

# Quantitative trait loci in pepper genome control the effective population size of two RNA viruses at inoculation

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## Abstract

Infection of plants by viruses is a complex process that involves several steps: inoculation into plant cells, replication in inoculated cells, cell-to-cell movement during leaf colonization and long-distance movement during systemic infection. The success of the different steps is conditioned by the effective viral population size ( $N_e$ ) defined as the number of individuals that pass their genes to the *next generation*.

During the infection cycle, the virus population will endure several bottlenecks leading to drastic reductions in  $N_e$  and to the random loss of some virus variants. If strong enough, these bottlenecks could act against selection by eliminating the fittest variants. Therefore, a better understanding of how plant affects  $N_e$  may contribute to the development of durable virus-resistant cultivars. We aimed to (i) identify plant genetic factors that control  $N_e$  at the inoculation step, (ii) understand the mechanisms used by the plant to control  $N_e$  and (iii) compare these genetic factors with other genes controlling virus life cycle and plant resistance durability.

The virus effective population size was measured in a segregating population of 152 doubled-haploid lines of *Capsicum annuum*. Plants were inoculated mechanically either with a *Potato virus Y* (PVY) construct expressing the green fluorescent protein (GFP), or a necrotic variant of *Cucumber mosaic virus* (CMV), the CMV-N strain of Fulton.  $N_e$  was assessed by counting the number of primary infection foci observed on inoculated cotyledons under UV light for PVY-GFP or the number of necrotic local lesions observed on inoculated leaves for CMV-N.

The numbers of primary infection foci and local lesions were correlated among the doubled-haploid lines ( $r=0.57$ ) and showed a high heritability ( $h^2=0.93$  and  $0.98$  for PVY and CMV, respectively). The effective population size of the two viruses was shown to be controlled by both common quantitative trait loci (QTLs) and virus-specific QTLs, indicating the contribution of both general and specific mechanisms.

The PVY-specific QTL colocalizes with a QTL that had previously been shown to be involved in PVY accumulation and capacity to break a major-effect resistance gene down.