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## **STRUCTURE AND INTRACELLULAR ACTIVITY OF THE DNA-CONTAINING HERPES SIMPLEX VIRUS**

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### **Annotation**

Herpes simplex viruses (HSV-1, HSV-2; Herpesvirus hominis) produce a variety of infections involving muco-cutaneous surfaces, the central nervous system (CNS), and—on occasion—visceral organs. Prompt recognition and treatment reduce the morbidity and mortality associated with HSV infections.

**Key words:** genome, virus, protein, intracellular, replication, receptor, synthesis.

The genome of HSV is a linear, double-strand DNA molecule (molecular weight,  $\sim 100 \times 10^6$  units) that encodes >90 transcription units with 84 identified proteins. The genomic structures of the two HSV subtypes are similar. The overall genomic sequence homology between HSV-1 and HSV-2 is  $\sim 50\%$ , whereas the proteome homology is  $>80\%$ . The homologous sequences are distributed over the entire genome map, and most of the polypeptides specified by one viral type are antigenically related to polypeptides of the other viral type. Many type-specific regions unique to HSV-1 and HSV-2 proteins do exist, however, and a number of them appear to be important in host immunity. These type-specific regions have been used to develop serologic assays that distinguish between the two viral subtypes. Either restriction endonuclease analysis of viral DNA or DNA sequencing can be used to distinguish between the two subtypes and among strains of each subtype. The variability of nucleotide sequences from clinical strains of HSV-1 and HSV-2 is such that HSV isolates obtained from two individuals can be differentiated by restriction enzyme patterns or genomic sequences. Moreover, epidemiologically related sources, such as sexual partners, mother-infant pairs, or persons involved in a common-source outbreak, can be inferred from such patterns.



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The viral genome is packaged in a regular icosahedral protein shell (capsid) composed of 162 capsomeres. The outer covering of the virus is a lipid-containing membrane (envelope) acquired as the DNA-containing capsid buds through the inner nuclear membrane of the host cell. Between the capsid and lipid bilayer of the envelope is the tegument. Viral replication has both nuclear and cytoplasmic phases. Attachment and fusion of the viral envelope and the cell membrane involve several ubiquitous heparin-like surface receptors. Replication is highly regulated. After fusion and entry, the nucleocapsid enters the cytoplasm and several viral proteins are released from the virion. Some of these viral proteins shut off host protein synthesis (by increasing cellular RNA degradation), whereas others “turn on” the transcription of early genes of HSV replication. These early gene products, designated  $\alpha$  genes, are required for synthesis of the subsequent polypeptide group, the  $\beta$  polypeptides, many of which are regulatory proteins and enzymes required for DNA replication. Most current antiviral drugs interfere with  $\beta$  proteins, such as the viral DNA polymerase enzyme. The third ( $\gamma$ ) class of HSV genes requires viral DNA replication for expression and constitutes most of the structural proteins specified by the virus. After replication of the viral genome and synthesis of structural proteins, nucleocapsids are assembled in the nucleus of the cell. Envelopment occurs as the nucleocapsids bud through the inner nuclear membrane into the perinuclear space. In some cells, viral replication in the nucleus forms two types of inclusion bodies: type A basophilic Feulgen-positive bodies that contain viral DNA and eosinophilic inclusion bodies that are devoid of viral nucleic acid or protein and represent a “scar” of viral infection. Enveloped virions are then transported via the endoplasmic reticulum and the Golgi apparatus to the cell surface.

HSV infection of some neuronal cells does not result in cell death. Instead, viral genomes are maintained by the cell in a repressed state compatible with survival and normal activities of the cell, a condition called latency. Latency is associated with transcription of only a limited number of virus-encoded proteins. Subsequently, the viral genome may become activated; its activation results in the normal pattern of regulated viral gene expression, viral replication, and viral release. The release of HSV from the neuron and its subsequent entry into epithelial cells result in viral replication in these cells, destruction of the cells, and the subsequent reappearance



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of virus on mucosal surfaces. This process is termed reactivation. Whereas infectious virus is rarely recovered from sensory or autonomic nervous system ganglia dissected from cadavers, maintenance and growth of the neural cells (as “explants”) in tissue culture result in production of infectious virions and in subsequent permissive infection of susceptible cells (cocultivation). The mechanisms by which latency is established, maintained, or broken are incompletely understood. Two RNA “latency-associated” transcripts that overlap the immediate early ( $\alpha$ ) gene products, called ICP-O, are found in abundance in the nuclei of latently infected neurons. Deletion mutants of this region that can become latent have been made. However, the efficiency of their later reactivation is reduced; thus these latency associated transcripts may play a role in maintaining rather than in establishing latency. Recent studies suggest that HSV-specific micro-RNAs in these and other regions of the viral genome may play an important role in virus maintenance in and release from neurons. CD8<sup>+</sup> T cells have been found in ganglia of experimental animals and humans and appear to influence the process of reactivation, possibly by inducing antiviral factors such as interferon (IFN)  $\gamma$ . At present, strategies to interrupt latency or to maintain molecular latency in neurons are not available. In experimental animals, ultraviolet light, systemic and local immunosuppression, and trauma to the skin or ganglia are associated with reactivation.

All in all, exposure to HSV at mucosal surfaces or abraded skin sites permits entry of the virus and initiation of its replication in cells of the epidermis and dermis. HSV infections are usually acquired subclinically. Whether clinical or subclinical, HSV acquisition is associated with sufficient viral replication to permit infection of either sensory or autonomic nerve endings.

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