Perspective

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Mechanisms Modulating Viral Polymerase-Mediated Mutations and Their Potential Consequences

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Description

During genome replication with the error-prone viral polymerase, viral evolution begins. Natural selection will select a small number of well-adapted variants from the mutants produced by this process, thereby regenerating the viral genome population. Errors caused by viral polymerase are thought to occur randomly. However, there is mounting evidence to suggest that viral polymerase-mediated mutations are dispersed throughout the viral genome in a variety of ways. Here, we look at research that backs up this idea and sheds light on mechanistic explanations for how specific parts of the virus's genome might affect errors caused by viral polymerase. It is possible that a predisposition to accumulate viral polymerase-mediated errors at specific loci in the viral genome will direct evolution to particular pathways, opening up new research avenues for comprehending the dynamics of viral evolution.

Significant Effects on Viral Evolutionary Dynamics

An evolving virus can be easily identified by its capacity to produce numerous genome variants in its offspring. The haze of viral genome relatives, at first depicted on account of RNA infections in view of the great mistake pace of viral RNA polymerases, is characterized as viral quasispecies. As will be discussed in the following reviews, this property has significant effects on viral evolutionary dynamics, regulating viral fitness, immune escape, drug resistance, virulence, host adaptation, and "social" interactions between variants. After the action of multiple filters or bottlenecks that reduce the size of the mutant spectrum population, specific errors or mutations are fixed by natural selection and then passed down. Because these regions of the genome are more resistant to mutation, they are more likely to accumulate mutations. In addition, portions of the viral particle are subjected to severe bottlenecks mediated by the immune system. These bottlenecks tend to select immune-escaping viruses that contain novel antigenic sites. As a result, there is a lot of nucleotide variability associated with these antigenic sites. The immune system's primary targets, viral Glycoproteins (GPs), evolve more quickly than other viral proteins. On the other hand, some internal proteins have domains that are more structurally and/or functionally constrained and are therefore shielded from neutralizing antibodies, making them less susceptible to mutations and more evolutionary stable.

Influence of the Viral Genome Properties on Host-Dependent Editing

However, there is mounting evidence that the viral genome template itself may be able to influence mutational events. Numerous RNA and DNA viruses have had their genomes altered by host enzymes. The influence of the viral genome properties on host-dependent editing is demonstrated by the fact that these host editing enzymes target specific genome locations that are enriched in particular base composition or RNA/DNA structures. There will be no further discussion of host editing enzymes here. Recent chemical mappings suggested that the extensive RNA structures in the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) genome could facilitate polymerase template switching. These structures co-occur with hotspots of template switching, which are characterized by insertion and deletion scars in the viral genome. In accordance with this observation, the *Bromoviridae* family's brome mosaic virus undergoes non-homologous recombination events that appear to be dependent on the viral genome's folding and primary sequence. These results add to earlier studies that suggested that the ribonucleoproteins of the influenza A virus might have RNA structures that triggered polymerase-jumps and encouraged the production of defective genome segments. As a result, a more precise investigation into how the genetic environment might affect mutation rates may benefit from the design of viable infectious viruses that allow for a portion of their genome to be free of selective pressures. An interesting challenge in viral evolutionary biology is to gain a better understanding of the mechanisms that control viral polymerase-mediated mutations and the possible effects these mechanisms could have on viral evolution.