

分子擬態が明らかにする巨大ウイルスと宿主の共進化関係

Molecular mimicry reveals the co-evolution relationship in giant viruses and their hosts

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研究成果概要

Background:

The relationship between viruses and their hosts represents an example of a molecular arms race. Under persistent evolutionary selective pressures, both parties have developed sophisticated strategies to secure their own survival advantages. Viruses continuously refine their infectivity and transmissibility, whereas hosts strive to evade viral invasion and deploy complex defense mechanisms. This intense reciprocal interaction drives the rapid evolution of viral functional molecules and the pronounced divergence of their sequences. Specifically, owing to their tolerance for genetic variation and inherently high mutation rates, many viral genes undergo extensive sequence diversification to achieve immune evasion and heightened host adaptation.

This molecular-level confrontation fundamentally shapes the characteristics of viral genomes. Sustained selective pressure for adaptive changes—particularly those enabling escape from host immune recognition—results in rapid sequence divergence and the erosion of detectable genetic signals. Consequently, traditional bioinformatic methods that rely on sequence homology (such as BLAST) often face systematic challenges in identifying distant viral homologs and reconstructing their evolutionary histories. This underscores the scientific necessity of moving beyond sequence-based analyses and leveraging conserved protein structural features to explore distant homology and potential biological functions.

Molecular mimicry is one of the most efficient and covert strategies employed by viruses in the evolutionary arms race. This strategy enables viruses to exploit structural similarity to imitate functional motifs or domains of host proteins, thereby hijacking or disrupting host cellular processes, including nucleic acid metabolism and the regulation of immune responses. Molecular mimicry has been widely recognized as a mechanism that can trigger autoimmune diseases, in which host responses to viral or microbial infection inadvertently cross-react with corresponding host antigens.

Molecular mimicry has been extensively studied in a variety of human-targeting viruses, such as members of the Poxviridae and Herpesviridae families[1]. Poxviruses, for example, encode molecules known as virokines and viroreceptors that mimic host cytokines, complement regulators, or other humoral inhibitors, effectively suppressing antiviral immunity or inflammatory processes[2]. These studies commonly focus on the mimicry of host short linear amino acid motifs (SLMs), which viruses leverage to evade immune detection. However,