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# Variation in virus effects on host plant phenotypes and insect vector behavior: what can it teach us about virus evolution?

Kerry E Mauck[1]

Virus infection can elicit changes in host plant cues that mediate vector orientation, feeding, and dispersal. Given the importance of plant cues for vector-mediated virus transmission, it is unlikely that selection is blind to these effects. Indeed, there are many examples of viruses altering plant cues in ways that should enhance transmission. However, there are also examples of viruses inducing transmission-limiting plant phenotypes. These apparently mal-adaptive effects occur when viruses experience host plant environments that also limit infectivity or within-host multiplication. The apparent link between virus effects and pathology argues for consideration of prior evolutionary relationships between viruses and host plants in order to understand how viruses might evolve to manipulate vector behavior via effects on host plant cues.

**Address**
Department of Environmental Systems Science, ETH Zürich, Schmelzbergstrasse 9, 8092 Zürich, Switzerland

Corresponding author: Mauck, Kerry E (kerry.mauck@usys.ethz.ch, kerry.mauck@ucr.edu)
[1] Future address: Department of Entomology, University of California, Riverside, 900 University Ave, Riverside, CA 92521, USA.

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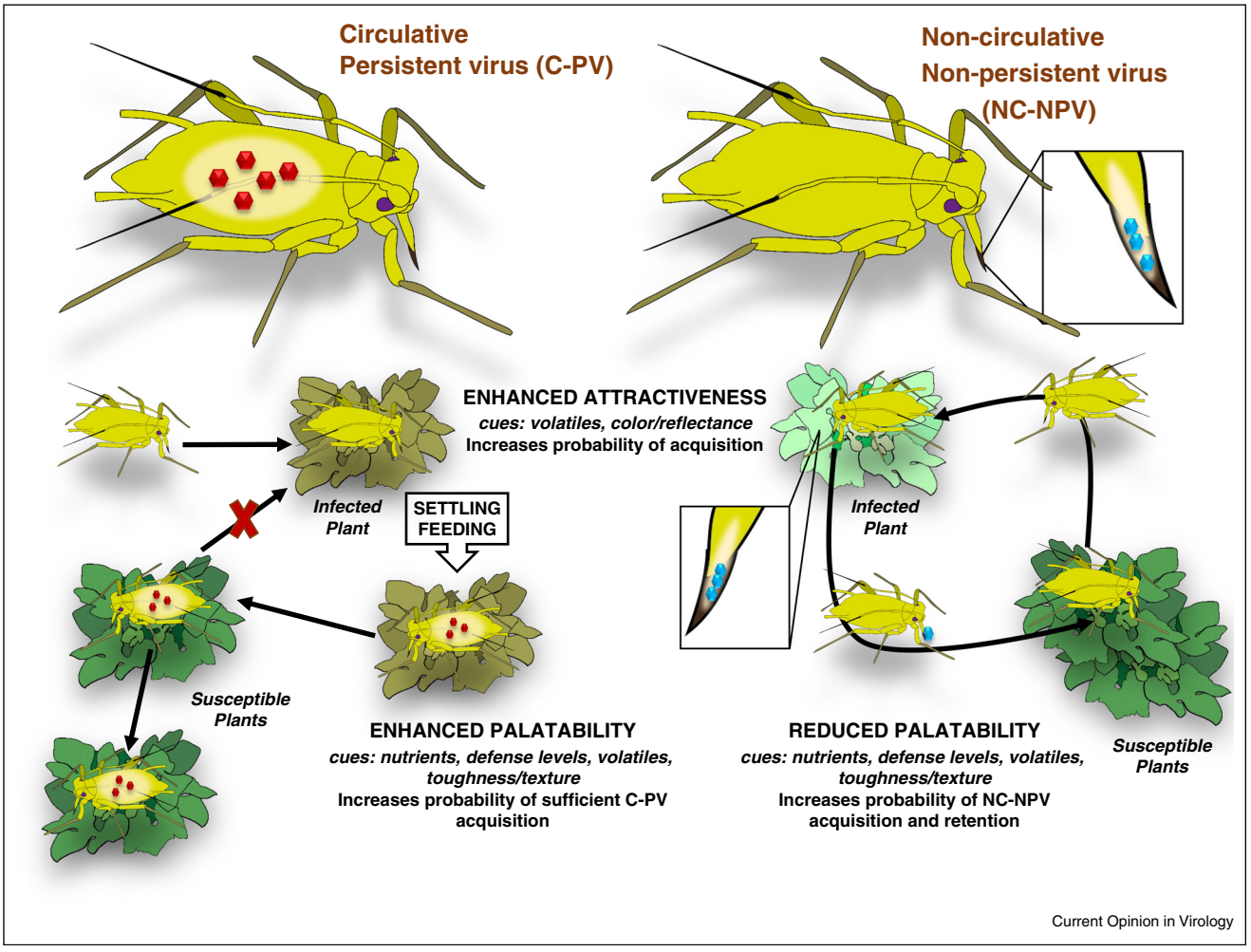
## Introduction

Virus infection can fundamentally alter the way that host plants interact with other organisms. In particular, many viruses change host plant phenotypes in ways that influence interactions with vectors [1–3,4•], with significant implications for virus transmission. These phenotypic changes involve alteration of visual or odor cues mediating vector orientation to plants (recruitment), quality or palatability cues mediating feeding behavior (virus acquisition), and effects on vector movement from infected to susceptible hosts (virus transmission) [3,4•]. In the case of viruses that also circulate and/or replicate in their vectors, phenotype changes that permit discrimination between infected and healthy plants can also interact with direct effects of the virus on vector physiology and behavior [5–9]. For example, virus-free aphids prefer to settle and feed on wheat infected with *Barley yellow dwarf virus* (BYDV), but once aphids have acquired the virus, their settling preferences change to favor healthy plants, facilitating virus spread [5]. This example, along with other recent studies reporting complex, transmission-conducive effects of viruses on plants and vectors [4•,10], support the hypothesis that such effects are the result of specific viral adaptations, and are not just by-products of pathology. However, much of this work has ignored natural genetic variation in virus and host plant populations. Among insect-vectored viruses, genetic diversity is generated by mutations that occur during replication (particularly for RNA viruses) coupled with biotic and abiotic heterogeneity in plant communities [11–14], and variation in vector competencies or host preferences [15]. Despite this diversity, most studies to date involve cultivated model host plants infected with virus strains originally isolated from monocultures and subsequently maintained in the laboratory [3,4•]. A more robust test of the adaptive significance of virus effects on plant phenotypes would be one that considers natural genetic variation in both the virus and the host plant, as well as the ecological context in which different virus isolates have evolved. This review highlights examples of recent progress toward this goal and synthesizes this work to gain insight into the factors shaping the evolution of virus effects on plant cues mediating plant–vector interactions.

## Expectations for virus effects on host plant phenotypes

Since the earliest reports of viruses influencing vectors via a shared host plant [16], there has been speculation about whether these effects constitute evidence of specific virus adaptations for manipulating plant phenotypes in ways that enhance transmission. Unlike clear cases of manipulation involving higher organisms (reviewed in [17,18]), for plant viruses it is often difficult to distinguish adaptive effects from by-products of infection because viruses alter suites of existing cues, such as volatile emissions or free amino acids [19,20], rather than inducing complex morphological [21•] or behavioral [18] changes. Nonetheless, given the importance of host cues for vectors, selection should tend to favor virus genotypes that alter plant phenotypes in ways that are generally conducive to transmission (no effect or a positive change) and disfavor virus genotypes that change plant phenotypes in ways that have clear negative effects on transmission [3,4•].

**Figure 1**




Expectations for transmission-mechanism specific effects of viruses on host plant phenotypes. Plant viruses can be either circulative or non-circulative. Circulative viruses are acquired during long-term feeding, usually in the phloem, after which they circulate within the vector (sometimes replicating) and migrate to specific tissues, such as salivary glands, from which they can be inoculated to multiple plants. Since long bouts of feeding are required for circulative virus acquisition and inoculation, it is expected that these viruses should have neutral to positive effects on plant attractiveness (to encourage vector contacts) and palatability or quality (to ensure uptake of a sufficient number of virions). Following virion acquisition, it is beneficial for the virus if the vector disperses from the infected plant and then exhibits a preference for healthy plants (as for C-PVs, shown above). In contrast, acquisition of most non-circulative viruses (particularly NC-NPVs, shown above) is favored by vectors making brief probes of non-vascular epidermal cells, then rapidly dispersing from infected plants to healthy plants. Non-circulative viruses are not retained internally, instead binding to specific regions of the mouthparts (e.g. NC-NPVs that adhere to aphid stylets, shown above) or foregut (most semi-persistently transmitted viruses, not depicted) for a few hours to a few days. This transmission mechanism should be facilitated by phenotypic changes to hosts that render them attractive to vectors, but less palatable following acquisition of plant cues and virions in order to encourage the rapid dispersal necessary for transmission. It is beneficial for a non-circulative virus if this shift in preference is temporary, as this will ensure that vectors do not permanently avoid infected hosts. For comprehensive reviews on each transmission mechanism, see [65,66].

One key line of evidence supporting the hypothesis that virus effects on plant cues are not mere by-products of infection is the apparent convergence of phenotypic effects across distantly-related pathogens transmitted in the same way [3,4•]. Viruses sharing a transmission mechanism will benefit from similar sequences of vector orientation, feeding, and dispersal behavior, and are thus expected to induce similar phenotypic shifts in host plants (Figure 1). This hypothesis has been recently discussed in two reviews [3,4•] which document clear patterns of congruency in virus effects based on shared transmission mechanism. But despite this broad pattern, individual reports of apparently mal-adaptive effects also exist (Table 1). This seems unlikely to be the result of publication bias, since mal-adaptive effects of a virus on its own transmission are still of ecological interest [22]. Rather, these reports may constitute evidence that viruses experience trade-offs in their ability to alter the phenotypes of multiple plant genotypes or species.