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The sustainable management of plant disease has two goals:

- □ Reducing severity and frequency of disease epidemics (immediate epidemiological goal),
- □ Reducing the rate of evolution of new patho-types (longer-term evolutionary).

Here, we model epidemiological and evolutionary dynamics of spore-producing pathogens in a homogeneous host population.



The model considers a continuum of different pathogen strains (denoted by their phenotypic value x). On quantitatively resistant hosts, pathogen exhibit a continuous distribution of their disease phenotype: all the pathogen strains cause infection but each with its own level of quantitative pathogenicity.



Figure 1. Grapevine infected by downy mildew.

Quantitative traits of pathogenicity of strain *x* : (*i*) $\beta(x)$, infection efficiency, (ii) $\tau(x)$, latent period, (iii) p(x), sporulation rate, (iv) l(x), infectious period.

Fitness.

The fitness of the pathogen with phenotypic value \boldsymbol{x} is given by the fitness function $\boldsymbol{\psi}$ above.

Evolutionary Strategy (ES).

The set of the fitness function maximum points is called the ES-set. And each element of that set is called an ES-

phenotypic value.



Evolutionary Stable Strategy (ESS): it's a phenotypic value such that, when the vast majority of the individuals has it, no rare mutant with a different phenotype can increase in numbers.

By using a suitable order, defined on the set of phenotypic values which maximize the fitness function, we characterize the ESS-phenotypic value.

MAIN RESULTS

□ The biological endemic steady state of the model, when it exists, is unique.

At the evolutionary equilibrium, the pathogen population is typically concentrated around the well-characterized ESS-phenotypic value (using the fitness function ψ).





Figure2: (Left) The fitness function ψ with respect to the phenotypic value space. The fitness function is maximized by a single phenotypic value x_2 and the phenotypic value x_1 is close to the maximum of the fitness function, i.e. $\psi(x_1) \approx$ $\psi(x_2)$ (as illustrated by the zoom in). In this case, x_2 is the ESS-phenotypic value. (Right) Dynamics of infectious tissues with respect to the phenotypic value space. Initially (i.e. at t = 0), the pathogen population is essentially concentrated around the phenotypic value x_1 . With the time and due to mutations in the space of phenotypic values, the pathogen population will typically concentrate around the ESS-phenotypic value x_2 . Notice that the concentration process around the phenotypic value x_2 is preceding by some co-existence of both phenotypes x_1 and *x*₂.

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Figure3: (Left) The fitness function ψ with respect to the phenotypic values space. The fitness function is maximized by two phenotypic value x_1 and x_2 . The phenotypic values x_1 and x_2 differ by their respective second derivative of the fitness function: $\psi''(x_2) > \psi''(x_1)$. According to a suitable order defined on the set of the fitness function maximum points, x_2 is then the ESS-phenotypic value. (Right) Dynamics of infectious tissues with respect to the phenotypic value space. Initially (i.e. at time t = 0), the pathogen population is essentially concentrated around the phenotypic value x_2 . Before reaching the ESS-phenotypic value x_2 , the pathogen population can evolve during long time around the phenotypic value x_1 followed by a co-existence of both phenotypes x_1 and x_2 . This is because in this current case the phenotypic value x_1 is much more close to the ESS-phenotypic value *x*² than in the case of **Figure 2**.

Further information

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