

Virus adaptation to low host density

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One of the most relevant factors affecting viral propagation is the density of accessible hosts, which determines the number of virus-host contacts and, consequently, the probability of new infections. The lower the host density, the longer the virus spends in the external environment between successive infections, thus increasing its probability of degradation due to physical-chemical variables. There should be a critical host density separating sustained propagation from extinction. In the latter situation, the virus is under a selective pressure to fix survival strategies under limited host availability. The study of these strategies is important from an evolutionary viewpoint, and it can significantly affect the epidemiological properties of a given virus, potentially turning local outbreaks into endemic propagation. In this work we have carried out an evolution experiment in which the bacteriophage Q β , a virus with an RNA genome that infects *Escherichia coli* was propagated in the presence of suboptimal host concentrations at either 37 °C (optimal temperature) or 43 °C (suboptimal temperature). As other RNA viruses, Q β replicates with very high error rate (between 10⁻⁴ and 10⁻⁶ errors per copied nucleotide) and has a quasispecies population structure.

Our results showed that the minimal host concentration compatible with sustained propagation of the virus depends

on temperature. After a certain number of generations, all lineages propagated at suboptimal host concentration and optimal temperature selected a mutation in a minor capsid protein whose phenotypic effect was to favor the entry of the virus into the cell, with no significant effects on other parameters characterizing the infectious cycle. In contrast to this, Q β adaptation to suboptimal host density at 43 °C took place through a different mutation, located in the virus protein involved in virus adsorption to the bacteria and also in virus release to the external environment once a progeny has been produced. Whereas at optimal temperature the time period that the virus remains within the bacteria was not modified, at 43 °C it was significantly longer. As a consequence, the virus spends less time in the external medium, a behavior similar to that shown by viruses that infect hyperthermophile microorganisms in nature. Although it is difficult to extrapolate our results to more complex situations, as it could be the spread of epidemics in the human population, they provide support for the idea that containment measures based on the reduction of contacts between people (the equivalent to reducing the number of hosts in our system) constitute a selective pressure that may lead to adaptive changes in viruses.