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A Retrospective Study of the Orthopoxvirus Molecular Evolution

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DESCRIPTION

Orthopoxviruses are a family of large, enveloped, double-stranded DNA viruses that cause significant disease in humans and animals. The most well-known member of this family is the variola virus, which causes smallpox. While smallpox has been eradicated, other orthopoxviruses, such as monkeypox and cowpox, continue to pose a threat. To better understand the progression and impact of orthopoxvirus infections, it is crucial to examine the stages involved in the viral life cycle and the subsequent host response. In this article, we will delve into the stages of orthopoxvirus infections, including viral entry, replication, assembly, release, and the immune response elicited by the host. The first stage of orthopoxvirus infection involves viral entry into the host cell. This process primarily occurs through direct contact with the virus, either through respiratory droplets, contact with infected skin lesions, or ingestion of contaminated materials. The orthopoxvirus enters the host cell through membrane fusion or endocytosis, facilitated by viral envelope proteins. Once inside the host cell, the viral particle disassembles, releasing the viral genome. After entering the host cell, the orthopoxvirus begins to replicate its genome and express viral genes. The viral genome is a large, linear, double-stranded DNA molecule that encodes numerous proteins essential for viral replication and evasion of the host immune response. The orthopoxvirus replicates in the cytoplasm of the host cell and utilizes the cellular machinery to transcribe and translate viral genes. The expression of early genes promotes viral DNA replication, while late genes encode structural proteins required for viral assembly. During the assembly stage, the newly synthesized viral components are orchestrated to form mature infectious virions. Orthopoxvirus assembly occurs in the cytoplasm and is a highly complex process involving the recruitment and assembly of viral proteins and the acquisition of a lipid envelope from the host cell. As the viral components assemble, immature virions undergo morphological changes, ultimately resulting in the formation of mature, infectious virions. These mature virions are then ready for release from the host cell. After assembly, the mature orthopoxvirus particles are released from the host cell. This can occur through cell lysis, in which the host cell ruptures due to an overwhelming viral burden, or by a process known as budding, where the virus acquires a lipid envelope from the host cell membrane and exits the cell without causing immediate cell death. Once released, the virus can infect neighboring cells or spread to other individuals through respiratory droplets, contact with infected materials, or direct contact with infected lesions. Simultaneously with viral replication and spread, the host immune system recognizes the presence of orthopoxvirus and initiates an immune response. The immune response consists of both innate and adaptive components aimed at controlling and eliminating the infection. The innate immune response involves the activation of various immune cells, such as natural killer cells, macrophages, and dendritic cells, which recognize the virus and release cytokines to recruit and activate other immune cells. The adaptive immune response involves the activation of T and B lymphocytes, which recognize specific viral antigens and mount a targeted immune response to eliminate the virus. The progression and clinical manifestations of orthopoxvirus infections can vary depending on the specific virus involved. Smallpox, caused by variola virus, has distinct clinical stages, including an incubation period, followed by an initial febrile prodrome and subsequent development of characteristic skin lesions. Monkeypox, another orthopoxvirus infection, presents with a similar clinical picture but is generally less severe than smallpox.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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