
alt-text: Fig. 2

Fig. 2
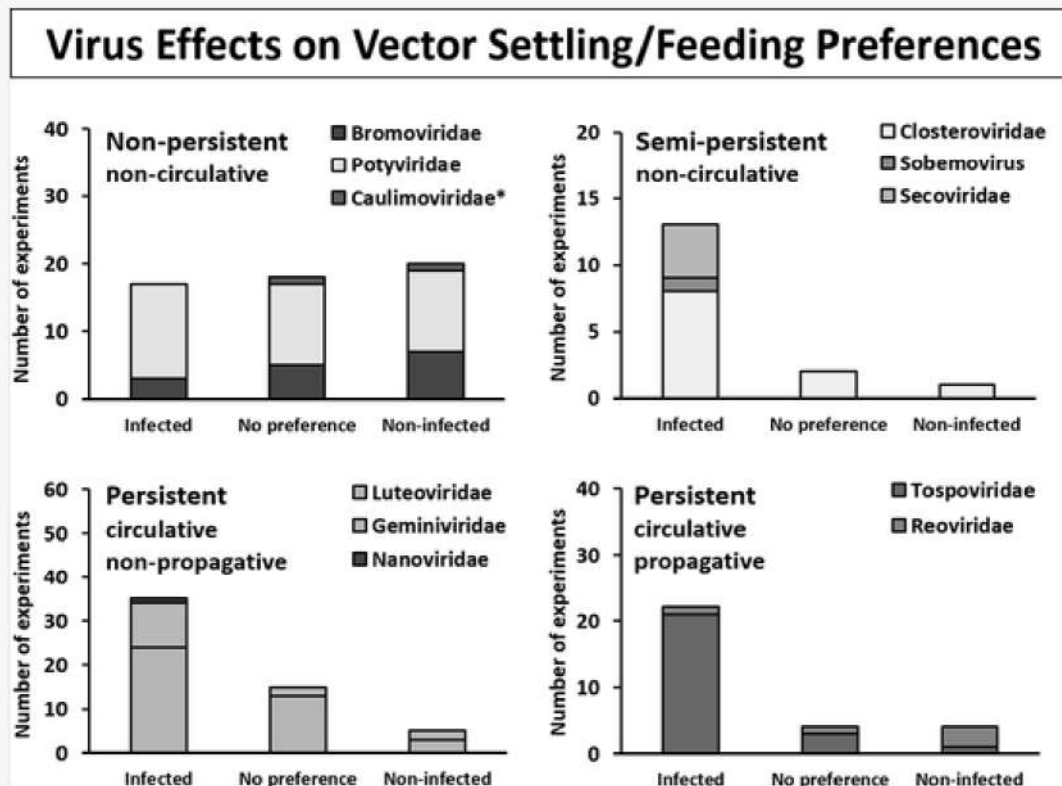
Summary of virus effects on the settling and/or feeding preferences of insect vectors. Adapted from the quantitative synthesis of plant virus manipulation literature reported in Mauck et al. (2018). Literature summarized in Mauck et al. (2018) is listed in the supplementary material.

attractiveness to vectors; non-persistent viruses had neutral to positive effects on host attractiveness, usually through exaggeration or elevation of already attractive visual and/or odor cues (Fig. 3) (Mauck et al., 2018). This one significant effect makes sense given our summary of competent vector species (Table 2) and field evidence discussed in Section 3. Changing cues that function over a distance, and that are broadly attractive, could result in differential attraction of most competent vectors regardless of whether they are able to colonize the infected host (Kring, 1972; Powell et al., 2006). Increasing vector contacts is generally beneficial for virus spread, especially when there may be concurrent selection pressure to facilitate transmission before the dominant host (crop) progresses in phenology and age-related resistance (Panter and Jones, 2002). However, manipulation of vector attraction by non-persistent viruses has only been explored in a handful of papers (Eigenbrode et al., 2002 ; Mauck et al., 2014, 2010; Tungadi et al., 2017; Wu et al., 2017) and attraction is the least-studied aspect of virus manipulation, with most experiments across all virus groups focusing on post-contact vector-host interactions (Mauck et al., 2018). But for non-persistent viruses, enhancements in general attractiveness to vectors may be the manipulation we are most likely to observe given the realities of how members of this complex pathogen group are transmitted in real-world scenarios (Section 3).

alt-text: Fig. 3

Fig. 3

Summary of virus effects on orientation preferences of insect vectors. Adapted from the quantitative synthesis of plant virus manipulation literature reported in Mauck et al. (2018). Literature summarized in Mauck et al. (2018) is listed in the supplementary material.

In contrast to non-persistently transmitted viruses, our compilation suggests that viruses having semi-persistent and persistent lifestyles interact with a limited suite of vector species that must feed on the infected host for extended durations to acquire virions (Tables 2&-3). For these pathogen groups, persistence in vectors for days to lifetimes increases the likelihood that each vector individual will provide extended transmission opportunities

extended ingestion often enhances transmission efficiency and/or retention durations (Jiménez-Martínez and Bosque-Pérez, 2004; Palacios et al., 2002) (Tables 2&-3). This requirement limits the breadth of possible vectors to those likely to feed on the plant for long periods of time. Consistent with this requirement, in our quantitative summary we found clear evidence that semi-persistent and persistent viruses enhance host palatability in ways that encourage vector settling and feeding (Mauck et al., 2018) (Fig. 2). Persistent virus infection in hosts also strongly enhanced vector attraction (Mauck et al., 2018) (Fig. 3). Long-term retention in the vector helps guarantee that the pay-off of these manipulations is potentially large once vectors disperse. This has been shown for persistent viruses across numerous theoretical studies modeling effects of manipulations on transmission by a single colonizing vector in a homogeneous host environment (e.g., monoculture) (Donnelly et al., 2019; Roosien et al., 2013; Shaw et al., 2019, 2017; Sisterson, 2008). Furthermore, recent empirical publications report that vector dispersal does occur reliably even though non-viruliferous vectors initially settle more readily on hosts infected with persistent and semi-persistent viruses (Fereres et al., 2016; Ingwell et al., 2012; Maluta et al., 2014; Moreno-Delafuente et al., 2013; Rajabaskar et al., 2014; Roosien et al., 2013). Collectively, our compilation, quantitative synthesis, and recent literature indicate that viruses relying on a small number of colonizing vector species for transmission are more likely to experience benefits from manipulating hosts and may thus be under stronger selection pressure to do so.

5 Conclusions, limitations, and knowledge gaps

We can safely assume that Tables 2 & 3 are incomplete, as there is no way of establishing when all competent vectors have been identified. And as discussed in Section 3, data on competent vector species for non-persistent viruses may be understated because biases in studying transmission of this group have existed since the persistent/non-persistent distinction was established. There may similarly be underestimations for virus species that are recently described, recently emerged as crop pathogens, or that have limited economic importance (Ghosh et al., 2015; Quito-Avila et al., 2012; Reinbold et al., 2003). Additionally, inability to resolve cryptic vector species complexes (e.g., for *Bemisia* whiteflies) may lead to spurious rejection of vector species as competent if concurrent molecular confirmation of the vector subspecies or biotype is not performed (De Barro et al., 2011; Milenovic et al., 2019). But even with somewhat incomplete data, when considered alongside multi-year field studies (Section 3), our compilation reveals important discrepancies in how we are studying the phenomenon of plant virus manipulation across persistent and non-persistent virus groups.

Pathogen-vector combinations studied for evidence of manipulation by persistent and semi-persistent virus species are mostly representative of the actual pathogen-vector interactions that occur in the field environment. Even in cases where a persistent virus species-has several possible vectors (e.g., the species *Barley yellow dwarf virus, genus Luteovirus, family Luteoviridae* [BYDV] having 17 vectors across all strains) studies reporting manipulation have used the most compatible virus strain x vector species combinations (Bosque-Pérez and Eigenbrode, 2011; dos Santos et al., 2016; Gildow, 1980; Ingwell et al., 2012; Jiménez-Martínez et al., 2004aJiménez-Martínez et al., 2004; Jiménez-Martínez et al., 2004bJiménez-Martínez et al., 2004; Medina-Ortega et al., 2009). This is important because the field environment is where manipulative traits are purported to have evolved. Given the abundance of studies on persistent viruses, and the clear requirements for vector-host compatibility, researchers need only choose the one or two colonizing vectors that are competent, efficient and abundant in the target crop host to reconstruct a laboratory pathosystem that is congruent with field realities.

However, it is troubling that this same strategy is employed when selecting vectors for virus manipulation studies with non-persistent viruses (especially those discussed in Section 3). Almost all studies with non-persistent

often a result of transmission by non-colonizing vectors. The identities of the most important vectors for the same virus species may vary by geographic region and across seasons within geographic regions (e.g., PVY spread is driven by Asteraceae specialist aphids in Europe and grass-feeding aphids in the U.S.). Furthermore, the diversity of possible vectors is far greater than that recorded thus far for most persistent and semi-persistent viruses. By ignoring these facts when selecting virus-vector combinations, researchers studying virus manipulation are certainly failing to approximate conditions under which manipulative traits are evolving, yet still claiming that phenotypic changes induced by non-persistent viruses are adaptive (Mauck et al., 2018, 2014a, [Instruction: This reference does not appear to be linked.]2010).

This realization can help researchers to clarify the evolution of manipulative traits in future work. For example, even though the number of studies is sparse, it seems that non-persistent viruses induce changes in hosts that enhance vector attraction (Fig. 3). Effects on cues mediating attraction of aphid vectors may substantially increase transmission because such changes are more likely to enhance visitation by multiple species, including those that do not colonize the infected host. Thus, to establish whether non-persistent viruses can evolve to manipulate hosts, it may be more prudent to focus on quantifying phenotypic changes that affect pre-alighting vector behavior. If changes in these cues cause non-colonizing vectors to differentially visit infected hosts in an otherwise homogeneous crop environment, it will increase the basic reproductive number (R_0) for viruses capable of inducing attractive host plant phenotypes. In contrast, for semi-persistent and persistent viruses, focusing on post-contact cues may be more appropriate, as the bottleneck for transmission of these pathogens lies in the vector ingesting sufficient quantities of virions to become viruliferous. The bigger question here is how arrested vectors ultimately disperse to spread the pathogen. A few studies indicate that vectors may change preferences following virion acquisition, adding a new dimension to persistent virus manipulation (Fereres et al., 2016; Ingwell et al., 2012; Maluta et al., 2014; Moreno-Delafuente et al., 2013; Rajabaskar et al., 2014; Roosien et al., 2013). Ultimately, a full understanding of the robustness and evolutionary significance of plant virus manipulation will require greater integration of experimental approaches with the entire transmission process, including relevant selection pressures experienced by viruses in agricultural and natural environments. To address these knowledge gaps, we propose the following focus areas as next steps:

1 Determine key vectors driving virus transmission across agroecological interfaces.

Annual monoculture environments are conducive for the evolution of manipulative traits in plant viruses because they are genetically uniform, phenologically synced, intensively managed to exclude pests, and, ultimately, dead ends for the virus if it is not transmitted elsewhere before harvest. All of these aspects will favor the evolution of manipulative traits that improve the odds of persisting across seasons, either in other crops, in vectors, or in unmanaged vegetation. But whether this selection pressure exists depends on which vector species or genotypes are visiting host plants in the field, and when they are doing it. We provided examples of the value of this information for management of PVY and non-persistent cucurbit viruses in Section 3 and discuss how it does, or does not, inform work on virus manipulation in Section 4. More studies are needed across a wider array of geographic areas and pathosystems to provide information on the breadth of vectors responsible for transmitting economically important viruses. These data will improve consideration of ecological aspects during design and interpretation of empirical studies on virus manipulation, assist in refining integrated pest management approaches, and provide a wealth of information on how temporal and spatial variation in agriculture impacts vectors and viruses (Claflin et al. 2019).

2 Study changes in virus-induced host phenotypes over time.

components within the context of host progression through phenological stages, a process that involves changes in the same metabolic and phytohormone-regulated pathways purported to be targeted by viruses with manipulative traits (Westwood et al. 2013, Mauck et al. 2014b, Casteel et al. 2015, Yuan and Zhang 2015, Iqbal et al. 2017, Bera et al. 2020). Studies thus far strongly suggest that putative virus manipulations of host phenotype are limited to specific host phenology x disease progression time points, which can be as small as one week (Blua and Perring 1992a; ~~Blua and Perring 1992b~~Blua and Perring, 1992; Blua et al. 1994; Werner et al. 2009; Higashi and Bressan 2013; Shi et al. 2016; Legarrea et al. 2015; Mann et al. 2008; Rajabaskar et al. 2013; Williams 1995; Shrestha et al. 2019). In some cases, vectors even exhibit opposing behavioral responses to the same virus-host combination at different time points in disease progression (e.g., Blua and Perring 1992, Rajabaskar et al. 2013, Shi et al. 2016). Implications of "temporally limited host manipulations" for virus spread are intriguing to consider, but are not well-integrated into theoretical models that attempt to quantify benefits of manipulation for viruses (McElhany et al. 1995, Sisterson et al. 2008, Roosien et al. 2013, Shaw et al. 2017, Shaw et al. 2019). Likewise, these dynamics are rarely considered alongside typical fluctuations in vector activity present in the field environments where manipulative traits are purported to have evolved.

3 Comprehensively evaluate the ecology of viruses before, during, and after undertaking empirical work on virus manipulation.

Viruses infect multiple hosts and can be transmitted by multiple vectors. These aspects must be considered during the experimental design phase of studies exploring whether virus-induced host phenotypes are likely to enhance transmission through effects on vector behavior. Doing so often reveals complexity underlying what appears to be a simple story at the surface level (Rajabaskar et al. 2013, Mauck et al. 2014a). For example, we previously reported induction of a transmission-conducive phenotype by the non-persistently transmitted cucumber mosaic virus (CMV) (species *Cucumber mosaic virus*, genus *Cucumovirus*, family *Bromoviridae*) infecting cultivated squash (Mauck et al. 2010). But CMV infects a wide variety of hosts across many plant families. When additional CMV isolates were tested for manipulative ability, we observed both transmission-conducive and transmission-limiting effects depending on the host species being infected (Mauck et al. 2014a). In the host where we detected transmission-conducive effects, only a very compatible, colonizing vector species responded to the shift in host phenotype, while a marginal colonizer had a neutral response (Mauck et al. 2014a). These findings reveal important trade-offs and limitations on the evolution of manipulative traits but were only evident when variation in host and vector associations were included in the experimental approach. In this manuscript, we consider virus-vector associations retroactively in the context of previously published studies that did not include the breadth of competent vectors as an ecological axis. The outputs of this exercise suggest that conclusions about many cases of manipulation may be premature and formed out of context. More value and insight into virus manipulation could be gained by considering virus ecology outside of the laboratory before study conception, data interpretation, and publication.

4 Include more marginal and non-colonizing vector species and genotypes.

Our synthesis indicates that studies exploring virus manipulation of host phenotypes and vector behavior tend to focus on the most compatible vector-host combinations. We highlighted downsides of this focus for our understanding of putative manipulations by non-persistently transmitted viruses. However, exclusion of marginal and non-colonizing vector species or genotypes from studies on persistently transmitted viruses also influences interpretations of putative manipulations by viruses in this group. Benefits of host phenotype manipulation for persistently transmitted viruses may extend beyond colonizing vectors if virus infections improve host

virus. For example, Davis et al. (2017) found that infection by the persistent, circulative bean leafroll virus (species *Bean leafroll virus*, genus *Luteovirus*, family *Luteoviridae*) reduced the degree of host fidelity (attraction and settling) exhibited by different genotypes of *Acyrthosiphon pisum* (pea aphids) specialized on pea or alfalfa, both of which are susceptible hosts for the virus. Thus, viruses which alter host phenotypes to favor feeding by a broader array of vector species or genotypes will create additional transmission opportunities that are not available to viruses lacking manipulative traits. These benefits, driven by marginal or non-colonizing vector species, could even be a primary selective force favoring manipulative traits under certain field conditions. Understanding the contributions of marginal and non-colonizing vectors to the transmission process, and evolution of manipulative traits, will require more studies to identify relevant vectors across diverse pathosystems and landscape structures.

Uncited reference[Instruction: These references appear in the tables. See response to Query 1 and associated attachment with tables.]s

Bath and Chapman (1968), Brown and Nelson (1989), Brown and Nelson (1988), Brown and Nelson (1986), Cabanas et al. (2013), Cadman (1954), Chen et al. (2004), Chen et al. (2014), Demler et al. (1996), Dubern (1994), Duffus (1963), Duffus et al. (1986), Firmino et al. (2009), Ghosh et al. (2016), Gray et al. (1991), Han et al. (2019), Harrington and Gibson (1989), Jiu et al. (2006), Lakshmi et al. (1995), Lecoq et al. (1992), Leonard and Holbrook (1978), Limburg et al. (1997), Maluta et al. (2017), Mehta et al. (1994), Nasu (1963), Nateshan et al. (1996), Nault et al. (1978), Pu et al. (2012), ~~Purcifull et al. (2020)~~Purcifull et al., 1984, Rochow and Carmichael (1979), Santos et al. (2003), Shikata et al. (1979), Shinkai (1967), Shinkai (1962), Slack and Fulton (1971), Sylvester (1956), Sylvester and Richardson (1969), Tamada (1970), Thekke-Veetil et al. (2017), Toloy et al. (2018), Van de Wetering et al. (1999), Wang et al. (1994), Wei et al. (2019), Wijkamp et al. (1996), Wisler et al. (1998), Zeidan and Czosnek (1991), Zettler et al. (1968) and Zhou and Tzanetakis (2013).

Acknowledgements

Appendix A Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.virusres.2020.197957.

References

> ⓘ The corrections made in this section will be reviewed and approved by a journal production editor. The newly added/removed references and its citations will be reordered and rearranged by the production team.

Adams, M.J., Antoniw, J.F., 2006. DPVweb: a comprehensive database of plant and fungal virus genes and genomes. Nucleic Acids Res. 34, D382-5. doi:10.1093/nar/gkj023.

interactions and the agro-ecological interface. Eur. J. Plant Pathol. 138, 529–547. doi:10.1007/s10658-013-0317-1.

Angelella, G.M., Egel, D.S., Holland, J.D., Nemacheck, J.A., Williams, C.E., Kaplan, I., 2015. Differential life history trait associations of aphids with non-persistent viruses in cucurbits. Environ. Entomol. 44, 562–573. doi:10.1093/ee/nvv058.

Angelella, G.M., Holland, J.D., Kaplan, I., 2016. Landscape composition is more important than local management for crop virus–insect vector interactions. Agric. Ecosyst. Environ..

Angelella, G., Nalam, V., Nachappa, P., White, J., Kaplan, I., 2018. Endosymbionts differentially alter exploratory probing behavior of a nonpersistent plant virus vector. Microb. Ecol. 76, 453–458. doi:10.1007/s00248-017-1133-5.

Angelella, G.M., Michel, A.P., Kaplan, I., 2019. Using host-associated differentiation to track source population and dispersal distance among insect vectors of plant pathogens. Evol. Appl. 12, 692–704. doi:10.1111/eva.12733.

Bak, A., Cheung, A.L., Yang, C., Whitham, S.A., Casteel, C.L., 2017. A viral protease relocalizes in the presence of the vector to promote vector performance. Nat. Commun. 8, 14493. doi:10.1038/ncomms14493.

Bath, J.E., Chapman, R.K., 1968. Influence of aphid stage on acquisition and inoculation phases of pea enation mosaic virus transmission. Ann. Entomol. Soc. Am. 61, 906–909. doi:10.1093/aesa/61.4.906.

Bedhomme, S., Lafforgue, G., Elena, S.F., 2012. Multihost experimental evolution of a plant RNA virus reveals local adaptation and host-specific mutations. Mol. Biol. Evol. 29, 1481–1492. doi:10.1093/molbev/msr314.

Bennett, C.W., Wallace, H.E., 1938. ~~Relation of the curly top virus to the Vector, Futettix Tenellus~~Relation of the curly top virus to the Vector, *Futettix Tenellus*. US Government Printing Office.

Bera, S., Blundell, R., Liang, D., Crowder, D.W., Casteel, C.L., 2020. ~~The oxylipin Signaling pathway is required for increased aphid attraction and retention on virus-infected plants~~The oxylipin signaling pathway is required for increased aphid attraction and retention on virus-infected plants. J. Chem. Ecol.. doi:10.1007/s10886-020-01157-7.

Blua, M.J., Perring, T.M., 1992. Alatae production and population increase of aphid vectors on virus-infected host plants. Oecologia 92, 65–70. doi:10.1007/BF00317263.

Blua, M.J., Perring, T.M., 1992. ~~Effects of zucchini yellow mosaic virus on colonization and feeding behavior of Aphis gossypii (Homoptera: Aphididae) Alatae~~Effects of zucchini yellow mosaic virus on colonization and feeding behavior of *Aphis gossypii* (Homoptera: Aphididae) Alatae. Environ. Entomol. 21, 578–585. doi:10.1093/ee/21.3.578.

Blua, M.J., Perring, T.M., Madore, M.A., 1994. Plant virus-induced changes in aphid population development and temporal fluctuations in plant nutrients. J. Chem. Ecol. 20, 691–707. doi:10.1007/BF02059607.

Myzus persicae (Sulzer) from infected to healthy plants under laboratory conditionsRate of spread of PVYn by alate *Myzus persicae* (Sulzer) from infected to healthy plants under laboratory conditions. Potato Res. 41, 335–344. doi:10.1007/BF02358966.

Boquel, S., Ameline, A., Giordanengo, P., 2011. Assessing aphids potato virus Y-transmission efficiency: A new approach. J. Virol. Methods 178, 63–67. doi:10.1016/j.jviromet.2011.08.013.

Boquel, S., Delayen, C., Couty, A., Giordanengo, P., Ameline, A., 2012. Modulation of aphid vector activity by potato virus Y on in vitro potato plants. Plant Dis. 96, 82–86. doi:10.1094/PDIS-06-11-0499.

Boquel, S., Zhang, J., Goyer, C., Giguère, M.-A., Clark, C., Pelletier, Y., 2015. Effect of insecticide-treated potato plants on aphid behavior and potato virus Y acquisition. Pest Manag. Sci. 71, 1106–1112.

Bosque-Pérez, N.A., Eigenbrode, S.D., 2011. The influence of virus-induced changes in plants on aphid vectors: Insights from luteovirus pathosystems. Virus Res. 159, 201–205. doi:10.1016/j.virusres.2011.04.020.

Bradley, R.H.E., 1963. Some ways in which a paraffin oil impedes aphid transmission of potato virus Y. Can. J. Microbiol. 9, 369–380. doi:10.1139/m63-045.

Bradley, R.H.E., Rideout, D.W., 1953. Comparative transmission of potato virus Y by four aphid species that infest potato. Can. J. Zool. 31, 333–341. doi:10.1139/z53-026.

Brown, J.K., Nelson, M.R., 1989. Characterisation of watermelon curly mottle virus, a geminivirus distinct from squash leaf curl virus. Ann. Appl. Biol. 115, 243–252. doi:10.1111/j.1744-7348.1989.tb03383.x.

Brown, J.K., Nelson, M.R., 1988. Transmission, host range and virus-vector relationships in chino del tomate virus, a whitefly-transmitted geminivirus from Sinaloa. Mexico. Plant Dis. 72, 866–869.

Brown, J.K., Nelson, M.R., 1986. Whitefly-borne viruses of melons and lettuce in Arizona. Phytopathology 76, 236–239.

A.[Instruction: The "A. " before "Brunt" should be removed. I cannot edit this reference for some reason.] Brunt, K. Crabtree, M. Dallwitz, A. Gibbs, L. Watson, E. Zurcher. Plant Viruses Online : Descriptions and Lists from the VIDE Database. Cab International. http://sdb.im.ac.cn/vide/refs.htm, 1997.

Buchen-Osmond, C., 2006. Index to ICTVdB virus descriptions. ICTVdB-The Universal Virus Database, version 4.

Cabanas, D., Watanabe, S., Higashi, C.H.V., Bressan, A., 2013. Dissecting the mode of maize chlorotic mottle virus transmission (Tombusviridae: Machlomovirus) by Frankliniella williamsi (Thysanoptera: Thripidae)Dissecting the mode of maize chlorotic mottle virus transmission (*Tombusviridae*: *Machlomovirus*) by *Frankliniella williamsi* (Thysanoptera: Thripidae). J. Econ. Entomol. 106, 16–24. doi:10.1603/ec12056.

Cadman, C.H., 1954. Studies in Rubus virus diseases: aphid transmission of raspberry leaf mottle virus. Ann. Appl. Biol. 41, 207–214. doi:10.1111/j.1744-7348.1954.tb00927.x.

F.O., Murphy, A.M., Arinaitwe, W., Pate, A.E., Cunniffe, N.J., Gilligan, C.A., 2020. Modelling and manipulation of aphid-mediated spread of non-persistently transmitted viruses. Virus Res. 277, 197845. doi:10.1016/j.virusres.2019.197845.

Carrière, Y., Degain, B., Hartfield, K.A., Nolte, K.D., Marsh, S.E., Ellers-Kirk, C., Van Leeuwen, W.J.D., Liesner, L., Dutilleul, P., Palumbo, J.C., 2014. Assessing transmission of crop diseases by insect vectors in a landscape context. J. Econ. Entomol. 107, 1–10.

Casteel, C.L., Yang, C., Nanduri, A.C., De Jong, H.N., Whitham, S.A., Jander, G., 2014. ~~The NIa-Pro protein of Turnip mosaic virus improves growth and reproduction of the aphid vector, Myzus persicae (green peach aphid)~~The NIa-Pro protein of turnip mosaic virus improves growth and reproduction of the aphid vector, *Myzus persicae* (green peach aphid). Plant J. 77, 653–663. doi:10.1111/tpj.12417.

Casteel, C., De Alwis, M., Bak, A., Dong, H., Steven, A.W., Jander, G., 2015. ~~Disruption of ethylene responses by Turnip mosaic virus mediates suppression of plant defense against the aphid vector, Myzus persicae~~Disruption of ethylene responses by turnip mosaic virus mediates suppression of plant defense against the aphid vector, *Myzus persicae*. Plant Physiol. 169, 209–216. doi:10.1104/pp.15.00332.

Castle, S.J., Mowry, T.M., Berger, P.H., 1998. ~~Differential settling by Myzus persicae (Homoptera: Aphididae) on various virus infected host plants~~Differential settling by *Myzus persicae* (Homoptera: Aphididae) on various virus infected host plants. Ann. Entomol. Soc. Am. 91, 661–667. doi:10.1093/aesa/91.5.661.

Chay, C.A., Gunasinge, U.B., Dinesh-Kumar, S.P., Miller, W.A., Gray, S.M., 1996. Aphid transmission and systemic plant infection determinants of barley yellow dwarf luteovirus-PAV are contained in the coat protein readthrough domain and 17-kDa protein, respectively. Virology 219, 57–65. doi:10.1006/viro.1996.0222.

Chen, C.C., Yeh, S.D., Hsu, H.T., 2004. ~~Acquisition, incubation and transmission of Watermelon silver mottle virus by Thrips palmi, in: XI International Symposium on Virus Diseases of Ornamental Plants 722~~Acquisition, incubation and transmission of watermelon silver mottle virus by *Thrips palmi*, in: XI International Symposium on Virus Diseases of Ornamental Plants 722. actahort.org 83–90.

Chen, W.-T., Tseng, C.-H., Tsai, C.-W., 2014. ~~Effect of watermelon silver mottle virus on the life history and feeding preference of Thrips palmi~~Effect of watermelon silver mottle virus on the life history and feeding preference of *Thrips palmi*. PLoS One 9, e102021. doi:10.1371/journal.pone.0102021.

Claflin, S.B., Jones, L.E., Thaler, J.S., Power, A.G., 2017. Crop-dominated landscapes have higher vector-borne plant virus prevalence. J. Appl. Ecol. 54, 1190–1198.

Claflin, S.B., Hernandez, N., Groves, R., Thaler, J.S., Power, A.G., 2019. Intra-annual variation and landscape composition interactively affect aphid community composition. Ecosphere 10, e02710. doi:10.1002/ecs2.2710.

Culver, J.N., Padmanabhan, M.S., 2007. Virus-induced disease: altering host physiology one interaction at a time. Annu. Rev. Phytopathol. 45, 221–243. doi:10.1146/annurev.phyto.45.062806.094422.

host selection behavior of specialized pea aphid (Acyrthosiphon pisum, Hemiptera: Aphididae) genotypesThe effects of bean leafroll virus on life history traits and host selection behavior of specialized pea aphid (*Acyrthosiphon pisum*, Hemiptera: Aphididae) genotypes. Environ. Entomol..

De Barro, P.J., Liu, S.-S., Boykin, L.M., Dinsdale, A.B., 2011. Bemisia tabaci: a statement of species status*Bemisia tabaci*: a statement of species status. Annu. Rev. Entomol. 56, 1–19. doi:10.1146/annurev-ento-112408-085504.

Demler, S.A., de Zoeten, G.A., Adam, G., Harris, K.F., 1996. Pea enation mosaic enamovirus: Properties and aphid transmission. In: Harrison, B.D., Murant, A.F. (Eds.), The Plant Viruses: Polyhedral Virions and Bipartite RNA Genomes. Springer US, Boston, MA, pp. 303–344. doi:10.1007/978-1-4899-1772-0_12.

Deshoux, M., Monsion, B., Uzest, M., 2018. Insect cuticular proteins and their role in transmission of phytoviruses. Curr. Opin. Virol. 33, 137–143. doi:10.1016/j.coviro.2018.07.015.

Djidjou-Demasse, R., Moury, B., Fabre, F., 2017. Mosaics often outperform pyramids: insights from a model comparing strategies for the deployment of plant resistance genes against viruses in agricultural landscapes. New Phytol. 216, 239–253. doi:10.1111/nph.14701.

Donnelly, R., Cunniffe, N.J., Carr, J.P., Gilligan, C.A., 2019. Pathogenic modification of plants enhances long-distance dispersal of non-persistently transmitted viruses to new hosts. Ecology e02725.. doi:10.1002/ecy.2725.

Döring, T.F., 2014. How aphids find their host plants, and how they don't. Ann. Appl. Biol. 165, 3–26.

Döring, T.F., Chittka, L., 2007. Visual ecology of aphids: a critical review on the role of colours in host finding. Arthropod Plant Interact. 1, 3–16. doi:10.1007/s11829-006-9000-1.

dos Santos, R.C., Peñaflor, M.F.G.V., Sanches, P.A., Nardi, C., Bento, J.M.S., 2016. The effects of Gibberella zeae, Barley yellow dwarf virus, and co-infection on Rhopalosiphum padi olfactory preference and performanceThe effects of *Gibberella zeae*, barley yellow dwarf virus, and co-infection on *Rhopalosiphum padi* olfactory preference and performance. Phytoparasitica 44, 47–54. doi:10.1007/s12600-015-0493-y.

Dubern, J., 1994. Transmission of African cassava mosaic geminivirus by the whitefly (Bemisia tabaci)Transmission of african cassava mosaic geminivirus by the whitefly (*Bemisia tabaci*). Tropical Science..

Duffus, J.E., 1963. Possible multiplication in the aphid vector of sowthistle yellow vein virus, a virus with an extremely long insect latent period. Virology 21, 194–202. doi:10.1016/0042-6822(63)90257-5.

Duffus, J.E., Larsen, R.C., Liu, H.Y., 1986. Lettuce infectious yellows virus—a new type of whitefly-transmitted virus. Phytopathology 76, 97–106.

Edwardson, J.R., Christie, R.G., 1991. CRC Handbook of Viruses Infecting Legumes. CRC Press. doi:10.1201/9781351071192.

leafroll virus attract and arrest the virus vector, Myzus persicae (Homoptera: Aphididae)Volatiles from potato plants infected with potato leafroll virus attract and arrest the virus vector, *Myzus persicae* (Homoptera: Aphididae). Proc. Biol. Sci. 269, 455–460. doi:10.1098/rspb.2001.1909.

Fereres, A., Blua, M.J., Perring, T.M., 1992. ~~Retention and transmission characteristics of Zucchini yellow mosaic virus by Aphis gossypii and Myzus persicae (Homoptera: Aphididae)~~Retention and transmission characteristics of zucchini yellow mosaic virus by *Aphis gossypii* and *Myzus persicae* (Homoptera: Aphididae). J. Econ. Entomol. 85, 759–765. doi:10.1093/jee/85.3.759.

Fereres, A., Collar, J.L., 2001. Analysis of non-circulative transmission by electrical penetration graphs. Virus-Insect-Plant Interactions. Academic Press, New York, NY, pp. 87–109.

Fereres, A., Moreno, A., 2009. Behavioural aspects influencing plant virus transmission by homopteran insects. Virus Res. 141, 158–168. doi:10.1016/j.virusres.2008.10.020.

Fereres, A., Peñaflor, M.F.G.V., Favaro, C.F., Azevedo, K.E.X., Landi, C.H., Maluta, N.K.P., Bento, J.M.S., Lopes, J.R.S., 2016. Tomato infection by whitefly-transmitted circulative and non-circulative viruses induce contrasting changes in plant volatiles and vector behaviour. Viruses 8. doi:10.3390/v8080225.

Firmino, A.C., Yuki, V.A., Moreira, A.G., Rezende, J.A.M., 2009. ~~Tomato yellow vein streak virus: relationship with Bemisia tabaci biotype B and host range~~Tomato yellow vein streak virus: relationship with *Bemisia tabaci* biotype B and host range. Sci. Agric. 66, 793–799.

Gadhave, K.R., Dutta, B., Coolong, T., Srinivasan, R., 2019. ~~A non-persistent aphid-transmitted Potyvirus differentially alters the vector and non-vector biology through host plant quality manipulation~~A non-persistent aphid-transmitted potyvirus differentially alters the vector and non-vector biology through host plant quality manipulation. Sci. Rep. 9, 2503. doi:10.1038/s41598-019-39256-5.

Garcêz, R.M., Chaves, A.L.R., Eiras, M., Meletti, L.M.M., de Azevedo Filho, J.A., da Silva, L.A., Colariccio, A., 2015. ~~Survey of aphid population in a yellow passion fruit crop and its relationship on the spread Cowpea aphid-borne mosaic virus in a subtropical region of Brazil~~Survey of aphid population in a yellow passion fruit crop and its relationship on the spread cowpea aphid-borne mosaic virus in a subtropical region of Brazil. Springerplus 4, 537. doi:10.1186/s40064-015-1263-5.

García-Arenal, F., Fraile, A., 2013. Trade-offs in host range evolution of plant viruses. Plant Pathol. 62, 2–9.

Garzo, E.I., Duque, M., Fereres, A., 2004. Transmission efficiency of different non-persistent viruses infecting melon by four aphid species. Span. J. Agric. Res. 2, 369–376. doi:10.5424/sjar/2004023-91.

Ghosh, A., Das, A., Lepcha, R., Mandal, B., 2016. Identification, distribution and temporal occurrence of aphids infesting large cardamom and their efficiency in transmitting large cardamom viruses in northeastern sub-Himalayan region. Australas. Plant Pathol. 45, 533–536.

Ghosh, A., Das, A., Vijayanandraj, S., Mandal, B., 2015. ~~Cardamom bushy dwarf virus infection in large cardamom alters plant selection preference, life stages, and fecundity of aphid vector, Micromyzus kalimpongensis (Hemiptera: Aphididae)~~Cardamom bushy dwarf virus infection in large cardamom alters

(Hemiptera: Aphididae). Environ. Entomol. 45, 178–184.

Gibbs, A.J., Fargette, D., Garcia-Arenal, F., Gibbs, M.J., 2010. Time - the emerging dimension of plant virus studies. J. Gen. Virol. 91, 13–22.

Gibbs, A.J., Ohshima, K., Phillips, M.J., Gibbs, M.J., 2008. The prehistory of potyviruses: Their initial radiation was during the dawn of agriculture. PLoS One 3, e2523. doi:10.1371/journal.pone.0002523.

Gibson, R.W., Rice, A.D., Sawicki, R.M., 1982. ~~Effects of the pyrethroid deltamethrin on the acquisition and inoculation of viruses by Myzus persicae~~Effects of the pyrethroid deltamethrin on the acquisition and inoculation of viruses by *Myzus persicae*. Ann. Appl. Biol. 100, 49–54. doi:10.1111/j.1744-7348.1982.tb07190.x.

Gildow, F.E., 1980. ~~Increased production of alatae by aphids reared on oats infected with Barley yellow dwarf virus~~Increased production of alatae by aphids reared on oats infected with barley yellow dwarf virus. Ann. Entomol. Soc. Am. 73, 343–347. doi:10.1093/aesa/73.3.343.

Gómez, P., Rodríguez-Hernández, A.M., Moury, B., Aranda, M.A., 2009. Genetic resistance for the sustainable control of plant virus diseases: breeding, mechanisms and durability. Eur. J. Plant Pathol. 125, 1–22. doi:10.1007/s10658-009-9468-5.

Govier, D.A., Kassanis, B., 1974. Evidence that a component other than the virus particle is needed for aphid transmission of potato virus Y. Virology 57, 285–286.

Gray, S.M., Power, A.G., Smith, D.M., Seaman, A.J., Altman, N.S., 1991. Aphid transmission of barley yellow dwarf virus: acquisition access periods and virus concentration requirements. Phytopathology 81, 539–545.

Guo, H., Gu, L., Liu, F., Chen, F., Ge, F., Sun, Y., 2019. ~~Aphid-borne viral spread Is enhanced by virus-induced accumulation of plant reactive oxygen species~~Aphid-borne viral spread is enhanced by virus-induced accumulation of plant reactive oxygen species. Plant Physiol. 179, 143–155. doi:10.1104/pp.18.00437.

Halbert, S.E., Irwin, M.E., Goodman, R.M., 1981. Alate aphid (Homoptera: Aphididae) species and their relative importance as field vectors of soybean mosaic virus. Ann. Appl. Biol. 97, 1–9. doi:10.1111/j.1744-7348.1981.tb02988.x.

Han, J., Nalam, V.J., Yu, I.-C., Nachappa, P., 2019. Vector competence of thrips species to transmit soybean vein necrosis virus. Front. Microbiol. 10, 431. doi:10.3389/fmicb.2019.00431.

Harrington, R., Gibson, R.W., 1989. Transmission of potato virus Y by aphids trapped in potato crops in southern England. Potato Res. 32, 167–174. doi:10.1007/BF02358229.

Harrington, R., Katis, N., Gibson, R.W., 1986. Field assessment of the relative importance of different aphid species in the transmission of potato virus Y. Potato Res. 29, 67–76. doi:10.1007/BF02361982.

Higashi, C.H.V., Bressan, A., 2013. ~~Infection rates and comparative population dynamics of Peregrinus maidis (Hemiptera: Delphacidae) on corn plants with and without symptoms of maize mosaic virus (Rhabdoviridae: Nucleorhabdovirus) infection~~Infection rates and comparative population dynamics of

virus (*Rhabdoviridae*: *Nucleorhabdovirus*) infection. Environ. Entomol. 42, 949–956. doi:10.1603/EN12321.

Ingwell, L.L., Eigenbrode, S.D., Bosque-Pérez, N.A., 2012. Plant viruses alter insect behavior to enhance their spread. Sci. Rep. 2, 1–6. doi:10.1038/srep00578.

Iqbal, N., Khan, N.A., Ferrante, A., Trivellini, A., Francini, A., Khan, M.I.R., 2017. Ethylene role in plant growth, development and senescence: interaction with other phytohormones. Front. Plant Sci. 8, 475. doi:10.3389/fpls.2017.00475.

Jiménez-Martínez, E.S., Bosque-Pérez, N.A., 2004. ~~Variation in barley yellow dwarf virus transmission efficiency by Rhopalosiphum padi (Homoptera: Aphididae) after acquisition from transgenic and non-transformed wheat genotypes~~Variation in barley yellow dwarf virus transmission efficiency by *Rhopalosiphum padi* (Homoptera: Aphididae) after acquisition from transgenic and non-transformed wheat genotypes. J. Econ. Entomol. 97, 1790–1796.

Jiménez-Martínez, E.S., Bosque-Pérez, N.A., Berger, P.H., Zemetra, R.S., 2004. ~~Life history of the bird cherry-oat aphid, Rhopalosiphum padi (Homoptera: Aphididae), on transgenic and untransformed wheat challenged with Barley yellow dwarf virus~~Life history of the bird cherry-oat aphid, *Rhopalosiphum padi* (Homoptera: Aphididae), on transgenic and untransformed wheat challenged with barley yellow dwarf virus. J. Econ. Entomol. 97, 203–212.

Jiménez-Martínez, E.S., Bosque-Pérez, N.A., Berger, P.H., Zemetra, R.S., Ding, H., Eigenbrode, S.D., 2004. ~~Volatile cues influence the response of Rhopalosiphum padi (Homoptera: Aphididae) to Barley yellow dwarf virus–infected transgenic and untransformed wheat~~Volatile cues influence the response of *Rhopalosiphum padi* (Homoptera: Aphididae) to barley yellow dwarf virus–infected transgenic and untransformed wheat. Environ. Entomol. 33, 1207–1216. doi:10.1603/0046-225X-33.5.1207.

Jiu, M., Zhou, X.-P., Liu, S.-S., 2006. ~~Acquisition and transmission of two begomoviruses by the B and a non-B Biotype of Bemisia tabaci from Zhejiang~~Acquisition and transmission of two begomoviruses by the B and a non-B Biotype of *Bemisia tabaci* from Zhejiang. China. Journal of Phytopathology 154, 587–591.

Kalleshwaraswamy, C.M., Verghese, A., Ranganath, H.R., Krishna Ku, N.K., Dinesh, M.R., 2005. ~~Role of transient aphid vectors on the temporal spread of Papaya ringspot virus in south India, in: I International Symposium on Papaya 740~~Role of transient aphid vectors on the temporal spread of papaya ringspot virus in south India, in: I International Symposium on Papaya 740. actahort.org 251–258.

Katis, N.I., Tsitsipis, J.A., Lykouressis, D.P., Papapanayotou, A., Margaritopoulos, J.T., Kokinis, G.M., Perdikis, D.C., Manoussopoulos, I.N., 2006. ~~Transmission of Zucchini yellow mosaic virus by colonizing and non-colonizing aphids in Greece and new aphid species vectors of the virus~~Transmission of zucchini yellow mosaic virus by colonizing and non-colonizing aphids in Greece and new aphid species vectors of the virus. J. Phytopathol. 154, 293–302. doi:10.1111/j.1439-0434.2006.01096.x.

Kennedy, J.S., 1951. Benefits to aphids from feeding on galled and virus-infected leaves. Nature 168, 825–826. doi:10.1038/168825a0.

attraction. Ann. Appl. Biol. 49, 1–21. doi:10.1111/j.1744-7348.1961.tb03587.x.

Kennedy, J.S., Booth, C.O., Kershaw, W.J.S., 1959. Host finding by aphids in the field: *Aphis fabae* Scop. (gynoparae) and *Brevicoryne brassicae* L.; with a re-appraisal of the role of host-finding behavior in virus spread. Ann. Appl. Biol. 47, 424–444.

Kennedy, J.S., Day, M.F., Eastop, V.F., 1962. A conspectus of aphids as vectors of plant viruses. Commonwealth Institute of Entomology., London.

Knoll, M., Weilguni, S., Koschier, E.H., Redl, H., Steinkellner, S., 2004. Seasonal fluctuation in the flight activity of potential PPV-vector aphids (Homoptera: Aphididae) in Austrian apricot tree orchards. Zeitschrift für Pflanzenkrankheiten und Pflanzenschutz/Journal of Plant Diseases and Protection 408–415.

Kring, J.B., 1972. Flight behavior of aphids. Annu. Rev. Entomol. 17, 461–492.

Lakshmi, K.V., Wightman, J.A., Reddy, D.V.R., Rao, G.V.R., Buiel, A.A.M., Reddy, D.D.R., 1995. Transmission of peanut bud necrosis virus by *Thrips palmi* in India. In: Parker, B.L., Skinner, M, Lewis, T (Eds.), Thrips Biology and Management. Springer US, Boston, MA, pp. 179–184.

Lecoq, H., Bourdin, D., Wipf-Scheibel, C., Bon, M., Lot, H., Lemaire, O., Herrbach, E., 1992. A new yellowing disease of cucurbits caused by a luteovirus, cucurbit aphid-borne yellows virus. Plant Pathol. 41, 749–761. doi:10.1111/j.1365-3059.1992.tb02559.x.

Lefeuvre, P., Martin, D.P., Elena, S.F., Shepherd, D.N., Roumagnac, P., Varsani, A., 2019. Evolution and ecology of plant viruses. Nat. Rev. Microbiol.. doi:10.1038/s41579-019-0232-3.

Legarrea, S., Barman, A., Marchant, W., Diffie, S., Srinivasan, R., 2015. Temporal effects of a begomovirus infection and host plant resistance on the preference and development of an insect vector, *Bemisia tabaci*, and implications for epidemics. PLoS One 10, e0142114. doi:10.1371/journal.pone.0142114.

Leonard, S.H., Holbrook, F.R., 1978. Minimum acquisition and transmission times for potato leaf roll virus by the green peach aphid. Ann. Entomol. Soc. Am. 71, 493–495. doi:10.1093/aesa/71.4.493.

Limburg, D.D., Mauk, P.A., Godfrey, L.D., 1997. Characteristics of beet yellows closterovirus transmission to sugar beets by *Aphis fabae*. Phytopathology 87, 766–771. doi:10.1094/PHYTO.1997.87.7.766.

Li, R., Weldegergis, B.T., Li, J., Jung, C., Qu, J., Sun, Y., Qian, H., Tee, C., van Loon, J.J.A., Dicke, M., Chua, N.-H., Liu, S.-S., Ye, J., 2014. Virulence factors of geminivirus interact with MYC2 to subvert plant resistance and promote vector performance. Plant Cell 26, 4991–5008. doi:10.1105/tpc.114.133181.

Lu, G., Zhang, T., He, Y., Zhou, G., 2016. Virus altered rice attractiveness to planthoppers is mediated by volatiles and related to virus titre and expression of defence and volatile biosynthesis genes. Sci. Rep. 6, 38581. doi:10.1038/srep38581.

virus transmission mechanism on plant virus disease epidemics. Phytopathology 90, 576–594. doi:10.1094/PHYTO.2000.90.6.576.

Maluta, N.K.P., Garzo, E., Moreno, A., Lopes, J.R.S., Fereres, A., 2014. Tomato yellow leaf curl virus benefits population growth of the Q Biotype of *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae). Neotrop. Entomol. 43, 385–392. doi:10.1007/s13744-014-0223-z.

Maluta, N.K.P., Garzo, E., Moreno, A., Navas-Castillo, J., Fiallo-Olivé, E., Lopes, J.R.S., Fereres, A., 2017. Stylet penetration activities of the whitefly *Bemisia tabaci* associated with inoculation of the crinivirus tomato chlorosis virus. J. Gen. Virol. 98, 1515–1520.

Mann, R.S., Sidhu, J.S., Butter, N.S., Sohi, A.S., Sekhon, P.S., 2008. Performance of *Bemisia tabaci* (Hemiptera: Aleyrodidae) on healthy and cotton leaf curl virus infected cotton. Fla. Entomol. 91, 249–255. doi:10.1653/0015-4040(2008)91[249:POBTHA]2.0.CO;2.

Markham, PG, Pinner, MS, Raccah, B, Hull, R, 1987. The acquisition of a caulimovirus by different aphid species: comparison with a potyvirus.. Annals of Applied Biology 111, 571–587.

Martin, B., Collar, J.L., Tjallingii, W.F., Fereres, A., 1997. Intracellular ingestion and salivation by aphids may cause the acquisition and inoculation of non-persistently transmitted plant viruses. J. Gen. Virol. 78, 2701–2705.

Mauck, K., Bosque-Pérez, N.A., Eigenbrode, S.D., De Moraes, C.M., Mescher, M.C., 2012. Transmission mechanisms shape pathogen effects on host–vector interactions: evidence from plant viruses. Funct. Ecol. 26, 1162–1175. doi:10.1111/j.1365-2435.2012.02026.x.

Mauck, K.E., Chesnais, Q., Shapiro, L.R., 2018. Evolutionary determinants of host and vector manipulation by plant viruses. Adv. Virus Res. 101, 189–250. doi:10.1016/bs.aivir.2018.02.007.

Mauck, K.E., De Moraes, C.M., Mescher, M.C., 2014. Evidence of local adaptation in plant virus effects on host–vector interactions. Integr. Comp. Biol. 54, 193–209. doi:10.1093/icb/icu012.

Mauck, K.E., De Moraes, C.M., Mescher, M.C., 2014. Biochemical and physiological mechanisms underlying effects of cucumber mosaic virus on host-plant traits that mediate transmission by aphid vectors. Plant Cell Environ. 37, 1427–1439. doi:10.1111/pce.12249.

Mauck, K.E., De Moraes, C.M., Mescher, M.C., 2010. Deceptive chemical signals induced by a plant virus attract insect vectors to inferior hosts. Proc. Natl. Acad. Sci. U. S. A. 107, 3600–3605. doi:10.1073/pnas.0907191107.

Mauck, K.E., Kenney, J., Chesnais, Q., 2019. Progress and challenges in identifying molecular mechanisms underlying host and vector manipulation by plant viruses. Current Opinion in Insect Science 33, 7–18. doi:10.1016/j.cois.2019.01.001.

yellow dwarf virus. Ecology 76, 444–457. doi:10.2307/1941203.

Medina-Ortega, K.J., Bosque-Pérez, N.A., Ngumbi, E., Jiménez-Martínez, E.S., Eigenbrode, S.D., 2009. ~~Rhopalosiphum padi (Hemiptera: Aphididae) responses to volatile cues from barley yellow dwarf virus-infected wheat~~*Rhopalosiphum padi* (Hemiptera: Aphididae) responses to volatile cues from barley yellow dwarf virus-infected wheat. Environ. Entomol. 38, 836–845. doi:10.1603/022.038.0337.

Mehta, P., Wyman, J.A., Nakhla, M.K., Maxwell, D.P., 1994. ~~Polymerase chain reaction detection of viruliferous Bemisia tabaci (Homoptera: Aleyrodidae) with two tomato-infecting geminiviruses~~Polymerase chain reaction detection of viruliferous *Bemisia tabaci* (Homoptera: Aleyrodidae) with two tomato-infecting geminiviruses. J. Econ. Entomol. 87, 1285–1290. doi:10.1093/jee/87.5.1285.

Milenovic, M., Wosula, E.N., Rapisarda, C., Legg, J.P., 2019. ~~Impact of host plant species and whitefly species on feeding behavior of Bemisia tabaci~~Impact of host plant species and whitefly species on feeding behavior of *Bemisia tabaci*. Front. Plant Sci. 10, 1. doi:10.3389/fpls.2019.00001.

Mondal, S., Wenninger, E.J., Hutchinson, P.J.S., Weibe, M.A., Eigenbrode, S.D., Bosque-Pérez, N.A., 2016. Contribution of non-colonizing aphids to potato virus Y prevalence in potato in Idaho. Environ. Entomol. 45, 1445–1462. doi:10.1093/ee/nvw131.

Moreno-Delafuente, A., Garzo, E., Moreno, A., Fereres, A., 2013. A plant virus manipulates the behavior of its whitefly vector to enhance its transmission efficiency and spread. PLoS One 8, e61543. doi:10.1371/journal.pone.0061543.

Murphy, A.F., Rondon, S.I., Moreno, A., Fereres, A., 2018. ~~Effect of potato virus Y presence in Solanum tuberosum (Solanales: Solanaceae) and Chenopodium album on aphid (Hemiptera: Aphididae) behavior~~Effect of potato virus Y presence in *Solanum tuberosum* (Solanales: Solanaceae) and *Chenopodium album* on aphid (Hemiptera: Aphididae) behavior. Environ. Entomol. 47, 654–659. doi:10.1093/ee/nvy041.

Mwando, N.L., Tamiru, A., Nyasani, J.O., Obonyo, M.A.O., Caulfield, J.C., Bruce, T.J.A., Subramanian, S., 2018. Maize chlorotic mottle virus induces changes in host plant volatiles that attract vector thrips species. J. Chem. Ecol.. doi:10.1007/s10886-018-0973-x.

Nasu, S., 1963. Studies on some leafhoppers and planthoppers which transmit virus diseases of rice plant in Japan. Bull. Kyushu Agric. Exp. Stn 8, 153–349.

Nateshan, H.M., Muniyappa, V., Swanson, M.M., Harrison, B.D., 1996. Host range, vector relations and serological relationships of cotton leaf curl virus from southern India. Ann. Appl. Biol. 128, 233–244. doi:10.1111/j.1744-7348.1996.tb07319.x.

Nault, L.R., Styer, W.E., Coffey, M.E., Gordon, D.T., Negi, L.S., Niblett, C.L., 1978. Transmission of maize chlorotic mottle virus by chrysomelid beetles. Phytopathology 68, 1071–1074.

Ng, J.C.K., Falk, B.W., 2006. Virus-vector interactions mediating nonpersistent and semipersistent transmission of plant viruses. Annu. Rev. Phytopathol. 44, 183–212. doi:10.1146/annurev.phyto.44.070505.143325.

transmissibility of cucumber mosaic virus mutants. Virology 332, 397–405. doi:10.1016/j.virol.2004.11.021.

Nguyen, H.D., Tomitaka, Y., Ho, S.Y.W., Duchêne, S., Vetten, H.-J., Lesemann, D., Walsh, J.A., Gibbs, A.J., Ohshima, K., 2013. Turnip mosaic potyvirus probably first spread to Eurasian brassica crops from wild orchids about 1000 years ago. PLoS One 8, e55336. doi:10.1371/journal.pone.0055336.

Palacios, I., Drucker, M., Blanc, S., Leite, S., Moreno, A., Fereres, A., 2002. Cauliflower mosaic virus is preferentially acquired from the phloem by its aphid vectors. J. Gen. Virol. 83, 3163–3171. doi:10.1099/0022-1317-83-12-3163.

Panter, S.N., Jones, D.A., 2002. Age-related resistance to plant pathogens. in: Advances in Botanical Research. 251–280. doi:10.1016/S0065-2296(02)38032-7.

Patton, M.F., Bak, A., Sayre, J., Heck, M., Casteel, C.L., 2020. A polerovirus, potato leafroll virus, alters plant-vector interactions using three viral proteins. Plant Cell Environ. 43, 387–399. doi:10.1111/pce.13684.

Perotto, M.C., Di Rienzo, J.A., Lanati, S., Panonto, S., Macchiavelli, R., Cafrune, E.E., Conci, V.C., 2014. Temporal and spatial spread of potyvirus infection and its relationship to aphid populations visiting garlic crops. Australas. Plant Pathol. 43, 623–630. doi:10.1007/s13313-014-0312-9.

Powell, G., 1992. The effect of mineral oil on stylet activities and potato virus Y transmission by aphids. Entomol. Exp. Appl. 63, 237–242. doi:10.1111/j.1570-7458.1992.tb01579.x.

Powell, G., Tosh, C.R., Hardie, J., 2006. Host plant selection by aphids: behavioral, evolutionary, and applied perspectives. Annu. Rev. Entomol. 51, 309–330. doi:10.1146/annurev.ento.51.110104.151107.

Pu, L., Xie, G., Ji, C., Ling, B., Zhang, M., Xu, D., Zhou, G., 2012. Transmission characteristics of Southern rice black-streaked dwarf virus by rice planthoppers. Crop Prot. 41, 71–76. doi:10.1016/j.cropro.2012.04.026.

~~Purcifull, D.E., Adlerz, W.C., Simone, G.W., Hiebert, E., Christie, S.R., n.d. Serological relationships and partial characterization of Zucchini yellow mosaic virus isolated from squash in Florida. Plant Disease 68, 240.~~Purcifull, D.E., Adlerz, W.C., Simone, G.W., Hiebert, E., Christie, S.R., 1984. Serological relationships and partial characterization of zucchini yellow mosaic virus isolated from squash in Florida. Plant Disease 68, 230–233.

Quito-Avila, D.F., Lightle, D., Lee, J., Martin, R.R., 2012. ~~Transmission biology of Raspberry latent virus, the first aphid-borne reovirus~~Transmission biology of raspberry latent virus, the first aphid-borne reovirus. Phytopathology 102, 547–553. doi:10.1094/PHYTO-12-11-0331.

Rajabaskar, D., Wu, Y., Bosque-Pérez, N.A., Eigenbrode, S.D., 2013. ~~Dynamics of Myzus persicae arrestment by volatiles from potato leafroll virus-infected potato plants during disease progression~~Dynamics of _Myzus persicae_ arrestment by volatiles from potato leafroll virus-infected potato plants during disease progression. Entomol. Exp. Appl. 148, 172–181.

plants is reversed after virus acquisition. Virus Res. 186, 32–37. doi:10.1016/j.virusres.2013.11.005.

Rand, F.V., Pierce, W.D., 1920. A coordination of our knowledge of insect transmission in plant and animal diseases. Phytopathology 10, 189–231.

Reinbold, C., Herrbach, E., Brault, V., 2003. ~~Posterior midgut and hindgut are both sites of acquisition of Cucurbit aphid-borne yellows virus in Myzus persicae and Aphis gossypii~~Posterior midgut and hindgut are both sites of acquisition of cucurbit aphid-borne yellows virus in *Myzus persicae* and *Aphis gossypii*. J. Gen. Virol. 84, 3473–3484. doi:10.1099/vir.0.19415-0.

Rochow, W.F., Carmichael, L.E., 1979. Specificity among barley yellow dwarf viruses in enzyme immunosorbent assays. Virology 95, 415–420.

Roosien, B.K., Gomulkiewicz, R., Ingwell, L.L., Bosque-Pérez, N.A., Rajabaskar, D., Eigenbrode, S.D., 2013. Conditional vector preference aids the spread of plant pathogens: results from a model. Environ. Entomol. 42, 1299–1308. doi:10.1603/EN13062.

Rousseau, E., Bonneault, M., Fabre, F., Moury, B., Mailleret, L., Grognard, F., 2019. Virus epidemics, plant-controlled population bottlenecks and the durability of plant resistance. Philos. Trans. R. Soc. Lond. B Biol. Sci. 374, 20180263. doi:10.1098/rstb.2018.0263.

Safari, M., Ferrari, M.J., Roossinck, M.J., 2019. Manipulation of aphid behavior by a persistent plant virus. J. Virol. 93. doi:10.1128/JVI.01781-18.

Sanjuán, R., Cuevas, J.M., Moya, A., Elena, S.F., 2005. Epistasis and the adaptability of an RNA virus. Genetics 170, 1001–1008. doi:10.1534/genetics.105.040741.

Santos, C.D.G., Ávila, A.C., de Resende, R. de O., 2003. Estudo da interação de um begomovírus isolado de tomateiro com a mosca branca. Fitopatol. Bras. 28, 664–673.

Saucke, H., Döring, T.F., 2004. Potato virus Y reduction by straw mulch in organic potatoes. Ann. Appl. Biol. 144, 347–355. doi:10.1111/j.1744-7348.2004.tb00350.x.

Schröder, M.L., Glinwood, R., Ignell, R., Krüger, K., 2017. The role of visual and olfactory plant cues in aphid behaviour and the development of non-persistent virus management strategies. Arthropod Plant Interact. 11, 1–13. doi:10.1007/s11829-016-9463-7.

Severin, H., 1931. ~~Modes of curly-top transmission by the beet leafhopper, Eutettix tenellus (Baker)~~Modes of curly-top transmission by the beet leafhopper, *Eutettix tenellus* (Baker). Hilgardia 6, 253–276.

Shaw, A.K., Igoe, M., Power, A.G., Bosque-Pérez, N.A., Peace, A., 2019. Modeling approach influences dynamics of a vector-borne pathogen system. Bull. Math. Biol.. doi:10.1007/s11538-019-00595-z.

Shaw, A.K., Peace, A., Power, A.G., Bosque-Pérez, N.A., 2017. Vector population growth and condition-dependent movement drive the spread of plant pathogens. Ecology 98, 2145–2157. doi:10.1002/ecy.1907.

virus, a new member of plant reovirus group. Japanese Journal of Phytopathology 45, 436–443.

Shinkai, A., 1967. Transmission of stripe and black-streaked dwarf viruses by a new planthopper. Ann Phytopathol Soc Jpn 33, 318.

Shinkai, A, 1962. Studies on insect transmission of rice virus diseases in Japan. Bull. Natl. Inst. Agric. Sci. Ser. C 14, 1–112.

Shi, X., Gao, Y., Yan, S., Tang, X., Zhou, X., Zhang, D., Liu, Y., 2016. Aphid performance changes with plant defense mediated by cucumber mosaic virus titer. Virol. J. 13, 70. doi:10.1186/s12985-016-0524-4.

Shrestha, D., McAuslane, H.J., Ebert, T.A., Cervantes, F.A., Adkins, S.T., Smith, H.A., Dufault, N., Webb, S.E., 2019. ~~Assessing the temporal effects of squash vein yellowing virus Infection on settling and feeding behavior of Bemisia tabaci (MEAM1) (Hemiptera: Aleyrodidae)~~Assessing the temporal effects of squash vein yellowing virus infection on settling and feeding behavior of *Bemisia tabaci* (MEAM1) (Hemiptera: Aleyrodidae). J. Insect Sci. 19. doi:10.1093/jisesa/iez036.

Sisterson, M.S., 2008. Effects of insect-vector preference for healthy or infected plants on pathogen spread: insights from a model. J. Econ. Entomol. 101, 1–8.

Slack, S.A., Fulton, J.P., 1971. Some plant virus-beetle vector relations. Virology 43, 728–729. doi:10.1016/0042-6822(71)90300-x.

Smith, O., Clapham, A., Rose, P., Liu, Y., Wang, J., Allaby, R.G., 2014. A complete ancient RNA genome: identification, reconstruction and evolutionary history of archaeological barley stripe mosaic virus. Sci. Rep. 4, 4003. doi:10.1038/srep04003.

Srinivasan, R., Alvarez, J.M., 2007. ~~Effect of mixed viral infections (potato virus Y–potato leafroll virus) on biology and preference of vectors Myzus persicae and Macrosiphum euphorbiae (Hemiptera: Aphididae)~~Effect of mixed viral infections (potato virus Y–potato leafroll virus) on biology and preference of vectors *Myzus persicae* and *Macrosiphum euphorbiae* (Hemiptera: Aphididae). J. Econ. Entomol. 100, 646–655. doi:10.1093/jee/100.3.646.

Srinivasan, R., Hall, D.G., Cervantes, F.A., Alvarez, J.M., Whitworth, J.L., 2012. ~~Strain specificity and simultaneous transmission of closely related strains of a Potyvirus by Myzus persicae~~Strain specificity and simultaneous transmission of closely related strains of a potyvirus by *Myzus persicae*. J. Econ. Entomol. 105, 783–791.

Steinger, T., Goy, G., Gilliand, H., Hebeisen, T., Derron, J., 2015. Forecasting virus disease in seed potatoes using flight activity data of aphid vectors. Ann. Appl. Biol. 166, 410–419. doi:10.1111/aab.12190.

Sylvester, E.S., 1956. Beet mosaic and beet yellows virus transmission by the green peach aphid. J. Am. Soc. Sugar Beet Technol. 9, 56–61.

Sylvester, E.S., 1954. Aphid transmission of nonpersistent plant viruses with special reference to the brassica nigra virus. Hilgardia 23, 53–98.

virus in an aphid vector - serial passage. Virology 37, 26–31. doi:10.1016/0042-6822(69)90302-X.

Tamada, T., 1970. Aphid transmission and host range of soybean dwarf virus. Japanese Journal of Phytopathology 36, 266–274. doi:10.3186/jjphytopath.36.266.

Thekke-Veetil, T., Khadgi, A., Johnson, D., Burrack, H., Sabanadzovic, S., Tzanetakis, I.E., 2017. First report of raspberry leaf mottle virus in blackberry in the United States. Plant Dis. 101 265–265. doi:10.1094/PDIS-07-16-1014-PDN.

Tjallingii, W.F., 1978. Electronic recording of penetration behavior by aphids. Entomol. Exp. Appl. 24, 521–530.

Toloy, R.S., Mituti, T., Freitas, D.M.S., Maluta, N.K.P., Silva, T.N.Z., Lopes, J.R.S., Fereres, A., Rezende, J.A.M., 2018. ~~Features of the relationship between Tomato severe rugose begomovirus and Bemisia tabaci MEAM1 reveal that the virus is acquired during a probe lasting only one minute~~Features of the relationship between tomato severe rugose begomovirus and *Bemisia tabaci* MEAM1 reveal that the virus is acquired during a probe lasting only one minute. Eur. J. Plant Pathol. 151, 541–547.

Tungadi, T., Groen, S.C., Murphy, A.M., Pate, A.E., Iqbal, J., Bruce, T.J.A., Cunniffe, N.J., Carr, J.P., 2017. Cucumber mosaic virus and its 2b protein alter emission of host volatile organic compounds but not aphid vector settling in tobacco. Virol. J. 14, 91. doi:10.1186/s12985-017-0754-0.

Uzest, M., Gargani, D., Dombrovsky, A., Cazevieille, C., Cot, D., Blanc, S., 2010. The "acrostyle": A newly described anatomical structure in aphid stylets. Arthropod Struct. Dev. 39, 221–229. doi:10.1016/j.asd.2010.02.005.

Van de Wetering, F., Van der Hoek, M., Goldbach, R., Mollema, C., Peters, D., 1999. ~~Variation in tospovirus transmission between populations of Frankliniella occidentalis (Thysanoptera: Thripidae)~~Variation in tospovirus transmission between populations of *Frankliniella occidentalis* (Thysanoptera: Thripidae). Bull. Entomol. Res. 89, 579–588.

Wang, R.Y., Gergerich, R.C., Kim, K.S., 1994. The relationship between feeding and virus retention time in beetle transmission of plant viruses. Phytopathology 84, 995.

Wang, R.Y., Ghabrial, S.A., 2002. ~~Effect of aphid behavior on efficiency of transmission of soybean mosaic virus by the soybean-colonizing aphid, Aphis glycines~~Effect of aphid behavior on efficiency of transmission of soybean mosaic virus by the soybean-colonizing aphid, *Aphis glycines*. Plant Dis. 86, 1260–1264. doi:10.1094/PDIS.2002.86.11.1260.

Wang, J., Bing, X., Li, M., Ye, G., Liu, S., 2012. Infection of tobacco plants by a begomovirus improves nutritional assimilation by a whitefly. Entomol. Exp. Appl. 144, 191–201. doi:10.1111/j.1570-7458.2012.01278.x.

Watson, M.A., 1936. Factors affecting the amount of infection obtained by aphis transmission of the virus Hy. III. Philos. Trans. R. Soc. Lond. B Biol. Sci. 226, 457–489. doi:10.1098/rstb.1936.0013.

Watson, M.A., Roberts, F.M., 1939. ~~A comparative study of the transmission of hyoscyamus virus 3, potato virus Y and cucumber virus 1 by the vectors Myzus persicae (Sulz), M. circumflexus (Buckton),~~

Y and cucumber virus 1 by the vectors *Myzus persicae* (Sulz), *M. circumflexus* (Buckton), and *Macrosiphum gei* (Koch). Proceedings of the Royal Society of London. Series B-Biological Sciences 127, 543–576.

Watson, M.A., Russell, E.J., 1938. ~~Further studies on the relationship between hyoscyamus virus 3 and the aphid Myzus persicae (Sulz.) with special reference to the effects of fasting~~Further studies on the relationship between hyoscyamus virus 3 and the aphid *Myzus persicae* (Sulz.) with special reference to the effects of fasting. Proceedings of the Royal Society of London. Series B - Biological Sciences 125, 144–170. doi:10.1098/rspb.1938.0018.

Webb, Sarah E, Adkins, Scott, Reitz, Stuart R, 2012. Semipersistent whitefly transmission of squash vein yellowing virus, causal agent of viral watermelon vine decline. Plant Disease 96, 839–844. doi:https://doi.org/10.1094/PDIS-09-11-0761.

Webster, B., 2012. The role of olfaction in aphid host location. Physiol. Entomol. 37, 10–18. doi:10.1111/j.1365-3032.2011.00791.x.

Wei, K.-K., Li, J., Ding, T.-B., Liu, T.-X., Chu, D., 2019. ~~Transmission characteristics of Tomato chlorosis virus (ToCV) by Bemisia tabaci MED and its effects on host preference of vector whitefly~~Transmission characteristics of tomato chlorosis virus (ToCV) by *Bemisia tabaci* MED and its effects on host preference of vector whitefly. J. Integr. Agric. 18, 2107–2114. doi:10.1016/S2095-3119(18)62080-5.

Werner, B.J., Mowry, T.M., Bosque-Pérez, N.A., Ding, H., Eigenbrode, S.D., 2009. Changes in green peach aphid responses to potato leafroll virus-induced volatiles emitted during disease progression. Environ. Entomol. 38, 1429–1438. doi:10.1603/022.038.0511.

Westwood, J.H., Groen, S.C., Du, Z., Murphy, A.M., Anggoro, D.T., Tungadi, T., Luang-In, V., Lewsey, M.G., Rossiter, J.T., Powell, G., Smith, A.G., Carr, J.P., 2013. A trio of viral proteins tunes aphid-plant interactions in Arabidopsis thaliana. doi:10.1371/journal.pone.0083066.

Wijkamp, I., van de Wetering, F., Goldbach, R., Peters, D., 1996. ~~Transmission of tomato spotted wilt virus by Frankliniella occidentalis: median acquisition and inoculation access period~~Transmission of tomato spotted wilt virus by *Frankliniella occidentalis*: median acquisition and inoculation access period. Ann. Appl. Biol. 129, 303–313. doi:10.1111/j.1744-7348.1996.tb05753.x.

Williams, C.T., 1995. ~~Effects of plant age, leaf age and virus yellows infection on the population dynamics of Myzus persicae (Homoptera: Aphididae) on sugarbeet in field plots~~Effects of plant age, leaf age and virus yellows infection on the population dynamics of *Myzus persicae* (Homoptera: Aphididae) on sugarbeet in field plots. Bull. Entomol. Res. 85, 557–567. doi:10.1017/S000748530003306X.

Wintermantel, W.M., Hladky, L.L., Cortez, A.A., 2009. A new expanded host range of cucurbit yellow stunting disorder virus includes three agricultural crops. Plant Dis. 93, 685–690. doi:10.1094/PDIS-93-7-0685.

Wisler, G.C., Duffus, J.E., Liu, H.-Y., Li, R.H., 1998. Ecology and epidemiology of whitefly-transmitted closteroviruses. Plant Dis. 82, 270–280. doi:10.1094/PDIS.1998.82.3.270.

Kang, L., Ding, S.-W., Xie, D., 2017. Viral effector protein manipulates host hormone signaling to attract insect vectors. Cell Res.. doi:10.1038/cr.2017.2.

Yuan, Z., Zhang, D., 2015. Roles of jasmonate signalling in plant inflorescence and flower development. Curr. Opin. Plant Biol. 27, 44–51. doi:10.1016/j.pbi.2015.05.024.

Zeidan, M., Czosnek, H., 1991. ~~Acquisition of tomato yellow leaf curl virus by the whitefly Bemisia tabaci~~Acquisition of tomato yellow leaf curl virus by the whitefly _Bemisia tabaci_. J. Gen. Virol. 72 (Pt 11), 2607–2614. doi:10.1099/0022-1317-72-11-2607.

Zettler, F.W., Edwardson, J.R., Purcifull, D.E., 1968. Ultramicroscopic differences in inclusions of papaya mosaic virus and papaya ringspot virus correlated with differential aphid transmission. Phytopathology 58, 332–335.

Zhang, T., Luan, J.-B., Qi, J.-F., Huang, C.-J., Li, M., Zhou, X.-P., Liu, S.-S., 2012. Begomovirus-whitefly mutualism is achieved through repression of plant defences by a virus pathogenicity factor. Mol. Ecol. 21, 1294–1304. doi:10.1111/j.1365-294X.2012.05457.x.

Zhou, J., Tzanetakis, I.E., 2013. Epidemiology of soybean vein necrosis-associated virus. Phytopathology 103, 966–971. doi:10.1094/PHYTO-12-12-0322-R.

Ziebell, H., Murphy, A.M., Groen, S.C., Tungadi, T., Westwood, J.H., Lewsey, M.G., Moulin, M., Kleczkowski, A., Smith, A.G., Stevens, M., Powell, G., Carr, J.P., 2011. Cucumber mosaic virus and its 2b RNA silencing suppressor modify plant-aphid interactions in tobacco. Sci. Rep. 1, 187. doi:10.1038/srep00187.

Highlights

- Plant viruses face pressure to escape dead end hosts in agricultural environments.
- Viruses may evolve to manipulate host phenotypes and vector behaviors.
- Manipulation should enhance transmission and the odds of persisting outside of crops.
- Virus transmission traits provide context for the evolution of virus manipulation.
- Virus-vector pairs in manipulation studies are not congruent with transmission traits.

Appendix A Supplementary data

The following are Supplementary data to this article:

Multimedia Component 1

Transmission mechanism	Phloem-limited?	Typical latent period	Optimum AAP/IAP duration	Retention duration & location	Replication in vector
Non-persistent, non-circulative	No	none	<1 minute	minutes to hours, stylet	No
Semi-persistent, non-circulative	Most	none	minutes to several hours	hours to days, stylet/foregut	No
Persistent, circulative, non-propagative	Most	24-48 hours	minutes to several hours	lifetime of vector, salivary glands	No
Persistent, circulative, propagative	Some	several days to > 1 week	minutes to several hours	lifetime of vector, salivary glands plus other locations	Yes

Table 1: Characteristics of arthropod-borne viruses with different transmission mechanisms. Non-persistent, semi-persistent, and persistent refer to the retention mechanism. Circulative and non-circulative refer to capacity to cross the gut barrier and enter the hemolymph, or not, respectively. Propagative and non-propagative refer to capacity to replicate in vector tissues, or not, respectively.

Transmission mechanism	Family	Genus	Virus species	Vector number	Minimum acquisition period	Enhanced by longer feeding?	References
	Bromoviridae	Alfamovirus	AMV	22	3 seconds	No	Edwardson & Christie 1991
		Cucumovirus	CMV	87	3 seconds	No	Edwardson & Christie 1991
Non-persistent non-circulative	**Potyviridae**	Potyvirus	BYMV	47	3 seconds	No	Edwardson & Christie 1991
		Potyvirus	PRSV	21	3 seconds	No	DPVweb[a], Zettler et al. 1968
		Potyvirus	PVY	26	3 seconds	No	DPVweb[a], Harrington & Gibson 1989
		Potyvirus	SMV	32	3 seconds	No	Edwardson & Christie 1991
		Potyvirus	SPFMV	4	3 seconds	No	ICTVdB Management (2006)
		Potyvirus	TuMV	89	3 seconds	No	Edwardson & Christie 1991
		Potyvirus	WMV	44	3 seconds	No	Edwardson & Christie 1991
		Potyvirus	ZYMV	8	3 seconds	No	DPVweb[a], Purcifull et al. 1984
	Caulimoviridae	Caulimovirus	CaMV†	27	<1 minute	Yes	DPVweb[a], Kennedy et al. 1962, Palacios et al. 2002
	Caulimoviridae	Caulimovirus	CaMV†	27	5-15 minutes	Yes	Markham et al. 1987, Palacios et al. 2002
Semi-persistent non-circulative	**Closteroviridae**	Closterovirus	BYV	22	20-30 min	Yes	DPVweb[a], Sylvester 1956, Limburg et al. 1997
		Crinivirus	LIYV	1	10 min	Yes	DPVweb[a], Duffus et al. 1986
		Closterovirus	RLMV	1	5-30 min	Yes	DPVweb[a], Thekke-Veetil et al. 2017, Cadman 1954
		Crinivirus	ToCV	1	20-30 min	Yes	Wisler et al. 1998, Maluta et al. 2017, Wei 2019
	Potyviridae	Ipomovirus	SqVYV	1	20-30 min	Yes	Webb et al. 2012
	Secoviridae	Comovirus	BPMV	6	2 hours	Yes	DPVweb[a], Slack & Fulton 1971, Edwardson & Christie 1991
		Sobemovirus*	SBMV	3	24 hours	Yes	Edwardson & Christie 1991; Wang et al. 1994
	Tombusviridae*	Machlomovirus	MCMV	7	3 hours	Yes	DPVweb[a], Nault et al. 1978, Cabanas et al. 2013

Table 2: Compiled transmission characteristics of viruses reported to have evolved manipulative traits. Included in this table are non-circulative viruses with non-persistent and semi-persistent retention mechanisms. * indicates virus taxa for which there is some evidence

of virion entry to hemolymph. † indicates virus taxa for which there is evidence of a bimodal or biphasic transmission mechanism that is dependent on the identity of the vector species (listed in two transmission categories). Virus species corresponding to abbreviations from top to bottom of column three of the table are as follows: *Alfalfa mosaic virus, Cucumber mosaic virus, Bean yellow mosaic virus, Papaya ringspot virus, Potato virus Y, Soybean mosaic virus, Sweet potato feathery mottle virus, Turnip mosaic virus, Watermelon mosaic virus, Zucchini yellow mosaic virus, Cauliflower mosaic virus, Beet yellows virus, Lettuce infectious yellows virus, Raspberry leaf mottle virus, Tomato chlorosis virus, Squash vein yellowing virus, Bean pod mottle virus, Southern bean mosaic virus, Maize chlorotic mottle virus.* [a] *DPVweb: (Adams & Antoniw 2006);* [b] *ICTVdB - The Universal Virus Database, version 4, April 2006.*

Transmission mechanism	Family	Genus	Virus species	Vector number	Minimum acquisition period	Enhanced by longer feeding?	References
	Geminiviridae	Begomovirus	CdTV	1	60 min	Yes	Brown & Nelson 1988
		Begomovirus	CLCuV	3	60 min	Yes	Edwardson & Christie 1991, Nateshan et al. 1996
		Begomovirus	CLCV	3	est. 60 minutes	Yes	Edwardson & Christie 1991
		Begomovirus	EACMV	1	3.5 hours	Yes	DPVweb[a], Dubern 1994
		Begomovirus	TbCSV	1	30-60 minutes	Yes	Jiu et al. 2006
		Begomovirus	ToMoV	1	<24h (est. 30-60 minutes)	Yes	ICTVdB Management (2006), Mehta et al. 1994
Persistent, circulative non-propagative		Begomovirus	ToSRV	1	1-30 minutes	Yes	ICTVdB Management (2006), Toloy et al. 2018
		Begomovirus	TYLCCNV	1	30-60 minutes	Yes	ICTVdB Management (2006), Jiu et al. 2006
		Begomovirus	TYLCV	1	15-60 minutes	Yes	DPVweb[a], Zeidan and Czosnek 1991
		Begomovirus	WCMoV	1	est. 30 minutes	Yes	Brown & Nelson 1986, Brown & Nelson 1989
		Begomovirus	ToRMV	1	15 min	Yes	Santos et al. 2003
		Begomovirus	ToYVSV	1	30 min	Yes	Firmino et al. 2009
	Luteoviridae	Luteovirus	BLRV	12	2 hours	Yes	Edwardson & Christie 1991
		Luteovirus / Polerovirus	B/CYDV	14	15-60 minutes	Yes	DPVweb[a], Rochow & Carmichael 1979, Gray et al. 1991
		Polerovirus	CABYV	2	<24h (est. 15-60 minutes)	Yes	Lecoq et al. 1992
		Enamovirus	PEMV	13	15-120 minutes	Yes	DPVweb[a], Bath & Chapman 1968, Edwardson & Christie 1991, Demler et al. 1996

Family	Genus	Virus	No.	Acquisition/retention time	Transmission	References
	Polerovirus	PLRV	2	5-60 minutes	Yes	DPVweb[a], Kennedy et al. 1962, Leonard & Holbrok 1978
	Polerovirus	TuYV	17	5 minutes	Yes	DPVweb[a] (J.E. Duffus, unpublished), Edwardson & Christie 1991
	Luteovirus	SbDV	1	30-60 min	Yes	Tamada 1970
Nanoviridae	Babuvirus	CBDV	1	< 24h (est. 30 min–4 hours)	Yes	Ghosh et al. 2015, Ghosh et al. 2016
Reoviridae	Phytoreovirus	RDV	5	1-5 minutes	Yes	DPVweb[a], Shinkai 1962, Nasu 1963
	Phytoreovirus	RpLV	1	24 hours	Yes	Quito-Avila et al. 2012
	Oryzavirus	RRSV	1	3 hours	Yes	DPVweb[a], Shikata et al. 1979
	Fijivirus	SRBSDV	1	5 minutes	Yes	DPVweb[a], Shinkai 1967, Pu et al. 2012
Tospoviridae	Orthotospovirus	GBNV	1	5 minutes	Yes	Lakshmi et al. 1995
	Orthotospovirus	INSV	1	4 hours	Yes	ICTVdB Management (2006); van de Wetering et al. 1999
	Orthotospovirus	SVNV	2	6 hours	Yes	DPVweb[a]; Zhou & Tzanetakis 2013; Han et al. 2019
	Orthotospovirus	TSWV	9	5 minutes	Yes	Edwardson & Christie 1991; Wijkamp et al. 1996
	Orthotospovirus	WSMoV	1	30 minutes	Yes	Chen et al. 2004 ; Chen et al. 2014
Rhabdoviridae	Nucleo-rhabdovirus	SYVV	1	2 hours	Yes	DPVweb[a], Sylvester & Richardson 1969, Duffus 1963

(Left margin label for bottom group: **Persistent circulative propagative**)

Table 3: Compiled transmission characteristics of viruses reported to have evolved manipulative traits. Included in this table are circulative viruses with persistent, circulative non-propagative and persistent, circulative propagative mechanisms. Virus species corresponding to abbreviations from top to bottom of column three of the table are as follows: *Chino del tomate virus, Cotton leaf curl virus, Chili leaf curl virus, East african cassava mosaic virus, Tobacco curly shoot virus, Tomato mottle virus, Tomato severe rugose virus, Tomato yellow leaf curl china virus, Tomato yellow leaf curl virus, Watermelon curly mottle virus, Tomato rugose mosaic virus, Tomato yellow vein streak virus, Bean leafroll virus, Barley yellow dwarf virus, Cucurbit aphid-borne yellows virus, Pea enation mosaic virus, Potato leafroll virus, Turnip yellows virus, Soybean dwarf virus, Cardamom bushy dwarf virus, Rice dwarf virus, Raspberry latent virus, Rice ragged stunt virus, Southern rice black-streaked dwarf virus, Groundnut bud necrosis virus, Impatiens necrotic spot virus,*

Soybean vein necrosis virus, Tomato spotted wilt virus, Watermelon silver mottle virus, Sowthistle yellow vein virus.