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The evolution of plant virus transmission pathways

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HIGHLIGHTS

- We use adaptive dynamics theory to study the evolution of plant viruses.
- Coexistence of infected and healthy plants is impossible in absence of pollen transmission.
- Evolutionary bistability may prevent vector transmission to replace pollen transmission.
- Evolutionary branching may lead to the coexistence of vector borne and non-vector-borne strains.

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ABSTRACT

The evolution of plant virus transmission pathways is studied through transmission via seed, pollen, or a vector. We address the questions: under what circumstances does vector transmission make pollen transmission redundant? Can evolution lead to the coexistence of multiple virus transmission pathways? We restrict the analysis to an annual plant population in which reproduction through seed is obligatory. A semi-discrete model with pollen, seed, and vector transmission is formulated to investigate these questions. We assume vector and pollen transmission rates are frequency-dependent and density-dependent, respectively. An ecological stability analysis is performed for the semi-discrete model and used to inform an evolutionary study of trade-offs between pollen and seed versus vector transmission. Evolutionary dynamics critically depend on the shape of the trade-off functions. Assuming a trade-off between pollen and vector transmission, evolution either leads to an evolutionarily stable mix of pollen and vector transmission (concave trade-off) or there is evolutionary bi-stability (convex trade-off); the presence of pollen transmission may prevent evolution of vector transmission. Considering a trade-off between seed and vector transmission, evolutionary branching and the subsequent coexistence of pollen-borne and vector-borne strains is possible. This study contributes to the theory behind the diversity of plant–virus transmission patterns observed in nature.

1. Introduction

Plant viruses naturally spread through three main transmission pathways: pollen, seed, and vector. Many plant viruses have vectors, providing the means for horizontal transmission from plant-to-plant (Gray and Banerjee, 1999; Bragard et al., 2013). Although various organisms serve as plant viral vectors, insects represent the most important group (Hull, 2014). Seed transmission (Sastry, 2013) serves as a major route for long-distance dissemination, provides an initial local source of inoculum for spread by vectors, and through vertical transmission enables virus survival at times when vector populations crash or go locally extinct. Just over 100 plant viruses are known to be seed-borne (Revers and Garcia, 2015). Virus transmission through pollen is also known (Mink, 1993; Card et al., 2007), which provides a pathway for an indirect form of vertical transmission, i.e. from an infected donor plant to the progeny of a healthy receptor plant. In addition pollen can provide a pathway for direct horizontal transmission. Finally, contact transmission can also occur, but there is little quantitative data on its occurrence in natural settings (Sacristán et al., 2011).

Vector transmission requires an active association with the virus, unlike passive transfer of infected pollen by insects. Specific interactions between virus and vector factors occur regardless of

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the type of virus/vector association, i.e. non-persistent (virus on mouth-parts/stylet of vector leading to short term transmission) or semi-persistent (movement of the virus to the foregut) (Ng and Falk, 2006; Blanc et al., 2011). For stylet-borne viruses the virus determinants for insect transmission reside on the viral coat protein (CP) (Ng and Perry, 2004). Some viruses, such as potyviruses, even have an additional virus-encoded protein, helper-component proteinase (Pirone and Blanc, 1996; Ng and Falk, 2006) which acts as a bridge for direct interaction with receptors on the aphid stylet as well as viral CP to facilitate vector transmission.

Plant viruses combine seed, pollen, and vector transmission pathways in a diverse and puzzling manner, in which it is difficult to discern consistent trends. Appendix A reports contrasting patterns as represented in extant virus species, documented from the literature, although restricted to the case of positive-sense single-stranded RNA (+ssRNA) viruses known to infect hosts with an annual life history. Some plant virus species have no known vector (Table A1), some others seem to be transmitted only by vectors (Table A2), while the majority combine seed/pollen and vector transmission (Table A3). Importantly, strains of the same virus species may be transmitted differently (Evans et al., 1970; Carroll, 1972; Stewart et al., 2005).

There may be a trade-off among modes of virus transmission. For instance, horizontal transmission rate is positively correlated with virulence (measured as the reduction in lifetime viable seed output of the host) in the *Barley stripe mosaic virus* (BSMV) – barley (*Hordeum vulgare*) system (Stewart et al., 2005). This suggests a trade-off between vertical (seed) and horizontal (vector) transmission in this virus species. Similarly in *Cucumber mosaic virus* (CMV) aphid (*Aphis gossypii*) transmission rate positively correlates with virus accumulation in tomato (*Lycopersicon esculentum* Mill.) (Escriu et al., 2000), whereas CMV virulence (measured as the negative effect of infection on plant fecundity) positively correlates with virus accumulation in *Arabidopsis thaliana* (Pagán et al., 2014). Thus when vector transmission correlates positively with virulence (negatively with fecundity) less seed transmission can occur suggesting a trade-off between seed and vector transmission. In addition, sequence variation in viral motifs may enhance and reduce different modes of virus transmission thus leading to a direct trade-off among transmission modes. For example, it was shown that a single amino-acid substitution in the coat protein (CP) coding region of *Soybean mosaic virus* (SMV) can both enhance aphid transmission rate and reduce seed transmission rate (p413-CP2mut; Jossey et al., 2013). More generally, there may be a trade-off between plant (pollen, seed) and animal (vector) transmission.

In this paper we do not address whether vector transmission preceded seed/pollen transmission or the reverse. Rather we concentrate on the mechanisms enabling seed/pollen and vector transmission to coexist in an evolutionarily stable manner, be it at the individual level or at the population level. Also, we investigate under what circumstances vector transmission is selected against pollen/seed transmission or the reverse. In particular, do climatic or latitudinal changes (longer growing seasons) select for vector or pollen/seed transmission (Jansen and Mulder, 1999; Garrett et al., 2006, 2009)? Also, does host adaptation or breeding for tolerance (lower virulence; Boots and Bowers, 1999; Jeger et al., 2006) select for vector or pollen/seed transmission?

To study the ecological and evolutionary interplay between plant virus transmission pathways, we developed a semi-discrete model taking into account vector (horizontal), seed (direct vertical) and pollen (horizontal) transmission pathways. For simplicity, we restricted the model to an annual plant population, with the rationale that in annual plants, fertilization and seed production is obligatory for population persistence. This led us to express an epidemiological invasion threshold based on the basic reproductive number of the virus, taking into account different combinations of the transmission pathways. Next we explored the circumstances under which vector transmission is selected against pollen/seed transmission or the reverse.

2. Ecological model

We focus on annual plants with indeterminate flowering. More specifically, we assume that seed germination and seedling emergence occur on a shorter time scale than vegetative plant growth, flowering and seed set/maturation. Flowering may occur at any time during the growth period, which therefore corresponds to the pollination period as well. At the end of the growth and pollination period, seeds drop, and eventually plant dies. Seeds that survive the overwintering period start a new cycle. We assume there is no seed bank.

There are three methods for viral transmission to a host plant: infected vectors, infected pollen and infected seeds. Vector acquisition of virus and inoculation of host plants may occur during the growth and pollination period. Vector transmission therefore overlaps with pollen transmission. We also assume that virus infection of the plant quickly becomes systemic. In particular, the virus is assumed to quickly spread from the vegetative tissues of the inoculated plant to the seeds.

Based on the transmission assumptions, a semi-discrete model is formulated (Mailleret and Lemesle, 2009; Fabre et al., 2012, 2015). We model the annual life cycle, t to $t+1$, in two parts, the growth and pollination period $t \to t+\tau$ and the survival and germination period $t+\tau \to t+1$. During the growth and pollination period $\tau < 1$ year, hereafter the growing season, a continuous-time model accounts for vector acquisition and inoculation of plants and pollen transmission. During the remainder of the year $1-\tau$, a discrete-time model accounts for seed survival and germination.

Let $H(t)$ and $I(t)$ denote the densities of healthy and infected plants, respectively, at time t, the beginning of the growing season. The total plant density is denoted as $T(t) = H(t) + I(t)$.

To keep the model simple, we assume the virus/vector association is non-persistent (Gray and Banerjee, 1999; Bragard et al., 2013) and leave vector dynamics implicit. Also, vector transmission is assumed to depend on the frequency of healthy plants, whereas pollen transmission is assumed to depend on the density (Appendix B; Thrall et al., 1995). Hence, the vector transmission rate per infected plant is $\beta H/T$ and the pollen transmission rate is αH. We refer to the parameter β as the vector transmission coefficient and to the parameter α as the pollen transmission coefficient.

More specifically, it is shown in Appendix B that one can express the vector transmission coefficient β as the product of five parameters: $\beta = \varepsilon \vartheta \Phi^2 U/\Lambda$, where ε is the probability that a viruliferous vector inoculates the virus to an uninfected plant, ϑ is the probability that a vector feeding on an infected plant acquires the virus, Φ^2 is the square of the vector feeding rate, U is the total vector density and finally, $1/\Lambda$ is the mean time during which transmission occurs. The parameter most subject to evolutionary pressure acting on β may be the acquisition rate ϑ since specific molecular interactions may occur between the virus and the vector receptors.

During the growing season, t to $t+\tau$, the healthy and infected plant densities are modeled as a system of differential equations with initial conditions $H(t)$ and $I(t)$,

$$\frac{\mathrm{d}H(s)}{\mathrm{d}s} = -\left(\alpha + \frac{\beta}{T(s)}\right) H(s) I(s), \quad \frac{\mathrm{d}I(s)}{\mathrm{d}s} = \left(\alpha + \frac{\beta}{T(s)}\right) H(s) I(s) \tag{1}$$

for $t \le s \le t+\tau$. Since the total plant density is constant, $T(s) = T(t)$, the healthy and infected plant densities at the end of the growing season can be easily computed (Appendix C):

$$H(t+\tau) = \frac{T(t)}{1 + \left(\frac{T(t)}{H(t)} - 1\right) \exp\left((\alpha T(t) + \beta)\tau\right)}$$
$$I(t+\tau) = T(t) - H(t+\tau). \tag{2}$$

For the remainder of the year, we model the dynamics as a simple difference equation. Let b_H and b_I denote the average number of