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Version définitive du manuscrit publié dans / Final version of the manuscript published in :
PNAS, 2007, vol.104, no.45, 17891-17896

Fig. 1

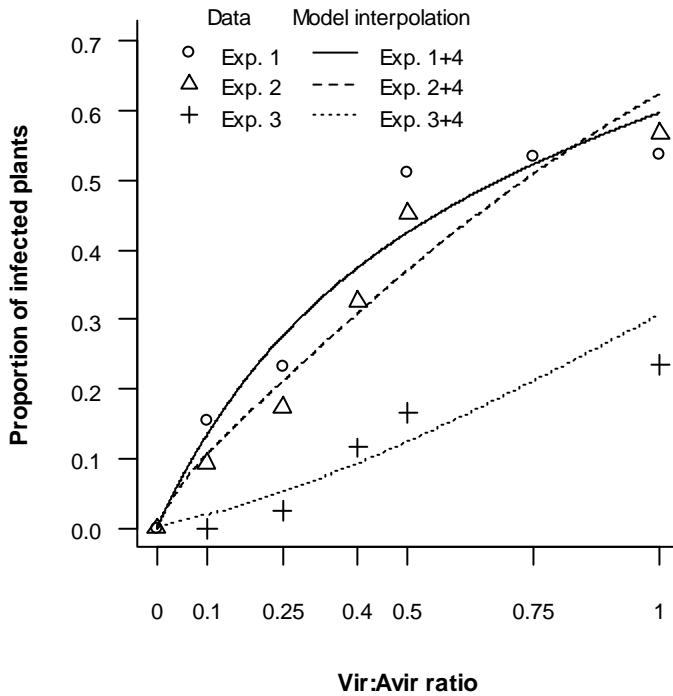
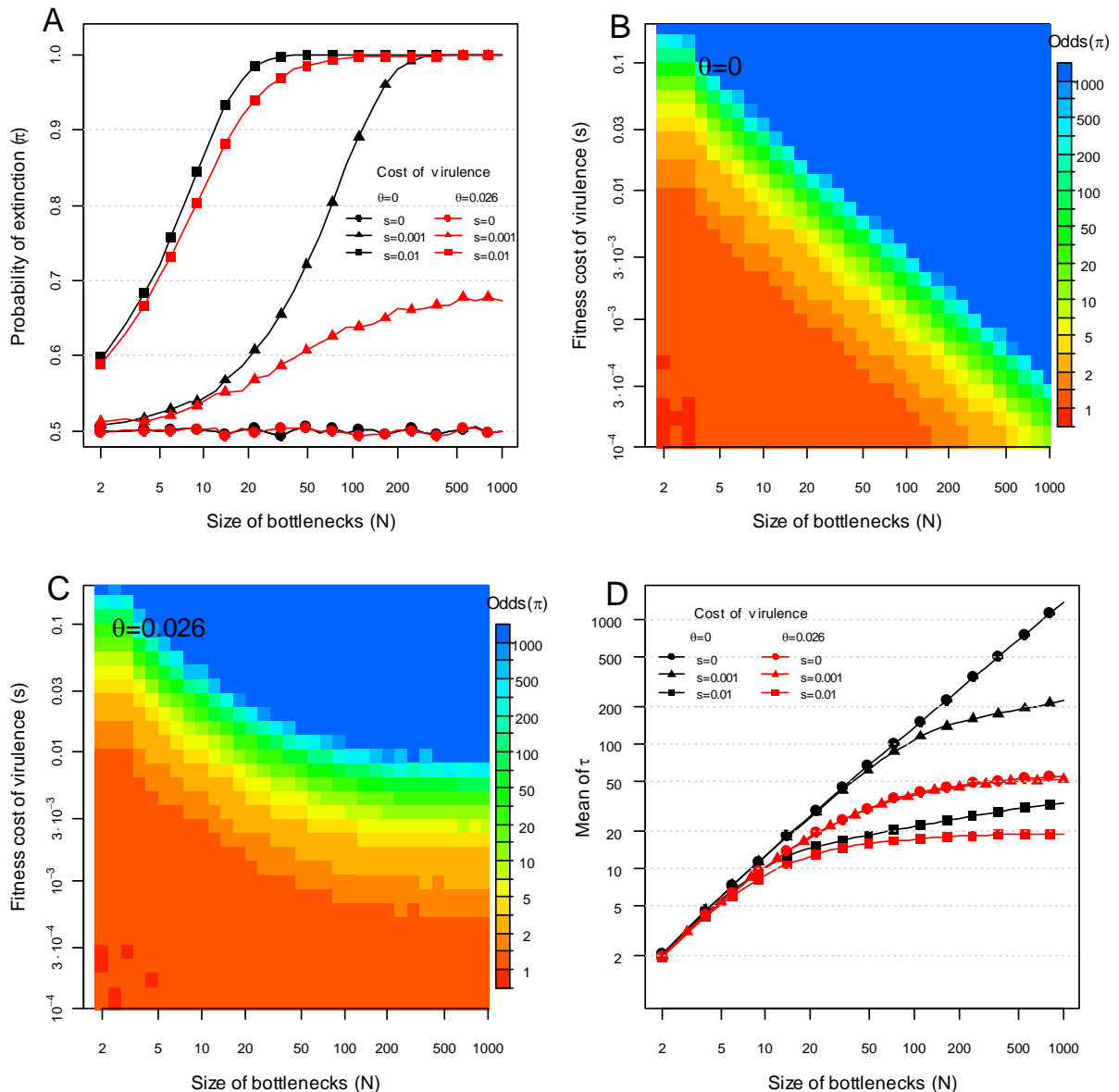


Fig.2



SI Methods

A stochastic virus transmission and plant infection model. The experimental results were modelled as the issue of three successive stochastic events. The first one describes virus acquisition. We assumed that Vir and Avir PVY particles have the same probability of being acquired and that the total number of viral particles acquired and further inoculated by an aphid to a plant follows a Poisson distribution with parameter λ_a . This means that the viral

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Moury, B., Fabre, F., Sénoussi, R. (2007). Estimation of the number of virus particles transmitted by an insect vector. Proceedings of the National Academy of Sciences of the United States of America, 104 (45), 17891-17896. , DOI : 10.1073/pnas.0702739104

particles that were acquired but not inoculated to the plants were not taken into account in our model. Hence, the number of transmitted Vir particles drawn from a c_i relative proportion of the Vir variant obeyed a Poisson distribution with parameter $\lambda_a \times c_i$. The Poisson assumption is plausible considering (i) that Vir and Avir particles were independently and homogeneously distributed in the acquisition medium and (ii) that they were indistinguishable by the aphids [1]. The second event deals with the inoculation process and introduces the number K of probes required by aphids to inoculate the acquired virus particles. The distribution of the number of inoculated virus particles according to the rank of the probe being unknown, we simply assumed that K follows a Poisson distribution with parameter κ . Hence, the number of viral particles inoculated during each aphid probe is Poisson distributed conditionally to K with parameter λ_a/K . Estimation of parameter κ was improved thanks to experiment 4 (Table 1), for which some of the aphids were only allowed to perform a single probing puncture. The third step concerns plant infection. With the kind of plant recessive resistance used, virulent variants do not trans-complement avirulent ones for infection of plants carrying the *pvr2^l* resistance gene, and avirulent variants do not impede these plants from being infected by the virulent ones [2,3]. Consequently, we assumed that the infection of a given plant depends only on the number of inoculated Vir particles and considered in the model that infection of that plant occurs if and only if the number of Vir particles inoculated is higher than, or equal to, a plant specific threshold value. To take into account the inter-plant heterogeneity due, for example, to variations in the inoculation site, this threshold was assumed to vary randomly between plants and to be Poisson distributed with parameter λ_p . Indeed, the threshold number of virus particles required for infecting a given plant could depend on a random number of obstacles (or cells) which should be successively overcome (or crossed) by the virus to yield a systemic infection, and can be derived via a Poisson process. Since PVY inoculations were carried out with two aphids per plant, two alternative models, “additive” and “non additive”

should be considered to describe plant infection. In the former model, infection of a given plant occurs if and only if the sum of the numbers of Vir particles transmitted by the two aphids is higher than, or equal to, the threshold for infection of that plant. Under the alternative model, infection takes place if and only if the number of Vir particles transmitted by at least one of the two aphids is higher than, or equal to, the threshold for infection. Previous experimental data obtained from inoculations of plants with 1, 5, 10 or 20 aphids per plant rejected the “additive” model and indicated that the “non additive” model was suitable for inoculations performed with up to 10 aphids per plant [4]. Consequently, we retained the “non additive” model for plant infection in the following analyses. When taking into account the multiple probes realized by aphids during inoculation, these probes were also considered to operate independently according to the “non additive” model for plant infection. In addition, since acquisition *in planta* was more efficient than acquisition *in vitro* (see Results), we defined λ_a^* as the number of virus particles transmitted after acquisition from PVY-infected leaves.

Formally, for any relative proportion c_i of the Vir PVY variant in the acquisition medium, let V_{ij} and R_{ij} be the number of Vir particles acquired by a single aphid and the threshold of Vir particles required to infect plant j , respectively. The infection status of plant j was modelled as a Bernoulli trial Z_{ij} (*i.e.* an experiment with only two possible outcomes), where $Z_{ij} = 1$ if the plant is infected (if $V_{ij} \geq R_{ij}$) and $Z_{ij} = 0$ if the plant is healthy. V_{ij} and R_{ij} are assumed to be independent and Poisson distributed with parameters $\lambda_a \times c_i$ and λ_p , respectively. The probability $p(Z_{ij} = 1)$ that plant j is infected expresses as follows:

$$p(Z_{ij} = 1) = 1 - \sum_{m=0}^{\infty} \exp(-\lambda_p) \frac{\lambda_p^m}{m!} \exp(-L\kappa(1 - \sum_{k=0}^m \frac{(\lambda_a c_i)^k}{k!})) \quad (1),$$

where L , the number of aphids used to inoculate the plants, is equal to one or two in our experiments.

Plants being independent, the four parameters λ_a , λ_a^* , κ and λ_p were estimated via maximum-likelihood techniques using the Nelder-Mead algorithm in the “R” statistical package [5] (<http://www.cran.org>) by maximizing the following log-likelihood function computed for several subsets S among experiments 1, 2, 3 and 4 (Tables 1 and 2):

$$\sum_{e \in S} \sum_{i=1}^{C_e} [I_{ie} \times \log[p(Z_{ije} = 1)] + H_{ie} \times \log[1 - p(Z_{ije} = 1)]] \quad (2),$$

where I_{ie} and H_{ie} denote the total numbers of infected and healthy plants, respectively, for the i^{th} Vir:Avir ratio in experiment e , and C_e is the total number of Vir:Avir ratios used in the corresponding experiment.

A simulation model to explore the effect of population bottlenecks on the extinction (or emergence) of plant virus variants.

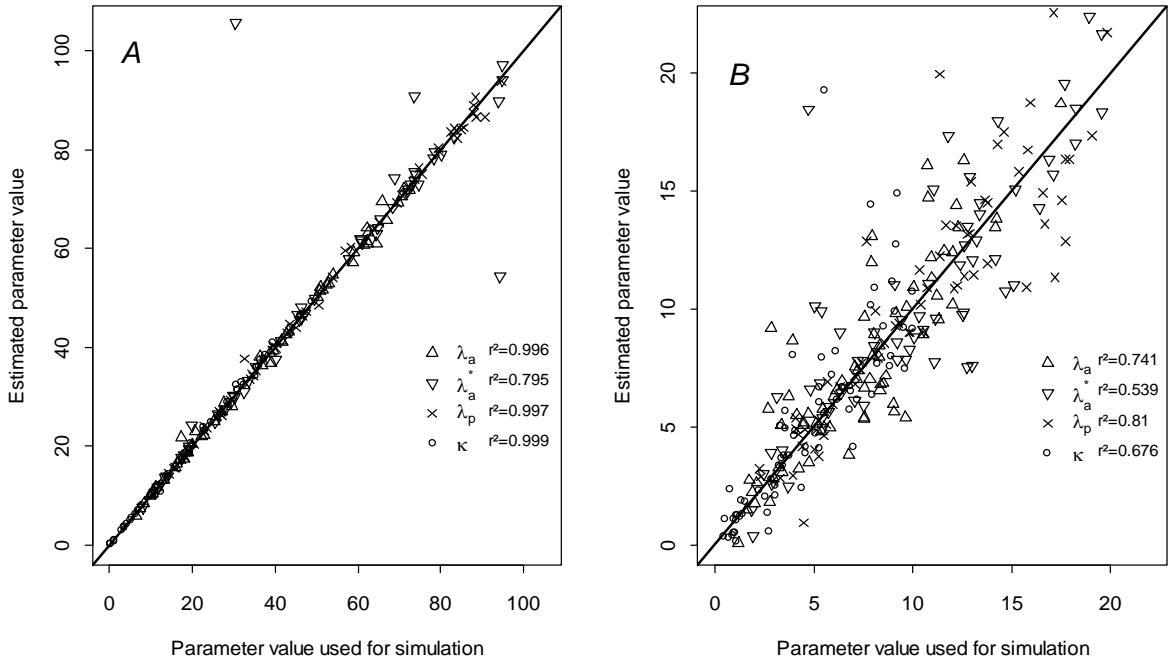
We consider a virus population composed of Vir and Avir individuals that evolve in successive steps including each a growth phase within plants followed by a sampling phase during which a fraction of the virus population is transmitted by aphids to a new plant. At step i , the within-plant growth dynamics of the virus population is described by a system of Lotka-Volterra’s equations adapted to the case of competing virus populations [6]:

$dN_{Vir,t}^i / dt = r_{Vir} \times N_{Vir,t}^i \times [K - N_{Vir,t}^i - (r_{Avir} / r_{Vir}) \times N_{Avir,t}^i] / K$ where (i) $N_{Vir,t}^i$ (respectively $N_{Avir,t}^i$) is the number of Vir (respectively Avir) virus particles at time t in an i^{th} plant (ii) r_{Vir} (respectively r_{Avir}), the growth rate of the Vir (respectively Avir) virus particles, is such that $r_{Vir} = r_{Avir} \times (1-s)$, s being a fitness cost associated to virulence in terms of replicative capacity and (iii) K is the plant carrying capacity. At $t=T$, when the virus population has reached K , N viruses are randomly sampled from plant i and transmitted to an $i+1$ healthy plant. Among these N viruses, a random number $N_{Vir,t=0}^{i+1}$ of Vir particles is drawn from a beta-binomial distribution with parameters N , $\alpha = p_T^i / \theta$ and $\beta = (1 - p_T^i) / \theta$ where

$p_T^i = N_{Vir,T}^i / (N_{Vir,T}^i + N_{Avir,T}^i)$. For the initial step, we fixed $p_T^0 = 0.5$. Note that $N_{Vir,t=0}^{i+1}$ has mean $N \times p_T^i \times (1 - p_T^i)$ and variance $N \times p_T^i \times (1 - p_T^i) \times (1 + N\theta) \times (1 + \theta)^{-1}$. Parameter θ measures the rate of aggregation of Vir and Avir particles within plants [7]. In the case $\theta=0$, $N_{Vir,t=0}^{i+1}$ is binomial distributed, *i.e.* viral particles are independently and homogeneously distributed into plants. When $\theta>0$, Vir and Avir viruses tend to be aggregated into plants. Steps of phases of population growth and sampling are repeated until the Vir variants are extinct. The probability of extinction of the Vir viruses and of the number of steps τ necessary to achieve this extinction were estimated from 25,000 simulations for each parameter set (N , s , θ). For all simulations, T , r_{Avir} and K were set to 20, 1 and 10^8 , respectively.

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SI Fig. S1



SI Fig. S2

