Viral evolution is a subfield of evolutionary biology and virology

Elena Pierre
Spain

Abstract

Viruses have short generation times, and many—in particular RNA viruses—have relatively high mutation rates (on the order of one point mutation or more per genome per round of replication). This elevated mutation rate, when combined with natural selection, allows viruses to quickly adapt to changes in their host environment. In addition, most viruses provide many offspring, so any mutated genes can be passed on to many offspring quickly. Although the chance of mutations and evolution can change depending on the type of virus (double stranded DNA, double stranded RNA, single strand DNA, etc.), viruses overall have high chances for mutations. Viral evolution is an important aspect of the epidemiology of viral diseases such as influenza (influenza virus), AIDS (HIV), and hepatitis (e.g. HCV). The rapidity of viral mutation also causes problems in the development of successful vaccines and antiviral drugs, as resistant mutations often appear within weeks or months after the beginning of a treatment. One of the main theoretical models applied to viral evolution is the quasispecies model, which defines a viral quasispecies as a group of closely related viral strains competing within an environment. Three classical hypotheses. Viruses are ancient. Studies at the molecular level have revealed relationships between viruses infecting organisms from each of the three domains of life, suggesting viral proteins that pre-date the divergence of life and thus infecting the last universal common ancestor. This indicates that some viruses emerged early in the evolution of life and that they have probably arisen multiple times. It has been suggested that new groups of viruses have repeatedly emerged at all stages of evolution, often through the displacement of ancestral structural and genome replication genes.

There are three classical hypotheses on the origins of viruses and how they evolved:

Virus-first hypothesis: Viruses evolved from complex molecules of protein and nucleic acid before cells first appeared on earth. By this hypothesis, viruses contributed to the rise of cellular life. This is supported by the idea that all viral genomes encode proteins that do not have cellular homolog's. The virus-first hypothesis has been dismissed by some scientists because it violates the definition of viruses, in that they require a host cell to replicate. Reduction hypothesis (degeneracy hypothesis): Viruses were once small cells that parasitized larger cells. This is supported by the discovery of giant viruses with similar genetic material to parasitic bacteria. However, the hypothesis does not explain why even the smallest of cellular parasites do not resemble viruses in any way. Escape hypothesis (vagrancy hypothesis): Some viruses evolved from bits of DNA or RNA that "escaped" from the genes of larger organisms. This doesn't explain the structures that are unique to viruses and are not seen anywhere in cells. It also does not explain the complex capsids and other structures of virus particles.

Virologists are in the process of re-evaluating these hypotheses.

Coevolution hypothesis (Bubble Theory): At the beginning of life, a community of early replicons (pieces of genetic information capable of self-replication) existed in proximity to a food source such as a hot spring or hydrothermal vent. This food source also produced lipid-like molecules self-assembling into vesicles that could enclose replicons. Close to the food source replicons thrived, but further away the only non-diluted resources would be inside vesicles. Therefore, evolutionary pressure could push replicons along two paths of development: merging with a vesicle, giving rise to cells; and entering the vesicle, using its resources, multiplying and leaving for another vesicle, giving rise to viruses. Chimeric-origins hypothesis: Based on the analyses of the evolution of the replicative and structural modules of viruses, a chimeric scenario for the origin of viruses was proposed in 2019. According to this hypothesis, the replication modules of viruses originated from the primordial genetic pool, although the long course of their subsequent evolution involved many displacements by replicative genes from their cellular hosts. By contrast, the genes encoding major structural proteins evolved from functionally diverse host proteins throughout the evolution of the virosphere. This scenario is distinct from each of the three traditional scenarios but combines features of the Virus-first and Escape hypotheses. One of the problems for studying viral origins and evolution is the high rate of viral mutation, particularly the case in RNA retroviruses like HIV/AIDS. A recent study based on comparisons of viral protein folding structures, however, is offering some new evidence. Fold Super Families (FSF's) are proteins that show similar folding structures independent of the

actual sequence of amino acids, and have been found to show evidence of viral phylogeny. Thus viruses have been found to be capable of being divided into 4 FSFs; based upon the three realms of bacterioviruses, archaeoviruses, and eukaryoviruses, together with a fourth FSF that seems to indicate that it pre-dated the separation of the three realms. The proteome of a virus, the viral proteome, still contains traces of ancient evolutionary history that can be studied today. The study of protein FSFs suggests the existence of ancient cellular lineages common to both cells and viruses before the appearance of the 'last universal cellular ancestor' that gave rise to modern cells. Evolutionary pressure to reduce genome and particle size may have eventually reduced viro-cells into modern viruses, whereas other coexisting cellular lineages eventually evolved into modern cells. Furthermore, the long genetic distance between RNA and DNA FSF's suggests that the RNA world hypothesis may have new experimental evidence, with a long intermediary period in the evolution of cellular life. A final exclusion of a hypothesis on the origin of viruses is difficult to make on earth because viruses and cells interact with each other everywhere today and very old rocks in which old traces of viruses could be found are probably rarely or no longer present on Earth. From an astrobiological point of view it has therefore already been proposed that on celestial bodies such as Mars not only cells but also traces of former virions or viroids should be actively searched for. If only traces of virions but no cells are found on another celestial body, this would be a strong indication of the virus-first hypothesis.