Improving the durability and efficiency of plant resistance deployment using eco-evolutionary modelling

Loup Rimbaud, Julien Papaix, Benoît Moury, Luke G. Barrett, Peter H. Thrall

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Rimbaud L[1], Papaïx J[2], Moury B[1], Barrett LG[3], Thrall PH[3]

[1]INRA, UR407 Pathologie Végétale, Avignon, France. [2]INRA, UR BioSP, Avignon, France. [3]CSIRO Agriculture & Food, Canberra, Australia

BACKGROUND Genetically-controlled plant resistance can reduce the damage caused by pathogens. However, pathogens have the ability to evolve and overcome the resistance. This often occurs very quickly after resistance is deployed, resulting in significant crop losses and continuing needs to breed new resistant cultivars. To tackle this issue, several strategies have been proposed to constrain the evolutionary potential of pathogen populations and thus increase resistance durability. These strategies mainly rely on using different combinations of resistance genes (e.g. qualitative and/or quantitative resistance) in time, space, or both (e.g. via gene pyramiding, cultivar rotations, cultivar mixtures, field mosaics). However, experimental assessment of the efficiency (i.e. ability to reduce disease impact) and the durability (i.e. ability to limit pathogen evolution and delay resistance breakdown) of different deployment strategies presents a major challenge.

MATERIALS & METHODS Therefore, we developed a spatially-explicit stochastic model [1] to assess the epidemiological and evolutionary outcomes of the major deployment options described above, for both qualitative (major resistance genes) and quantitative resistance (e.g. QTLs affecting different pathogen life-history traits). In addition, we analyzed the impact of landscape organization (as defined by the proportion of fields cultivated with a resistant cultivar, and their spatial aggregation) and epidemiological or evolutionary parameters (e.g. mutation probability, cost of infectivity).

RESULTS Our main results on resistance to rusts indicate that evolutionary and epidemiological control are not necessarily correlated [2], and that no deployment strategies is universally optimal [3].

DISCUSSION & CONCLUSION The model was first parameterized for cereal resistance to rusts (caused by fungi of the genus *Puccinia*), and is destined to be applied to other pathosystems including viruses of vegetable crops, especially Potato virus Y on pepper (*Capsicum spp.*).

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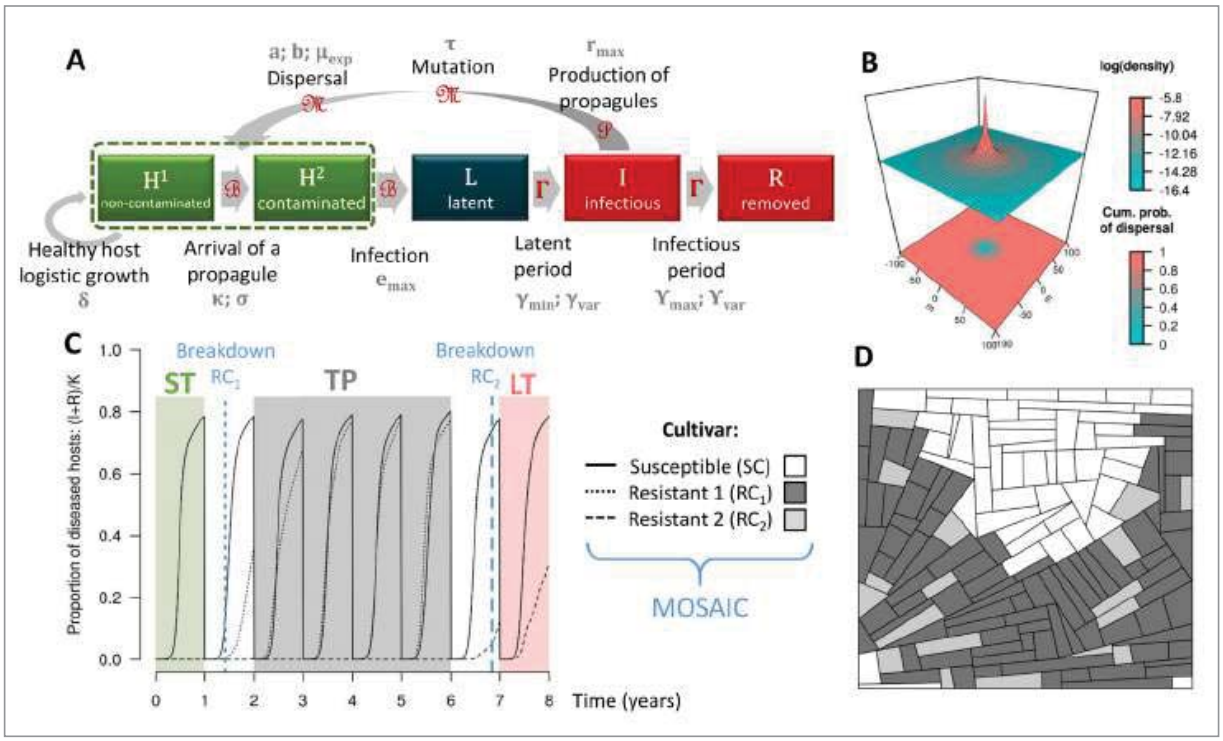

Figure 1. Model overview. (A) Model architecture. Healthy hosts can be contaminated by propagules and may become infected. Following a latent period, infectious hosts produce new propagules which may mutate and disperse across the landscape. At the end of the infectious period, infected hosts become epidemiologically inactive. Qualitative resistance prevents transition to the latent infected state (L). Green boxes indicate healthy hosts which contribute to crop yield and host growth, in contrast to latent hosts (dark blue box) and diseased hosts (i.e. symptomatic, red boxes). Model parameters associated with epidemiological processes are indicated in grey. Distributions used to simulate stochasticity in model transitions are indicated in red; B: binomial, Γ: gamma, P: Poisson, M: multinomial. Host growth is deterministic. (B) Two-dimensional representation of the power-law dispersal kernel calibrated for rust pathogens. Top panel indicates the logarithm of the probability to disperse from the origin to any point of the landscape; bottom panel indicates the cumulative probability of dispersing over a given distance. (C-D) Example of simulation with two major resistance genes deployed as a mosaic: (C) dynamic of diseased hosts; (D) landscape. Blue vertical lines indicate the durability of the two resistant cultivars. These lines delineate the three periods used to compute epidemiological outputs from AUDPC: short-term (ST, green area), transitory period (TP, grey) and long-term (LT, red).

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