



Impact of ploidy and pathogen life cycle on resistance durability

Méline Saubin, Stéphane de Mita, Fabien Halkett

► To cite this version:

Méline Saubin, Stéphane de Mita, Fabien Halkett. Impact of ploidy and pathogen life cycle on resistance durability. 18th Ecology and Evolution of Infectious Diseases, Jun 2021, (Online), France. hal-03336582

HAL Id: hal-03336582

<https://hal.science/hal-03336582>

Submitted on 7 Sep 2021

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Context and main question

Host adaptation is a major component of the evolution of pathogens. In agriculture, hosts with qualitative resistances are often deployed. Therefore, studying host adaptation provides insights on resistance durability. **A major component of host deployment strategies is the proportion of resistant hosts in the landscape (propR).** Previous studies focused primarily on asexual haploid pathogens. **What is the impact of the ploidy (haploid or diploid) and the pathogen's life cycle (with or without host alternation) on resistance durability?** To answer this question, we modelled pathogen population dynamics and genetic evolution at the virulence (infectivity) locus. The avirulence (infectivity) gene exists at two possible states: the dominant avirulent *Avr* allele and the recessive virulent *avr* allele.

Conclusion

Impact of the ploidy:

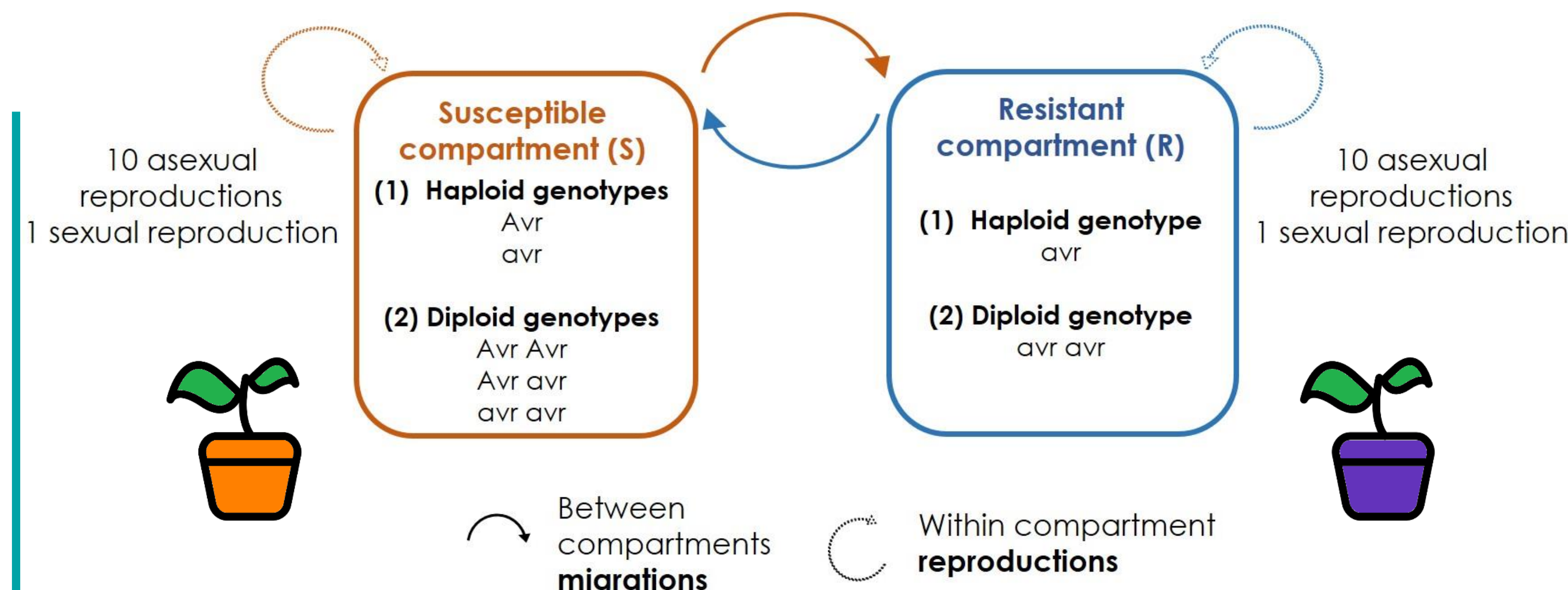
→ Ploidy has a strong impact on evolutionary trajectories, with much **greater stochasticity** and delayed times of resistance breakdown **for diploids**. This result emphasizes the importance of **genetic drift** in this system: as the virulent *avr* allele is recessive, positive selection on resistant hosts only applies to homozygous individuals, which may lead to population collapse at low frequencies of the virulent allele.

Impact of the life cycle:

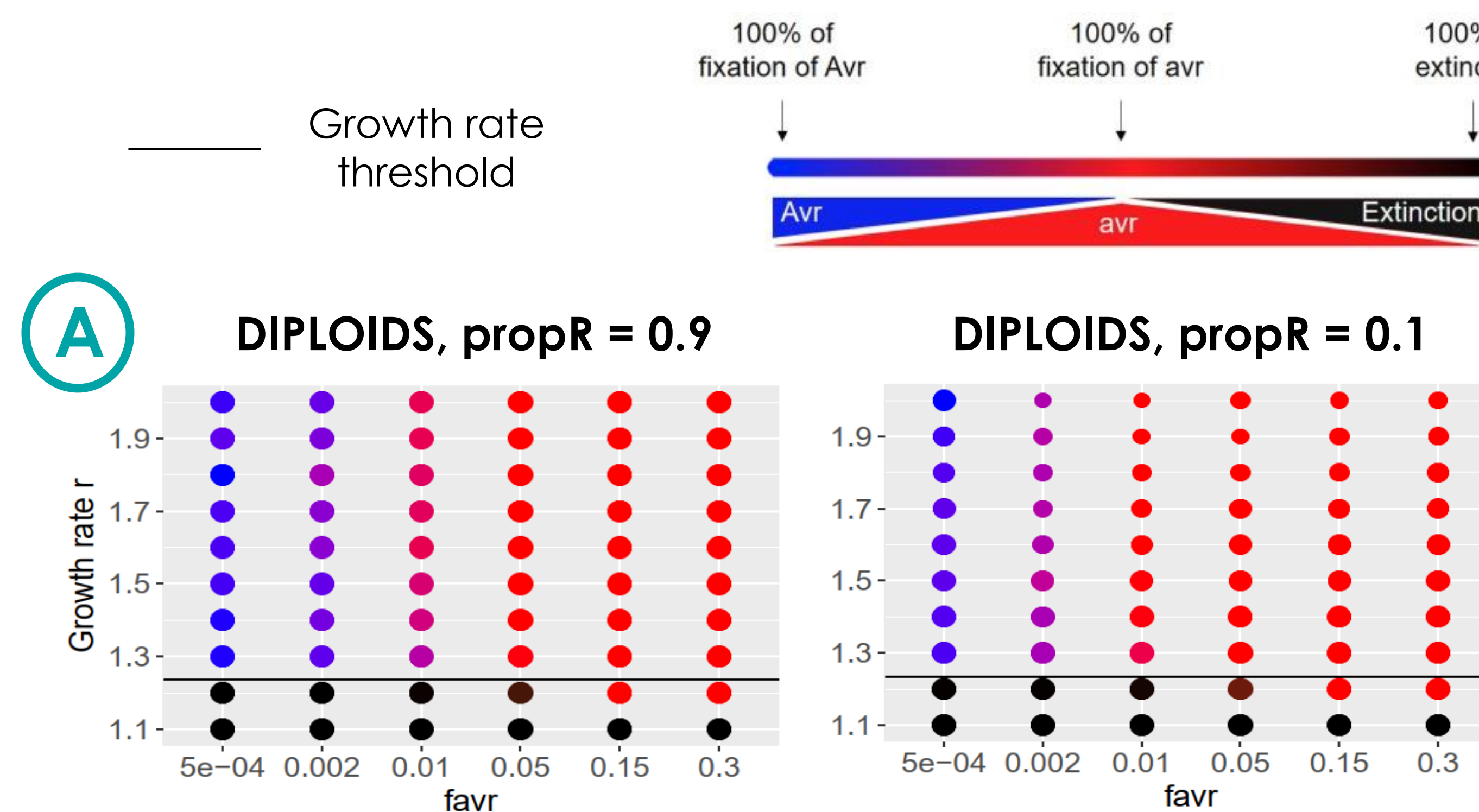
→ **With host alternation**, the probability that the pathogen population collapses strongly increases with the proportion of resistant hosts in the landscape. Therefore, resistance breakdown events occurring at high proportions of resistant hosts frequently amount to **evolutionary rescue**.

→ Life cycles correspond to two selection regimes: **without host alternation (soft selection)** the resistance breakdown is mainly driven by the migration rate. Conversely, **host alternation (hard selection)** resembles an all-or-nothing game, with stochastic trajectories caused by the recurrent allele redistributions on the alternate host.

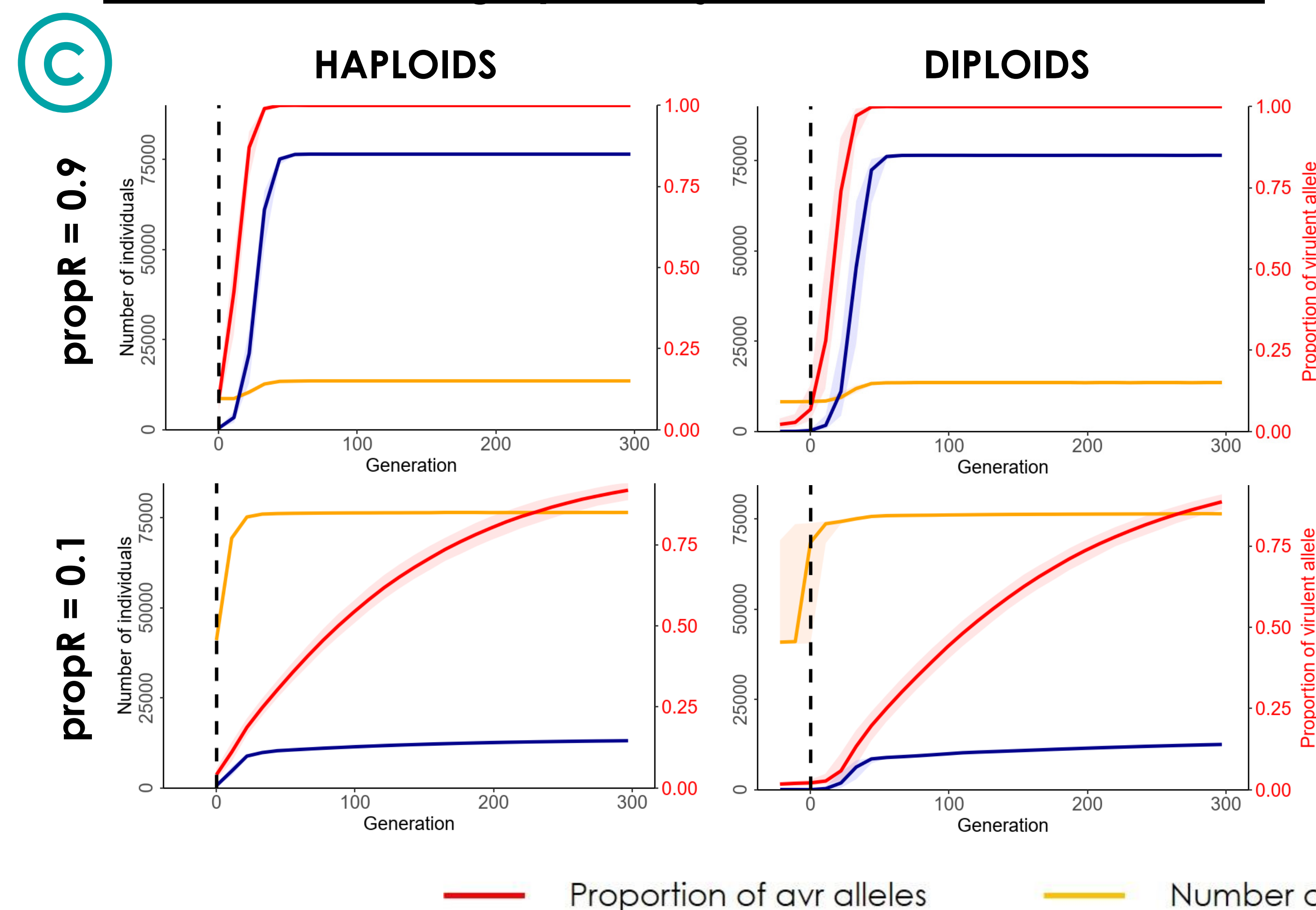
Life cycle without host alternation



Simulation results at equilibrium among replicates

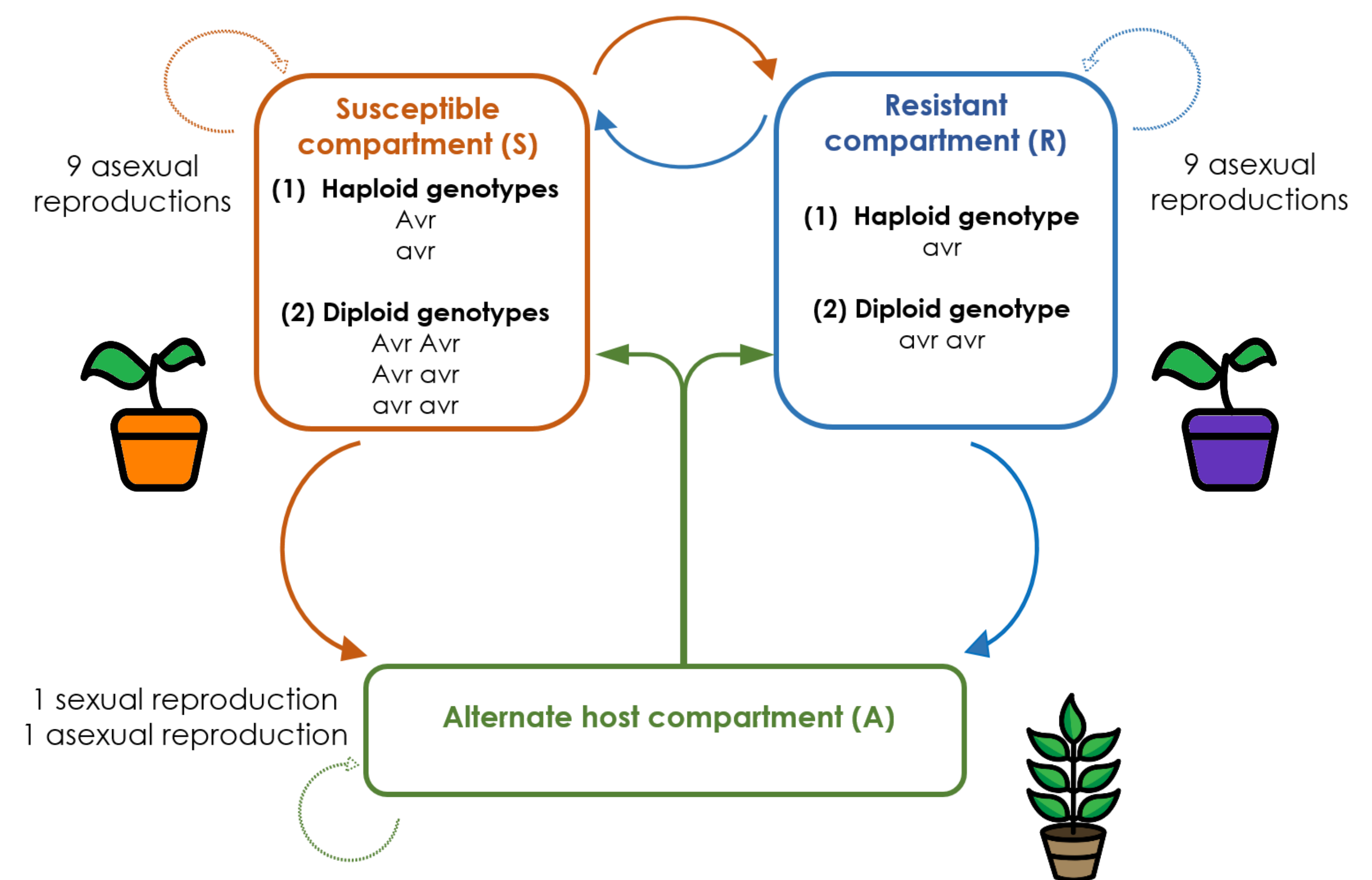


Allelic and demographic trajectories since the invasion

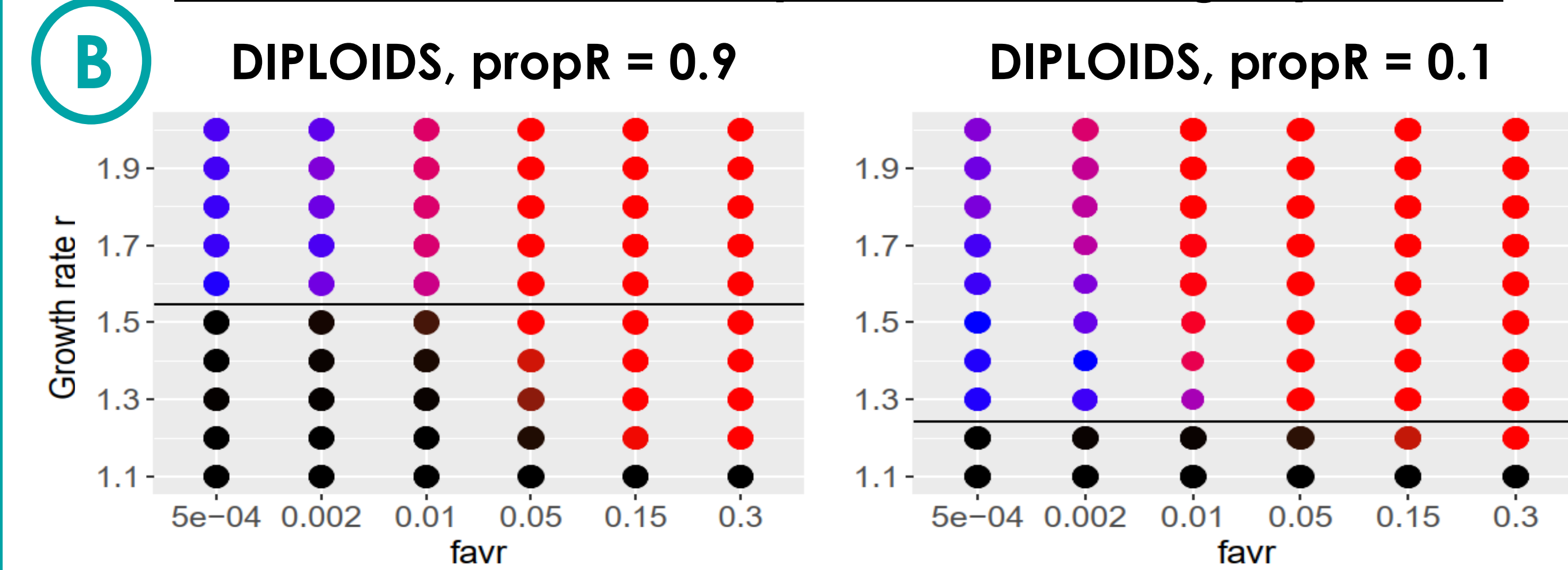


Generation 0 corresponds to the generation of invasion of the R compartment. Colored intervals correspond to the 95% confidence intervals.

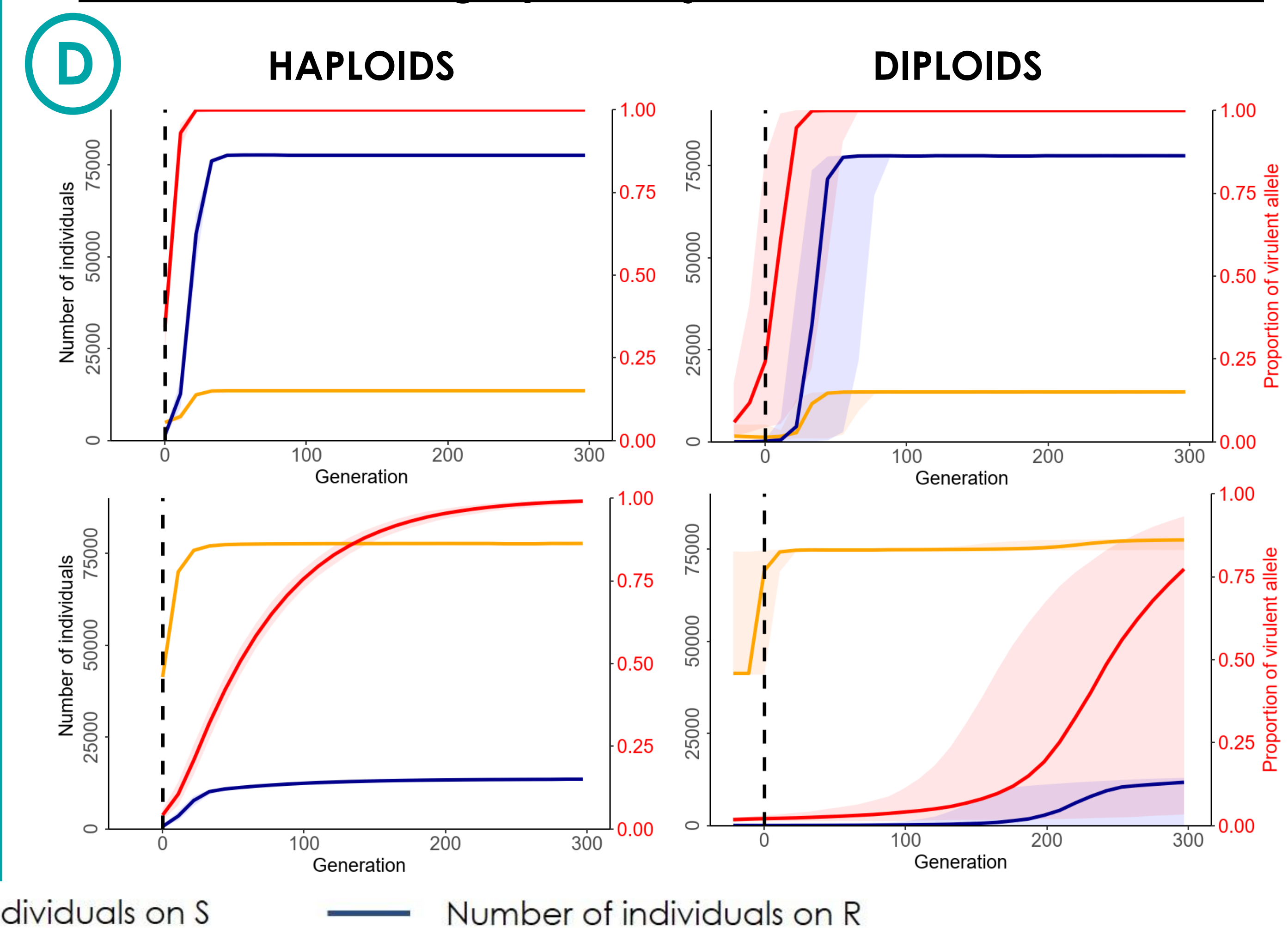
Life cycle with host alternation



Simulation results at equilibrium among replicates



Allelic and demographic trajectories since the invasion



Parameters

propR : proportion of resistant hosts in the landscape

favr: initial proportion of virulent *avr* alleles in the population

Results

- 3 equilibria (A, B):
- 1) Fixation of the avirulent *Avr* allele
 - 2) Fixation of the virulent *avr* allele
 - 3) Extinction of the pathogen population

→ Growth rate threshold (A, B) below which we observe either **extinction** of population or fixation of *avr* in cases of evolutionary rescue

→ Higher probabilities of extinction for diploids **with** host alternation (A, B)

→ Higher dependency on propR **with** host alternation (A, B, C, D)

→ Higher stochasticity in the resistance durability for diploids **with** host alternation (C, D)

→ Higher speed of increase in the virulent *avr* allele for high propR (C, D)