Experimental evolution of virulence

Understanding emergence of infectious diseases: Linking genomic evolution to the evolution of virulence of a phytovirus during the course of adaptation to its environments

ABSTRACT

Studies on the molecular mechanisms of virus adaptation could provide insights on the process underlying their emergence. However, such studies are scarse not only for viruses but alos for any other organisms. In this work, we propose to characterize adaptive mutations arising in viral populations, evolving either in constant or variable environments, and to evaluate their phenotypic expression.

Since the viral environment is mainly governed by the host, we designed an experimental protocol in which populations of Cauliflower mosaic virus were transmitted from plant to plant in two homogeneous environments (Arabidopsis thaliana or Nicotiana bigelovii), and also in a variable environment (alternating in both species). One single viral genotype was used to found all viral populations that evolved in parallel for each of these three treatments. During the time of the contract, we designed one technique to quantify viral DNA standardized by host DNA (targeting the number of actin gene). Moreover, we designed a new technique allowing a non-destructive quantification of the deleterious effect of viral infection on the development of the plant (through the measurement of the evolution of leaf area).

After five passages from plant to plant (±150 viral generations), we established the consensus full-length sequence of each viral populations and detected several fixed mutations in several populations. However, after ten passages, we did not observe these same mutations anymore. Moreover, evaluation of within-host viral load and virulence (i.e. reduction of leaf area due to viral infection) of viral populations that were passages from plant to plant did not allow us to reveal any particular pattern that would be a signature of adaptation (high variance within and among populations). It thus seems that mutations accumulating within viral populations are transitory. Taken all together, these results suggests that in our conditions, the effective size of populations of plant viruses (Ne) is relatively low leading to genetic drift as the predominant force compared to selection. Another hypothesis suggests that the time for accumulation of adaptive mutations is longer than spontaneous mutations and that it would be necessary to continue serial transfers of viruses in order to be able to observe contrasted patterns between the different environments.

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PERSPECTIVES

Evaluate the effective population size and mutation rate in different hosts