The evolution of plant virus transmission pathways

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Plant viruses

Examples

- *Potato Virus Y* (PVY)
  - considerable losses to e.g. the South African potato industry
  - transmitted in a non-persistent manner by aphids
- *African Cassava Mosaic Virus* (ACMV)
  - one of the most detrimental diseases affecting food supply in Africa
  - transmitted in a persistent manner by the whitefly *B. tabaci*

Plant viruses transmission pathways

- **Seed** (vertical) transmission

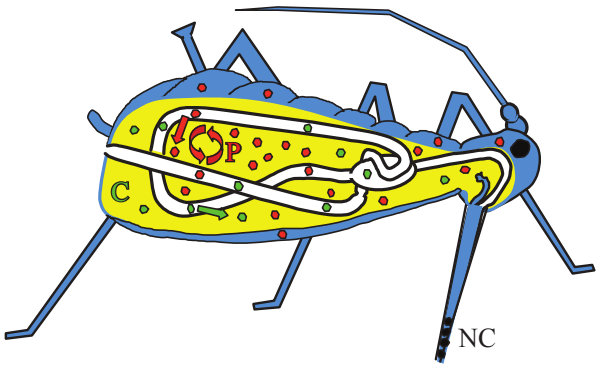
  can be *perfect* (success rate is 100%) or *imperfect*

- **Pollen** (horizontal and/or vertical) transmission

  i.e. from an infected donor plant to a healthy receptor plant and/or its progeny

- **Vector** (horizontal) transmission

  requires an active association with the virus, unlike passive transfer of infected pollen by insects.



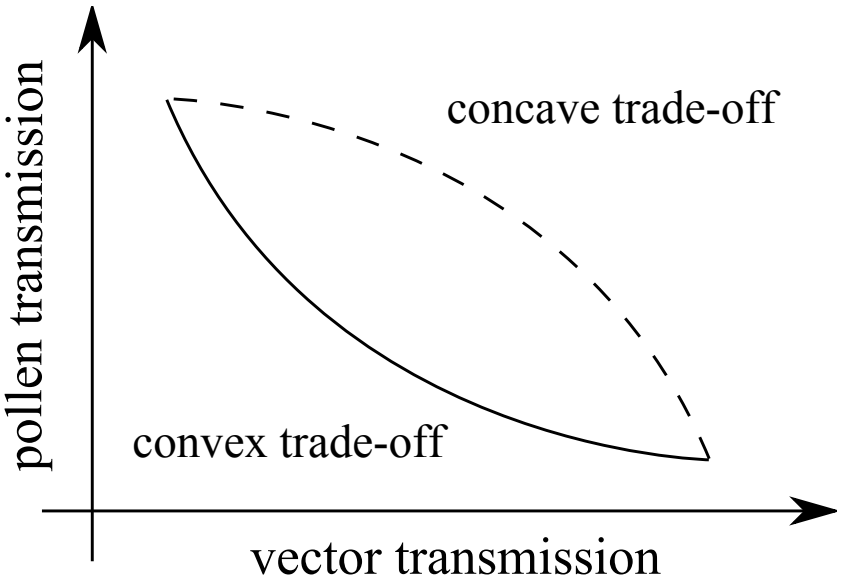
- Non-circulative (virus on mouth parts/stylet) often *non-persistent*
- Circulative/propagative (movement of the virus to the foregut) often *persistent*

[1]

[1] Froissart et al (2010)

Trade-offs

Negative correlations between horizontal and vertical transmission e.g.



as reported in *Barley Stripe Mosaic Virus* (BSMV), *Cucumber Mosaic Virus* (CMV), and *Soybean mosaic virus* (SMV)[1].

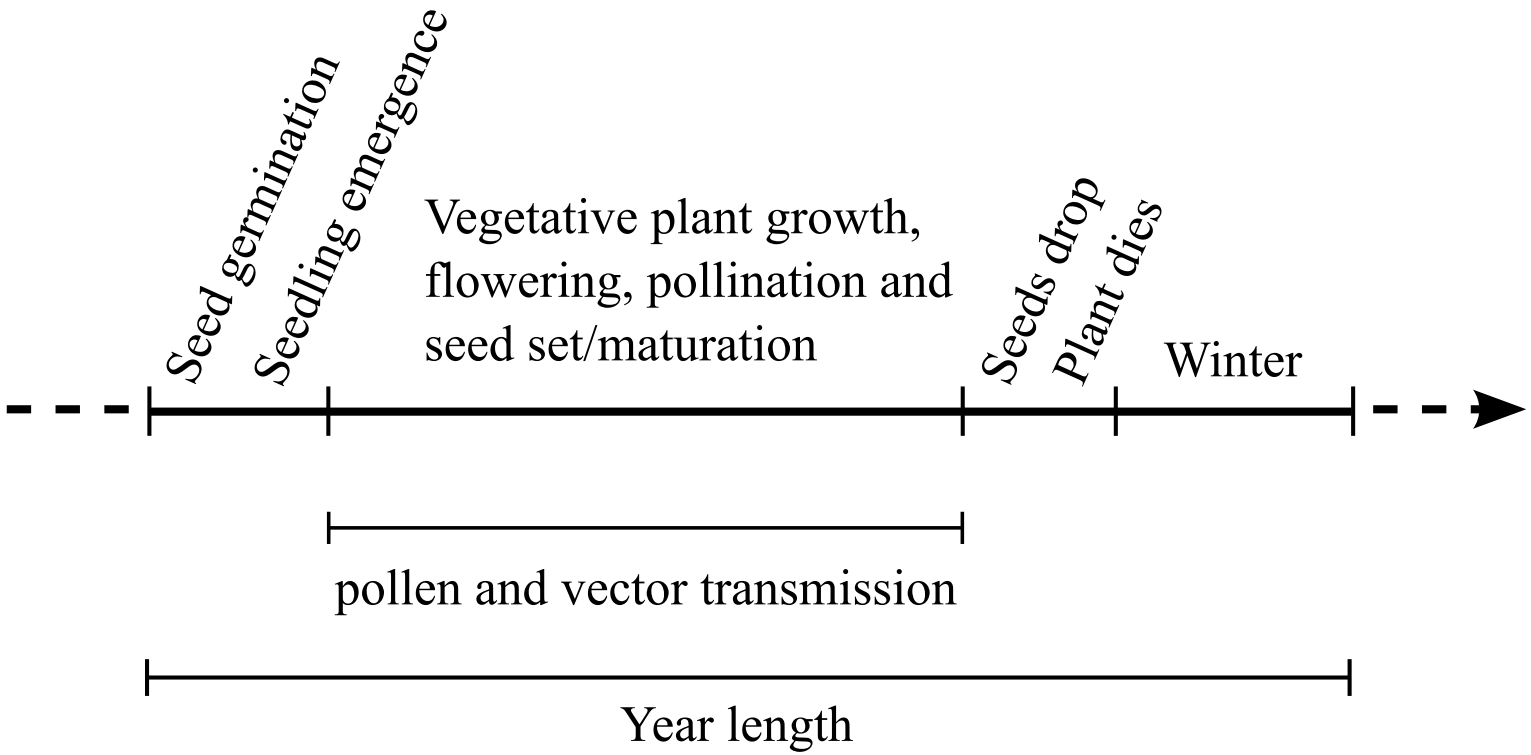
[1] Escriu et al 2000; Stewart et al 2005; Pagán et al 2014; Jossey et al 2013

Questions

- We do not address whether vector transmission preceded seed/pollen transmission during the course of evolution.
- Rather we concentrate on the mechanisms enabling seed/pollen and vector transmission to coexist in an evolutionarily stable manner.
- We address the questions:
  (i) Can vector transmission invade and replace pollen transmission?
  (ii) Can evolution lead to the coexistence of multiple virus transmission pathways?
- We consider 2 trade-offs:
  (i) pollen versus vector transmission
  (ii) seed versus vector transmission

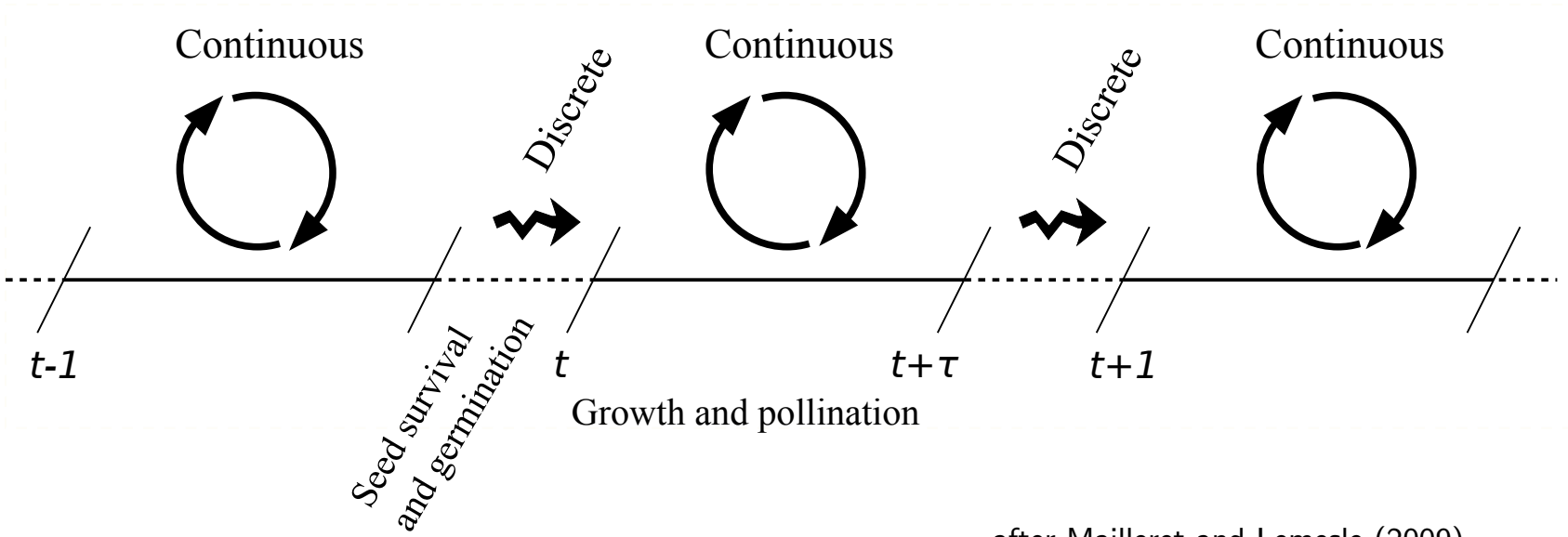
Modelling assumptions

- annual plant host, indeterminate flowering, no seed bank
- infection quickly systemic, non-persitent vector transmission



Semi-discrete model

- We assume that seed survival and germination occur on a shorter time scale than growth and pollination
- $t = 0, 1, 2, \ldots$ is the time in years
- $\tau < 1$ is the length of the growing season



after Mailleret and Lemesle (2009)

- horizontal infections occur continuously during the season

Continuous-time in-season epidemiological dynamics

- non-persistent transmission $\rightarrow$ implicit vector dynamics
- H and I are the densities of healthy and infected plants
- $T = H + I$ is the total plant density
- αH is the pollen transmission rate (density-dependent)
- $\beta H/T$ is the vector transmission rate (frequency-dependent)

For $t \leq s \leq t + \tau$,

$$\begin{aligned} \frac{\mathrm{d}H(s)}{\mathrm{d}s} &= -\left(\alpha + \tfrac{\beta}{T(s)}\right) H(s)I(s)\,, \\ \frac{\mathrm{d}I(s)}{\mathrm{d}s} &= +\left(\alpha + \tfrac{\beta}{T(s)}\right) H(s)I(s)\,. \end{aligned}$$

Discrete-time inter-seasonal dynamics

- b_H and b_I are number of seeds per healthy and infected plants
- We assume $b_H > b_I \geq 1$ (so the plant population persists)
- p is the seed transmission probability
- λ is a seedling competition coefficient

From $t + \tau$ to $t + 1$

$$H(t+1) = \frac{b_H H(t+\tau) + (1-p) b_I I(t+\tau)}{1 + \lambda T(t+\tau)}$$

$$I(t+1) = \frac{p b_I I(t+\tau)}{1 + \lambda T(t+\tau)}$$

- We then assume $p = 1$ (perfect vertical transmission)

Epidemiological points

- A disease-free population can be invaded by infected individuals iff

$$\mathcal{R}_0 = \frac{b_I}{b_H} \exp\left(+\left(\beta + \alpha\frac{b_H - 1}{\lambda}\right)\tau\right) > 1$$

- A fully infected population can be invaded by non-infected individuals iff

$$\overline{\mathcal{R}_0} = \frac{b_H}{b_I} \exp\left(-\left(\beta + \alpha\frac{b_I - 1}{\lambda}\right)\tau\right) > 1$$

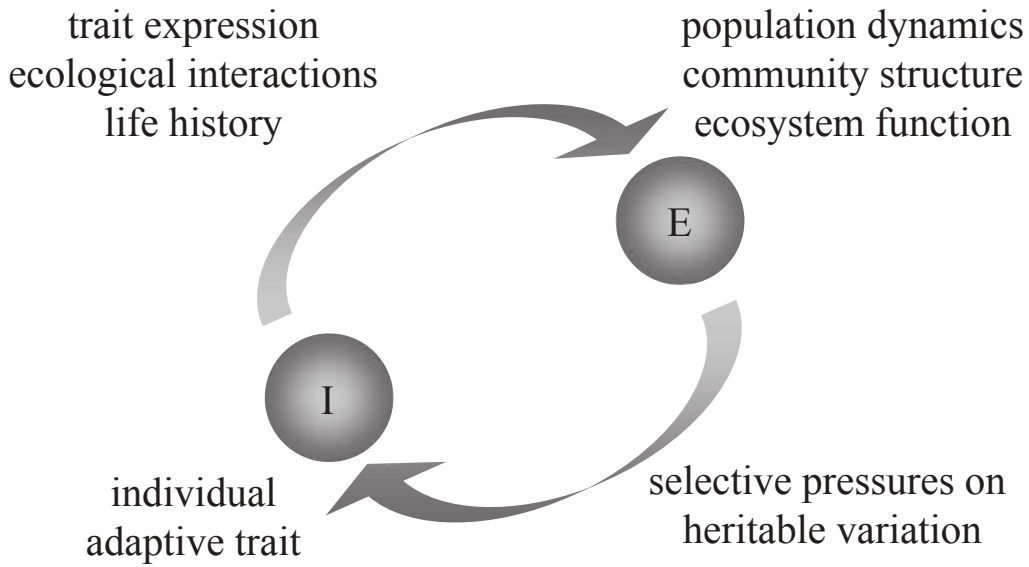
- Coexistence of infected and healthy plants is possible iff

$$\mathcal{R}_0 > 1 \quad \text{and} \quad \overline{\mathcal{R}_0} > 1$$

which requires $\alpha > 0$ (nonzero pollen transmission).

Eco-evolutionary feedback loop

- vector (frequency-dependent) transmission is expected to be selected against pollen (density dependent) transmission at low population density, and conversely
- population density in turn depends on virus characteristics, which creates an eco-evolutionary feedback loop, and may lead to polymorphism



from Ferrière and Legendre (2013)

Evolutionary invasion analysis

Adaptive Dynamics[1], a way to model phenotypic evolution

- consider a **resident** population I_1 at ecological equilibrium
- challenge it with a small **mutant** sub-population $I_2 \ll I_1$
- $x_1 = (\alpha_1, \beta_1, b_1)$ is the resident phenotype
- $x_2 = (\alpha_2, \beta_2, b_2)$ is the mutant phenotype

Mutant invasion condition:

$$\log\left(\frac{I_2(1)}{I_2(0)}\right) = \underbrace{\log\left(\frac{b_2}{b_1}\right)}_{\substack{\text{seed} \\ \text{transmission}}} + \underbrace{(\alpha_2 - \alpha_1)\tilde{H}_1\tau}_{\substack{\text{pollen} \\ \text{transmission}}} + \underbrace{(\beta_2 - \beta_1)\frac{\tilde{H}_1}{\hat{T}_1}\tau}_{\substack{\text{vector} \\ \text{transmission}}} > 0$$

where $\tilde{H}_1$ and $\hat{T}_1$ are mean healthy host and total host densities at equilibrium shaped by the resident phenotype x_1.

[1] Metz et al 1992; Dieckmann and Law 1996; Geritz et al. 1998; Diekmann 2004.

Pollen *versus* vector transmission

- To model a **trade-off between pollen and vector transmission**, we assume seed transmission is a constant ($b_1 = b_2 = b_I$) and let

$$\beta_i = f(\alpha_i)\,,\ \ i = 1, 2\,,$$

with f a decreasing function: $f'(\alpha) < 0$.

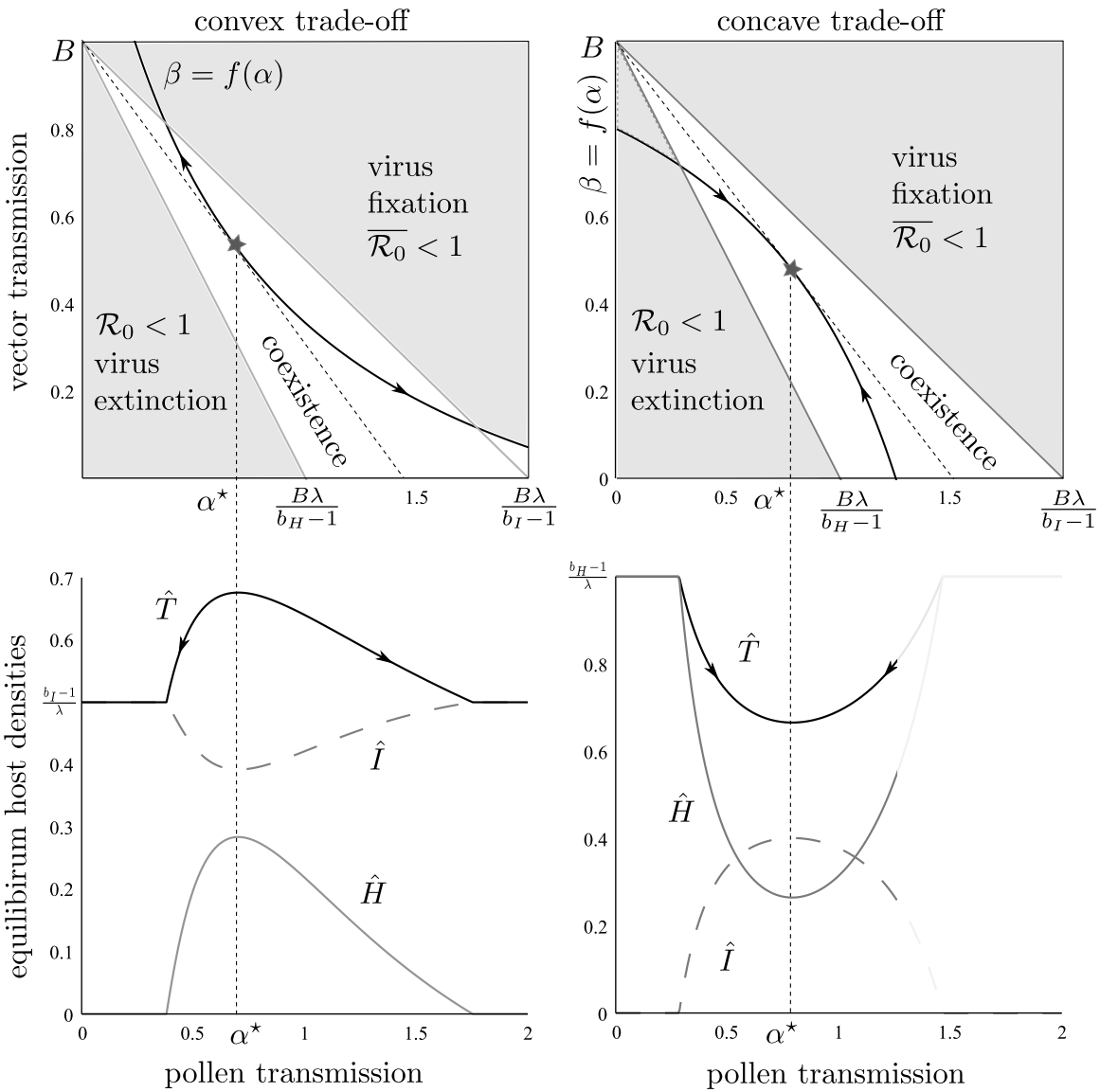
- **Invasion fitness proxy** (sign-equivalent to the invasion condition):

$$s(\alpha_1, \alpha_2) = (\alpha_2 - \alpha_1)\,\hat{T}(\alpha_1) + f(\alpha_2) - f(\alpha_1)\,,$$

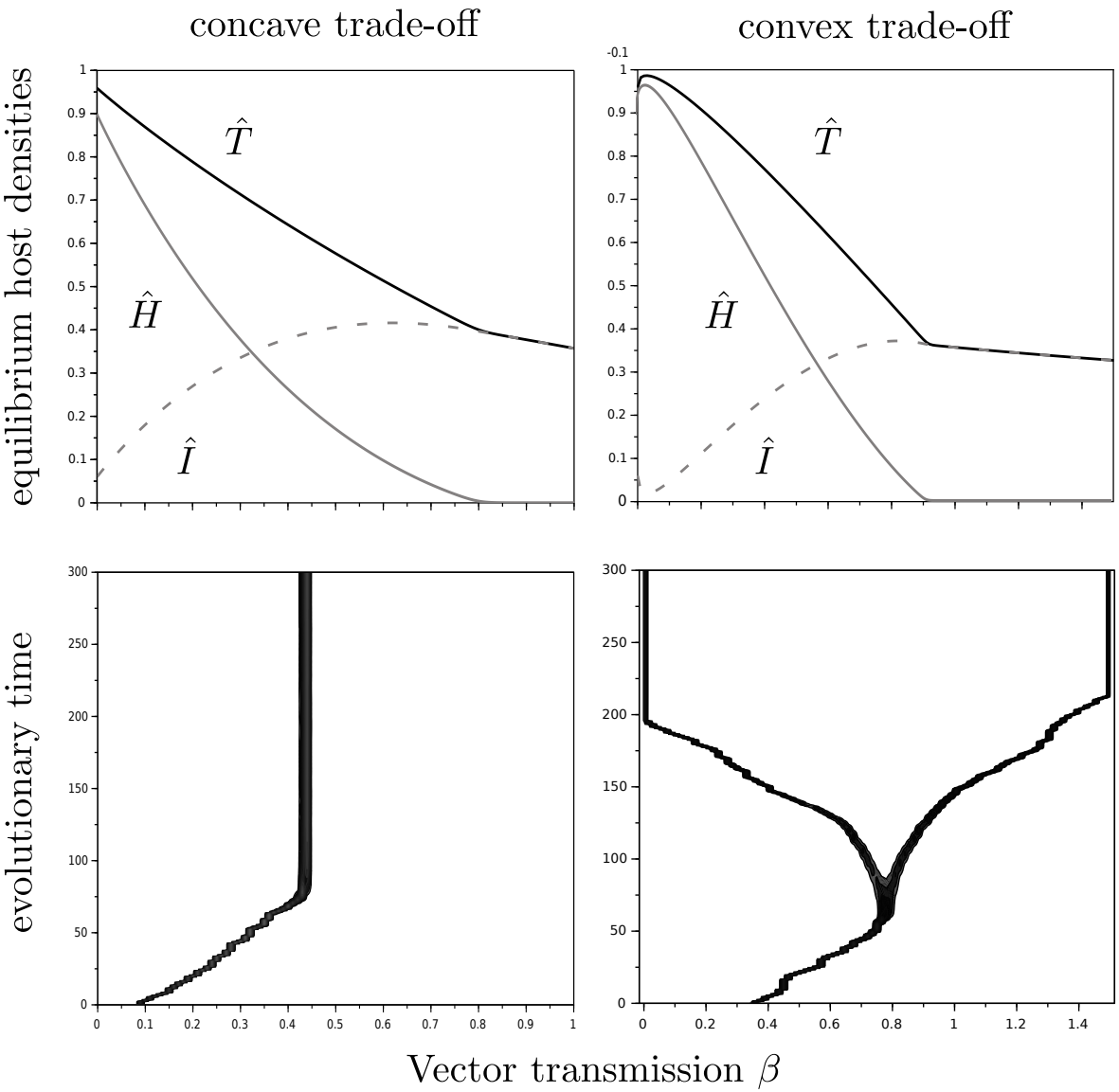
where $\hat{T}(\alpha_1) = \hat{T}_1$ is the total host density at equilibrium as shaped by the resident trait α_1.

- The dynamics of $s(\alpha_1, \alpha_2)$ as a function of α_2 determine the evolutionary trajectory.

Pollen *versus* vector transmission: optimization principle



Seed *versus* vector transmission: evolutionary branching



Eco-evolutionary insights

- Coexistence of healthy and infected plants requires nonzero pollen transmission
- Mixing vector and pollen transmission may be evolutionarily stable
- **Pollen** *versus* vector trade-off
  - Evolution minimizes total host density
  - Convex trade-off: evolutionary bistability is possible (min pollen/max vector or the reverse)
- **Seed** *versus* vector trade-off
  - No optimization principle
  - Convex trade-off: **evolutionary branching** is possible (coexistence of vector-borne and non-vector-borne variants)
- Convex trade-off: evolutionary bistability may prevent vector transmission to replace pollen transmission

Limits and perspectives

- Our model fits viruses specific to **annual plants** in temperate zones
  A perennial (tropical) system would require a continuous-time model
- Extending our study to persistent vector transmission would require making **vector dynamics** explicit
  This would increase the model complexity, but may lead to more general results in the 'pollen *versus* vector' trade-off case
- We assumed that plant viruses are parasites ($b_H > b_I$), and that vertical transmission was perfect ($p = 1$)

  Considering beneficial viruses ($b_I \geq b_H$; e.g. Rossinck, 2011) together with imperfect vertical transmission ($p \leq 1$) will allow us to address questions related to **viral symbiosis evolution**

Thank you

Investigative Workshop Vectored Plant Viruses, March 17-19, 2014

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Vector dynamics and frequency-dependent transmission

- I and V are the infected plant and viruliferous vector densities
- T and U are total plant and vector densities (assumed constant)
- Φ is the vector feeding frequency
- ϑ and ε are the vector probabilities to acquire and transmit the virus
- Λ is the rate at which the vector loses transmission ability

$$\frac{\mathrm{d}I(s)}{\mathrm{d}s} = \Phi V(s)\frac{T - I(s)}{T}\varepsilon\,,$$
$$\frac{\mathrm{d}V(s)}{\mathrm{d}s} = \Phi(U - V(s))\frac{I(s)}{T}\vartheta - \Lambda V(s)\,.$$

Non-persistent transmission simplifies vector dynamics

Let

$$s^* = \Phi\varepsilon s\,, \quad I^* = \frac{I}{T}\,, \quad V^* = \frac{V}{U}\,.$$

The dimensionless vector-plant model simplifies to

$$\frac{\mathrm{d}I^*}{\mathrm{d}s^*} = \frac{U}{T}V^*(1-I^*)\,,$$
$$\varepsilon\frac{\mathrm{d}V^*}{\mathrm{d}s^*} = \vartheta(1-V^*)I^* - \frac{\Lambda}{\Phi}V^*\,.$$

Assuming $\varepsilon \ll 1$ (low transmission probability), we apply the quasi-steady state approximation to the second equation to yield

$$V^* = \frac{I^*}{I^* + \frac{\Lambda}{\vartheta\Phi}} \approx \frac{\vartheta\Phi}{\Lambda}I^*\,,$$

since $\Lambda \gg \vartheta\Phi I^*$ (non-persistent virus). Letting $\beta = \varepsilon\vartheta\Phi^2 U/\Lambda$ yields

$$\frac{\mathrm{d}I(s)}{\mathrm{d}s} \approx \frac{\beta}{T}I(s)(T - I(s))\,.$$

Optimization principle in the pollen vs vector trade-off case

- Let $B = \log\left(b_H/b_I\right)/\tau$; using the fact that $\hat{T}(\alpha) = (B - f(\alpha))/\alpha$, the mutant **invasion fitness** proxy reads

$$s(\alpha_1, \alpha_2) = \alpha_2 \hat{T}(\alpha_1) - B + f(\alpha_2)\,.$$

- Since invasion fitness is both 1-dimensional and monotonous in the environmental variable $\hat{T}$, there is an optimization principle (Metz et al 2008; Gyllenberg and Service 2011)
- An evolutionary singular point $\alpha^\star$ is such that $\hat{T}'(\alpha^\star) = 0$
- **Evolution minimizes total host density** T
- We also have $\hat{T}''(\alpha) = -f''(\alpha)/\alpha$, hence the **convexity of the trade-off function** determines whether $\alpha^\star$ is an attracting or repelling point of the evolutionary dynamics