Why do viruses make aphids winged?

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Abstract

Aphids are hosts to diverse viruses and are important vectors of plant pathogens. The spread of viruses is heavily influenced by aphid movement and behavior. Consequently, wing plasticity (where individuals can be winged or wingless depending on environmental conditions) is an important factor in the spread of aphid-associated viruses. We review several fascinating systems where aphid-vectored plant viruses interact with aphid wing plasticity, both indirectly by manipulating plant physiology and directly through molecular interactions with plasticity pathways. We also cover recent examples where aphid-specific viruses and endogenous viral elements within aphid genomes influence wing formation. We discuss why unrelated viruses with different transmission modes have convergently evolved to manipulate wing formation in aphids and whether this is advantageous for both host and virus. We argue that interactions with viruses are likely shaping the evolution of wing plasticity within and across aphid species, and we discuss the potential importance of these findings for aphid biocontrol.

Resumen: ¿Por qué los virus hacen que los áfidos desarrollen alas?

Los áfidos albergan diversos virus y son vectores de importantes patógenos de plantas. La propagación de virus está fuertemente influenciada por el movimiento y el comportamiento de los áfidos. En consecuencia, la plasticidad de las alas (en la cual algunos individuos desarrollan alas dependiendo de las condiciones ambientales) es un factor importante en la propagación viral asociada a los áfidos. En este documento revisamos varios ejemplos fascinantes en los que virus de plantas transmitidos por áfidos interactúan con la plasticidad fenotípica de las alas, indirectamente manipulando la fisiología de la planta y directamente a través de interacciones moleculares con los mecanismos de plasticidad fenotípica del áfido. También describimos ejemplos recientes que demuestran como algunos virus específicos de áfidos y elementos virales endógenos localizados en los genomas de áfidos influyen en la formación de alas. Últimamente, discutimos por qué virus no relacionados con diferentes modos de transmisión han evolucionado convergentemente para manipular la formación de alas en áfidos y si este fenómeno es beneficioso para el insecto y el virus. Nosotros objetamos que las interacciones con virus están probablemente influenciando la evolución intra- e interespecífica de la plasticidad de las alas en áfidos, y discutimos el potencial de estos hallazgos para el control biológico de los áfidos.

Key Words: aphids, viruses, plasticity, biocontrol

Wing development is a phenotypically plastic trait in many insects, allowing for a rapid response to changing environmental conditions (Simpson et al., 2011). In some species, discrete winged and wingless morphs are produced in response to stimuli such as population density, food availability, or host plant quality (referred to as 'polyphenism'). Winged morph individuals, termed 'alates,' have fully developed flight muscles and wings but suffer reduced reproductive output compared with unwinged morphs (Yang and Pospisilik, 2019, Hayes et al., 2019). Because of this tradeoff between fecundity and dispersal, wing plasticity must be finely tuned to environmental conditions (Nettle and Bateson, 2015).

Plastic wing production is orchestrated at multiple levels, starting with the perception of environmental cues, signaling pathways, and then the physical production of wings. Aphids, in particular, are a model system for studying the ecology and molecular biology of insect wing plasticity (Brisson and Stern, 2006). Some aphid species exhibit trans-generational wing plasticity where signals of crowding trigger the production of offspring that eventually develop wings. In other species, developing juveniles directly sense the environment (Muller et al., 2001). The molecular mechanisms of wing plasticity have been most thoroughly investigated in pea aphids (*Acyrthosiphon pisum*), where signals of crowding trigger a decrease in ecdysone signaling in adult asexual females (Vellichirammal et al., 2017), which further elicits changes in insulin signaling in developing embryos leading to winged offspring (Grantham et al., 2020). Investigations in other aphid species have also implicated microRNAs (miRNAs) in the regulation of wing plasticity, potentially through their post-transcriptional action on insulin and insulin-like signaling pathways (Shang et al., 2020, Li et al., 2022). Collectively, the roles of neuro-endocrine signaling pathways and epigenetic mechanisms in wing plasticity seem likely to be fundamental across aphids and other insects (reviewed in Zhang et al., 2019).

Winged individuals migrate to new habitats, and wing plasticity is therefore critical for aphid movement and dispersal. Winged aphids have, perhaps unsurprisingly, been shown to be important in the transmission of viruses associated with aphids (Table 1 and references therein). The role of winged aphids in viral spread has been best studied in agriculturally relevant plant pathogens that are vectored by aphids. For example, a longitudinal study of *Plum pox virus* (PPV) transmission using data collected across hundreds of orchards showed that long-range dispersal by winged aphids is needed to explain PPV spread within and among orchards (Pleydell et al., 2018). Epidemiologically, plant virus spread is correlated with the number of

vector visits per plant per day, and therefore an increase in the number of winged aphids feeding on virus-infected plants is an important factor driving viral transmission (Madden et al., 2000).

Interestingly, unrelated viruses have evolved to manipulate wing plasticity both through changes in plant volatile organic compounds (VOCs) and/or host plant quality and through direct interactions with aphid wing plasticity pathways (Jayasinghe et al., 2021, Mauck et al., 2010). In this article, we review these systems and discuss why viruses with different transmission modes may have evolved to manipulate wing formation in aphids and whether this is advantageous for hosts and/or viruses. We argue that interactions between viruses and wing plasticity are likely widespread across the aphid phylogeny and are shaping the evolution of plasticity within and across species. We also highlight the importance of studying these interactions in a broader ecological and evolutionary context and the potential implications for aphid biocontrol.

Table 1. Examples of links between viruses and winged aphids.

Virus	Viral family (genome)	Aphid species studied	Host plant	Transmission mechanism	Wing induction	Reference
Cucumber mosaic virus (CMV)	Bromoviridae (ss(+)RNA)	*Myzus persicae*	*Nicotiana tabacum*	non-persistent	The number of winged aphids is higher on CMV-infected plants	(Shi et al., 2016)
Cucumber mosaic virus (CMV) and sat-derived small RNA (Y-sat)	Bromoviridae (ss(+)RNA) and satellite RNA (satRNA)	*Myzus persicae*	*Nicotiana tabacum*	non-persistent	CMV + Y-sat infected plants turn leaves yellow, which preferentially attracts aphids; aphids fed on plants that harbor Y-sat develop wings	(Jayasinghe et al., 2021)
Potato virus Y (PVY)	Potyviridae (ss(+)RNA)	Multiple, including *Rhopalosiphum padi* and *Aphis fabae*	*Solanum tuberosum*	non-persistent	PVY epidemics correlate with an elevated number of winged aphids	(Sigvald, 1989)
Turnip mosaic virus (TuMV)	Potyviridae (ss(+)RNA)	*Myzus persicae*	*Nicotiana benthamiana*	non-persistent	Winged aphids are more abundant on TuMV-infected plants than on control plants	(Casteel et al., 2014)
Zucchini yellow mosaic virus (ZYMV)	Potyviridae (ss(+)RNA)	*Aphis gossypii*	*Cucurbita pepo*	non-persistent	Early stages of ZYMV infection stimulate wing formation in field experiments	(Blua and Perring, 1992)
Beet western yellows virus (BWYV)	Luteoviridae (ss(+)RNA)	*Myzus persicae*	*Beta vulgaris*	persistent	Winged aphids are more likely to be found on virus-infected sugar beet leaves than on healthy leaves	(Macias and Mink, 1969)
Barley yellow dwarf virus (BYDV)	Luteoviridae (ss(+)RNA)	*Sitobion avenae* and *Rhopalosiphum padi*	*Avena byzantin*	persistent	Aphids reared on BYDV-infected oats are more likely to mature as winged adults	(Gildow, 1980)
Pea enation mosaic virus (PEMV)	Luteoviridae (ss(+)RNA)	*Acyrthosiphon pisum*	*Pisum sativum*	persistent	Adults crowded onto PEMV-infected plants produce more winged progeny	(Hodge and Powell, 2010)

Potato leafroll virus (PLRV)	Luteoviridae (ss(+)RNA)	*Myzus persicae*	*Solanum tuberosum*	persistent	Winged aphids preferentially colonize PLRV-infected plants	(Eigenbrode et al., 2002)
Dysaphis plantaginea densovirus (DplDNV)	Parvoviridae (dsDNA)	*Dysaphis plantaginea*	*Plantago longifolia*	insect specific	DplDNV infection in asexual clones produces a high percentage of winged offspring	(Ryabov et al., 2009)
Myzus persicae densovirus (MpDNV)	Parvoviridae (dsDNA)	*Myzus persicae*	*Nicotiana benthamiana*	insect specific	MpDNV infection correlates with winged aphid formation	(Pinheiro et al., 2019)

Vectored viruses are indirectly linked with winged aphids through host plant manipulation. Winged aphids find host plants using visual and olfactory cues (Döring, 2014). Plant leaf color and VOCs are important cues in aphid host-finding behavior (Chapman et al., 1981). Interestingly, viruses have evolved to take advantage of these aspects of aphid biology by altering plant physiology to attract winged aphids (Figure 1A). For example, some aphid-vectored plant viruses change the color of infected leaf tissue to light green or yellow by targeting plant chloroplast structure and function (Li et al., 2016). The lighter leaf color attracts winged aphids (Hodge and Powell, 2008) which then become exposed to the virus. Infected plants have also been shown to produce elevated emissions of attractive VOCs through changes in plant secondary metabolism (Safari Murhububa et al., 2021). Winged aphids are more sensitive to VOCs than unwinged individuals (Zhang et al., 2021), and increasing attractive VOC emissions increases colonization by winged aphids (Mauck et al., 2010, Mauck et al., 2012, Safari Murhububa et al., 2021).

After attraction to a new plant, host selection (whether an aphid remains on a plant after landing) can also be manipulated by viruses. Importantly, the direction in which this occurs varies across viruses and even among the same virus infecting different host plants. Many aphid-vectored viruses are localized only on the mouthparts of their aphid vector (e.g., families *Bromoviridae* and *Potyviridae*). These are acquired in just a few seconds after probing and are thought to make the aphid infectious for short periods of time (Gray and Banerjee, 1999, Ng and Perry, 2004). Important to the transmission of many of these ‘non-persistent’ viruses is the need for rapid dispersal of aphids to uninfected plants. Some viruses have evolved to quickly deter winged aphids, potentially by reducing host plant quality (Shi et al., 2016) and/or by modifying plant defenses against herbivores like the salicylic acid (SA) and jasmonic acid (JA) plant defensive pathways (Wu and Ye, 2020). Together, the viral-induced attraction of winged aphids through color and/or VOCs and the subsequent deterrence of selection by the aphid is referred to in the literature as the ‘attract and deter’ phenotype (Figure 1A).

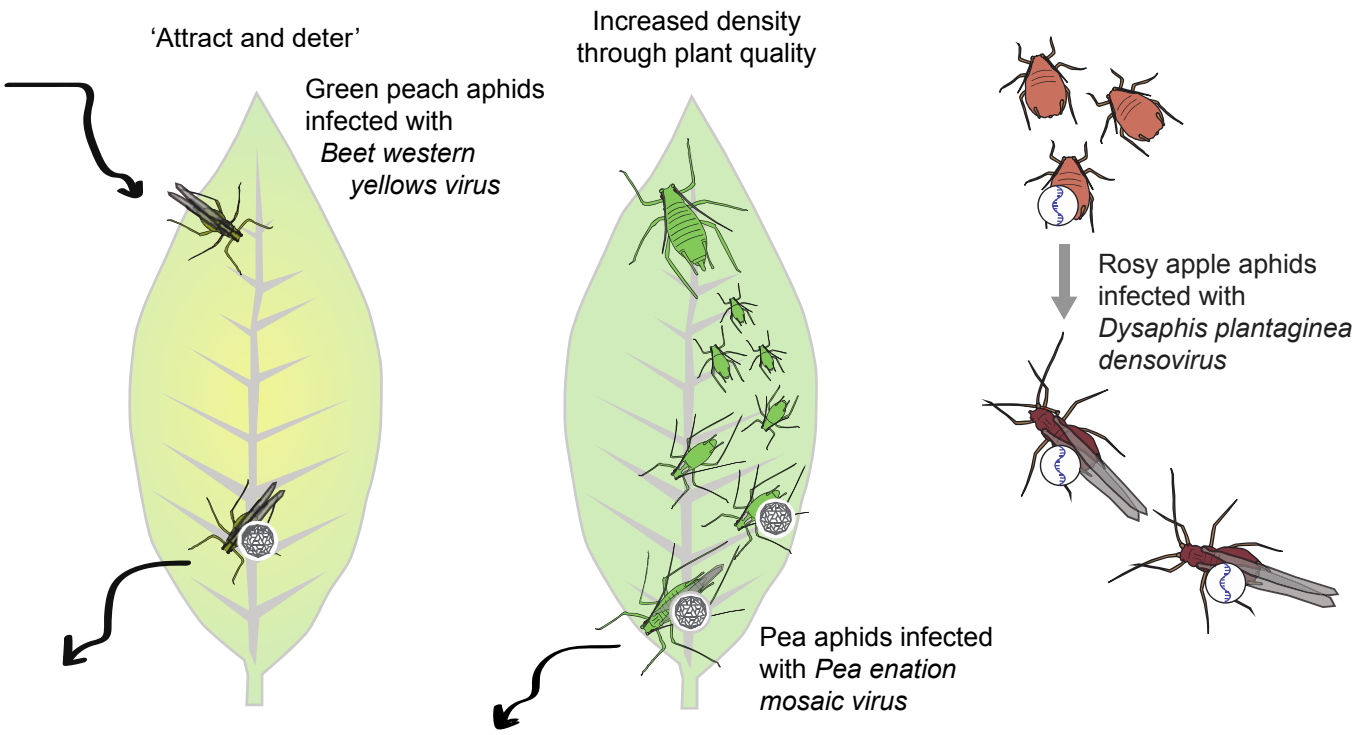

Figure 1: Indirect (A) and direct (B) interactions between viruses and aphid wing plasticity. This figure shows three examples of how viruses interact with aphid wing formation. Panel A shows two ways in which aphid-vectored plant viruses manipulate winged aphids indirectly through a host plant. The 'attract and deter' phenotype refers (left) to viruses (e.g. *Beet western yellow virus* vectored by *Myzus persicae*) that attract and quickly infect winged aphid vectors, and then deter winged aphids to disperse via altered host plant chemistry. Other viruses (e.g. *Pea enation mosaic virus* infecting *Acyrthosiphon pisum*) increase host plant quality (middle) leading to higher aphid density and winged aphid production. Viruses have also been shown to directly manipulate aphids (right) to form wings or produce winged offspring by interacting with aphid wing plasticity pathways (e.g. *Dysaphis plantigena densovirus* infecting *Dysaphis plantigena*).

In contrast, other aphid-vectored plant viruses increase host plant quality for hosts, prolonging aphid feeding after infection (Figure 1A). This has been found both in non-persistent viruses (e.g., *Potato virus Y* and *Zucchini yellow mosaic virus* (Boquel et al., 2010)) and among 'persistent' viruses (i.e., family *Luteoviridae*) that require a longer time to become infectious before transmission to new hosts (Ng and Perry, 2004, Gray and Banerjee, 1999). A possible mechanism for this manipulation involves the reduction of plant defense signaling (Mauck et al., 2012, Bosque-Perez and Eigenbrode, 2011) and changes in the concentrations of the plant's

amino acids, sugars, and proteins (Alexander and Cilia, 2016). For example, *Turnip mosaic virus* (TuMV) promotes aphid settling on TuMV-infected plants by suppression of callose deposition, an important plant defense (Howe and Jander, 2008, Campbell et al., 1986). TuMV infection also increases free amino acid content of the phloem, a major source of nitrogen for aphids (Casteel et al., 2014). Higher fecundity is achieved by aphids feeding on TuMV-infected plants, which, presumably, can lead to wing induction due to overcrowding. Similarly, the persistent *Turnip yellows virus* (TuYV) relies on aphid settlement for sustained periods on the plants for successful virus acquisition and inoculation. Viral infection leads to decreased expression of genes implicated in salicylic acid biosynthesis (Chesnais et al., 2022).

Plant viruses directly manipulate winged aphid formation. The examples of viral manipulation of winged aphids discussed so far include indirect interactions between viruses and winged aphids mediated through host plants. However, some viruses also directly manipulate wing formation in aphids. This was recently described in *Cucumber mosaic virus* (CMV), a non-persistent virus that is vectored by dozens of aphid species and is capable of infecting a wide range of plant hosts (Jacquemond, 2012). CMV is sometimes associated with a satellite RNA called Y-sat, which induces bright yellow symptoms in host plants and attracts aphids (Shimura et al., 2011). A recent study found that green peach aphids (*Myzus persicae*) fed as 1-day-old first instar larvae on yellow plants harboring CMV and Y-sat subsequently developed wings (Jayasinghe et al., 2021). The wing induction was shown to be a result of sequence complementarity between a Y-sat-derived piwi-RNA (piRNA) and an aphid microRNA (miRNA) called miR-9b (Figure 2). The aphid miR-9b regulates the expression of genes regulating wing formation (Shang et al., 2020). Adult aphids infected as first instars with CMV + Y-sat had higher expression levels of aphid genes that influence wing plasticity (Jayasinghe et al., 2021), including an ABC transporter that is thought to influence wing formation through insulin signaling (Shang et al., 2020).

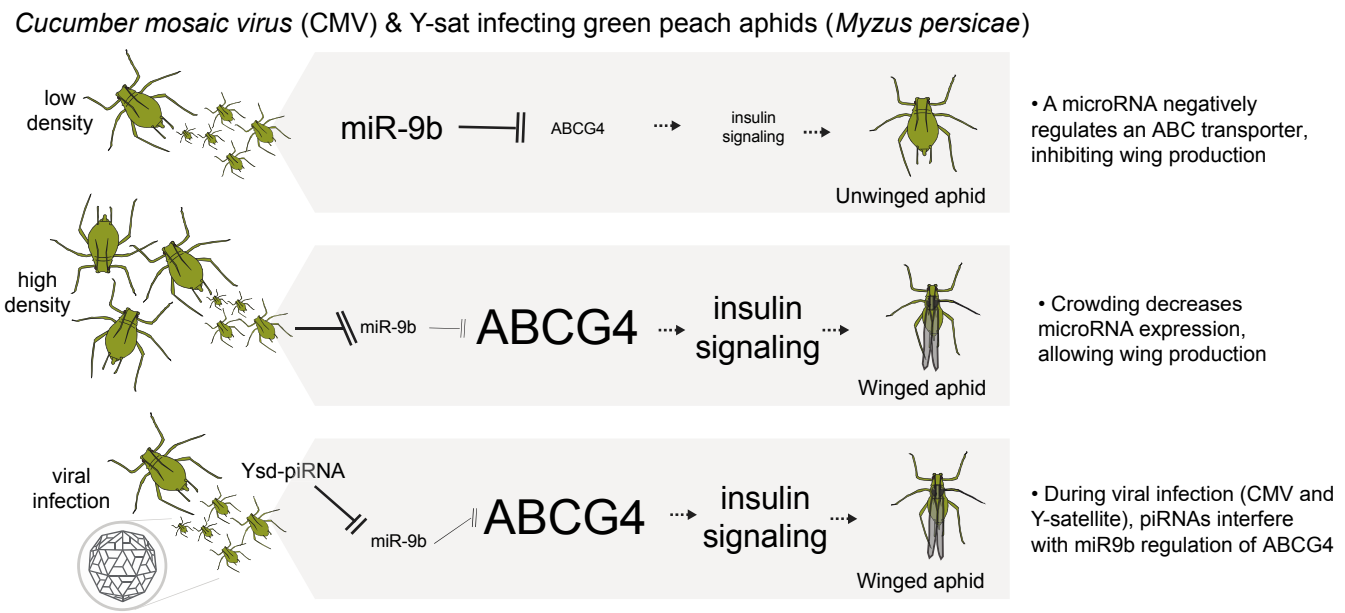

Figure 2: *Cucumber mosaic virus* and its satellite virus in *Myzus persicae*. This figure illustrates the molecular mechanism of direct viral induction of wing formation by CMV in green peach aphids. The top panel shows virus-free aphids in low-density conditions, which have relatively high levels of expression of a miRNA that negatively regulates insulin signaling and does not lead to wing development. The middle panel shows aphids in high-density conditions, which reduces miRNA expression and allows wing development. The bottom figure shows aphids infected with CMV + Y-sat, which interferes with the aphid miRNA, regulating insulin signaling, and leading to wing formation.

Though this direct manipulation of aphid wing plasticity has only been studied mechanistically in one aphid-vectored plant pathogen, this is likely a more widespread phenomenon across aphid-virus interactions. In many previous studies (Table 1), the link between viral infection and the presence of winged aphids has been assumed to be the result of indirect manipulation through host plants, but some of these viruses may instead be directly inducing wings through unexplored mechanisms. Future studies should focus on viruses that have demonstrated impacts on aphid growth and reproduction (e.g., *Potyviruses* and *Luteoviruses* (Bosque-Perez and Eigenbrode, 2011, Gadhave et al., 2019)). Proteins encoded by the genomes of CMV, Cauliflower mosaic virus (CaMV), Barley stripe mosaic virus (BSMV), Tomato yellow leaf curl virus (TYLCV), and Cotton leaf curl Multan virus (CLCuMuV) are known to interact with the autophagy and ubiquitin-proteasome degradation pathways (Li et al., 2018, Ismayil et al., 2020, Yang et al., 2018, Cheng and Wang, 2017, Hafrén et al., 2017), which could affect insect metabolism and thereby manipulate nutrient absorption and wing development.

Insect specific viruses and endogenous viral elements directly induce wing formation. In addition to plant viruses, a group of insect-specific densoviruses (DNVs, family *Parvoviridae*)

have also been directly linked to wing formation in aphids (Figure 1B). Rosy apple aphids (*Dysaphis plantaginea*) infected with *Dysaphis plantaginea densovirus* (DplDNV) were shown to produce winged offspring in response to crowding and poor host plant quality, while virus-free aphids produced no winged offspring, suggesting the virus is inducing winged offspring formation (Ryabov et al., 2009). Relatedly, *Myzus persicae densovirus* (MpDNV) infection titers were found to be correlated with wing induction in *M. persicae*, though this study did not directly test for a causal link between MpDNV and wing induction (Pinheiro et al., 2019). The underlying molecular mechanisms of wing induction by densoviruses are not yet known but could involve direct molecular interactions of small RNAs with wing plasticity pathways or effects of an aphid immune response to viral infection on wing induction.

Remarkably, some densoviral genes have become incorporated into many aphid genomes, and these endogenous viral elements (EVEs) play a role in wing formation. In pea aphids, two genes with close homology to a DplDNV non-structural protein (NS1) are up-regulated in response to crowding and play a functional role in wing induction (Parker and Brisson, 2019). The underlying molecular mechanisms are unknown, but EVEs often function in insect genomes as cis-regulatory DNA elements such as enhancers or promoters and can produce small RNAs as targeted immunity against cognate viruses (Blair et al., 2020). Many aphid species in addition to *A. pisum* have transcribed densoviral EVEs (Clavijo et al., 2016, Liu et al., 2011), but the effects on wing formation in these other species are unknown.

Is direct viral induction of wings advantageous for aphids? The examples shown above demonstrate that multiple, unrelated viruses with different life history strategies have convergently evolved to manipulate wing plasticity in their aphid vectors and/or hosts. Multiple authors have argued that viral manipulation of wing induction is advantageous for aphids both as vectors of plant pathogens (Ray and Casteel, 2022) and as hosts (Ryabov et al., 2009). These arguments revolve around the potential benefits to aphids of increased dispersal and host range expansion. DplDNV, in particular, was shown to facilitate dispersal of *D. plantaginea* under both laboratory and field conditions (Ryabov et al., 2009), and has been cited in the literature as providing a conditional benefit to its host and therefore acting as a mutualist (e.g. Roossinck, 2011).

We think these arguments are potentially problematic for several reasons. First, studies make the assumption that because DplDNV is vertically transmitted, wing induction must be advantageous for aphids because providing a benefit to hosts is needed for the virus to spread. However, the extent to which DplDNV affects aphid survival and fecundity, whether it provides benefits like resistance to pathogenic viruses (as is seen in other insect-Densovirus systems (Johnson and Rasgon, 2018)), and the extent to which DplDNV is also horizontally transmitted remain to be explored. Second, DplDNV is described as being required for wing induction and, therefore, essential for *D. plantaginea* movement and dispersal. But little is known about the phenotypic or molecular mechanisms of wing induction in this aphid species and whether dependence on the virus for wing induction is a feature or an evolved response to viral manipulation. Like other phenotypically plastic traits, wing plasticity exhibits genetic variation, where genotypes differ in their sensitivity to environmental cues. Aphid clonal lineages and populations differ in their response to environmental cues triggering wing formation (Grantham et al., 2016, Sentis et al., 2019, Parker et al., 2021). One possibility is that it is difficult to induce winged offspring in virus-free *D. plantaginea* because of evolution with manipulative Densoviruses, *i.e.,* the aphid has had to evolve to compensate for viral wing induction through changes to its own plasticity pathways. More broadly, it is currently unclear how viral manipulation is shaping the evolution of wing plasticity within and across aphid species, but it seems likely that the 'optimal' proportion of winged offspring born to an aphid differs from the perspective of aphid fitness and viral transmission.

Is viral wing induction advantageous for viruses? Across numerous studies, there is another common assumption that viruses benefit from manipulating winged aphid behavior or from triggering wing formation. For example, there is a long-standing hypothesis that manipulating plants to quickly deter winged aphids is beneficial for non-persistent viruses. Rapidly infected aphids remain infectious for brief periods of time, and therefore deterring aphids quickly is thought to increase transmission (Mauck, 2016, Carr et al., 2018). In contrast, when non-persistent viruses encourage prolonged feeding, the assumption is that this strategy inhibits transmission and reflects a non-adaptive mismatch between virus, vector, and/or plant (Mauck et al., 2014).

However, recent studies have challenged these ideas. For example, a mathematical model of non-persistent viruses vectored by aphids found that deterring winged aphids might be a self-

limiting strategy because it leads to a decrease in vector population size, leading to lower epidemic sizes. Facilitating reproduction and the development of winged aphids could lead to greater long-range virus transmission and larger-scale epidemics (Donnelly et al., 2019). Mechanistic studies of viral manipulation have also challenged these hypotheses. For example, CMV is expected to benefit from deterring winged aphids as a non-persistent virus, and studies have found that in the absence of Y-sat the virus does decrease the quality of its host plant through increased plant defense signaling (Shi et al., 2016). However, when co-infected with Y-sat, there appears to be no reduction in photosynthesis or aphid fitness from CMV infection (Jayasinghe et al., 2021), potentially allowing winged aphids to settle on plants and produce more offspring that will eventually become winged due to direct manipulation by Y-sat. These studies highlight the complexity of interactions between viruses and aphid wing plasticity and the difficultly of ascribing adaptive value to wing induction for both aphids and virus.

Disrupting wing formation may provide a means of biocontrol. Aphid control has historically relied on the use of chemical insecticides. However, routine applications of insecticides often exhibit unintended effects on the environment and non-target organisms, while target pests rapidly evolve resistance (Bragard et al., 2013, Bass et al., 2014). As an alternative, genetically modified 'aphid-resistant' plants that express toxins or double-strand RNA inducing RNA interference in aphids have been developed (Yu et al., 2014). This approach has showed promising results in laboratory and semi-field settings. However, in larger agricultural settings, the effectiveness is constrained by the dose-dependent efficiency of the target molecules (natural environmental stress constrains the production of non-plant molecules), dsRNA stability, the effectiveness of target gene selection across multiple generations (evolution of resistance or detoxification mechanisms), and even limited unintended off-target effects on beneficial insects (Zhang et al., 2013, Kolliopoulou et al., 2020).

A deeper understanding of how viruses interact with aphid wing plasticity could lead to new approaches to minimizing viral disease transmission. For example, when crops are infected with plant pathogenic viruses linked to aphid wing induction, the use of 'decoy' plants that are more attractive to winged aphids could reduce viral spread within the harvest season (Moffett, 2016). Next, dsRNA could be deployed as a sprayable RNA pesticide (Zhang et al., 2022) along with virus-derived RNAs that show sequence complementarity with the aphid microRNAs to disrupt wing development, suppress aphid migration, and control virus spread. The deployment of

insect-specific viruses that have no consequences to crops along with these dsRNA delivery systems that generate defective wings could further prevent aphid dispersal and constrain aphid reproduction. Similarly, densovirus-induced control has been shown to have great potential in other insect systems (Johnson and Rasgon, 2018), and aphid specific densoviruses could be used as a tool to limit aphid dispersal parallel to genetically modified effectors that, upon delivery and expression, limit the expression of genes necessary for ingestion and digestion or that intervene in vector competence.

Conclusions. Unrelated viruses have evolved to influence aphid wing plasticity indirectly and directly. This manipulation has important effects on viral transmission and dispersal. Future work is needed to uncover the molecular mechanisms of wing manipulation in different aphid-virus systems, and to determine if and when wing induction is beneficial for hosts and viruses. There is a clear need to exchange ideas between entomologists and virologists to motivate better understanding of how viruses influence insect development. The decreasing costs of next-generation sequencing are enabling high-throughput characterization of viruses in natural populations, leading to a deeper understanding of the hidden roles of viruses in many insect phenotypes. A particularly important and unanswered question is whether viruses are contributing to the extensive phenotypic variation in wing plasticity found in natural aphid populations. We think it is likely that viruses are shaping the evolution of wing plasticity within and among species, and this will be an important consideration for future studies of this important model of phenotypic plasticity.

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