



Evolutionary dynamics of virulence spread: effects of life cycle, genetic determinism and host resistance deployment

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Evolutionary dynamics of virulence spread: effects of life cycle, genetic determinism and host resistance deployment.

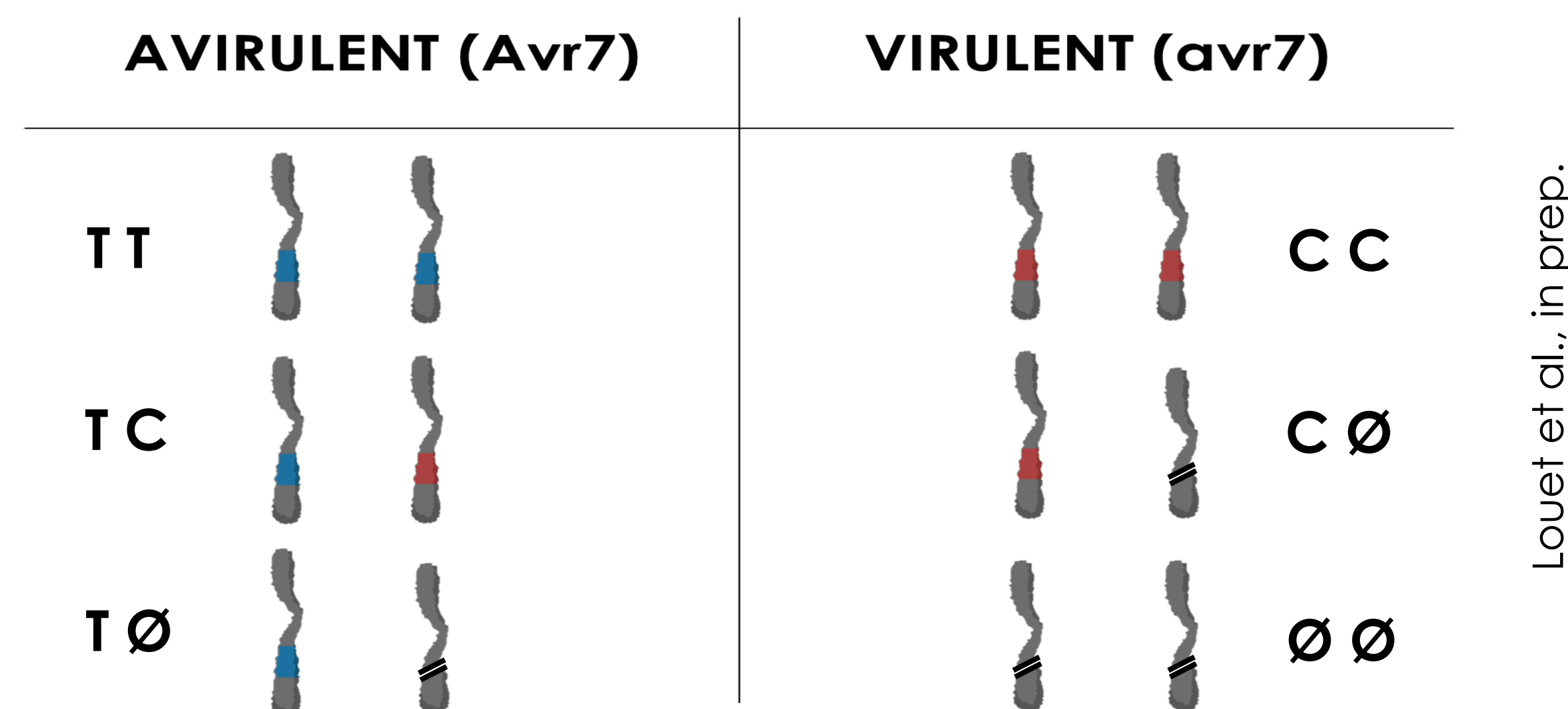
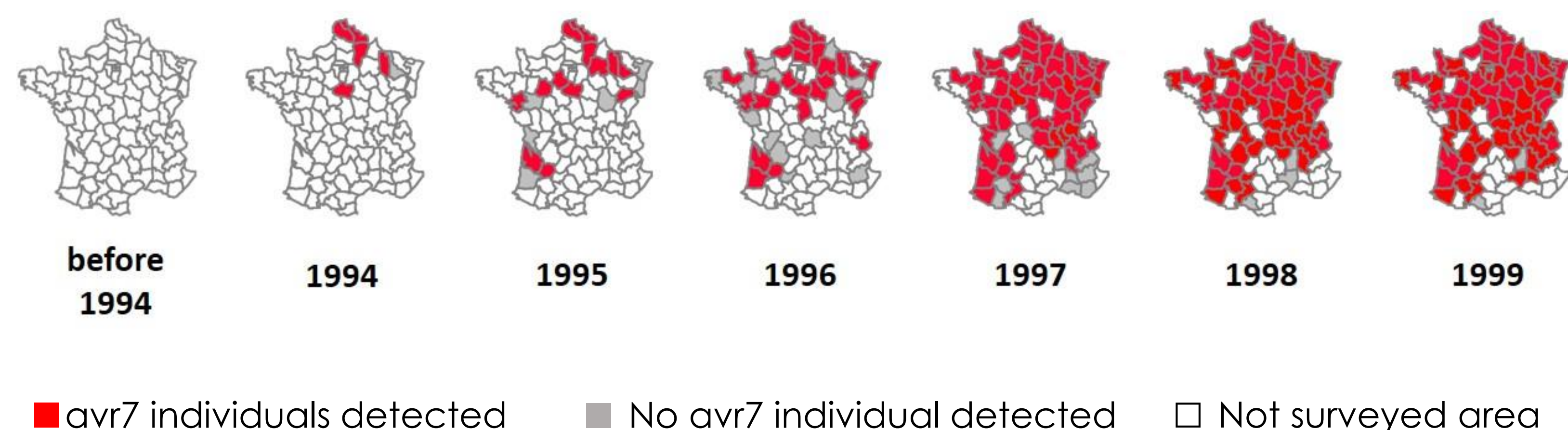
M. Saubin, C. Louet, S. De Mita, A. Persoons, A. Andrieux, J. Pétrowski, B. Fabre, P. Frey, F. Halkett

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Context

Melampsora larici-populina is a basidiomycete fungus (diploid) responsible for **poplar rust**. This partially asexual organism has its sexual stage on larch (*Larix* sp.) and its asexual stage on poplar (*Populus* sp.), a life cycle that amounts to cyclical parthenogenesis in insects.

A poplar cultivar 'Beaupré' (R7) was selected to be completely resistant to *M. larici-populina* and planted widely in northern France between 1982 and 1996. A **major resistance breakdown event** occurred in 1994 when virulent rust genotypes (avr7) emerged (Xhaard et al., 2011). Beaupré revealed to be highly susceptible to avr7 pathogen genotypes, and this rapid breakdown led to **a replacement of the pathogen population** by a new genetic group (Persoons et al., 2017).



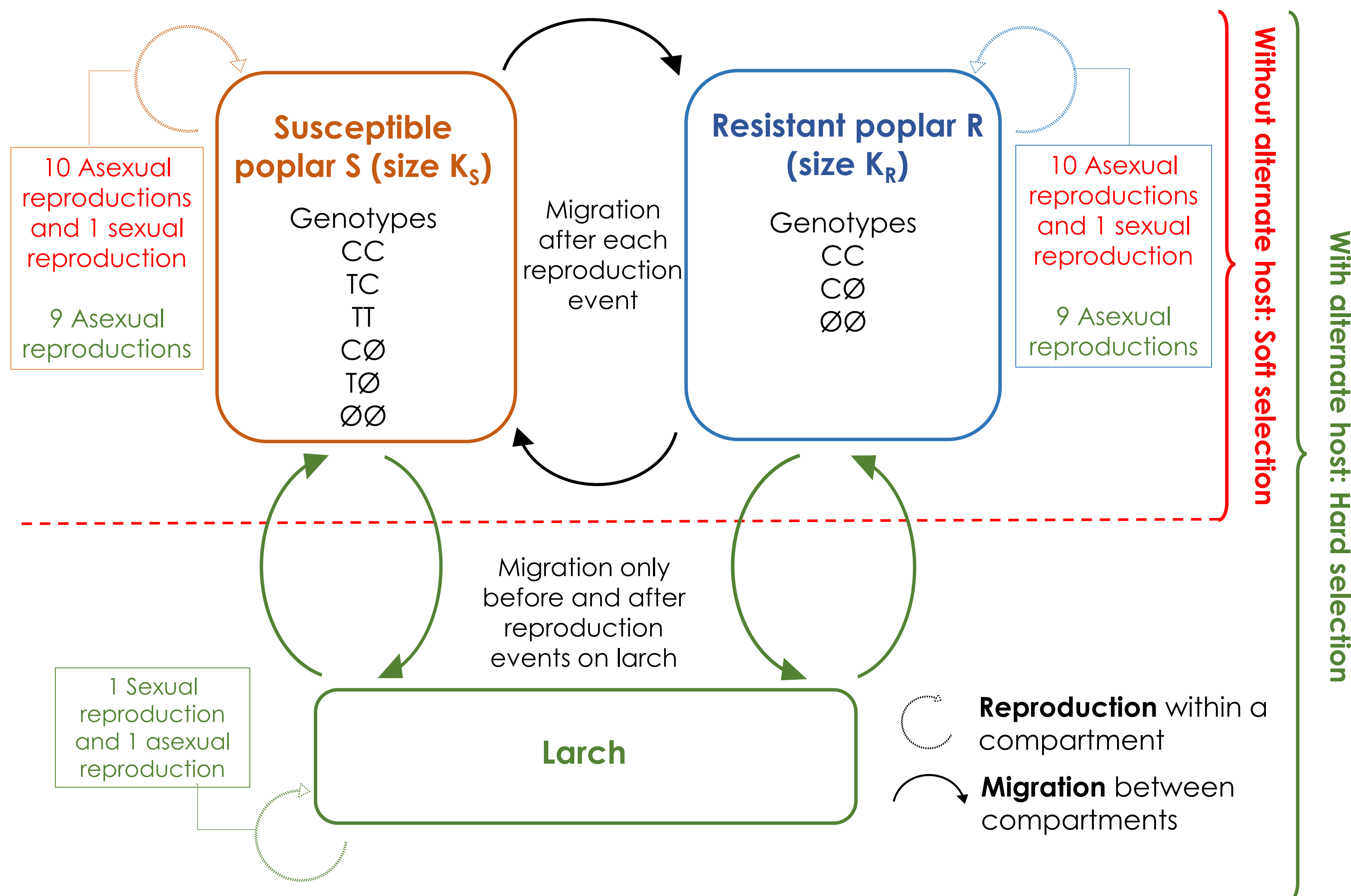
A candidate locus for the avr7 gene was identified in *M. larici-populina* (Persoons et al., in prep., Maupetit et al., in prep.). The **virulent allele is recessive** and there are two independent causal mutations at the same locus: **a single nucleotide polymorphism** (T → C) and **the deletion** of this locus.

Research goal: To study the dynamics of virulence allele fixation both theoretically and empirically, by

- (1) Modeling virulence fixation in different evolutionary scenarios.
- (2) Tracing back the polymorphism evolution at the candidate locus before, during and after the resistance breakdown.

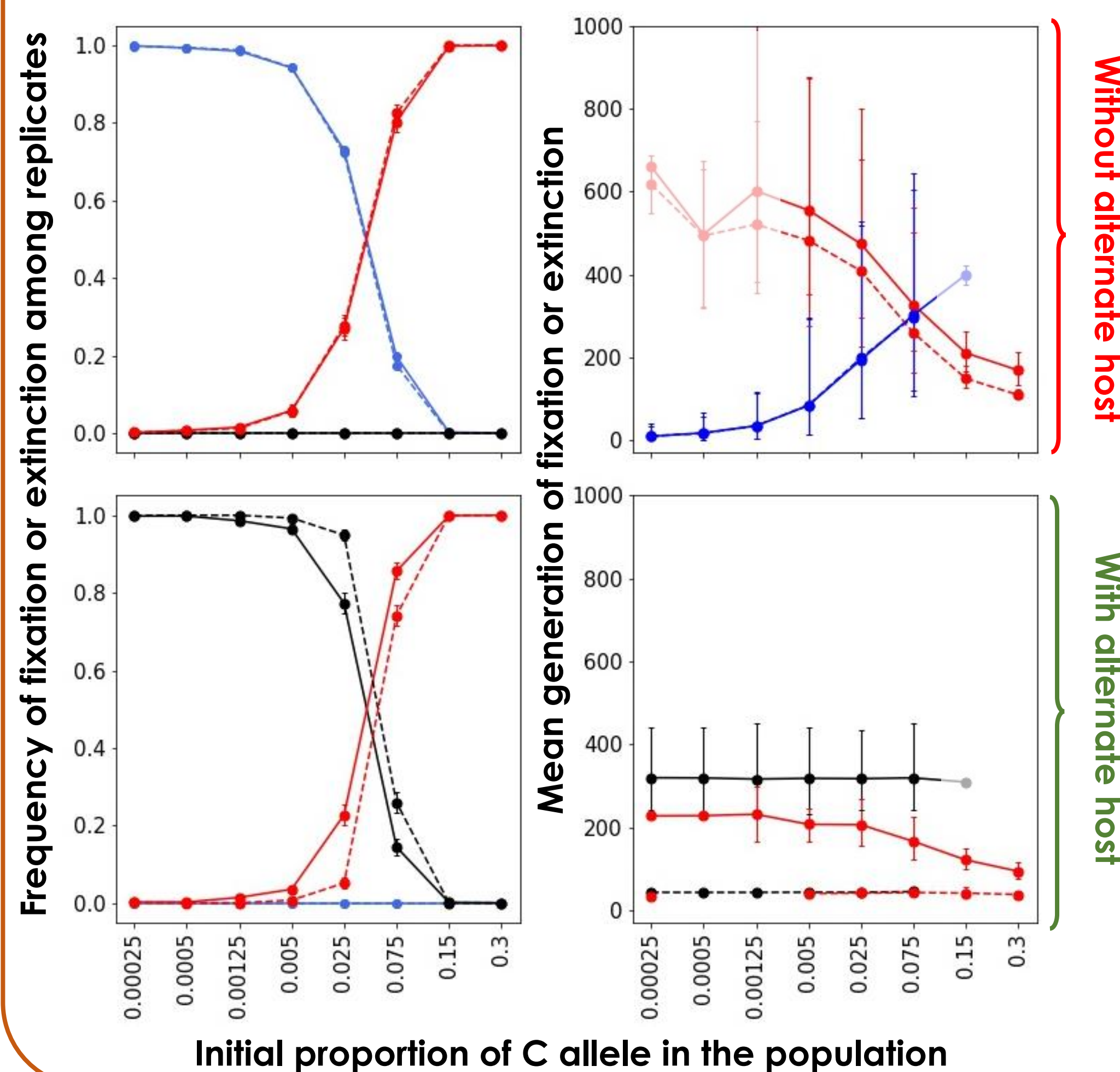
(1) Modeling evolutionary trajectory of a partially asexual organism

Two principal models: with and without alternate host



First simulations were run with **1000 replicates** using the library SIMUPOP (Peng et al., 2005) on PYTHON, **without the deletion**:

- Extinction of population
 - Fixation of allele T
 - Fixation of allele C
- $K_S = K_R = 10\,000$
- $K_S = 10\,000, K_R = 90\,000$

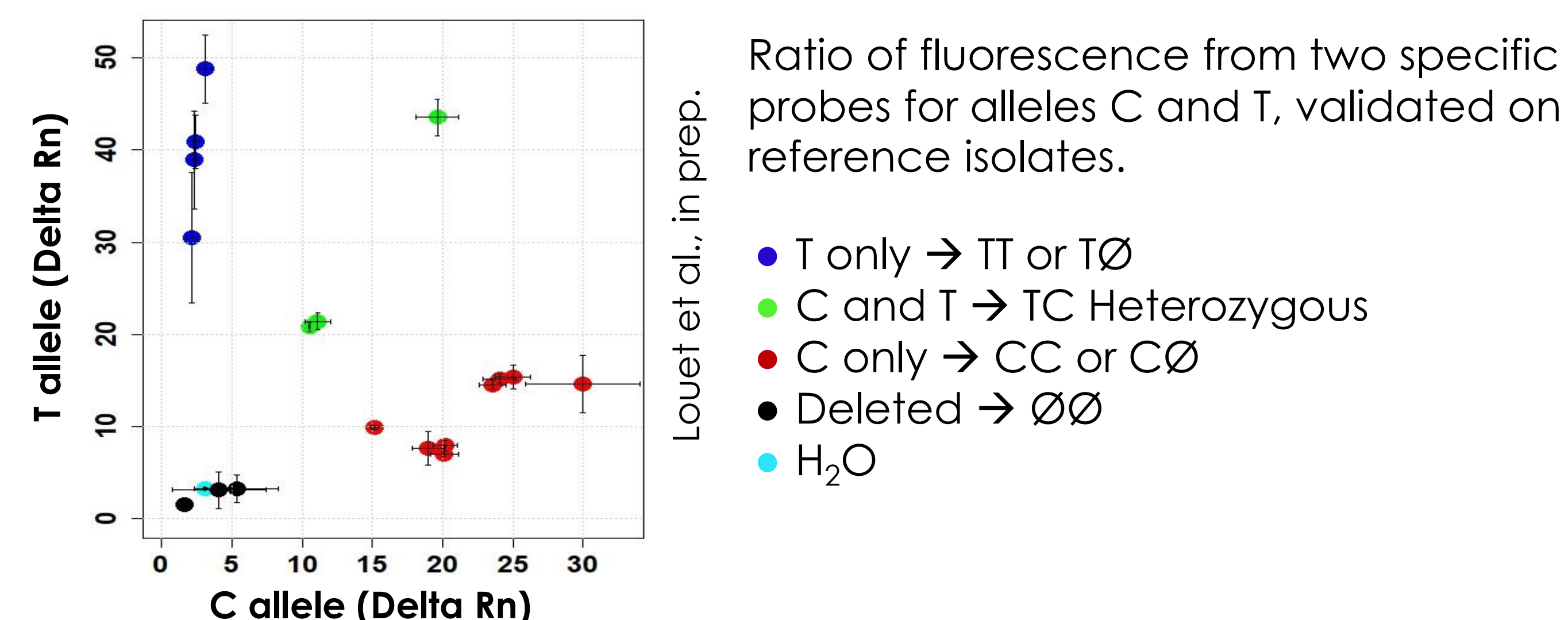


- **Independence of compartment sizes** on the frequency of fixation.
- **No extinction** of population but a **slower fixation of C allele** compared to simulations with alternate host.
- **Polymorphism** is more **protected**.

- Either **fixation of C allele** or **extinction** of the population.
- **Strong influence of compartment sizes** on the dynamics of fixation and extinction.

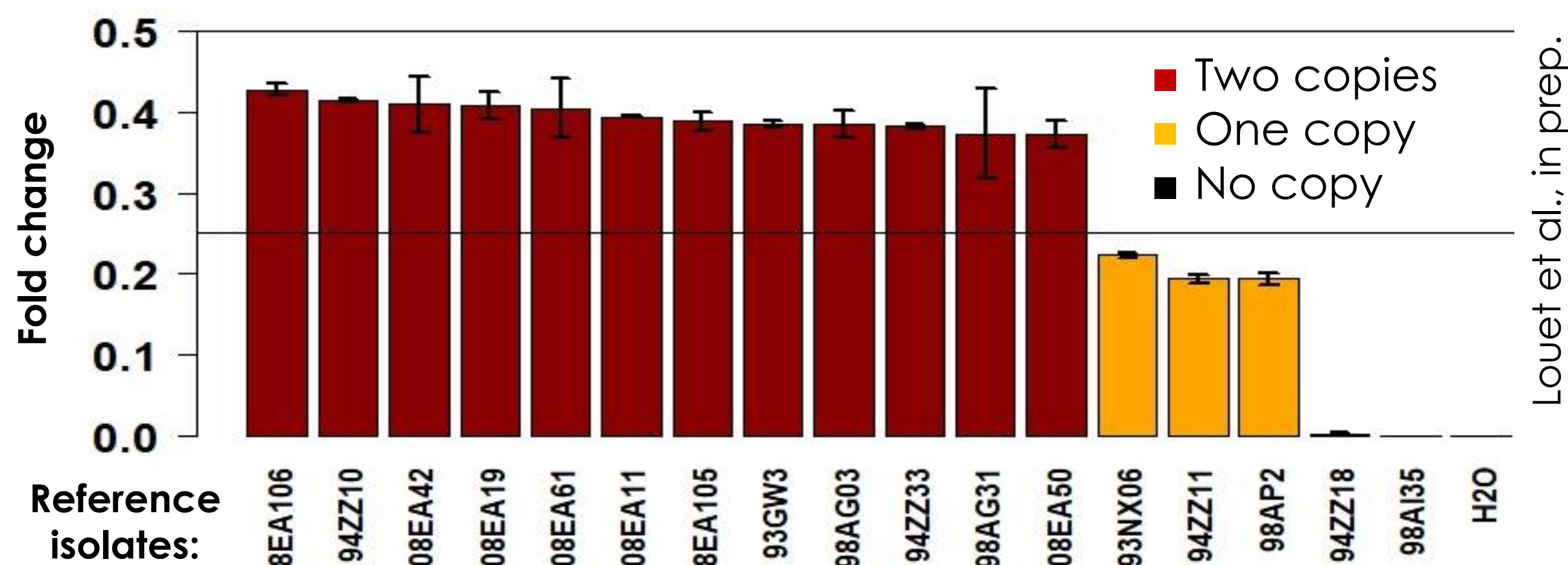
(2) Experimental characterization of the polymorphic avirulence locus (Avr7) of M. larici-populina

Detection of the recessive C allele at the candidate locus



Detection of the deletion at the candidate locus

Relative quantification with α -tubulin as reference gene, present in two diploid copies (4 copies) in *M. larici-populina*'s genome, validated on reference isolates.



Temporal evolution of polymorphism will be studied by characterizing around 300 isolates collected from 1988 to 2016.

Conclusions and perspectives

- **Larch plays a decisive role in virulence fixation:** it increases both the probability of extinction and the speed of fixation.
- The **heterozygous state is highly vulnerable**, being affected by genetic drift only (not selected on resistant cultivars). There is still to enrich the modeling part by taking into account both SNP and deletion and investigate in parallel the empirical evolution of virulence alleles dynamics.
- Hypothesis: **the combined presence of the deletion and the mutation conferring virulence may have led to such a rapid and complete replacement of the pathogen population.** This hypothesis will be tested and our model results will be linked to the effective polymorphism evolution of the studied population.

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