Impact of quantitative plant resistance on within-host viral demo-genetic dynamics

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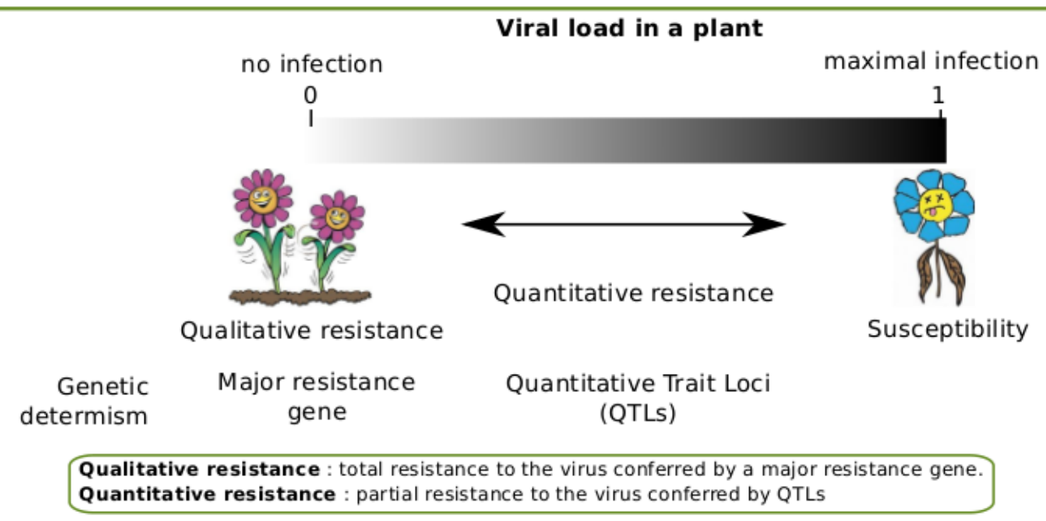
Introduction

The deployment of virus-resistant plants often leads to the emergence of **resistance-breaking** (RB) mutants that suppress the yield benefit provided by the resistance.

Although breakdowns are well known for **qualitative resistances**, they are still poorly understood for **quantitative resistances**.

Furthermore, it has been proved for several pathosystems that **combining qualitative** and **quantitative resistances** can **increase the sustainability** of the **qualitative resistance** (Palloix *et al.* 2009, New Phytol, Brun *et al.* 2010, New Phytol).

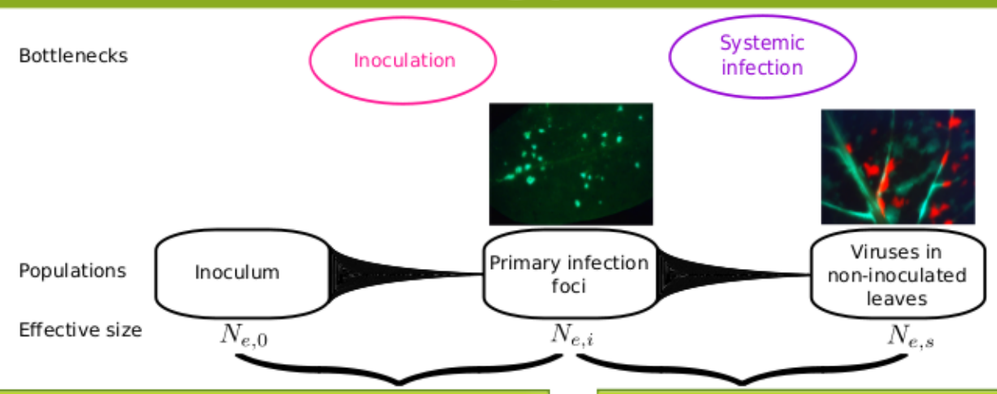


Qualitative resistance : total resistance to the virus conferred by a major resistance gene.
Quantitative resistance : partial resistance to the virus conferred by QTLs

Objective : To analyze the **effect of quantitative resistances**, in terms of genetic drift and selection, on the **sustainability** of **qualitative resistances** by coupling experimental and modelling approaches.

Quantitative resistances are associated to a **decrease** of the **fixation rate** of **RB mutants**. Two possible explanations :
- **decrease** of the **selection differential**
- **increase** of the **genetic drift** (not studied in the context plant resistances to pathogens)

Experimentation : strength of genetic drift on viruses during plant infection



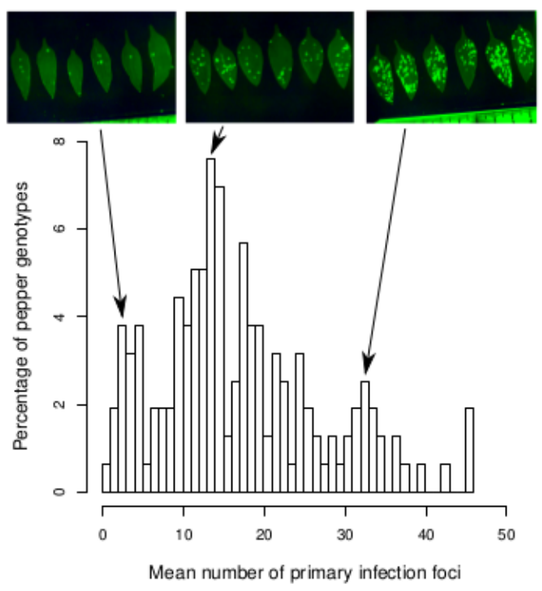
Exp 1 : genetic drift due to inoculation

One virus particle is responsible for each primary infection focus. Hence, effective population size N_e following inoculation is well estimated by the number of primary infection foci. (Zwart *et al.* 2011, PLoS Pathog)

Strategy :

Inoculation of 158 pepper genotypes carrying a **major resistance gene** (pvr2[3]) and several combinations of quantitative trait loci (**QTLs**), with Potato virus Y (**PVY**) strains carrying the Green Fluorescent Protein (**GFP**) fluorescent marker.

Counts of the primary infection foci.



We observed a **large variability** in the **number of primary infection foci** depending on the pepper genotype.

Objective :

Detection of **QTLs** associated to **effective population size** variation after **inoculation**.

Exp 2 : genetic drift due to systemic infection

Strategy :

Inoculation of pepper genotypes exhibiting **contrasted effective population size** following **inoculation** (cf. Exp 1) with a **1:1 mix** of **PVY-GFP** and **PVY-mCherry**, carrying a green and a red fluorophore, respectively.

Observations :

- t_1 : relative **frequency** of **primary infection foci** initiated by **PVY-GFP** and **PVY-mCherry** within **inoculated leaves**.

- t_2 : relative **frequency** of **PVY-GFP** and **PVY-mCherry** within **systemically infected leaves** (area of PVY-GFP and PVY-mCherry).

We will assess the **strength** of **genetic drift** due to **systemic infection** by comparing t_1 and t_2 frequencies.

Objective :

Detection of **QTLs** associated to **genetic drift** variation due to **systemic infection**.

What is the most appropriate estimator of the genetic drift due to systemic infection ?

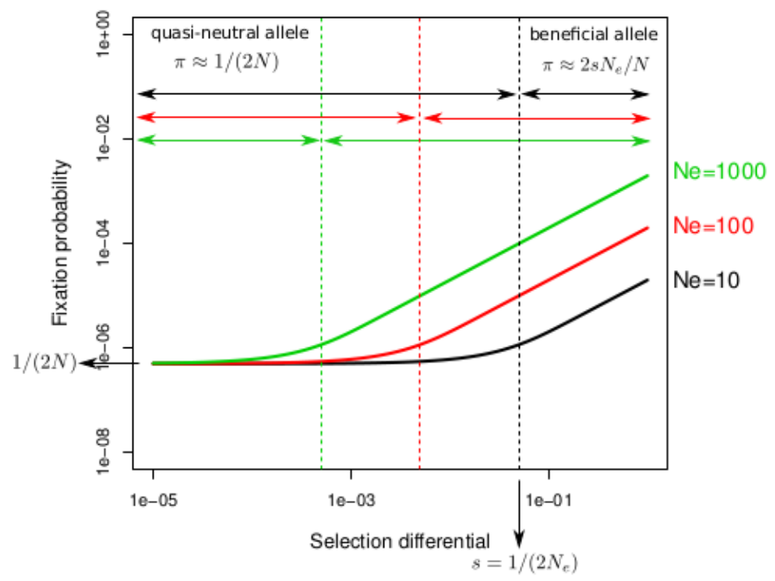
Is the estimation more accurate if we take more time points ?

How can we estimate the genetic drift due to systemic infection if selection cannot be ignored ?

Modelling : impact of genetic drift and selection on the durability of qualitative resistances

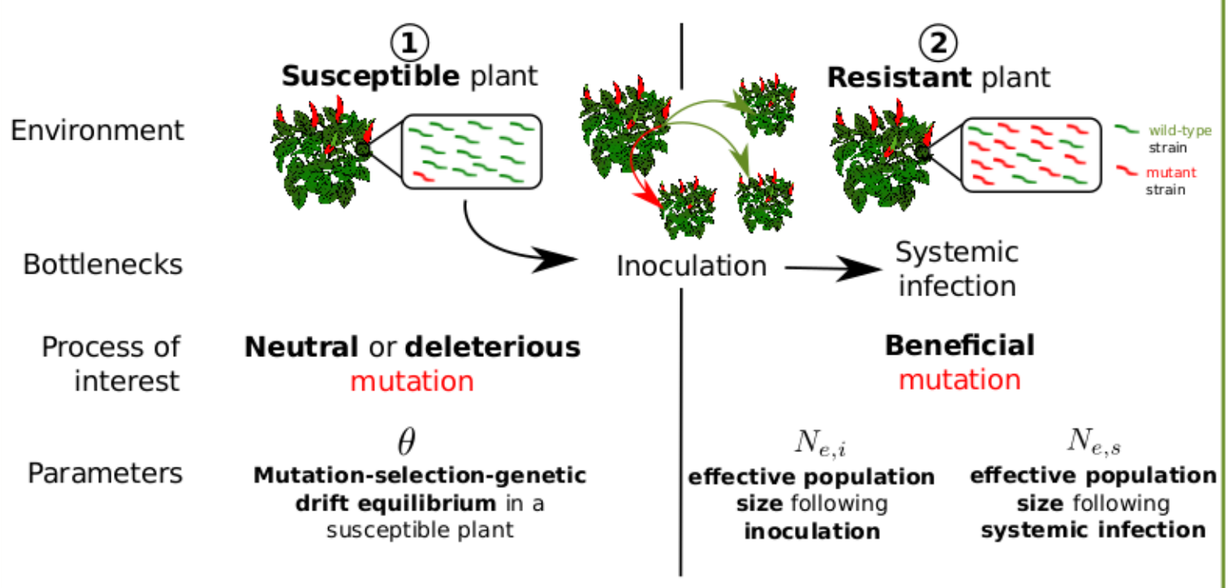
Ohta & Kimura (1970, Genetics) proposed a formula to approximate the **fixation probability** for a **beneficial mutation**, under the hypotheses :
- of diploid individuals, with sexual reproduction, panmixia, and a large and constant population size,
- of discrete generations, and one mutant at a time (moderate mutation rate).

$$\pi = \frac{1 - exp(-4spN_e)}{1 - exp(-4sN_e)}$$



Strategy :

To study the emergence of RB mutants when resistant plants are deployed.



Objectives :

To determine the **fixation probability** π of **benefical mutations** for **viruses** (haploid, varying size, high mutation rate (clonal interference), etc.).

$$\pi = f(\theta, N_{e,i}, N_{e,s}, \Delta s)$$

with Δs : selection differential between wild-type and mutant strains

Prospects

1. Experimentation and modelling : to follow the demo-genetic dynamics of PVY variants in different pepper genotypes by high-throughput sequencing, and to disentangle **genetic drift** and **selection** effects by fitting models to these data (Fabre *et al.* 2012, PLoS Pathog).

2. Experimental evolution : to record the **appearance** of virus **beneficial mutations** (against qualitative resistances) and their increase in frequency with time.

3. Modelling : to design and study an **epidemiological model** at the **landscape scale**, accounting for the **demo-genetic dynamics** of viruses at the **within-host scale**.