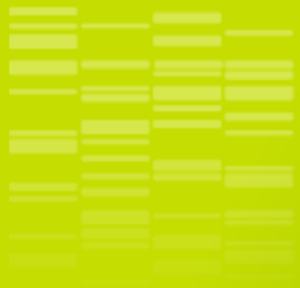

Emmerging spatial structures drive pathogen evolution in coevolving plant-pathogen systems

Julien Papaïx, Jeremy Burdon, Christian Lannou, Peter Thrall

Introduction

Importance of spatial and temporal heterogeneities in environmental conditions in determining the coexistence and evolutionary trajectory of populations and species.

In homogeneous conditions, when species interact, migration rates of one of them affect the heterogeneity of selection on the other:
$\Rightarrow$ the spatial scale at which species interact is important in determining coevolutionary trajectories.

How the geographical structure of coevolution may shape spatial patterns of variation in the coevolving species ?

Model

General considerations

Stochastic and discrete-time SEI metapopulation model in which plant and pathogen populations are inter-connected via dispersal of propagules.

The **host is composed by two fixed genotypes** with no mutations between them but the **pathogen evolves gradually** by small steps. Pathogen adaptation on the two host genotypes is constrained by a gaussian trade-off.

Infected plants do not produce seeds anymore.

Genetic interaction between the host and the pathogen impacts **only the infection efficacy**.

Seeds and spores that fail to establish are removed which implicitly imposes a **cost of dispersal**.

Model

Plant dynamics

Reproduction: S plants produce r_H seeds each day ($\sim$ Pois(r_H)),

Dispersal: seeds migrate from population i to population j with probability μ_{ij}^H computed from a Weibull function ($\sim$ Multi(nb seeds, $[\mu_{i1}^H ...]$)),

Establishment: seeds get established in population i with probability $\pi_i^H = 1 - \sum_h (S_{ih} + E_{ih} + I ih)/K_i$ ($\sim$ Bin(nb seeds i, π_i^H)),

Survival: S plants die with probability d^H ($\sim$ Bin(nb plants i, d^H)).

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Model

Pathogen dynamics

Latency: E plants remain latent during τ days ($\sim$ Bin(nb E, $1 - e^{-1/\tau}$)),

Reproduction: I plants produce r^P spores per day during T days after which they are removed ($\sim$ Pois(r^P), $\sim$ Bin(nb I, $1 - e^{-1/T}$)),

Mutation: Spores can mutate from genotype p to genotype p' with probability $m_{pp'}$ ($\sim$ Multi(nb spores, $[m_{p1}...]$)),

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Contamination: Spores contaminate a S plant with probability $\pi_i^P(x)$, where x is the proportion of S plants and $\pi^P(.)$ can take different shapes ($\sim$ Bin(nb S i, $\pi_i^P(x)$)),

Infection: Contaminated plants become infected (E) with a probability e_{ph}, the infection efficacy ($\sim$ Bin(nb S cont, e_{ph})).

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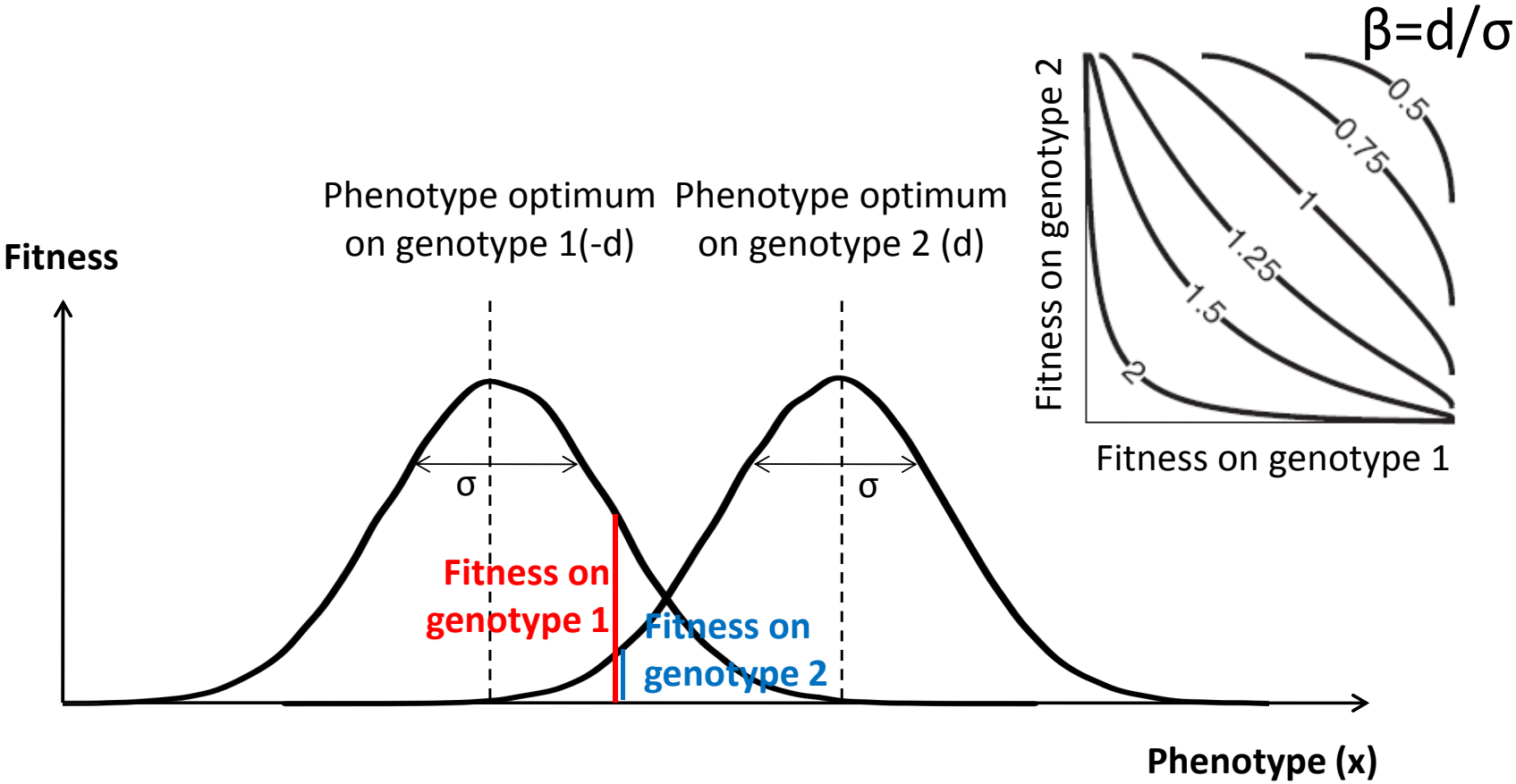
Trade-off function



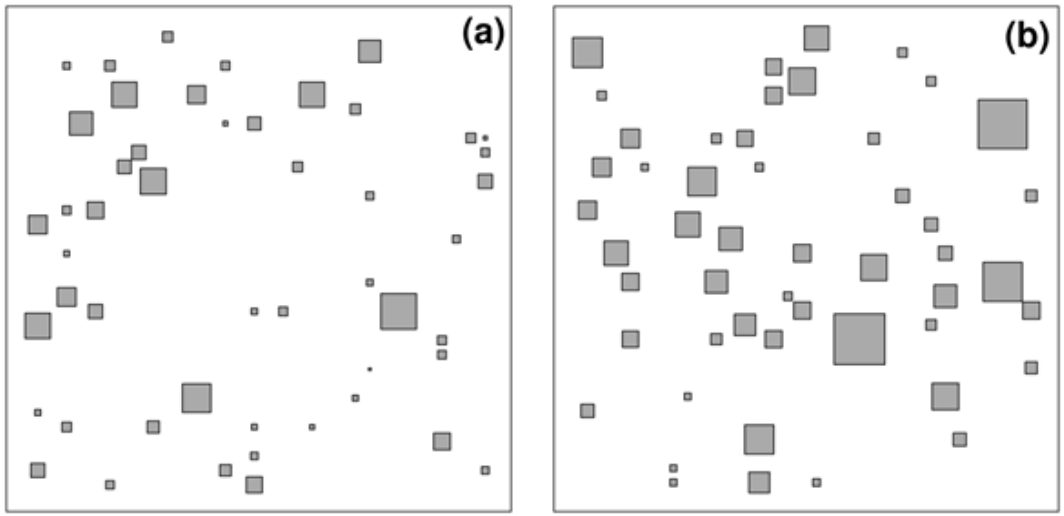
Model

Population network

The metapopulation consists of a network of $N = 50$ populations that covers a total proportion q of the environment.

Populations are located randomly via a homogeneous Poisson point process with surfaces drawn from a log-normal distribution.

Environmental conditions are homogeneous.



Simulation experiment

Parameters of interest:

- host and pathogen mean dispersal distances (μ_0^H and μ_0^P)
- trade-off shape (β)

The convergence was checked on a subset of simulations.

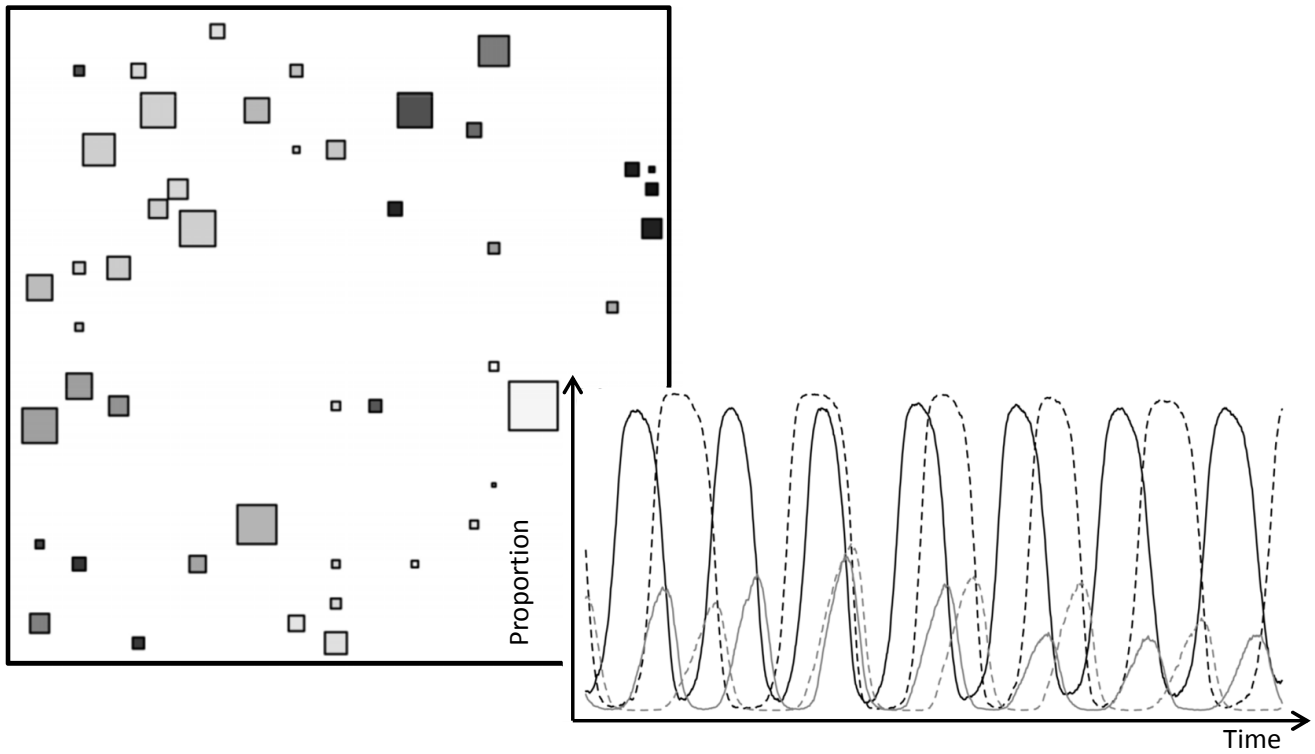
The system started with the two host genotypes present in all patches and with the pathogen population composed of a unique genotype with generalist features.

Sensitivity to r^P, T, τ, $\pi(.)$, b and q was assessed with no qualitative consequences.

Outputs

Host spatial structure

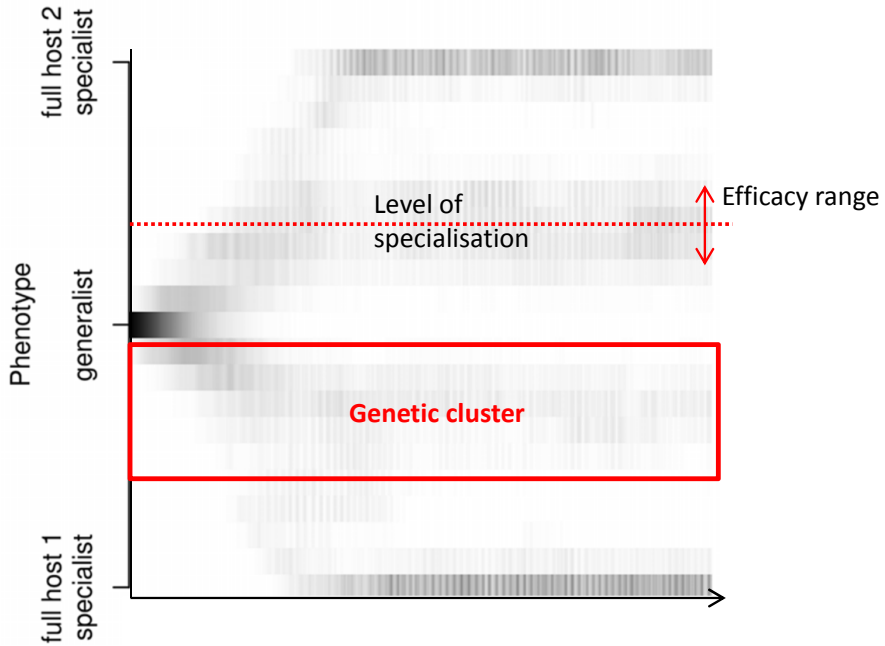
1. Global metapopulation synchrony: mean correlation among global and local dynamics.
2. Synchrony as a function of distance: spatial spline centred correlogram among local population dynamics.



Outputs

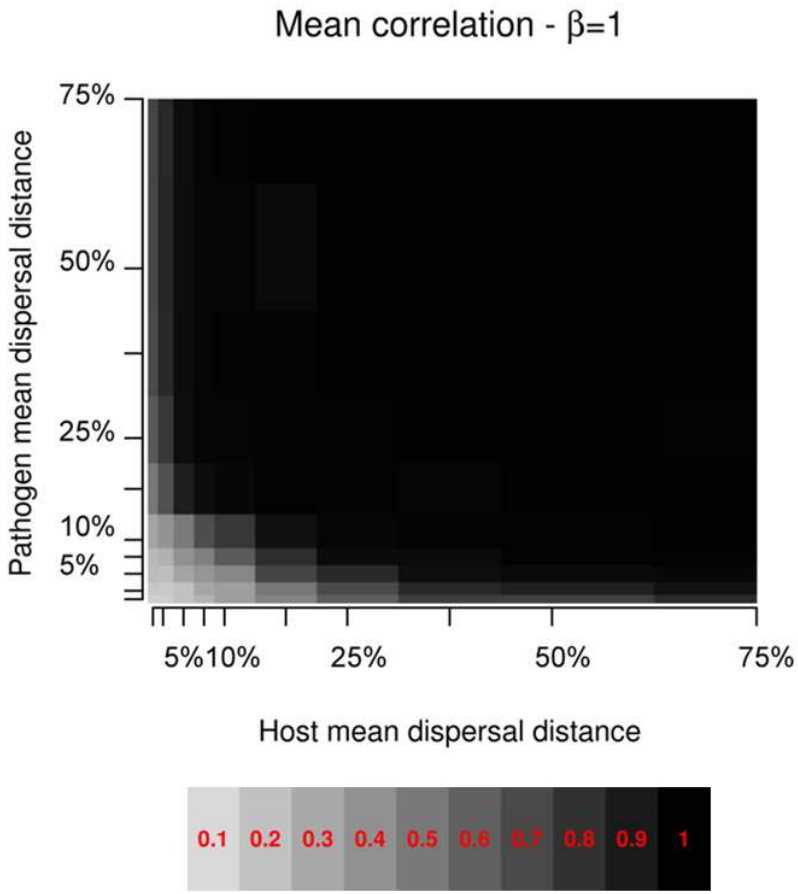
Pathogen evolutionary trajectories

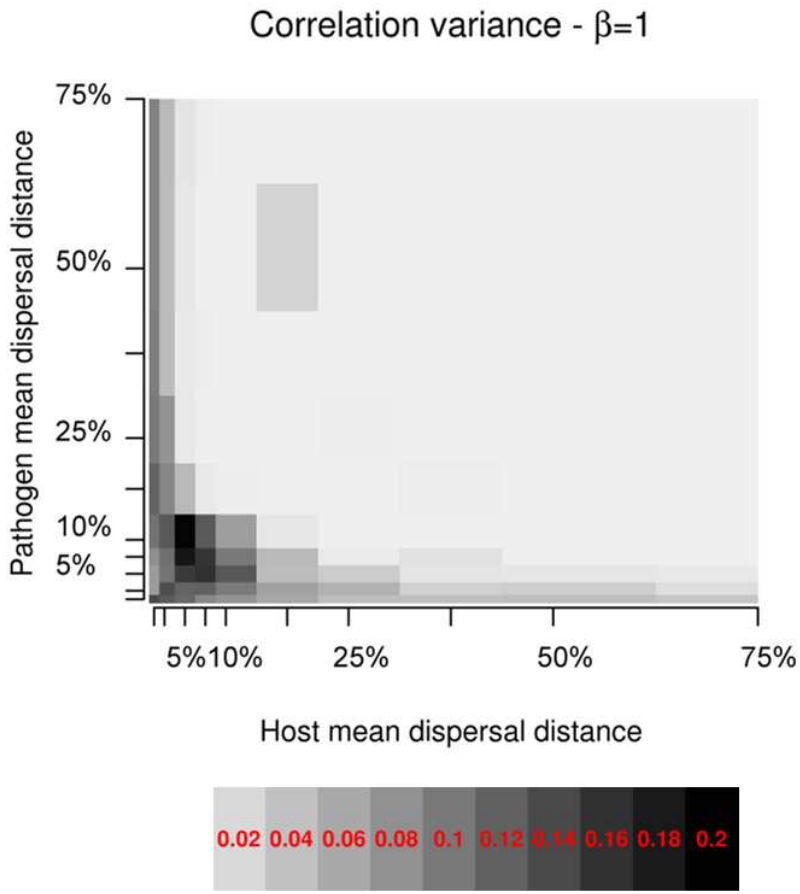
1. Coexistence among genetic clusters: number of stable genetic clusters that persisted.
2. Level of specialisation of genetic clusters: median of the infection efficacy over genotypes and time.
3. Efficacy range: $(2.5\%, 97.5\%)$ quantile interval of the infection efficacy over genotypes and time.



Results: spatial structure

Global synchrony in the host metapopulation dynamics





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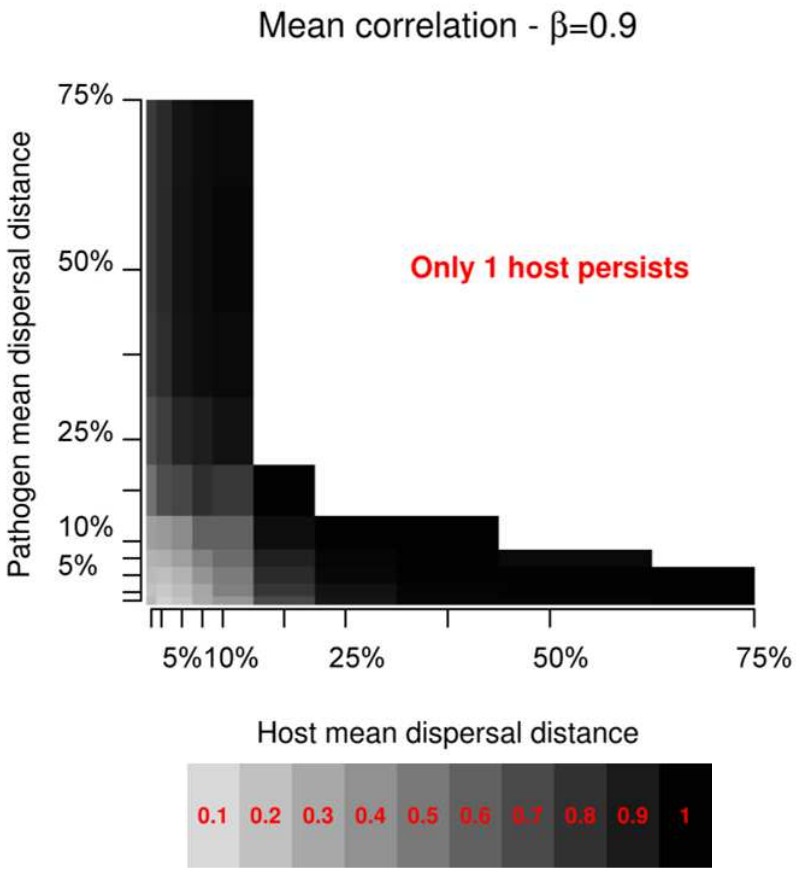

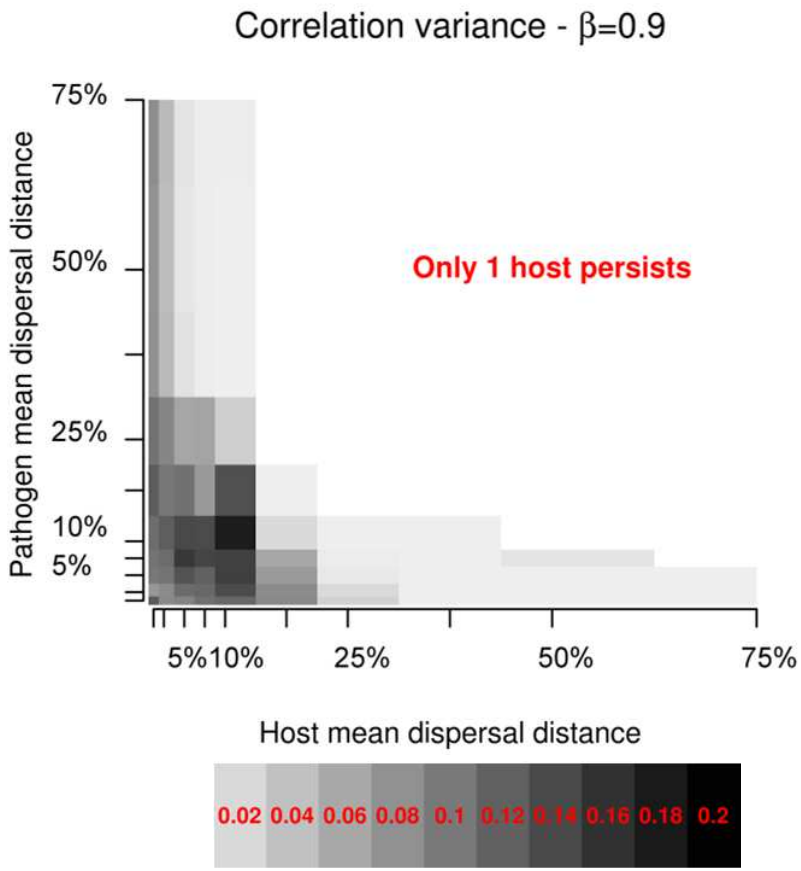

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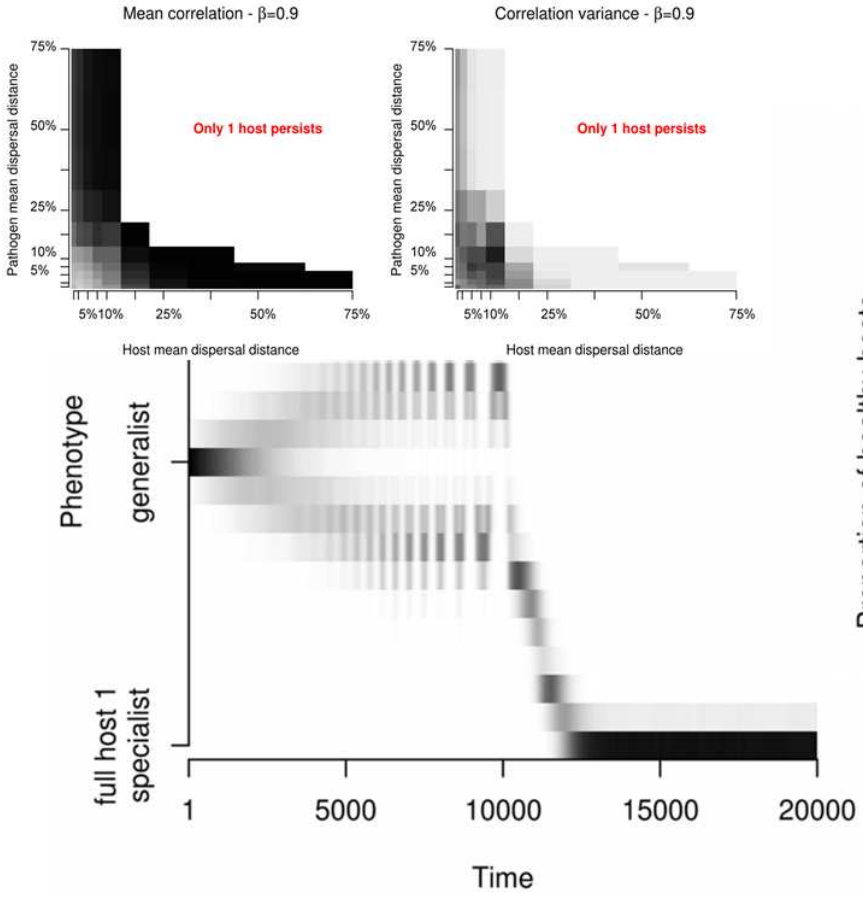
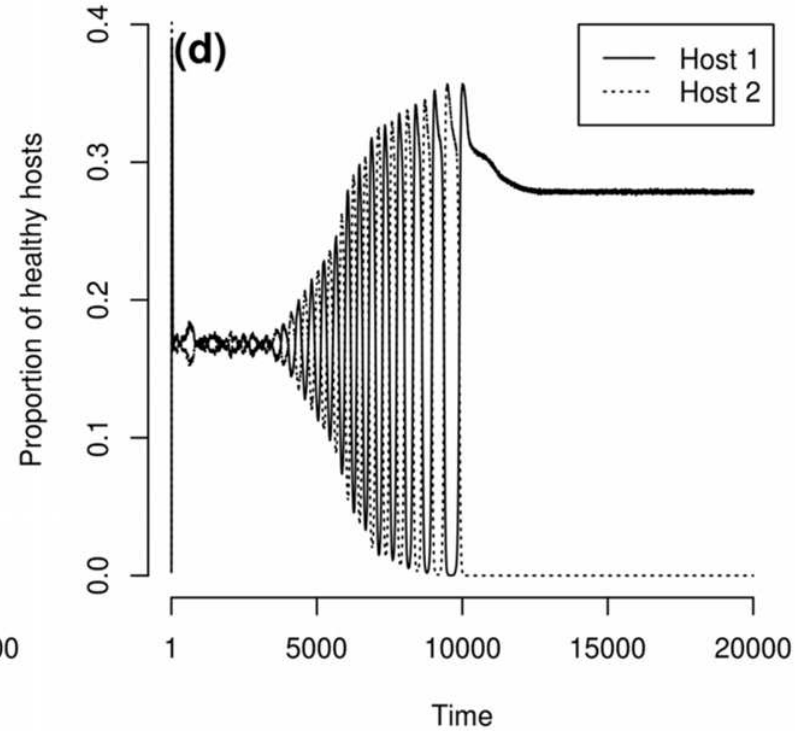
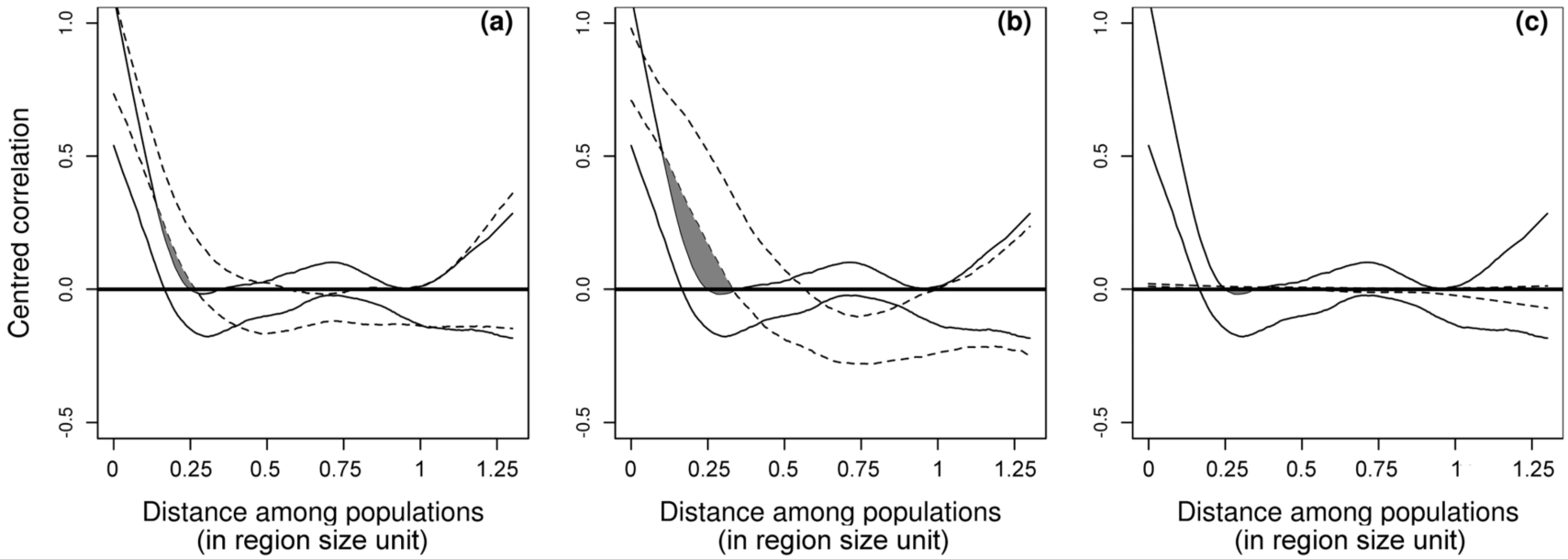




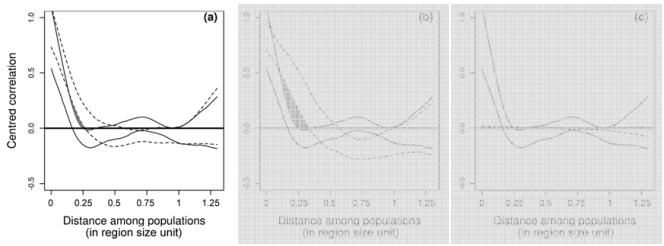
Results: spatial structure

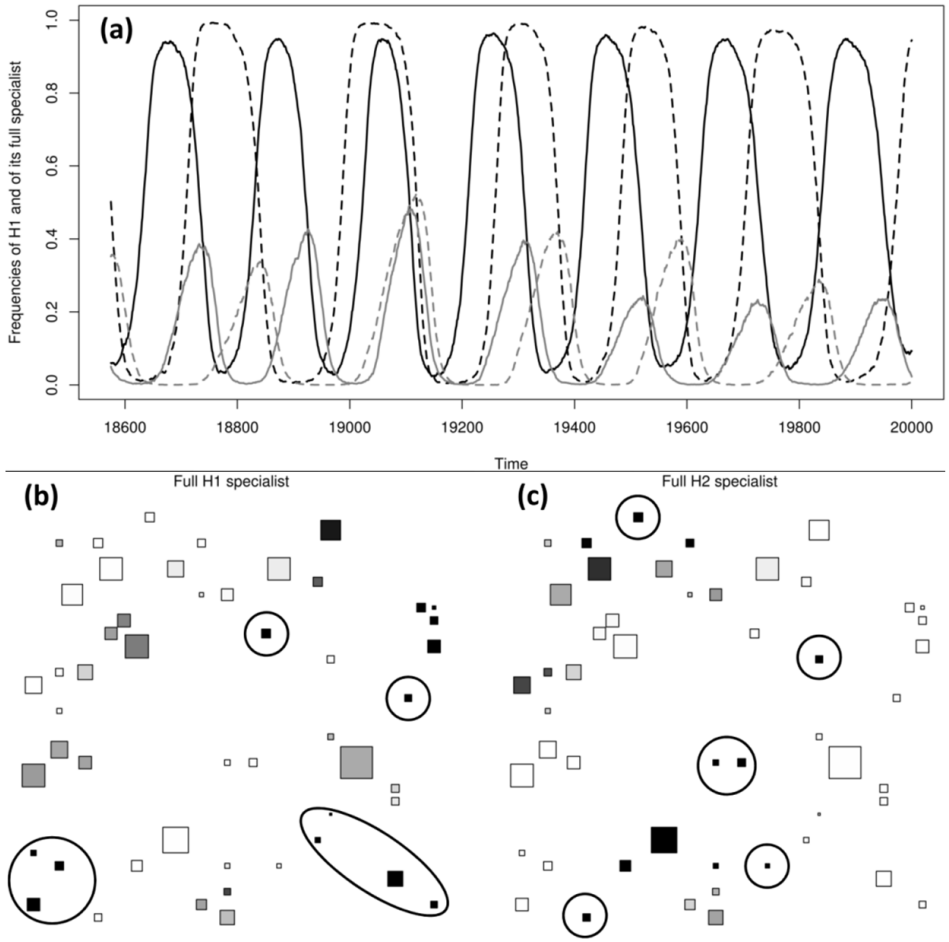
Synchrony among host populations as a function of distance



Results: spatial structure

An example





Results: spatial structure

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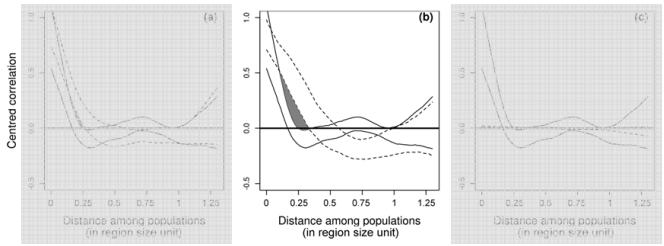
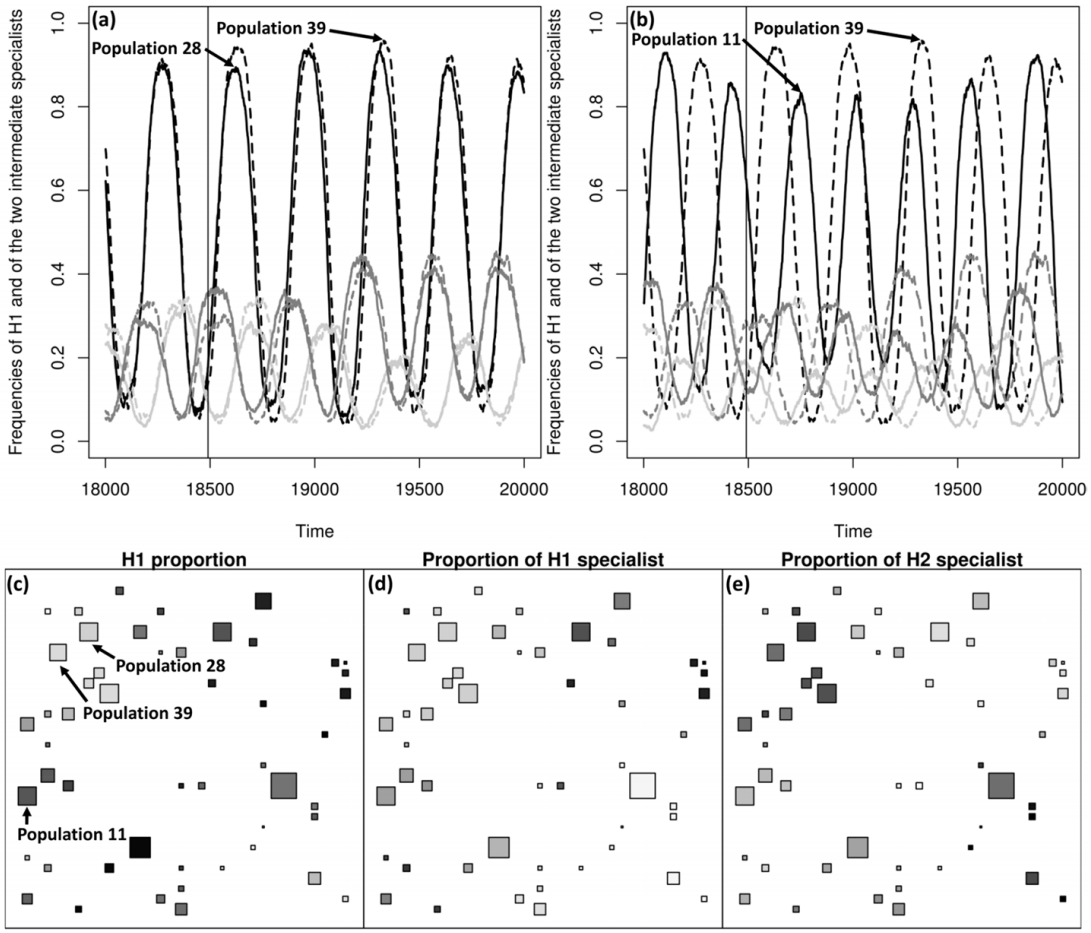
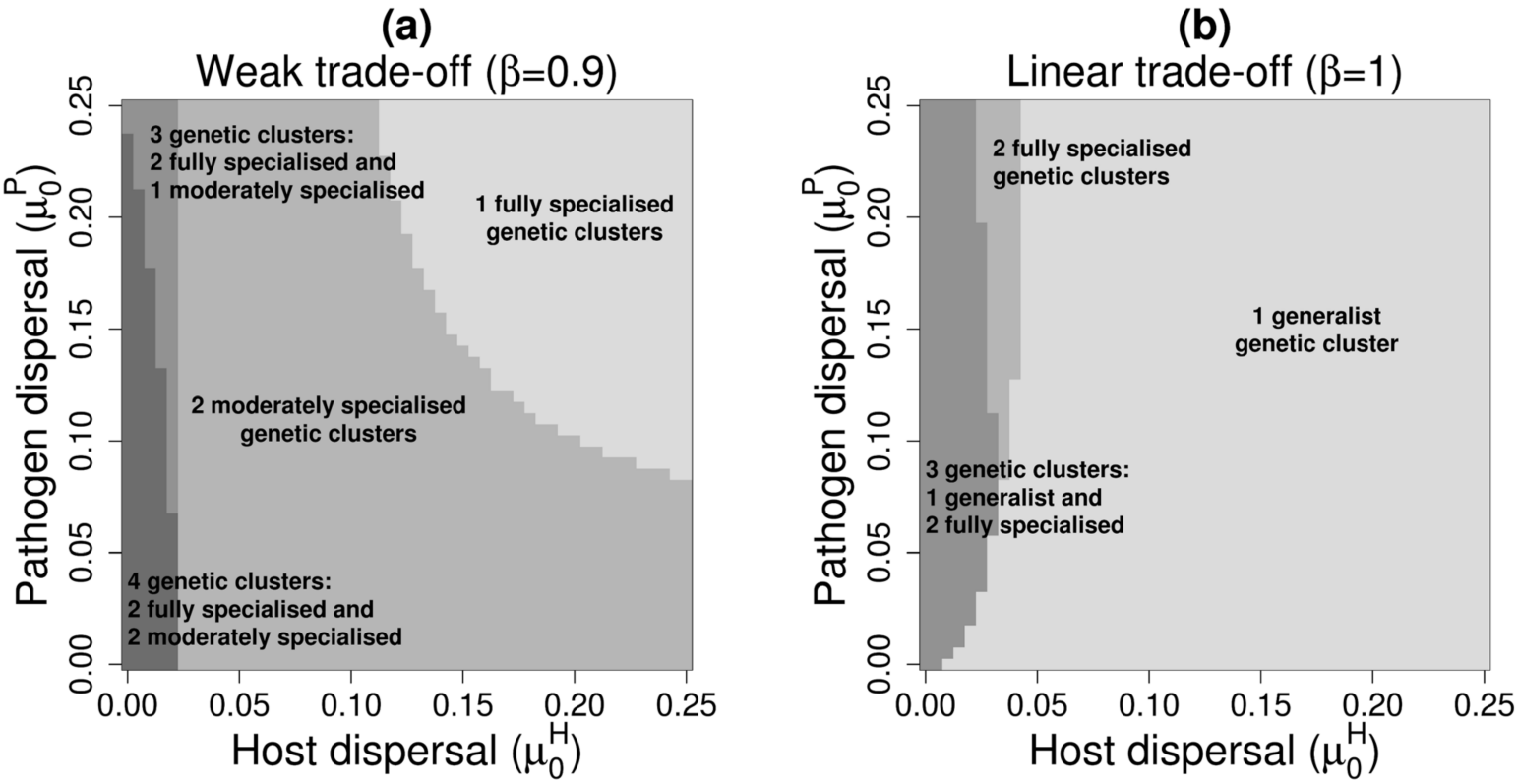




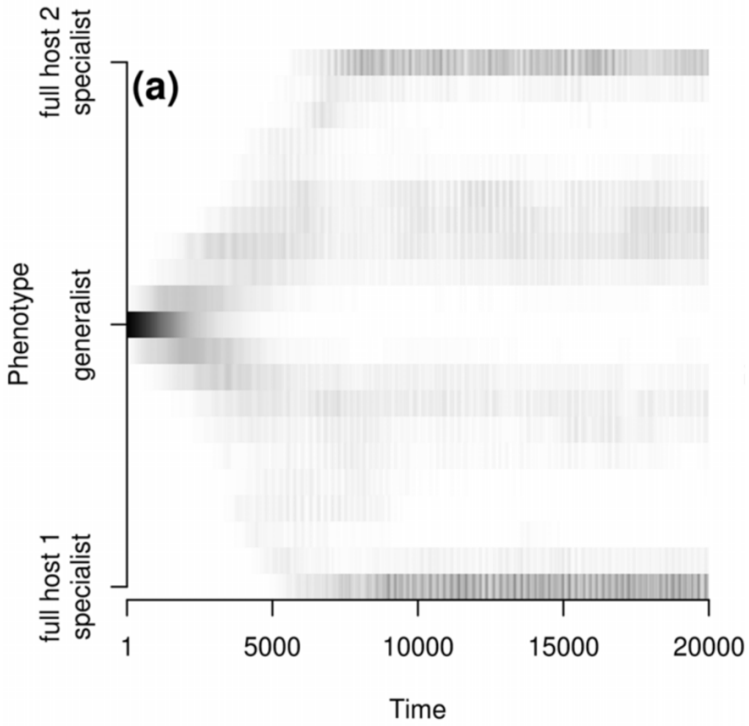
Results: coexistence among pathogen genetic clusters

Global persistence



Results: coexistence among pathogen genetic clusters

Dynamics and spatial repartition



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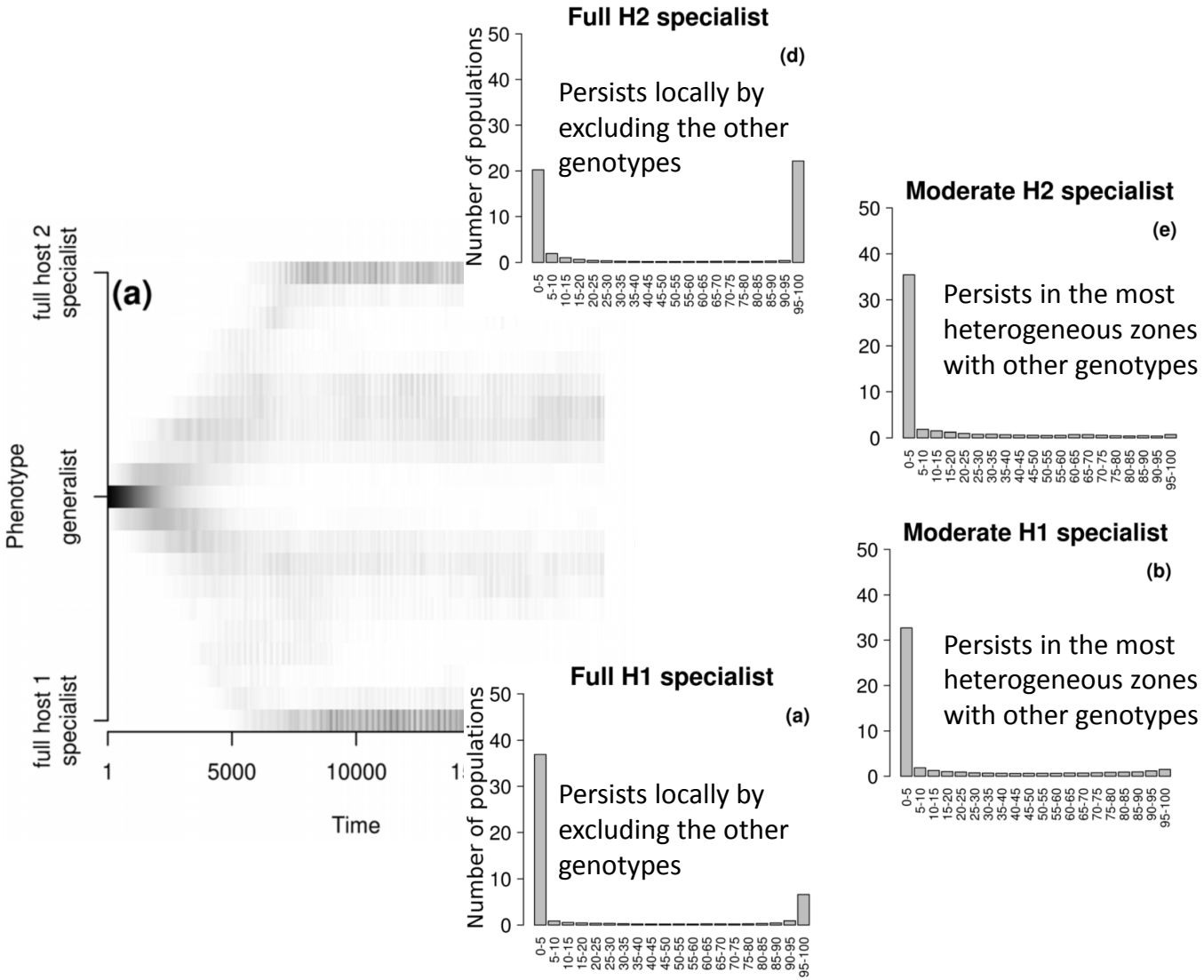
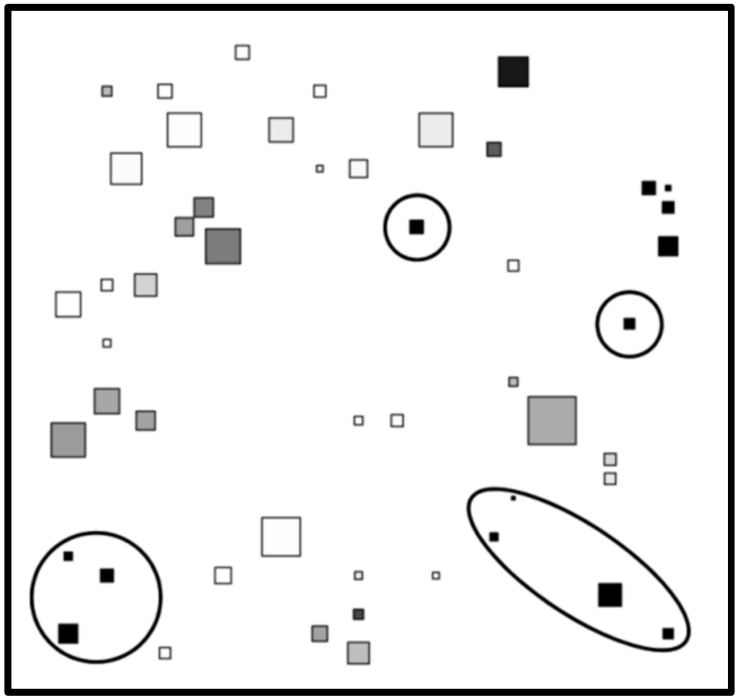


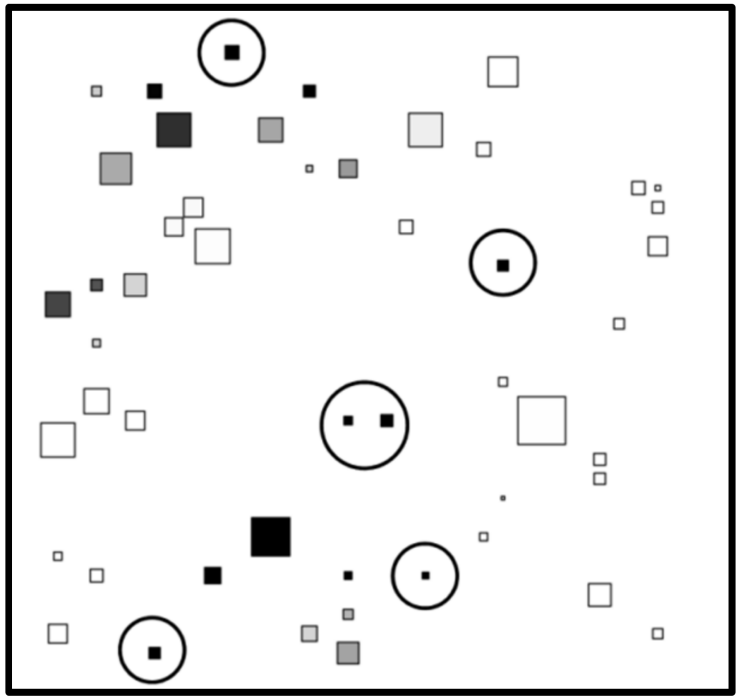
Results: coexistence among pathogen genetic clusters

Dynamics and spatial repartition

Proportion of H1 specialist

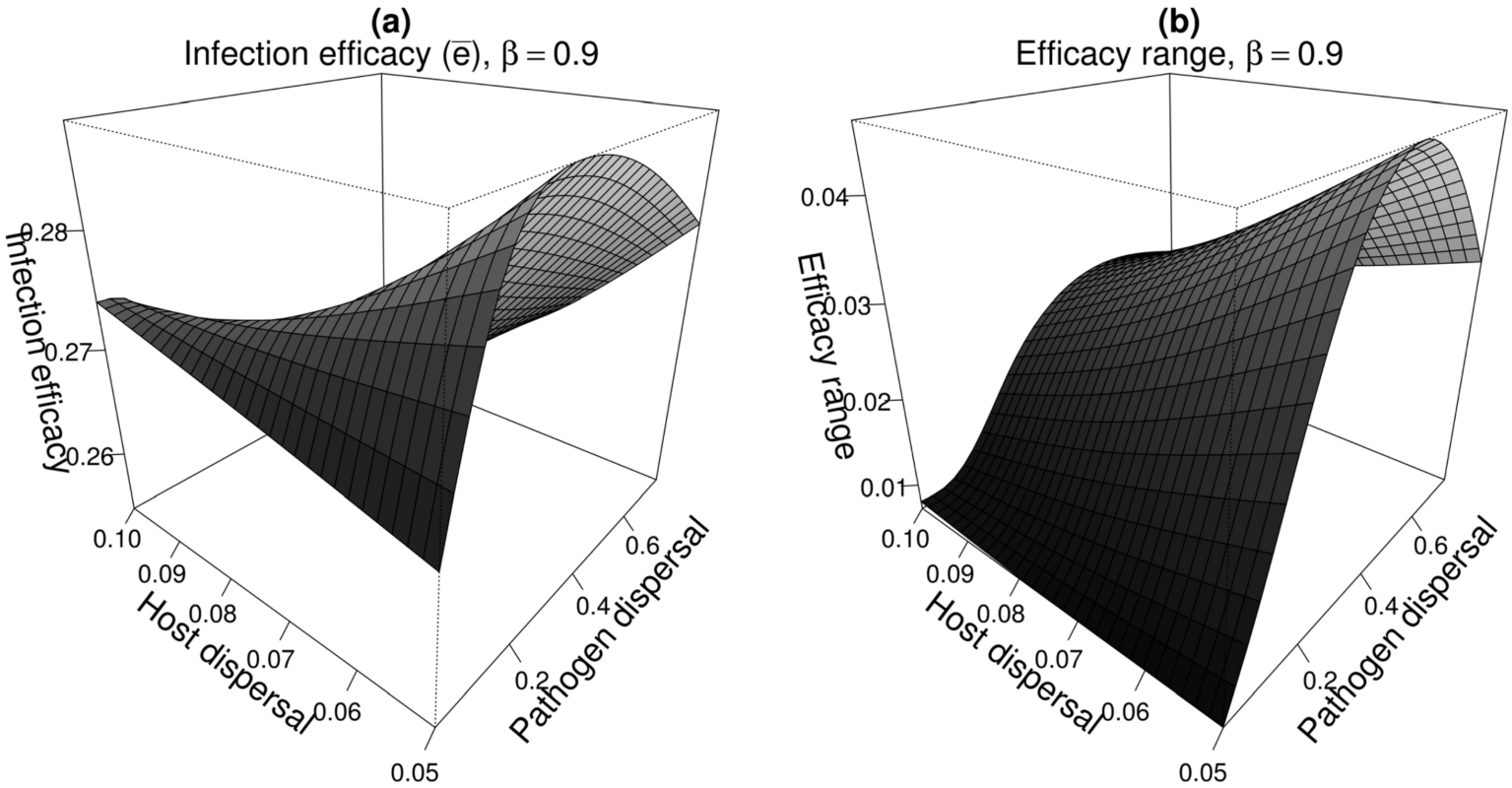


Proportion of H2 specialist



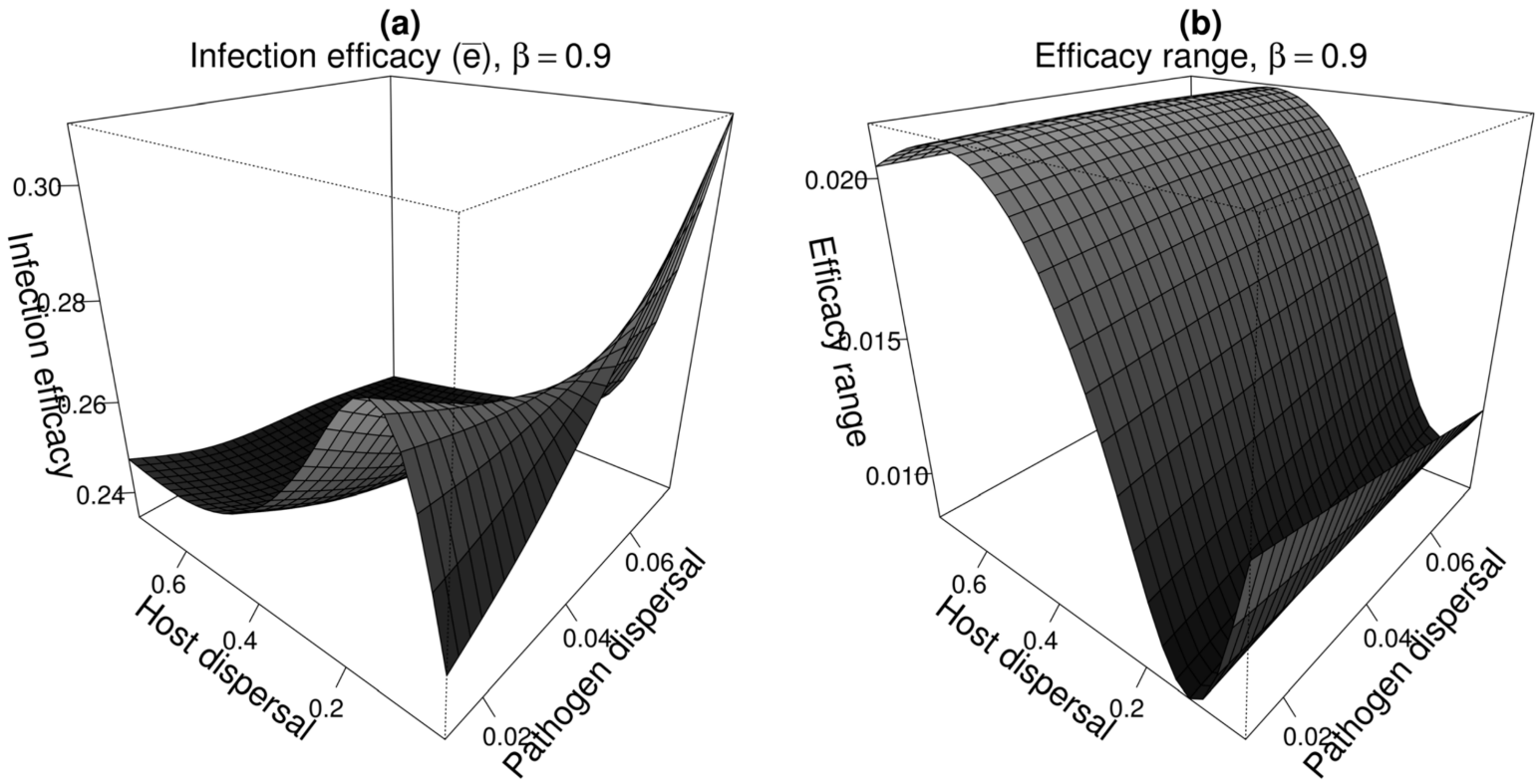
Results: charateristics of the specialists

Effect of pathogen dispersal



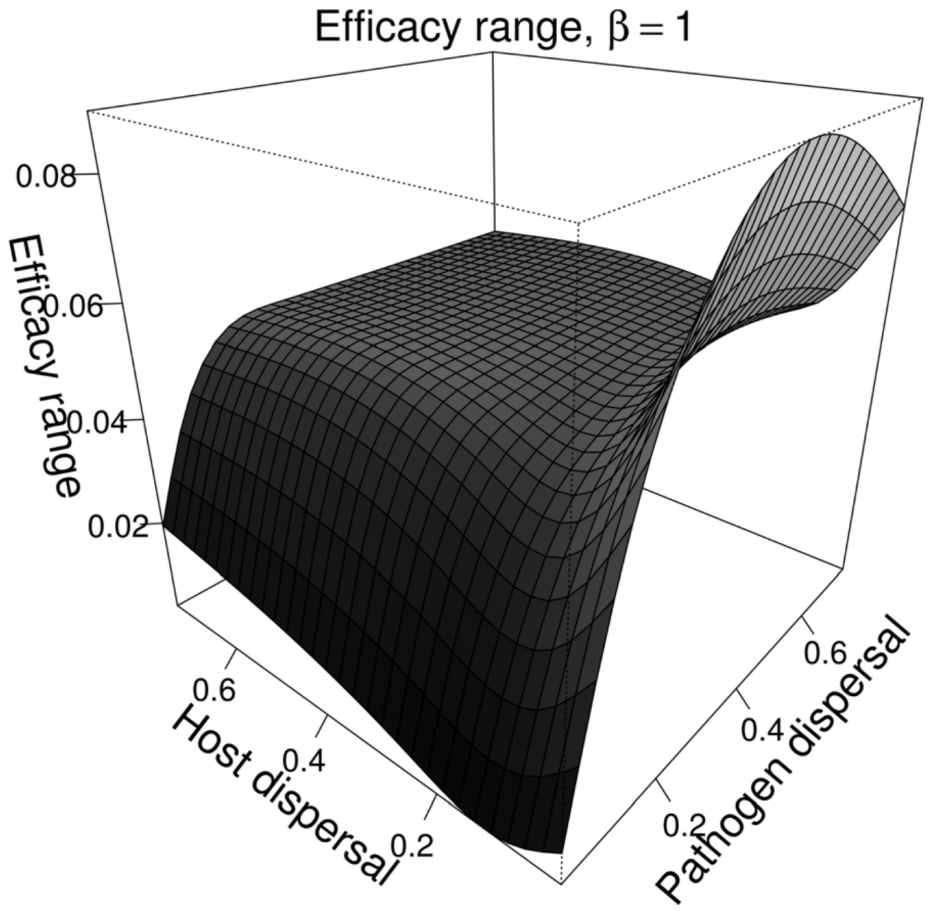
Results: charateristics of the specialists

Effect of host dispersal



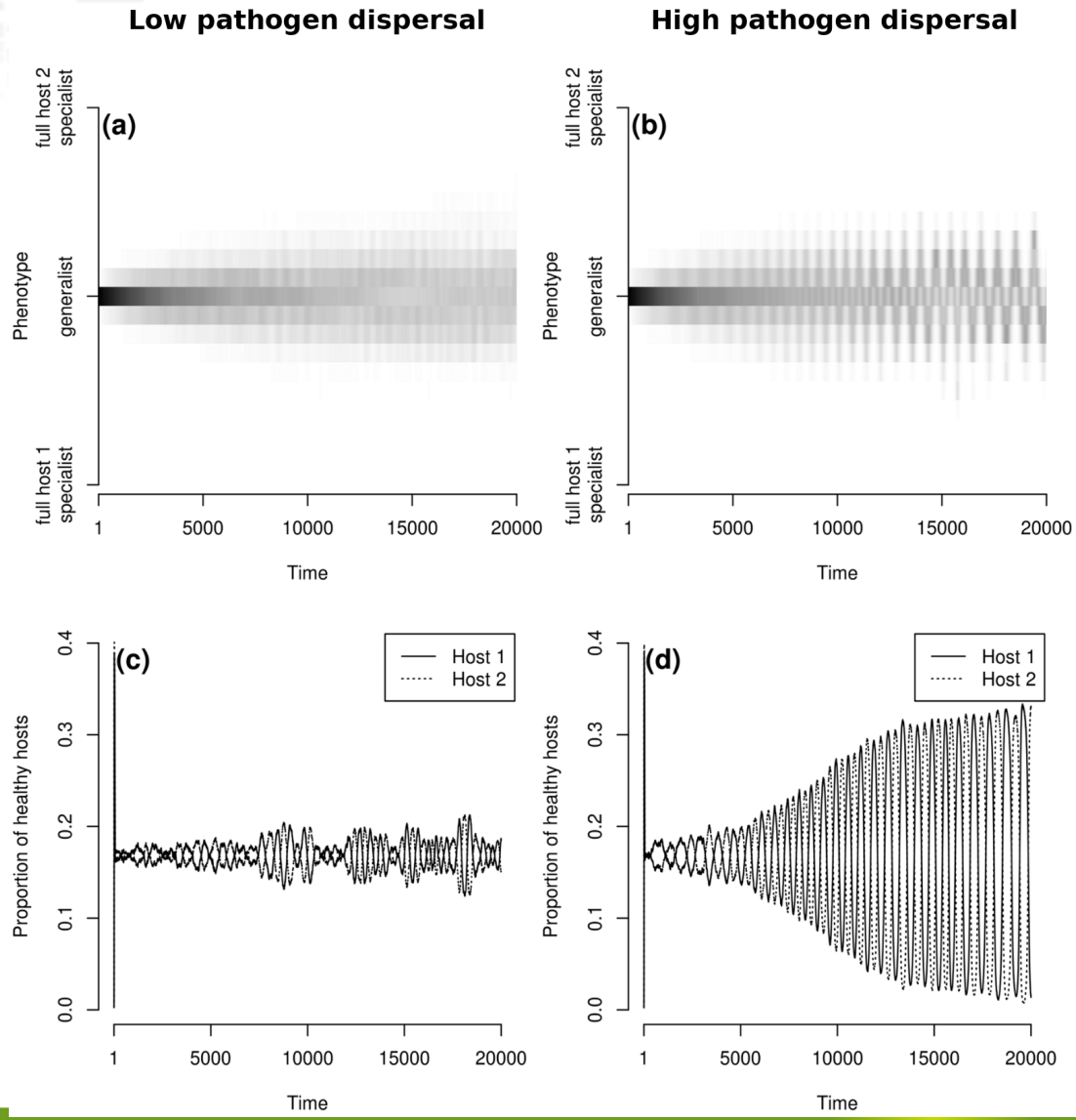
Results: charateristics of the generalist

Effect of dispersal on variability



Results: charateristics of the generalist

An example



Discussion

Key results:

- high level of pathogen diversity can evolve,
- specialisation level depends on dispersal,
- higher host dispersal can lead to higher pathogen specialisation

Spatial structure of the host population and synchrony among host populations explained by host and pathogen dispersal clearly relate to pathogen metapopulation diversity and pathogen specialisation.

MERCI !