Vector-Mediated Transmission of Plant Pathogens
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CHAPTER 1

Chemical Ecology of Aphid-Transmitted Plant Viruses

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Most described plant viruses require vectors for transmission between host plants. The epidemiology of such viruses is therefore largely dependent upon the population dynamics, long- and short-range dispersal, and host-selection and feeding behaviors of the vectors. This dependency sets the stage for complex direct and indirect interactions involving the plant, virus, and vector. Thus, the ecology and evolution of virus, vector, and host plant are closely intertwined.

To varying degrees, these three-way interactions can be mediated by chemistry; i.e., they can fall within the realm of chemical ecology, broadly defined to include ecological interactions mediated by biogenic organic compounds, whether the effects are primarily behavioral or physiological (Jones, 1988). Since plant viruses cannot produce or respond to metabolites directly, a chemical ecology of vector-transmitted plant viruses must be indirect, resulting from the effects of virus infection on the host plants and the responses to these changes by vectors and other organisms in the ecological community.

Examination of the chemical ecology of vector-transmitted plant viruses is just beginning. Most of the published literature concerns aphid-transmitted viruses, which reflects their predominance; aphids are vectors for 35% of all described plant viruses (Gray and Banerjee, 1999) and 50% of all insect-transmitted viruses (Nault, 1997; Ng and Perry, 2004). In this chapter, we review this literature and identify emerging themes and needs for continuing research in this area. Chemically mediated interactions among other insect-transmitted plant pathogens and their hosts and vectors—e.g., *Tomato spotted wilt virus* (*Tospovirus: Bunyaviridae*) and thrips (Belliure et al., 2005, 2008); phytoplasma and psyllids (Mayer et al., 2008); Dutch elm disease and bark beetles (McLeod et al., 2005; and Chapter 5, this volume)—are beyond the scope of this chapter, but some principles we examine may apply to them as part of a broader field of the chemical ecology of vector-transmitted pathogens.

Effects of Virus-Infected Hosts on Aphid Performance and Behavior

In a seminal paper, J. S. Kennedy (1951) reported that *Aphis fabae* Scopoli (Hemiptera: Aphidae) colonies grew more rapidly and individual aphids produced more offspring on leaves of various ages of their host plant, *Beta vulgaris* L., infected with an undetermined virus compared with noninfected control plants. Aphid reproduction was 1.4 times greater on the infected plants, leading to greater crowding and increased emigration. Professor Kennedy (1951; page 825) somewhat conservatively stated, "The epidemiological and evolutionary consequences, for both virus and vector, invite further attention… ."

Since this report, the effects of virus-infected plants on aphid vectors have been investigated in several systems. Positive (Ajayi, 1986; Araya and Foster, 1987; Baker, 1960; Costa et al., 1991; Ellsbury et al., 1985; Fereres et al., 1989; Hodgson, 1981; Jiménez-Martínez et al., 2004b; Markkula and Laurema, 1964; McIntyre et al., 1981; Srinivasan et al., 2008), neutral (Hodgson, 1981; McIntyre et al., 1981), and negative (Donaldson and Gratton, 2007; Hodge and Powell, 2008; Jiménez-Martínez and Bosque-Pérez, 2009) effects of virus-infected plants on aphid life history have been reported. Virus-infected host plants can also result in increased production of alate (winged) forms in aphids (Blua and Perring, 1992a; Gildow, 1980; Hodge and Powell, 2010; Montllor and Gildow, 1986). These virus-related changes in vector biology have implications for virus spread, which depends upon the abundance and mobility of vectors.

In addition to life history, aphid behavior can also be influenced by the virus infection status of the host plant. Frequently, more aphids settle on infected plants than on noninfected ones (Ajayi and Dewar, 1983; Blua and Perring, 1992b; Eckel and Lampert, 1996; Fereres and Moreno, 2009; Fereres et al., 1999; Macias and Mink, 1969). For example, wheat (*Triticum aestivum* L.) plants infected with *Barley yellow dwarf virus* (BYDV) or *Cereal yellow dwarf virus* (*Luteoviridae*) are preferentially colonized or elicit preferential settling relative to noninfected wheat plants by several of the aphid species that transmit these viruses (Ajayi and Dewar, 1983; Jiménez-Martínez et al., 2004a; Medina-Ortega et al., 2009). As is the case for aphid life history, however, aphid behavioral responses to virus-infected plants

vary among and within pathosystems. In some pathosystems, infected plants do not affect aphid behavior. For example, Fereres et al. (1999) found that *Myzus persicae* (Sulzer) alighted with equal frequency and remained for equal amounts of time on soybean (*Glycine max* L.) infected with *Cucumber mosaic virus* (CMV) (*Cucumovirus: Bromoviridae*) and noninfected control plants. In other pathosystems, responses are complex, with evidence of attractiveness of virus-infected plants to aphids that is not associated with their sustained feeding and colonization by aphids (Carmo-Sousa et al., 2014; Mauck et al., 2010b). Within pathosystems, plant responses to virus infection, and associated aphid reactions, can vary with disease progression (Blua et al., 1994; Werner et al., 2009), with age of inoculation (Rajabaskar et al., 2013b), and among host species (Power, 1996), genotypes, or varieties (Jiménez-Martínez et al., 2004a; Rajabaskar et al., 2013a). Furthermore, there is recent evidence that aphid behavior in response to virus-infected plants is altered after the aphid acquires the virus (Ingwell et al., 2012; Rajabaskar et al., 2014).

The potential interactions among aphid-transmitted plant viruses, their host plants, and vectors and associated species are summarized in Figure 1.1. Understanding the ecology, evolution, and potential applications of these interactions is facilitated by knowledge of the mechanisms that mediate them. This review of the chemical aspects of these interactions is intended to contribute to that understanding.

Chemical Factors Affecting Aphid Performance

Virus-infection-induced changes in plant nutritional quality

Plants infected with viruses have been reported to contain greater concentrations of amino acids in whole plant tissue (e.g., Ajayi, 1986; Markkula and Laurema, 1964; McMenemy et al., 2012) and in extruded phloem sap (Blua et al., 1994). Mauck et

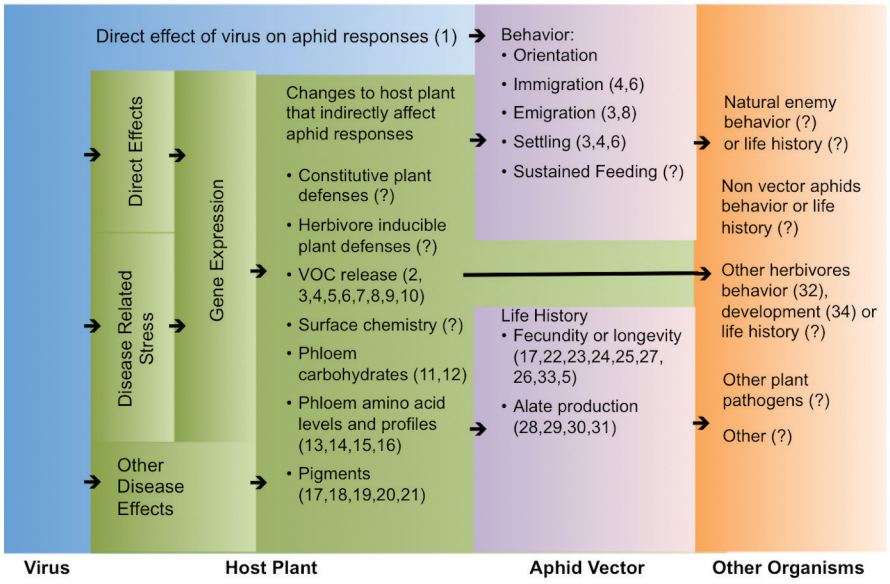

Fig. 1.1. Chemical ecology of plant–virus–aphid interactions. This schema summarizes the effects viruses can have on the chemical ecology of their host plants and the aphids that transmit viruses. These include the mechanisms whereby viruses can affect aphid vectors directly upon acquisition or indirectly through the host plant. Effects on the host plant can be indirect as a result of stress and disease reactions or direct through virus influence on specific gene-expression patterns within the plant. Effects on host plant chemistry can affect the life history or behavior of the aphid. Finally, other organisms that either compete with or prey on aphid vectors as well as other plant pathogens can potentially be influenced by virus-induced changes in host plant chemistry or indirectly through effects of plants on aphid behavior and life history. VOC = volatile organic compound. Publications reviewed in this chapter and relevant to each type of effect are indicated on this figure as follows: 1, Ingwell et al., 2012; 2, Alvarez et al., 2007; 3, Eigenbrode et al., 2002; 4, Jiménez-Martínez et al., 2004a; 5, Mauck et al., 2010b; 6, Medina-Ortega et al., 2009; 7, Ngumbi et al., 2007; 8, Srinivasan et al., 2006; 9, Rajabaskar et al., 2013a; 10, Rajabaskar et al., 2013b; 11, Fereres et al., 1990; 12, Feibig et al., 2004; 13, Ajayi, 1986; 14, Blua et al., 1994; 15, Blua and Perring, 1992b; 16, Markkula and Laurema, 1964; 17, Ajayi and Dewar, 1983; 18, Döring and Chittka, 2007; 19, Irwin and Thresh, 1990; 20, Macias and Mink, 1969; 21, Shimura et al., 2011; 22, Araya and Foster, 1987; 23, Baker, 1960; 24, Costa et al., 1991; 25, Ellsbury et al., 1985; 26, Jiménez-Martínez et al., 2004b; 27, Blua and Perring, 1992a; 28, Coon and Pepper, 1968; 29, Gildow, 1980; 30, Hodge and Powell, 2010; 31, Montllor and Gildow, 1986; 32, Mauck et al., 2010a; 33, Castle and Berger, 1993; 34, Carmo-Souza et al., 2014; 35, Casteel et al., 2015; 36, de Oliveira et al., 2014; 37, Kersch-Becker and Thaler, 2014; 38, Mauck et al., 2014; 39, Mauck et al., 2015a; 40, Mauck et al., 2015b; 41, Salvaudon et al., 2013; and 42, Wu et al., 2014. ? = Hypothesized or potential effects have not been reported or studied experimentally. The cited works pertain only to aphid-transmitted viruses, but similar ones have been observed or are possible in viruses dependent upon other vectors. Research on the direct and indirect effects of plant viruses on plant characteristics is beyond the scope of this chapter, so references are not provided for these effects. (© APS)

al. (2014) found that CMV infection disrupted amino acid profiles in *Cucurbita pepo* L. Since nitrogen is limiting for phloem-feeding insects (Douglas, 1993), these increased amino acids in phloem sap may explain improved aphid performance (growth and reproduction) on plants infected with some viruses. There is, however, no definitive evidence that improved aphid performance on virus-infected plants results from changes in amino acids in phloem. Indeed, amino acid composition appears in general not to affect aphid performance greatly (e.g., Weibull and Melin, 1990), evidently because of the capacity of endosymbionts to compensate for amino acid imbalances (Douglas, 1998; Hansen and Moran, 2011; Sandström and Moran, 1999). Furthermore, individual amino acids respond differently to virus infection, increasing or decreasing in concentration or remaining unaffected, with complex implications for aphid performance (Blua et al., 1994; Fiebig et al., 2004).

There is also evidence that virus infection increases soluble carbohydrate concentrations in whole plant tissue in BYDV-infected barley (*Hordeum vulgare* L.) plants (Fereres et al., 1990) and in *C. pepo* infected by CMV (Mauck et al., 2014), but carbohydrates are not considered limiting for aphids (Chapman, 1998). Furthermore, other work indicates a decrease in soluble carbohydrates in the phloem of wheat plants infected with the MAV isolate of BYDV (Fiebig et al., 2004).

Greater production of alates on virus-infected plants (Blua and Perring, 1992a; Gildow, 1980, 1983; Hodge and Powell, 2010) may also be related to virus-induced changes in plant nutritional quality, but evidence on this point is inconclusive. Alate production is associated with reduced nutritional quality and crowding within aphid colonies but also with intrinsic clocks and external factors such as day length (Braendle et al., 2006; Dixon, 1998; Muller et al., 2001). Hodge and Powell (2010) found that alate production of the pea aphid, *Acyrthosiphon pisum* (Harris), increased on *Pea enation mosaic virus* (*Enamovirus*)-infected pea (*Pisum sativum* L.) but only in combination with enhanced crowding that occurred within the clip cages used in their bioassays. Alate production by *Rhopalosiphum padi* (L.) was inconsistently associated with virus infection status of its host plant (Fiebig et al., 2004). Thus, it appears that the effects of virus infection on alate production are complex and depend upon context.

How virus infection influences the nutritional quality of plants is not well understood. Plant stress in general can impair protein synthesis, increasing amino acid concentrations (Brodbeck and Strong, 1987), but idiosyncratic effects on individual amino acids (Blua and Perring, 1992a; Fiebig et al., 2004) indicate that there are specific effects of virus infection on amino acid biosynthetic pathways yet to be elucidated.

Virus-infection-induced changes in plant defenses

Changes to plants after infection, including their chemical and physical defenses, are also important. Plants possess several transduction pathways inducible in response to herbivory, pathogen attack, and abiotic stresses, leading to changes in plant defensive chemistry (De Vos et al., 2007; Holopainen and Gershenzon, 2010; Walling, 2000). The jasmonic acid (JA)-dependent pathway typically is activated by herbivore feeding and physical stress, while the salicylic acid (SA)-dependent pathway typically is activated by pathogen attack. These pathways can interact negatively or positively, a phenomenon known as "cross-talk" (Bostock, 2005; Bostock et al., 2001; Rodriguez-Saona et al., 2005; Spoel et al., 2003; Stout et al., 2006) such that the net effects on plant defenses can depend upon the types of attackers acting simultaneously. In response to feeding by phloem-feeding insects, both JA-dependent and SA-dependent pathways can be induced, with complex implications for resulting induced defenses (Kaloshian and Walling, 2005; Walling, 2008). In a few cases, the metabolic pathways or specific chemical defensive factors induced are known (De Vos et al., 2007; Kim et al., 2008; Pieterse and Dicke, 2007; Pontoppidan et al., 2003; Smith and Boyko, 2007; Walling, 2009), and these seem to be predominantly JA-dependent. Through negative cross-talk, virus infection may suppress inducible defenses that otherwise limit aphid performance on their host plants. SA-dependent induction pathways that can be triggered by virus infection, e.g., *Turnip crinkle virus* (*Carmovirus: Tombusviridae*) in *Arabidopsis thaliana* (L.) Heynh. (Koornneef and Pieterse, 2008), are associated with attenuation of the JA-mediated defenses against insects. Lewsey et al. (2010) reported that a protein encoded by CMV when expressed in Arabidopsis suppresses 90% of the genes regulated by JA, an effect that could compromise a plant's capacity to defend against insects, including aphids. *Turnip mosaic virus* (TuMV) infection suppresses callose deposition in Arabidopsis, and this effect has been linked to the ethylene signaling pathway in the plant, accounting for suppression of an infected plant's defense against aphids (Casteel et al., 2015). Alternatively, positive cross-talk could lead to virus-induced elevation in defenses effective against aphids, potentially explaining the reduced performance of aphids on virus-infected plants in some pathosystems, e.g., the *C. pepo*–CMV–aphid (*M. persicae* and *Aphis gossypii* Glover) (Mauck et al., 2010b); potato (*Solanum tuberosum* L.)–*Potato virus Y* (PVY) (*Potyvirus: Potyviridae*)–*M. persicae* (Castle and Berger, 1993); and soybean–*Alfalfa mosaic virus* (*Alfamovirus: Bromoviridae*) and *Bean pod mottle virus* (*Comovirus: Comoviridae*)–*Aphis glycines* Mastumura (Donaldson and Gratton, 2007) pathosystems. The specific defenses involved in each of these cases are unknown.

Chemical Factors Affecting Aphid Behavioral Responses to Virus-Infected Plants

Visual cues influencing settling or colonization

Aphids use visual cues during host selection, at least as a basis for alighting on potential hosts (Döring and Chittka, 2007; Fereres et al., 1999; Kennedy et al., 1961; Kring, 1972). Although animal responses to plant color typically are not included in chemical ecology, we include them here because of their importance for aphids and because, to a large degree, plant color depends upon the pigments present in plant tissues. Colors with relatively strong reflectance in longer wavelengths (approximately 520–580 nm), appearing yellow to humans, elicit dropping of aphids from the air column in controlled wind-tunnel experiments and field studies (Döring and Chittka, 2007; Irwin and Thresh, 1990). Yellow pan traps are more effective than other colors at trapping aphids in the field and are widely used for this purpose (Coon and Pepper, 1968). Since virus-infected plants often appear yellow, this has long been considered a potential basis for aphids preferentially settling on virus-infected vs. non-infected plants (Ajayi and Dewar, 1983; Macias and Mink, 1969). Aphids evidently integrate spectral information rather than re-

sponding to specific wavelengths (Döring and Chittka, 2007) and so potentially respond to complex color cues that might be associated with virus infection. There is, however, little evidence that aphids discriminate among hosts on the basis of color in general (Fereres and Moreno, 2009; Kennedy et al., 1961) and no definitive studies showing that the color of virus-infected plants alone influences aphid settling behavior. For example, flight tunnel experiments that suggest visual attraction of *Sitobion avenae* and *Metopolophium* (reported as *Macrosiphum*) *dirhodum* to BYDV-infected wheat plants (Ajayi and Dewar, 1983) did not control for the potential effect of volatile cues on aphid behavior.

The mechanisms whereby virus infection influences plant color are poorly understood. Yellowing is widely associated with virus symptoms, presumably related to infection-induced plant stress, accelerated senescence, and direct injury to tissues that deplete chlorophyll and reveal other phytopigments. However, Shimura et al. (2011) demonstrate that a virus satellite RNA associated with CMV directly affects a chlorophyll biosynthesis gene in *Nicotiana tabacum* L., partly accounting for the yellow symptoms in this infection. Thus, the basis for yellowing of some virus-infected plants is potentially caused by direct interactions between the virus and the host genome.

Virus-induced volatiles and aphid responses

In contrast to the uncertainty about the relative importance of visual cues, the role of volatile organic compounds (VOCs) in aphid discrimination based on virus infection status of the plant has been demonstrated in several pathosystems. In bioassays conducted in darkness in order to eliminate visual cues, aphids preferentially settle on infected vs. noninfected plants (Alvarez et al., 2007; Castle et al., 1998; Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a; Srinivasan et al., 2006). This discrimination remains even if aphids are separated from the plants by a screen preventing access to gustatory or tactile cues (Alvarez et al., 2007; Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a; Mauck et al., 2010b; Medina-Ortega et al., 2009; Srinivasan et al., 2006), implicating virus-induced volatiles (VIVs) as the active cues. Discrimination based on VOCs is consistent with the established role of VOCs in aphid host selection (De Vos and Jander, 2010; Jones, 1944; Medina-Ortega et al., 2009; Ngumbi et al., 2007; Nottingham et al., 1991; Pettersson et al., 1996; Pickett and Glinwood, 2007; Pickett et al., 1992; Visser et al., 1996) and evidence that pathogens alter VOC release by plants (Cardoza et al., 2002; Holopainen and Gershenzon, 2010; Huang et al., 2003; Preston et al., 1999).

The initial discovery that aphids respond differently to VOCs from virus-infected and noninfected plants was made in the potato–*Potato leafroll virus* (PLRV) (*Polerovirus: Luteoviridae*)–*M. persicae* pathosystem (Eigenbrode et al., 2002). *M. persicae* is the principal vector of PLRV (Harrison, 1984). Prior work (Castle et al., 1998) demonstrated that apterous *M. persicae* preferentially settled upon leaflets of intact potato plants (cultivar Russet Burbank) infected with PLRV compared with those infected with two other viruses, *Potato virus X* (PVX) (*Potexvirus: Potexviridae*) and *Potato virus* (PVY) or noninfected plants. Castle et al. (1998) used bioassays in which aphids moved freely among several potato leaflets attached to virus-infected or control plants and positioned in contact with a common platform. Within 12 h (during scotophase), *M. persicae* apterae preferentially settled on PLRV-infected leaves compared with other treatments (Castle et al., 1998).

Using a bioassay similar to that employed by Castle et al. (1998), Eigenbrode et al. (2002) showed that preferential settling by *M. persicae* on leaflets of PLRV-infected potato cultivar Russet Burbank compared with sham-inoculated controls (exposed to feeding by aphids that were not carrying the virus) or PVY- and PVX-infected plants was detectable within 1 h of initiating the bioassay, showing that the response was relatively rapid and evidently did not require sustained feeding. Differential settling by the aphids could be detected in a dual-choice version of this bioassay conducted in darkness (Fig. 1.2A). When a fine screen was employed to prevent aphids from contacting the leaf surfaces, aphids again settled preferentially over PLRV-infected leaflets compared with other treatments (Fig. 1.2B), indicating that volatile cues were involved. To establish the role of VOCs, headspace VOCs from PLRV-infected and sham-inoculated potato plants were trapped onto SuperQ resin and eluted for testing in bioassays. VOCs from headspace of PLRV-infected plants applied to paper models of leaves elicited

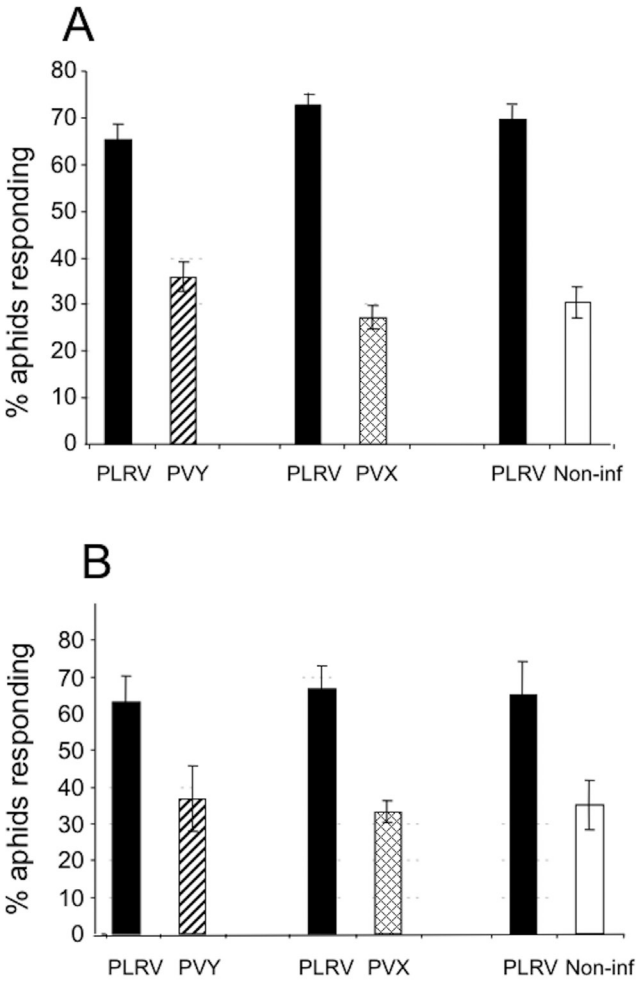

Fig. 1.2. Settling behavior of *Myzus persicae* in dual-choice experiments. **A,** Leaflets of plants infected with *Potato leaf roll virus* (PLRV), *Potato virus X* (PVX), or *Potato virus Y* (PVY) and noninfected controls. **B,** The same pairs of treatments with screen preventing aphids from contacting the plants and conducted in darkness to eliminate visual cues. Data are the number of aphids settling on the leaflets (A) or above the leaflets on the screen (B) after 1 h. All bioassays were dual-choice tests. Differences between the pairs are significant based on a *t*-test ($P < 0.01$) for all tests. (Redrawn from Eigenbrode et al., 2002)

greater settling than did paper models treated with VOCs from headspace of sham-inoculated controls (data not shown). This was true whether VOCs were applied in leaf equivalents (representing the different concentrations found in headspace of infected and noninfected plants; see below) or at equal concentrations, suggesting that the cue depended upon both quantitative and qualitative changes in the headspace.

To facilitate statistical comparisons among several treatments, an emigration bioassay measured the rate at which aphids move away from leaves or models placed under the screen (emigration) (Eigenbrode et al., 2002). The focus on emigration was consistent with observations indicating that arrestment rather than attraction contributed to the greater settling over infected leaflets in the potato–PLRV–*M. persicae* pathosystem (Eigenbrode et al., 2002). For this bioassay, 30 late-instar apterous aphids were placed on a screen directly above the target (living leaf or paper model) and the number emigrating was measured at intervals. The data were fitted to an exponential function from which an emigration rate was calculated and compared among treatments by using analysis of variance. Emigration rate from PLRV-infected leaflets was significantly lower than emigration rates from other treatments (Eigenbrode et al., 2002).

Subsequently, similar effects were detected in PLRV–*M. persicae* pathosystems with different host plants: potato cultivar Kardal (Alvarez et al., 2007), the noxious weed hairy nightshade, *Solanum sarrachoides* Sendtn. (Srinivasan et al., 2006), a genotype of *Solanum nigrum* L. (E. Ngumbi, S. D. Eigenbrode, H. Ding, and N. A. Bosque-Pérez, *unpublished data*), and a genotype of *Nicotiana benthamiana* Domin. (S. D. Eigenbrode, A. Karasev, and J. Kuhl, *unpublished data*). The results suggest that the phenomenon is robust and general for PLRV and its vector, *M. persicae*.

A similar phenomenon potentially occurs in the wheat–BYDV–*R. padi* pathosystem. In prior work, Ajayi and Dewar (1983) recorded greater populations of *S. avenae* and *M. dirhodum* in plots of BYDV-infected wheat, barley, and oats (*Avena sativa* L.) than in noninfected plants in the field during two seasons. In a wind tunnel bioassay, these authors detected greater settling onto leaves of infected barley and oats by alates of both aphid species.

To examine the basis for these effects, Jiménez-Martínez et al. (2004a) studied the response of *R. padi* to BYDV-infected wheat plants (cultivar Lambert). For bioassays, groups of 40 apterae were placed equidistant from sets of leaves of each of two treatments (virus-infected or sham-inoculated) positioned approximately 75 mm apart. Aphids could contact these leaves, but the bioassay was conducted in the dark to eliminate visual cues. The locations of aphids were monitored every 10 min for 2 h using a red light. The bioassay confirmed a significant preference for BYDV-infected plants. In a similar bioassay, but with a screen to prevent aphids from contacting the leaves (as in the PLRV bioassay described above), approximately twice as many *R. padi* apterae were found near the infected leaves of the cultivar Lambert compared with noninfected or sham-inoculated plants, implicating VIVs as the active cues (Jiménez-Martínez et al., 2004a).

For the wheat–BYDV–*R. padi* pathosystem, immigration rather than emigration bioassays better detected aphid responses (Medina-Ortega et al., 2009). Immigration assays were performed by placing 30 aphids per treatment approximately 70 mm away from the center and on one side of the arena. Wheat leaves or paper models were placed under the screen approximately 50 mm from the center of the arena on the side of the arena opposite the aphids. Aphids observed directly above the leaves were considered immigrants and removed from the arena at each observation and counted. In these bioassays, *R. padi* immigration rates were greater to wheat plants infected with the PAV strain of BYDV than to sham-inoculated controls, but unlike *M. persicae* in the PLRV pathosystem, emigration rates from virus-infected and noninfected plants did not differ (Medina-Ortega et al., 2009). The precise behavioral basis for the differential behavior of *R. padi* in response to BYDV-infected plants and *M. persicae* in response to PLRV-infected plants has yet to be determined.

The third example in which aphid responses to VIVs have been detected is the *C. pepo*–CMV pathosystems, with two aphid species, *M. persicae* and *A. gossypii*. Using a bioassay in which aphids were separated from *C. pepo* leaves by a screen, Mauck et al. (2010b) showed that apterae of each of these aphid species were attracted to VIVs from CMV-infected plants, but after the initial colonization, they dispersed to preferentially colonize virus-free plants rather than infected ones. An olfactometer was used in the bioassay to demonstrate that the aphid responses were to plant volatiles. Since CMV can be acquired within seconds by a probing aphid, the initial attraction followed by dispersal should enhance the spread of this virus and could represent an adaptive, two-part "deception" of vectors by CMV (Mauck et al., 2010b).

One other prior study sought evidence for effects of VOCs from infected plants on aphids. Fereres et al. (1999) found that *M. persicae* and *Rhopalosiphum maidis* (Fitch) were unresponsive to volatiles from soybean plants infected with *Soybean mosaic virus* (*Potyvirus: Potyviridae*). This study did not use VOCs from intact plants, however, but VOCs from whole-plant extracts, so its ecological relevance is uncertain.

Bioassay methods. The arenas for bioassays employed to examine the chemical ecology of aphid responses to virus-infected plants are depicted in Figure 1.3. The general approach is to bioassay individual aphids or groups of aphids by placing them at intermediate positions between or among treatments and allowing them to settle. Often the method allows aphids to acclimate after being introduced and before encountering stimuli. For example, in the method used by Castle et al. (1998) and in adaptations of this method (e.g., Mauck et al., 2010b), aphids climb a rod before reaching a platform or screen on which they can select among treatments. A modification of this approach (Srinivasan et al., 2006) allowed the aphids to climb to the platform within a tube to eliminate the need to negotiate a transition from the lower to the upper surface of the platform. Aphids can be placed in small depressions or within vials that can be removed immediately before the bioassay, allowing them to move.

All of the studies focusing on VOCs have employed bioassays in static air. Although aphids are able to respond to odors in moving-air devices such as Y-tube olfactometers (Pettersson, 1970; Visser and Piron, 1998), the still-air bioassay may better approximate conditions for apterous aphids walking among potential host plants. In these bioassays, the aphids are separated from the source (living plant material and individual or mixtures of VOCs) by a screen that prevents contact with the plant surface. The screen and source can be placed so that the aphids walk on its lower surface with the leaf material or odor source above them (Mauck et al., 2010b) or so that the aphids

walk on its upper surface with leaf material or odor source below (Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a; Ngumbi et al., 2007; Medina-Ortega et al., 2009; Werner et al., 2009). In a third approach, the source and screen are positioned above a plastic walking platform on which their behavior can be observed (Alvarez et al., 2007). Similar methods have been used to test the activity of trapped headspace VOCs (Eigenbrode et al., 2002), pure compounds (Medina-Ortega et al., 2009; Ngumbi et al., 2007), or synthetic blends (Ngumbi et al., 2007) dissolved in mineral oil and applied to paper models. The bioassays to study responses to VIVs from living plants or trapped or synthetic headspace VOCs have typically been carried out in darkness to eliminate visual cues, requiring use of red light to make the observations (Alvarez et al., 2007; Jiménez-Martínez et al., 2004a; Ngumbi et al., 2007). Alternatively, a screen that is sufficiently opaque to prevent detection of visual cues from the plants by the aphids can be employed to ensure that the plants are receiving sufficient photosynthetically active radiation to produce VOCs at relevant rates (Mauck et al., 2010b).

Studies of VOCs have either used choice tests, in which aphids are presented leaves of virus-infected plants and sham-inoculated controls (Alvarez et al., 2007; Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a), or consisted of bioassays to quantify immigration toward a source or emigration away from a source of VOCs. The appropriate type of bioassay appears to differ depending upon the pathosystem. For example, *M. persicae* individuals do not exhibit strong immigration responses to odor sources while walking on a screen, but *R. padi* individuals do. In either case, it has proved possible to model aphid immigration or emigration on the basis of observations performed at intervals, fit these models to exponential (Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a; Medina-Ortega et al., 2009; Werner et al., 2009) or linear (Alvarez et al., 2007) functions, and compare the slopes as estimates of immigration rates in response to treatments.

Bioassays with alate aphids, in which a type of wind tunnel is used that maintains aphids in a stationary position within an air column and the compensatory air flow is used as a measure of the aphid's tendency to continue flying, have been conducted to study host selection by aphids (Kennedy and Booth, 1963; Nottinghham and Hardie, 1993). In one instance, this type of apparatus was used to test aphid responses to infected hosts (see Ajayi and Dewar, 1983), but it has not been used to decipher responses to chemical or visual cues from infected plants in iso-

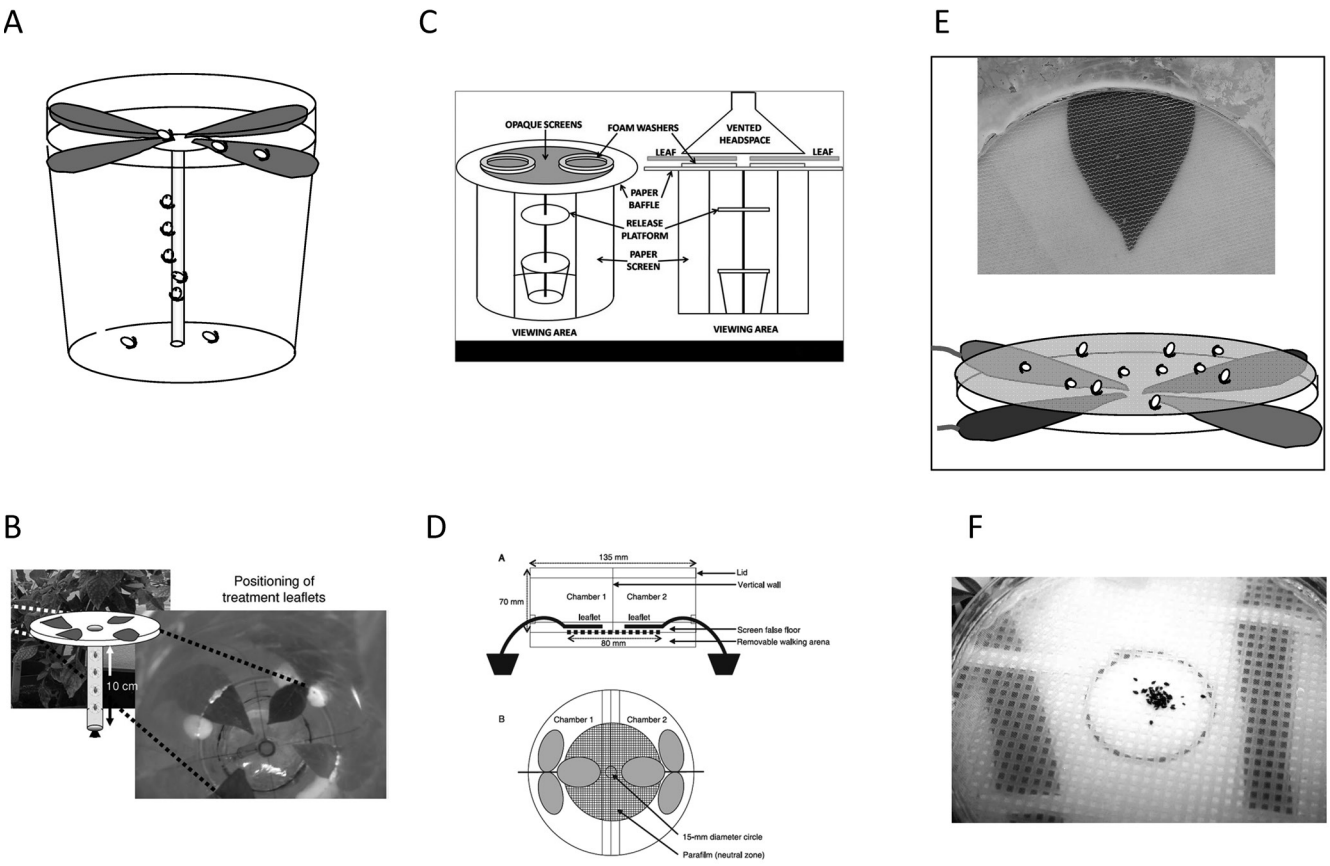

Fig. 1.3. Approaches to testing effects of virus-induced volatiles on aphid behavior. **A,** Bioassay in which aphids climb a rod to gain a platform in contact with leaves from host plants differing in infection status. **B,** Modification of A in which aphids climb within a tube to gain the platform. **C,** Modification of A for a dual-choice bioassay in which aphids walk on a screen beneath leaves from plants in different treatments. The screen is opaque to eliminate visual cues, and the cone provides ventilation to prevent buildup of headspace volatile organic compounds (VOCs) in the arena. **D,** Dual-choice arena in which aphids walk on a platform beneath leaves that are separated from the bioassay chamber by a screen. Upper chamber is divided to minimize mixing of VOCs from treatments. **E,** Aphids on a screen above test leaves, as used for dual-choice and emigration or immigration bioassays (Eigenbrode et al., 2002; Ngumbi et al., 2007; Werner et al., 2009). Upper panel, *M. persicae* on screen above a potato leaflet (photo courtesy B. Werner); lower panel, arrangement with four leaves. **F,** Dual-choice test with *Rhopalosiphum padi* and wheat. (A, redrawn from Castle et al., 1998; B, adapted, by permission of the publisher, from Srinivasan et al., 2006; C, adapted, by permission of the publisher, from Mauck et al., 2010b; D, reproduced, by permission of the publisher, from Alvarez et al., 2007; F, reproduced, by permission of the publisher, from Jiménez-Martínez et al., 2004a, and Medina-Ortega et al., 2009)

lation. In other work to measure responses of winged aphids to virus-infected plants, the insects were either placed on a platform from which they could disperse to host plants (Fereres et al., 1999; Medina-Ortega et al., 2009; Srinivasan et al., 2006) or provided the opportunity to move among host plants in different treatments within a cage (Mauck et al., 2010b). There is a need to devise better bioassays to measure alate responses to virus-infected plants in order to examine their capacity to discriminate on the basis of visual, olfactory, and gustatory cues.

All of the published bioassay methods in which plant material was used have ensured that leaves or leaflets of the plants remain attached to plants during bioassays to avoid potential confounding effects of plant responses to injury after removal and effects of acceptability of the plants for aphids following disruption of phloem pressure.

Characterizing VIVs. In some systems, headspace VOCs from virus-infected and noninfected plants have been compared chemically (Eigenbrode et al., 2002; Jiménez-Martínez et al., 2004a; Mauck et al., 2010b; Werner et al., 2009). In these studies, virus infection is consistently associated with greater overall concentration of VOCs in headspace and some shifts in relative concentrations of individual VOCs. In no case have unique compounds been detected in headspace of infected plants, so the effect can be regarded as quantitative rather than qualitative. For example, the headspace from PLRV-infected potatoes 4 weeks after inoculation contained nearly double (1.9 fold) the concentration of total components detectable by gas chromatography–mass spectrometry compared with the headspace of noninfected controls or sham-inoculated controls (Eigenbrode et al., 2002). Compounds with substantial increases following PLRV infection included green leaf volatiles, monoterpenes, short-chain alcohols and aldehydes, alkanes, and sesquiterpenes. On the basis of nonoverlapping standard errors, PLRV-infected plants produced higher concentrations of 14 of the 21 components detected, ranging from 1.6 fold (β-sesquiphellandrene) to fivefold (2-hexen-1-ol) relative to noninfected plants. The relative composition of the blend also was affected. For example (E)-2-hexen-1-ol was elevated nearly sevenfold, while cubebene was essentially unchanged by PLRV infection (Eigenbrode et al., 2002). In contrast, plants infected with PVY and PVX as part of the same study exhibited increases in just a few compounds: both caused increases in (E)-2-hexen-1-ol, PVY increased myrcene, and PVX increased nonane (Eigenbrode et al., 2002).

For BYDV-infected wheat (cultivar Lambert), the overall concentration of headspace VOCs increased approximately threefold and all compounds were elevated to some degree (Jiménez-Martínez et al., 2004a). (Z)-3-Hexenyl acetate was elevated more than threefold and nonanal approximately sevenfold, while dodecane was elevated just 1.5 fold and caryophyllene twofold in BYDV-infected wheat compared with noninfected plants (Jiménez-Martínez et al., 2004a).

Infection of *C. pepo* by CMV elicited a general increase in all 38 VOCs detected in plant headspace compared with controls (sham-inoculated plants) (Mauck et al., 2010b). Most compounds were increased by CMV infection, but some (e.g., (E)-β-ocimene and methyl benzoate) tended to be reduced in infected-plant headspace. The authors concluded that the overall effect of CMV infection was to increase the release of a blend of VOCs that was qualitatively similar to that of virus-free plants, thereby providing a stronger stimulus to aphids engaged in host finding (Mauck et al., 2010b), but whether the differences in relative concentrations contributed to the aphid responses was not tested.

We are not aware of any other published studies that have reported on the entire VOC blend of virus-infected plants, although work is ongoing in several laboratories at the time of this writing. Preston et al. (1999) detected an increase in methyl salicylate from *Tobacco mosaic virus* (*Tobamovirus*)-infected *N. tabacum* plants but did not examine other VOCs in headspace of the infected plants.

Aphid responses to individual VOCs and VIV blends. Two studies have examined aphid responses to individual VOCs and synthetic blends of these compounds that had previously been determined to be involved in aphid responses. Ngumbi et al. (2007) found that a synthetic blend mimicking VIVs from PLRV-infected potato plants elicited arrestment by *M. persicae,* whereas individual compounds comprising this blend did not elicit a response. The tested compounds were electrophysiologically active, as determined by electroantennography; each elicited significant depolarization of intact aphid antennae when applied to the antenna at ecologically relevant concentrations (Ngumbi et al., 2007). Removing any one of the components of the blend or any class of compounds (green leaf volatiles, monoterpenes, and sesquiterpenes) eliminated or strongly reduced the behavioral activity of the blend (E. Ngumbi, S. D. Eigenbrode, H. Ding, and N. A. Bosque-Pérez, *unpublished*). The result was confirmed by using synthetic blends and trapped natural blends separated by fractionating gas chromatography (Fig. 1.4). Thus, the VOC blend from PLRV-infected plants is critical for eliciting the observed response from aphids, as has been reported for many other arthropod–plant interactions (D'Alessandro and Turlings, 2006; Dickens, 2000; van Wijk et al., 2008; Zhang et al., 1999).

For the wheat–BYDV–*R. padi* pathosystem, a synthetic blend of five of the compounds most strongly elevated in the headspace of BYDV-infected wheat plants (nonanal, (Z)-3-hexenyl acetate, decanal, caryophyllene, and undecane) applied to paper leaf models was more attractive to *R. padi* apterae than a synthetic blend of the same VOCs tested at a concentration and in ratios representative of headspace of noninfected plants (concentration approximately one-half that of the infected blend mimic) (Medina-Ortega et al., 2009). Each of these compounds was attractive individually to the aphids in a bioassay, but a behavioral dose response was not detected across a range of concentrations bracketing ecologically relevant ones. Thus, the effect of concentration on the aphid response was detected for these compounds only when tested together as a blend (Medina-Ortega et al., 2009). The differential responses of *R. padi* to BYDV-infected wheat plants require further research but, as in the potato–PLRV–*M. persicae* system, is evidently dependent upon the blend of VOCs from the infected plants.

Some aphid species respond behaviorally to specific VOC classes or individual VOC characteristic of their host plants (Chapman et al., 1981; Dilawari and Atwal, 1989; Hardie et al., 1994; Nottingham et al., 1991), but such reports remain rare. It appears likely that many aphids respond to VOC blends from hosts during host selection. Most of the VOCs in these blends, and those investigated as components of VIVs, are widespread or ubiquitous in plant headspace. For example, Webster et al. (2008, 2010) have shown that certain blends of widely occurring plant headspace VOCs are attractive for *A. fabae*. In another system (aphids in the genus *Neuquenaphis* on its host trees, *Nothofagus* spp.), several compounds were found to be active

for aphid discrimination among hosts, but the ratios of these compounds in blends were important (Quiroz et al., 1999; Russell et al., 2004). Theory suggests that single volatile compounds (or other chemical cues) are more likely to be used by specialist insects, whereas blends may be more important for generalist insects (Bernays and Chapman, 1994; Egan and Funk, 2006; Vargas et al., 2005), such as the aphids that have been studied for their responses to virus-infected plants. It is possible that pathosystems in which the vectors are specialists involve specific virus infection-induced cues.

Chemical cues other than VIVs in aphid responses to virus-infected plants

Although most published work has focused on VOCs (VIVs), during host selection, aphids have access to multiple cues from infected plants, including gustatory cues, other chemicals detectable on contact, and postingestive cues. In the *Luteoviridae* systems, responses of the aphids in bioassays isolating the effect of VOCs and in those that permit aphids to assess plants using multiple cues are similar (Castle et al., 1998; Eigenbrode et al., 2002; Ingwell et al., 2012; Jiménez-Martínez et al., 2004a; Srinivasan et al., 2006), so although the contributions of these other cues may be negligible, they are unknown. In the *C. pepo*–CMV system (Mauck et al., 2010b), although both *M. persicae* and *A. gossypii* were attracted or arrested by VOCs from CMV-infected plants, after contact with the plants the aphids dispersed more rapidly from infected plants, indicating that cues accessible after contact or feeding were deterrent or repellent. As additional data accumulate, a range of complex responses to the several cues from infected plants is likely to be discovered.

To detect activity of other types of cues in a PLRV pathosystem, Ngumbi et al. (2007) examined the importance of VOCs in the response of *M. persicae* to PLRV-infected plants using antennectomized aphids. Antennae were severed just above the second segment in order to remove all rhinaria (odor-detecting

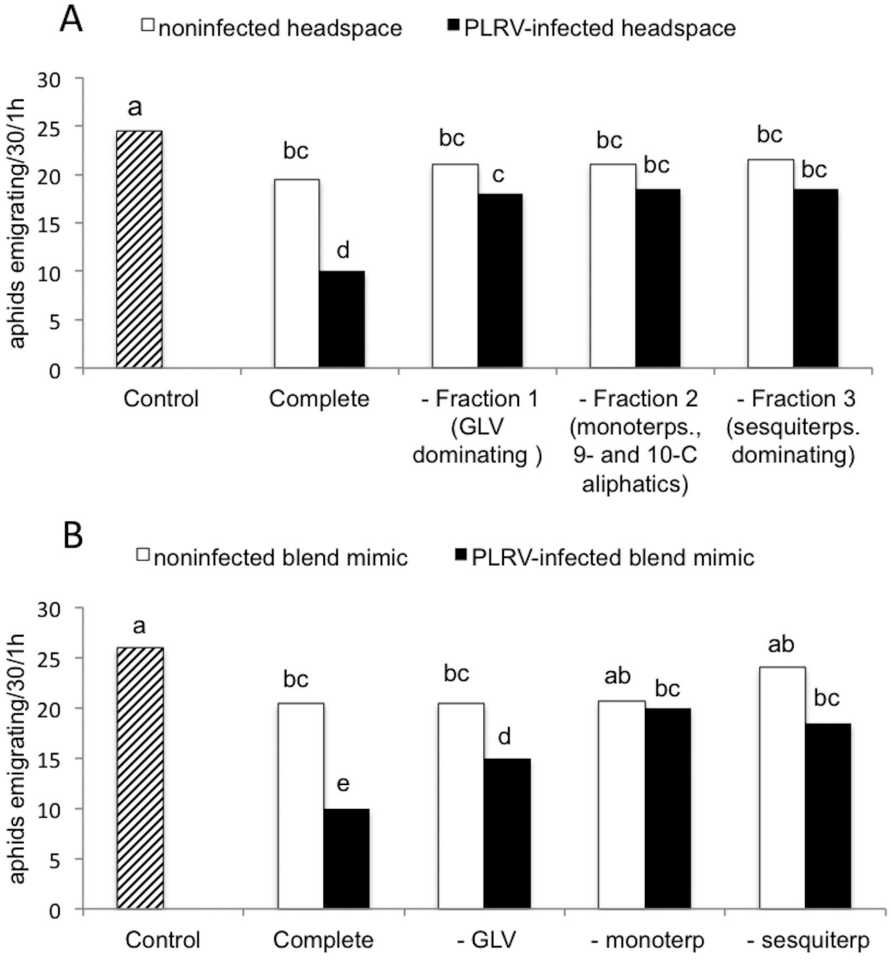

Fig. 1.4. Responses of *Myzus persicae* to intact blends and partial blends of headspace volatile organic compounds (VOCs) from *Potato leaf roll virus* (PLRV)-infected plants and sham-inoculated controls. Bioassays were conducted by applying materials to paper leaf models and testing for emigration in arenas as shown in Figure 1.3E. **A,** Trapped headspace VOCs from PLRV-infected plants and sham-inoculated controls. VOCs of intact blends and blends with fractions were removed using preparative gas chromatography. Predominant compounds in removed fractions are indicated. **B,** Synthetic blend composed of principal components in headspace VOCs and prepared to mimic concentrations found in headspace of the plants. Fractions were also prepared with key VOC classes removed as indicated. In both A and B, data are the number of aphids emigrating from directly above the source over a 1-h period. Columns with the same letters are not significantly different. GLV = green leaf volatiles. (E. Ngumbi, S. D. Eigenbrode, H. Ding, and N. A. Bosque-Pérez, *unpublished data;* © APS)

organs), while sham-operated aphids had removed only the final few segments, which are not known to carry olfactory sensilla. The aphids were allowed to recover after surgery until they began feeding and reproducing. In a dual-choice bioassay, only the sham-operated aphids and not the antennectomized aphids discriminated between PLRV-infected and noninfected potato plants after 12 h (Fig. 1.5), indicating that VIVs alone are responsible for discrimination during initial settling by the aphids in this system.

Electronic feeding monitors have detected differences in the feeding behavior on infected vs. noninfected hosts. *Schizaphis graminum* (Rondani) and *R. padi* exhibited a shorter time to first contact of phloem and more consistent feeding after phloem contact on oat plants infected with a BYDV isolate compared with controls (Montllor and Gildow, 1986). Carmo-Sousa et al. (2014) showed that *A. gossypii,* the aphid vector of CMV, responded to cues or characteristics of CMV-infected cucumber (*Cucumis sativus* cv. Marumba) by altered feeding behavior as detected by electrical penetration graph (EPG). The aphids settled preferentially on the CMV-infected plants, but after feeding for a short period, they began to preferentially emigrate from these plants. During this time, EPG signals detected a decrease in active feeding on infected vs. uninfected plants. The basis for this response has not been determined, but it could indicate aphid behavioral responses to levels of nutrients or defenses present in the phloem. Alternatively, indirect effects of the infection on phloem accessibility could contribute to this. Since these effects were detectable soon after initiation of probing, it is possible that they contribute to settling responses of the aphids. More work is needed to examine these effects.

Variation in VIVs and other cues and aphid responses to these cues

Plants at a single stage of development or age after inoculation and aphids that are not carrying the viruses (nonviruliferous) were used in most of the bioassays described above. In the field, however, plants at various stages of infection and both viruliferous and nonviruliferous individual aphids will be present. Studies indicate that the effects of plant viruses on plants and aphids are dynamic. Blua and Perring (1992b) found that late-stage *Zucchini yellow mosaic virus* (*Potyvirus: Potyviridae*)-infected *C. pepo* (4 weeks after inoculation) were not recognized as hosts by *A. gossypii* alates, whereas plants 2 weeks after inoculation were colonized preferentially by alates over noninfected controls. Werner et al. (2009) detected greater arrestment of *M. persicae* on a screen above leaflets of PLRV-infected potato plants 4 and 6 weeks after inoculation than above plants 2, 8, or 10 weeks after inoculation. Headspace VOCs from infected plants also changed with disease progression in this study. The total concentration of headspace VOCs of PLRV-infected plants increased throughout the infection process relative to sham-inoculated controls. Most of this increase resulted from increasing sesquiterpenes, while green leaf volatiles increased only slightly. Monoterpenes from PLRV-infected plants peaked at 4 weeks after inoculation and declined to concentrations below those from sham-inoculated plants by 8 weeks after inoculation (Ngumbi et al., 2007). The abundance of sesquiterpenes later in the infection process (Werner et al., 2009) may render the blend from PLRV-infected plants less arrestant than controls, whereas during the middle stage of infection, when a greater proportion of monoterpenes is present, a more balanced blend that is more arrestant is present. This is consistent with evidence that the VOC blend from infected plants is required to arrest *M. persicae* emigration (Ngumbi et al., 2007). Given that PLRV disease symptoms visibly progress in severity with duration of infection, it may not be surprising that effects upon the aphid vector are also dynamic, with implications for virus spread (Blua and Perring, 1992b). Finally, Rajabaskar et al. (2013b) varied the time of inoculation of potato plants with PLRV from 3 to 5 weeks after transplant from tissue culture. Earlier inoculation dates elicited greater arrestment by *M. persicae* on PLRV-infected plants compared with noninfected plants, while the later inoculation date elicited an opposite response.

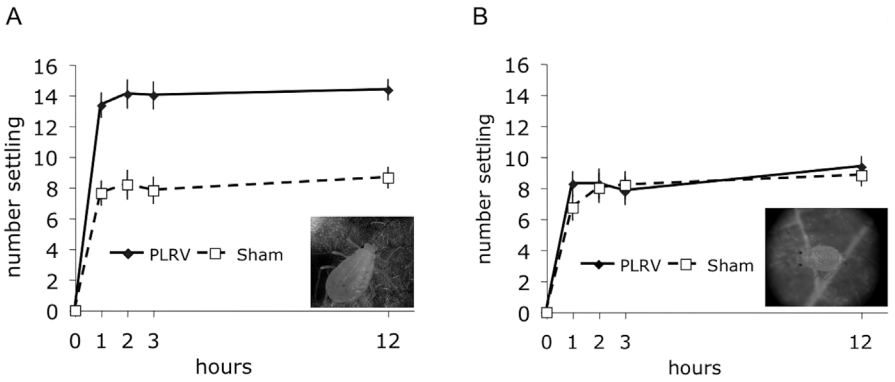

Fig. 1.5. Evidence that volatile organic compounds are critical for *Myzus persicae* discrimination between *Potato leaf roll virus* (PLRV)-infected plants and sham-inoculated controls on the basis of behavior of antennectomized aphids. Data are from two separate experiments, and each experiment utilized a dual-choice bioassay following the method of Castle et al. (1998). The data show the number of aphids settling on PLRV-infected plants or sham-inoculated controls at intervals up to 12 h. In the first experiment **(A),** aphids had the terminal segment of the antenna removed as a sham operation. In the second **(B),** aphids had the entire antenna distal to the second segment removed. The surgeries were performed with microelectronic wire cutters. Aphids were allowed to recover for 72 h after surgery before being used in the bioassays. Intact aphids settled more often on infected plants beginning at 1 h ($P < 0.001$), while antennectomized aphids did not discriminate between the PLRV-infected plants and sham-inoculated controls. (E. Ngumbi, S. D. Eigenbrode, H. Ding, and N. A. Bosque-Pérez, *unpublished data;* © APS)

These patterns in aphid behavior were related to differences in the VOC profile from the plants.

The effects of virus infection on behavior of vectors may also differ among positions within a single infected plant. This has been demonstrated in three studies in the PLRV–potato–*M. persicae* pathosystem. VOCs from youngest and oldest leaves of infected plants do not differ in attraction or arrestment of *M. persicae* compared with equivalent leaves from sham-inoculated plants, whereas VOCs from leaves from middle nodes of infected plants are more attractive than VOCs from comparable leaves of sham-inoculated plants (Alvarez et al., 2007; Werner et al., 2009). These patterns may be related to the spatial and temporal dynamics of virus titer within the infected plant or to relative importance of localized vs. systemic responses of the plant to virus infection. The aphid responses in bioassays indicate there are positional differences in total VOCs, the VOC blend, or both released from PLRV-infected plants. Rajabaskar et al. (2013b) found that the relative arrestment of *M. persicae* by infected plants and VOC release was greater for lower and middle leaflets than for upper leaflets of plants inoculated at 1 and 3 weeks after transplant, while the reverse in the positional effect was observed in plants inoculated at 5 weeks after transplant.

Changes in aphid responses to VIVs after virus acquisition

Effects of virus acquisition on vectors could have importance for virus spread. Evidence is accumulating for such effects of propagative plant viruses on their vectors (reviewed in Gutiérrez et al., 2013), although none in this review involves aphids or responses to host plant chemistry. Levin and Irwin (1995) reported that tethered flight durations of *R. padi* alates reared on BYDV-PAV-infected oat plants decreased compared with those reared on noninfected plants, an effect that accelerates dispersal of the virus. Similarly, changes in the responsiveness of the aphids to volatile cues from infected plants after virus acquisition could be important epidemiologically. *M. persicae* reared continuously on *Physalis floridana* Rydb. infected with PLRV were less likely to emigrate from the vicinity of potato leaflets than were nonviruliferous aphids, but a 2-day acquisition access period on infected *P. floridana* had no effect on aphid behavior (Werner, 2006). Medina-Ortega et al. (2009) found that viruliferous *R. padi* apterous aphids did not discriminate among the headspace of BYDV-infected and sham-inoculated plants of two wheat cultivars, whereas nonviruliferous aphids preferentially immigrated to BYDV-infected cultivar Lambert wheat plants compared with other wheat treatments. Ingwell et al. (2012) detected a clear settling preference by *R. padi* from a BYDV-infected colony for noninfected wheat plants, while *R. padi* from a noninfected colony preferentially settled on BYDV-infected wheat plants. Similarly, Rajabaskar et al. (2014) showed that *M. persicae* from a colony reared on PLRV-infected *P. floridana* preferentially settled on noninfected potato plants, while aphids from a virus-free colony settled on PLRV-infected plants. The dynamics could also be detected in response to trapped headspace or synthetic blends of VOCs from infected and noninfected plants.

Whether the effects of virus acquisition on the insect vector behavior are direct, i.e., the results of the acquired virus particles per se, or indirect as a result of vector exposure to infected plants is unknown in most systems because the infectious vectors have always acquired the virus by feeding on infected plants (Moreno-Delafuente et al., 2013; Shrestha et al., 2012; Stafford et al., 2011). Ingwell et al. (2012), however, showed definitively that virus particles alone can alter behavior of an aphid vector. *R. padi* that acquired BYDV by feeding through a membrane on an artificial diet containing virus particles preferred to settle on noninfected plants, while membrane-fed controls preferentially settled on BYDV-infected plants. Similar direct effects may occur in other systems and can be verified, as Ingwell et al. (2012) did by administering virus via membrane feeding (Gray, 2008; Ingwell et al., 2012; Mowry and Ophus, 2006; van den Heuvel et al., 1991) or by direct injection (Tamborindeguy et al., 2008)

R. padi is not a host of the virus. (BYDV, like other luteoviruses, does not replicate within its vectors.) The direct effect observed in the BYDV pathosystem (Ingwell et al., 2012) is therefore in support of a vector manipulation hypothesis (VMH), which applies specifically to systems in which the vector is a dispersal agent, but not a reproductive host, of the virus (Ingwell et al., 2012).

Influence of Chemical Factors on the Epidemiology of Aphid-Transmitted Viruses: Models of Virus Spread

The behavioral responses of aphids to chemical characteristics of virus-infected plants can influence virus epidemiology and disease ecology, but the possible complexities are substantial. Potentially, differential colonization in turn can result from differences in immigration, emigration, or both and can result from changes in rates or probabilities of movement, orientation, and other responses. Movements by and relative abundance of alate and apterous forms of the vector have distinctive implications for the scale and dispersion of secondary infections within a field or landscape. Furthermore, greater abundance of aphid vectors on infected plants or a behavioral preference by the vectors for infected plants does not necessarily enhance the spread of the virus throughout a plant population if, for example, the vectors do not move to healthy plants.

Because of these complexities, understanding how vector responses to infected plants potentially influence virus spread and epidemiology has depended primarily on models rather than experimental work. These models can indicate important behavioral and developmental parameters and guide experimental work toward better understanding of the mechanisms. One of the first models to examine the effects of vector preference for virus-infected plants (McElhany et al., 1995) was a simulation patterned after the wheat–BYDV–*R. padi* pathosystem. The simulation allowed settling preference by vectors for infected and noninfected plants to be varied. The model showed that relative rate of spread of a vector-transmitted virus is greater if vectors preferentially settle on infected plants compared with noninfected plants, but only at the early stages of an infestation when infected plants are relatively rare. As infected plants become more prevalent, continued rate of spread is greater if vectors preferentially settle on noninfected plants relative to infected ones. Overall, a preference for infected plants translates into a relatively rapid initial spread of the virus after colonization by vectors but a slower subsequent spread and lower prevalence of infected plants (McElhany et al., 1995). Sisterson (2008) expanded upon this modeling framework and included "orientation preference," which is equivalent to preference as

modeled by McElhany et al. (1995), and a second behavioral parameter, the time in residence once a plant was encountered, termed "feeding preference" by Sisterson (2008). Feeding preference for healthy plants increased virus spread regardless of prevalence of infected plants. If both parameters were varied, a range of predictions was obtained. Arrestment, shown to be chemically mediated in some of the experimental work (e.g., Eigenbrode et al., 2002), may have an effect similar to that of the modeled parameter feeding preference. Attraction to infected plants and VIVs (e.g., Mauck et al., 2010b; Medina-Ortega et al., 2009) may represent orientation preference. The behaviors exhibited by *M. persicae* and *A. gossypii* in response to CMV, i.e., attraction to VIVs followed by reduced feeding (Mauck et al., 2010b), may represent a positive orientation preference and a negative feeding preference. In Sisterson's simulation model (Sisterson, 2008), this combination promotes relatively fast spread compared with all other combinations of feeding preference and orientation preference. Better inferences concerning a chemical ecology of aphid-transmitted viruses could be obtained with bioassays explicitly designed to measure the modeled parameters or by developing models designed to incorporate the behavioral effects that have been measured.

Virus acquisition by aphids can alter their responsiveness to infected hosts and to VIVs. Most models of virus spread as influenced by vector behavior have omitted such effects (McElhany et al., 1995; Sisterson, 2008), but such changes in responsiveness could be important, as discussed but not modeled by McElhany et al. (1995). For example, if vectors are attracted to infected hosts only until virus acquisition takes place, as shown by Ingwell et al. (2012) and Rajabaskar et al. (2014) for a BYDV and PLRV pathosystem, respectively, this could accelerate virus spread. Using a deterministic model of disease spread, incorporating vector preferences for infected and noninfected plants dependent on whether or not the vector is inoculative, Roosien et al. (2013) showed that a change in preference by the vector from noninfected to infected plants after virus acquisition can greatly accelerate spread.

Sisterson's (2008) models of virus spread divide vector behavior into two phases, an orientation preference during host location and initial settling onto a potential host and a feeding preference that occurs during sustained ingestion. These phases would be mediated by different chemical cues, with orientation mediated by olfactory cues and feeding mediated by those cues and others accessible to the aphid during sustained ingestion. The models do not make explicit whether the orientation behavior represents behavior by walking aphids dispersing through a canopy or by alates but presumably could represent either, with implications for secondary spread of plant viruses at different spatial scales. Walking between plants is an important mechanism for aphid dispersal (Hodgson, 1991) and hence an important component of secondary spread (Badenhausser, 1994; Bailey et al., 1995; Boiteau, 1997; Gourmet, 1994; Hanafi et al., 1989; Irwin and Thresh, 1990; Syller, 1996; Thackray et al., 2009; Williams et al., 1998). Modeling potentially can incorporate both alate and apterous behavior in response to infection status of host plants for a more comprehensive understanding of the effects of each on virus spread.

Sisterson (2008) also incorporated vector population size into his models, showing that the importance of vector behavior diminishes as vector population size increases. Since virus-infected plants also can alter vector performance, and therefore potential population size, the net effects of vector responses to infected plants are likely complex in any system. Models of the epidemiology of vector-transmitted plant viruses can be improved to capture more of the pertinent behavioral and ecological dynamics of these systems that are potentially mediated by chemistry. These include interactions in genetically complex plant populations, effects on vector alate production in response to nutrients and defenses, and effects involving higher trophic levels (Jeger et al., 2004).

Research Directions in the Chemical Ecology of Aphid-Transmitted Viruses

The field of study is nascent. Additional research is merited to address several key issues relating to the biochemistry, ecology, evolution, and potential application of these phenomena.

Biochemical and molecular mechanisms of VIV induction

The study of the molecular mechanisms of induced changes in plant chemistry in response to biotic and abiotic stresses is an active field that is beginning to explore the effects of plant virus infections on plant responses at the molecular level. The mechanisms by which plant virus infection alters plant chemistry remain relatively unexplored, but some evidence indicates these responses are unique. The pattern of elevation of VOCs in PLRV-infected plants includes compounds from most major classes of VOCs (Eigenbrode et al., 2002; Werner et al., 2009). This is in contrast to VOC production in potato after wounding or treatment with methyl jasmonate, in which sesquiterpenes, a homoterpene, and some alkanes were primarily affected (S. D. Eigenbrode, J. Lorenzen, and H. Ding, *unpublished*), or shortly after feeding by Colorado potato beetles, in which sesquiterpenes, monoterpenes, and methyl salicylate were elevated (Bolter et al., 1997). Similarly, infections of wheat by BYDV (Jiménez-Martínez et al., 2004a) and *C. pepo* by CMV elicit a broad-spectrum induction of VOCs that seems not to be the product of one specific biochemical pathway (Mauck et al., 2010b). Thus, the pattern of VOC elevation by these viruses is not consistent with the jasmonate-dependent wounding induction pathway or with elevation in response to pathogens (salicylate dependent), suggesting a unique induction mechanism or involvement of several pathways. Preliminary work examining the changes in phytohormones in potato leaves soon after inoculation with PLRV (S. D. Eigenbrode, H. Ding, and E. Schmelz, *unpublished*) shows an increase in methyl salicylate compared with sham-inoculated plants after 48 h, which subsides by 72 h, and a trend toward reduced methyl jasmonate in the virus-inoculated leaves. Phytohormone levels were not tracked beyond that point, but most VIVs have been measured 2 weeks or longer after inoculation, so future work is needed to track phytohormones as virus infections progress. Casteel et al. (2015) demonstrated that changes in plant defenses affecting aphids induced by TuMV infection are ethylene dependent but that induction of SA or JA is not required for this response.

As virus titer increases within the plant, the level of disruption of plant metabolism presumably changes qualitatively and quantitatively, with effects on VOCs. Jiménez-Martínez et al. (2004a) detected a positive correlation between aphid immigration response to VOCs and virus titer in one of the wheat lines tested. Experiments conducted with PLRV and *N. benthamiana*

have shown that sequence variation in one of the open reading frames of an isolate of this virus elicits different headspace profiles and aphid responses (S. D. Eigenbrode, J. Kuhl, A. Karasev, M. Dibble, and H. Ding, *unpublished data*). Whether these sequence variations influence virus titer, plant reaction to infection, or both remains to be determined. The full array of tools and approaches available to elucidate plant responses to stresses should be employed to understand how plant virus infections alter plant gene expression and metabolism leading to the altered VOCs and changes in defenses that have been observed. Suitable molecular models, such as viruses affecting Arabidopsis, have already shown promise in elucidating these effects (Casteel and Jander, 2013; Casteel et al., 2015; DeVos and Jander, 2010).

Evolution of VIVs

The potential for evolution involving plant viruses, their vectors, and host plants has long fascinated biologists (Belliure et al., 2005, 2008; Blua and Perring, 1992a,b; Bosque-Pérez and Eigenbrode, 2011; Castle and Berger, 1993; Castle et al., 1998; Gutiérrez et al., 2013; Hodge and Powell, 2008, 2010; Ingwell et al., 2012; Kennedy, 1951; Malmstrom et al., 2011; Mauck et al., 2010b; McElhany et al., 1995; Musser et al., 2003; Powell et al., 2006; Power, 1991; Chapters 3, 5, and 15, this volume). As outlined elsewhere in this volume (Chapters 5 and 15), the physiology of aphid–virus interactions is finely tuned. It should not be surprising if fine-tuning occurs at the ecological level as well. Specifically, it is possible to view plant virus effects on host plants as evidence for the host manipulation hypothesis (HMH), which posits that parasites have been selected to manipulate the phenotype of their hosts such that their transmission to new hosts is facilitated (Lefèvre and Thomas, 2008; Poulin, 1995, 2000; Thomas et al., 2005). Virus effects on vectors may be examples of the VMH, which posits that plant pathogens evolve strategies that enhance their spread to new hosts through their effects on mobile vectors (Ingwell et al., 2012).

For plant viruses and their vectors, some patterns may be consistent with HMH or VMH. It is conceivable that distinct syndromes exist depending upon the specifics of vector ecology, virus mode of transmission, and host plant ecology. For example, members of *Luteoviridae* studied to date generally increase both the quality (Araya and Foster, 1987; Castle and Berger, 1993; Fereres et al., 1989; Jiménez-Martínez et al., 2004b; Miller and Coon, 1964) and attractiveness (Castle et al., 1998; Jiménez-Martínez et al., 2004a; Medina-Ortega et al., 2009; Ngumbi et al., 2007; Srinivasan et al., 2006; Werner et al., 2009) of the host plant for the vector, although there are exceptions (Fiebig et al., 2004; Power, 1996). This combination of effects has not been reported for other viruses (Castle et al., 1998; Eigenbrode et al., 2002; Mauck et al., 2010b).

On the basis of a meta-analysis of 224 experiments from 55 published studies, Mauck et al. (2012) found that aphid vector attraction preference for infected plants was predominant among all aphid-transmitted viruses, regardless of transmission mode. Preference for settling and continued feeding on infected plants, however, was predominant only among aphids with persistently transmitted viruses. Further, they found that aphid performance was enhanced on plants infected with persistently transmitted viruses, while the reverse was the case for nonpersistently transmitted viruses. These patterns can be explained in evolutionary terms. Members of *Luteoviridae* are circulative and persistently transmitted and rely on a narrow range of vector species, conditions that may favor a virus genotype that improves vector performance and attractiveness in contrast to viruses that are nonpersistently transmitted or rely on a broader range of vectors, often including aphids for which the plant is not a viable host. Persistently transmitted viruses also require extended phloem feeding, necessitating several hours for acquisition and transmission (Nault, 1997). In contrast, the rapidity with which nonpersistently transmitted viruses can be acquired and transmitted—as little as a few seconds of probing—should select for attraction of vectors but not necessarily for continued feeding or population growth. Although the patterns detected by Mauck et al. (2012) are significant and appear robust, the available literature is still relatively sparse, covering only 55 studies and 224 experiments. More knowledge of the chemical ecology of diverse pathosystems is required to verify these patterns and evaluate the plant traits on which they depend. Study of comparable viruses or virus strains differing in requirements for vector transmission could help confirm associations. The various mechanisms whereby viruses have been found to influence their host plant defensive systems such that vectors may gain an advantage strongly suggest that these effects have been shaped by natural selection (Casteel and Jander, 2013). Better understanding of the mechanisms by which virus infection alters plant characteristics and how vectors respond physiologically and behaviorally to plant viruses and virus-infected plants will help decipher the role of natural selection in shaping these patterns.

Mechanisms governing changes in aphid responses after virus acquisition

Evidence shows that aphid preferences for plants change after virus acquisition (Ingwell et al., 2012, Rajabaskar et al., 2014), but it has not been determined how these changes in vector preference are mediated. In the BYDV–wheat–*R. padi* system, the effect is known to be at least in part direct, such that virus particles present within the aphid alter its responsiveness to host cues. In addition, in this system and in the PLRV–potato–*M. persicae* system, conditioning of the vectors by exposure to and feeding on infected plants could also contribute to dynamic preferences. Additional research is needed to characterize these mechanisms.

Ecology, epidemiology, and application

Several research needs can be identified to achieve a longer-term goal to understand the chemical ecology of plant viruses and to apply this understanding to reduce the impact of the diseases they cause in agriculture (Bosque-Pérez and Eigenbrode, 2011).

First, the evidence that VIV production and aphid responses are temporally dynamic following infection (Medina-Ortega et al., 2009; Rajabaskar et al., 2013b; Werner et al., 2009) requires further study. Such dynamics could influence disease spread, but the effects need to be studied under conditions more representative of those potentially occurring in the field.

Second, much of the work on mechanisms has focused on VIVs, but a wider suite of cues potentially come into play (Carmo-Sousa et al., 2014; Casteel et al., 2014, 2015; Mauck et al., 2014; Wu et al., 2014). Bioassays should examine these effects and potential cues.

Third, our work (Rajabaskar et al., 2013b; Werner et al., 2009) and that of others (Alvarez et al., 2007) has shown that

VIV release in potato varies not only with disease progression but among parts within the plant and throughout disease progression. Further studies should assess how this variability arises, how widespread it may be in other pathosystems, and its implications for virus epidemiology.

Fourth, better links are required between controlled laboratory bioassays and processes that occur at the plot and field levels. VOC-mediated movements among entire plants or within the canopy need to be studied to validate modeled predictions of vector preferences on virus spread within plant populations. Much of the work has focused on apterous aphids, which are important for virus spread (Badenhausser, 1994; Bailey et al., 1995; Gourmet, 1994; Hanafi et al., 1989; Irwin and Thresh, 1990; Syller, 1996; Thackray et al., 2009; Thomas et al., 1997; Williams et al., 1998), but behavior of alates is critical for establishment of new disease foci within and among fields (Irwin and Thresh, 1990) and also may be influenced by VIVs and other cues. Alates may respond to different cues than do apterous aphids. Although alatae and apterae seem to respond similarly to plant odors (Pickett et al., 1992), differences in responsiveness of morphs have been detected (Park et al., 2000), and alates can discriminate in response to shorter-range (e.g., Phelan and Miller, 1982) and longer-range (e.g., Nottingham et al., 1991) cues that could be altered by virus infection.

Fifth, as reviewed herein, viruliferous aphids can differ from nonviruliferous aphids in their responsiveness to host plant chemistry (Ingwell et al., 2012; Medina-Ortega et al., 2009; Rajabaskar et al., 2014; Werner, 2006). Such changes are important for virus spread (Roosien et al., 2013) and should be considered in models of virus spread and as potential factors that can come under selection as part of host or vector manipulation by viruses.

Sixth, in addition to VIVs, other chemical cues, including surface chemistry, phloem, and nonphloem tissue chemistry accessible after contact with the plant, may contribute to aphid responses to virus-infected plants but have yet to be elucidated. Similarly, better understanding of how virus infection alters host nutritional quality is needed.

Seventh, the effects of VIVs and other virus-induced changes in plant chemistry on the ecological community other than the virus vectors merit further attention (see Fig. 1.1). The effects of VIVs and other induced plant volatiles on parasitoids and predators potentially influence vector populations and behavior and alter virus spread (Jeger et al., 2012; Mauck et al., 2015b). Mauck et al. (2010a) reported that females of the squash bug *Anasa tristis* (DeGeer) (Hemiptera: Coreidae) preferentially oviposit on "healthy" *C. pepo* over CMV-infected plants. Virus infection can also alter the performance of nonvector herbivores. Kersch-Becker and Thaler (2014) found that a caterpillar (*Trichoplusia ni* (Hübner)) (Lepidoptera: Noctuidae) and beetle larva (*Leptinotarsa decemlineata* (Say)) (Coleoptera: Chrysomelidae) had greater relative growth rates on tomatoes infected with a strain of PVY (NTN) than on sham-inoculated controls. Aphid tending by fire ants (*Solenopsis invicta*) (Hymenoptera: Formicidae) increases the incidence of aphid-transmitted viruses in tomato (Cooper et al., 2005). Aphid parasitoids respond to aphid-induced VOCs (Du et al., 1998; Tentelier et al., 2005), and thus they potentially respond to VIVs. Virus-infected plants affect natural enemies indirectly if aphids on these plants differ in quality for predators or parasitoids (Mauck et al., 2015a). Such potential effects, all of which fall within the purview of chemical ecology of aphid-transmitted viruses, invite investigation.

Concluding Remarks

Most ecological interactions, whether intraspecific or interspecific and involving multiple taxa, are mediated to some extent by chemicals that modify organismal performance and behavior. The field of chemical ecology includes many examples of the discovery of chemical dimensions of interactions not previously understood to be chemically mediated. A growing literature attests to the existence of chemically mediated interactions involving plant viruses, the aphids that transmit these viruses, and their host plants. It is intriguing that this chemical ecology exists despite the inability of plant viruses on their own to generate chemical signals or toxins or to respond to them directly. Although the field is nascent and patterns are just emerging, a chemical ecology of such pathosystems appears likely to provide opportunities for discovery of unique mechanisms in both natural and managed systems as well as novel applications for crop protection.

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