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Herpes Simplex Viruses 1 and 2: Virology, Pathogenesis, Epidemiology, and Clinical Management: A Comprehensive Review

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Abstract

Herpes Simplex Virus types 1 and 2 (HSV-1 and HSV-2) are ubiquitous human pathogens belonging to the *Alphaherpesvirinae* subfamily. They establish lifelong infections characterized by periods of latency and reactivation, leading to a significant global health burden. HSV-1 is traditionally associated with orofacial infections (cold sores), while HSV-2 is the primary cause of genital herpes. However, changing sexual practices have led to an increasing proportion of genital herpes cases attributable to HSV-1. A substantial feature of both viruses is asymptomatic shedding, which is a major driver of transmission. This review provides a detailed examination of the classification, structure, and genomic organization of HSV. We delve into the molecular mechanisms of viral replication, immune evasion, and the establishment of latency in sensory ganglia. The clinical manifestations, ranging from mucocutaneous ulcers to life-threatening encephalitis and neonatal herpes, are comprehensively described. Global epidemiological trends are analyzed, highlighting the disparities in seroprevalence between regions and demographic groups. Furthermore, the paper discusses current diagnostic methodologies, including viral culture, serological type-specific assays, and the gold-standard molecular PCR tests. Finally, we review the available antiviral treatments, preventive strategies, and the ongoing challenges in vaccine development. The aim of this review is to consolidate current knowledge on HSV, emphasizing its complex biology and significant public health impact.

Keywords: Herpes Simplex Virus, HSV-1, HSV-2, Genital Herpes, Orofacial Herpes, Viral Latency

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Introduction

Infection with the herpes simplex virus, commonly known as herpes, can be due to either herpes simplex virus type 1 (HSV-1) or herpes simplex virus type 2 (HSV-2). HSV-1 is mainly transmitted by oral to oral contact to cause infection in or around the mouth (oral herpes). HSV-2 is almost exclusively sexually transmitted, causing infection in the genital or anal area (genital herpes). However, HSV-1 can also be transmitted to the genital area through oral-genital contact to cause genital herpes. Both oral herpes infections and genital herpes infections are mostly asymptomatic but can cause mild symptoms or painful blisters or ulcers at the site of infection.

HSV-1 is a highly contagious infection, which is common and endemic throughout the world. Most HSV-1 infections are acquired during childhood, and infection is lifelong. The vast majority of HSV-1 infections are oral herpes (infections in or around the mouth, sometimes called or labial, oral-labial or oral-facial herpes), but a proportion of HSV-1 infections are genital

herpes (infections in the genital or anal area).In 2012, an estimated 3.7 billion people under the age of 50, or 67% of the population, had HSV-1 infection. Estimated prevalence of the infection was highest in Africa (87%) and lowest in the Americas (40-50%).With respect to genital HSV-1 infection, 140 million people aged 15-49-years were estimated to have genital HSV-1 infection worldwide in 2012, but prevalence varied substantially by region. Most genital HSV-1 infections are estimated to occur in the Americas, Europe and Western Pacific, where HSV-1 continues to be acquired well into adulthood. In other regions, for example in Africa, most HSV-1 infections are acquired in childhood, before the age of sexual debut. Oral herpes infection is mostly asymptomatic, and the majority of people with HSV-1 infection are unaware they are infected. Symptoms of oral herpes include painful blisters or open sores called ulcers in or around the mouth. Sores on the lips are commonly referred to as "cold sores." Infected persons will often experience a tingling, itching or burning sensation around their mouth, before the appearance of sores. After initial infection, the blisters or ulcers can periodically recur. The frequency of recurrences varies from person to person. HSV-2 infection is widespread throughout the world and is almost exclusively sexually transmitted, causing genital herpes. HSV-2 is the main cause of genital herpes, which can also be caused by herpes simplex virus type 1 (HSV-1). Infection with HSV-2 is lifelong and incurable. Genital herpes caused by HSV-2 is a global issue, and an estimated 417 million people worldwide were living with the infection in 2012. Prevalence of HSV-2 infection was estimated to be highest in Africa (31.5%), followed by the Americas (14.4%). It was also shown to increase with age, though the highest numbers of people newly-infected were adolescents. More women are infected with HSV-2 than men; in 2012 it was estimated that 267 million women and 150 million men were living with the infection. This is because sexual transmission of HSV is more efficient from men to women than from women to men. Genital herpes infections often have no symptoms, or mild symptoms that go unrecognized. Most infected people are unaware that they have the infection. Typically, about 10-20% of people with HSV-2 infection report a prior diagnosis of genital herpes (Thompson *et al.*, 2014).

Genome Nature

Herpes simplex virus is a dsDNA virus and it is non-segmented. The envelope is derived from the nuclear membrane of the infected cell. The genome (Figs. 1 & 2) possesses 75 genes, arranged and without long range ordering. Introns are present in only a few genes. Two classes of genetic entities necessary for virus DNA replication have been characterized: cis-acting sequences, which include origin of replication and packaging signals (McGeoch, 1987).

Classification

Herpes simplex virus can be classified using the binomial classification.
It belongs to group 1{dsDNA}
Order: Herpesvirales
Family: Herpesviridiae
Sub family: Alpha-herpesvirinae
Genus: *Simplex virus*
Species: Herpes *simplex virus 1(HSV-1)*
Herpes simplex virus 2(HSV_2)
Vernacular name: HSV 1-Cold Sores
HSV 2-Herpes or genital herpes (World Health Organisation, 2017).

Structure of Herpes Simplex Virus

The structure of herpes viruses consists of a relatively large double-stranded, linear DNA genome encased within an icosahedral protein cage called the capsid, which is wrapped in a lipid bilayer called the envelope. The envelope is joined to the capsid by means of a tegument. This complete particle is known as the virion. HSV-1 and HSV-2 each contain at least 74 genes (or open reading frames, ORFs) within their genomes (McGeoch *et al.*, 2006*)* although speculation over gene crowding allows as many as 84 unique protein coding genes by 94 putative ORFs (Rajcani *et al.*, 2004). These genes encode a variety of proteins involved in forming the capsid, tegument and envelope of the virus, as well as controlling the replication and infectivity of the virus.

Proteins

It has recently been proposed that the herpes simplex virus (HSV) protein ICP0 has cytoplasmic roles in blocking antiviral signaling and in promoting viral replication in addition to its well-known proteasome-dependent functions in the nucleus. However, the mechanisms through which it produces these effects remain unclear. While investigating this further, we identified a novel cytoplasmic interaction between ICP0 and the poorly characterized cellular protein WDR11. During an HSV infection, WDR11 undergoes a dramatic change in localization at late times in the viral replication cycle, moving from defined perinuclear structures to a dispersed cytoplasmic distribution. While this relocation was not observed during infection with viruses other than HSV-1 and correlated with efficient HSV-1 replication, the redistribution was found to occur independently of ICP0 expression, instead requiring viral late gene expression. We demonstrate for the first time that WDR11 is localized to the *trans*-Golgi network (TGN), where it interacts specifically with some, but not all, HSV virion components, in addition to ICP0. Knockdown of WDR11 in cultured human cells resulted in a modest but consistent decrease in yields of both wild-type and ICP0-null viruses, in the supernatant and cell-associated fractions, without affecting viral gene expression. Although further study is required, we propose that WDR11 participates in viral assembly and/or secondary envelopment (Thompson *et al.*, 2014).

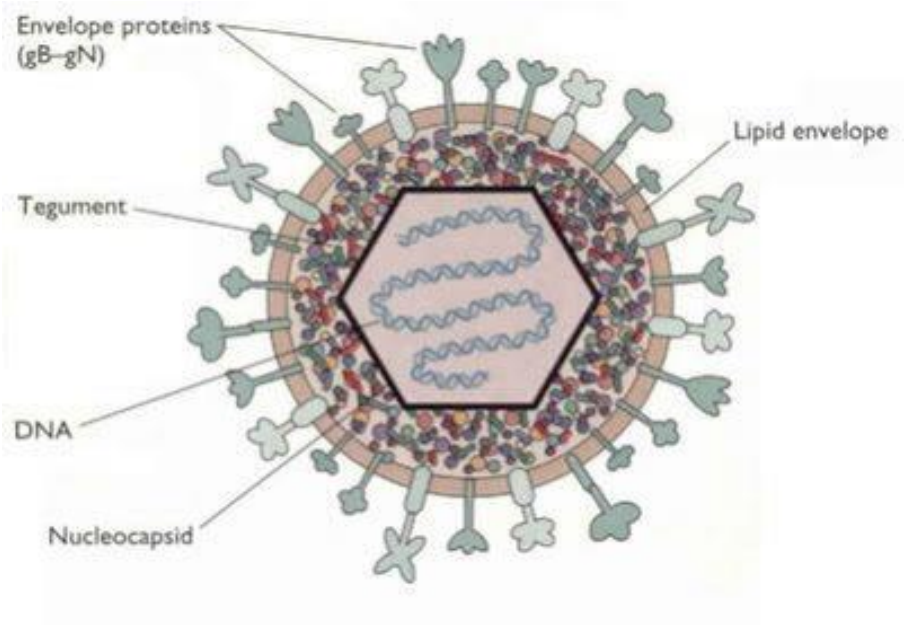

Figure 1: Herpes simplex virus genome
Source: Acharya (2010)

Genetic Bases.

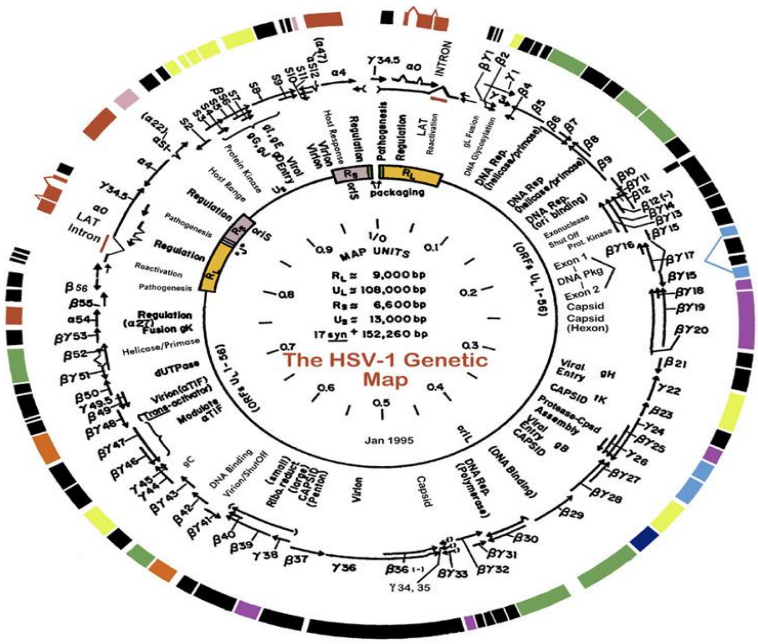

Figure 2: Herpes simplex virus
Source: Edwagher (2003)

Genes for Adaptation

Herpes Simplex Virus type 1 encodes several genes essential for viral DNA replication and for the replication of origin-containing plasmid DNA (Schaffer *et al*., 1973; Weller *et al*., 1983; Marchetti *et al*., 1988; Wu *et al*., 1988). Among these, the *UL30* gene encodes a 136 kDa viral DNA polymerase (Pol) (Purifoy *et al*., 1977; Challberg *et al*., 1980), while the *UL42* gene encodes a 65 kDa double-stranded DNA-binding protein (UL42) (Parris *et al*., 1988). Within infected cells, the products of these two genes interact to form a heterodimeric complex (Pol/UL42) (Gallo *et al*., 1988).

Recombinant bicistronic viruses have been used to overexpress Pol and UL42 as individual subunits, enabling detailed analysis of the effect of UL42 on Pol

activity (Hernandez & Lehman, 1990; Gottlieb *et al*., 1990). Biochemical studies demonstrate that although Pol possesses intrinsic catalytic activity, this activity is markedly enhanced when complexed with UL42 (Hernandez & Lehman, 1990; Gottlieb *et al*., 1990; Gallo *et al*., 1989). The enhancement occurs because UL42 increases the processivity of Pol (Gottlieb *et al*., 1990; Hernandez & Lehman, 1990).

Further evidence indicates that the Pol/UL42 complex is more resistant than Pol alone to salt-dependent increases in Km values for activated calf thymus DNA, and that the Vmax of Pol/UL42 rises with increasing salt concentration (Hart & Boehme, 1992). Although it is established that Pol and UL42 associate to form a replication-competent complex capable of efficiently replicating the 153 kbp viral genome, the precise mechanism of complex formation remains unclear. Co-immunoprecipitation studies have shown that the C-terminal region of Pol (amino acids 1008–1235) constitutes the UL42-binding domain (Digard & Coen, 1990; Digard *et al*., 1993a; Tenney *et al*., 1993a). Similarly, the Pol-binding domain of UL42 has been mapped to amino acids 1–315, suggesting that its C-terminal region is not essential for binding (Tenney *et al*., 1993b). In this study, proteolytic digestion of UL42 was employed to further investigate the nature of this interaction.

Pathogenicity

The virus multiples in the epithelium of the mouth of the throat, as it destroys the cells. The blisters that forms contains large number of infection virions. Some Epithelial cells fuse together, producing large, multinucleated giant cells. Nuclei of infected cells characteristically contain a staining area called an intranuclear body, which is the site of earlier viral replication. Some virions are carried by vessels to the nearby lymph nodes; response develops and quickly limits the infection. Some of the virions enter the sensory nerve in the area. Viral DNA persists in these nerve cells in a non –infections replicating form. This latent virus can from time to time become infections, be carried by the nerves to skin or mucous membranes and produce recurrent disease or stresses that can precipitate recurrences include menstruation, sunburn and any illness associated with fever (McGraw, 2007).

Virulent gene

Strains of HSV-1 display virulence patterns ranging from no disease to lethal encephalitis. It is likely that virulent differences are due to effects of multiple genes and the combination of allels carried by a given strain of virus. A significant qualitative trait locus (QTL) on chromosome 16 was found to associate with both percent mortality and HSK severity. This QTL maps close to a human QTL and the gene proposed to be associated with the frequency of recurrent herpetic labials (cold sores) also chr x epistatically associated with chr 16. Those host genetic locus modulate the severity of both herpetic disease in the nervous system and herpetic stromal keratitis (Thompson *et al.*, 2014).

Replication of Herpers simplex virus

The replication of HSV occurs primarily in epithelial cells during productive infection and in neurons during latency, highlighting its unique ability to establish lifelong persistence in the host.

1. Attachment and Entry

The replication cycle begins with the attachment of HSV to host cell surface receptors. Viral glycoproteins, including gB, gC, gD, gH, and gL, interact with host receptors such as nectin-1, herpesvirus entry mediator (HVEM), and heparan sulfate proteoglycans. This interaction mediates fusion of the viral envelope with the plasma membrane, allowing the capsid and tegument proteins to enter the cytoplasm (Iheukwumere *et al*., 2025a).

2. Transport to the Nucleus and Uncoating

Once in the cytoplasm, the capsid is transported along microtubules to the nuclear pore complex, where the viral DNA is released into the host nucleus. Tegument proteins such as VP16 play critical roles in initiating transcription of immediate-early (IE) genes by recruiting host transcriptional machinery (Iheukwumere *et al*., 2025b).

3. Gene Expression and Transcription

HSV gene expression occurs in a regulated cascade:

- **Immediate-early (α genes):** These genes encode regulatory proteins (e.g., ICP0, ICP4, ICP27) that activate subsequent gene expression (Iheukwumere *et al*., 2025c).
- **Early (β genes):** These genes encode proteins required for viral DNA replication, such as DNA polymerase and thymidine kinase (Iheukwumere *et al*., 2025d).
- **Late (γ genes):** These genes encode structural proteins for capsid formation, glycoproteins, and other virion components.

4. DNA Replication

HSV DNA replication begins at specific origins of replication (OriS and OriL) and involves a rolling-circle mechanism, producing long concatemeric DNA strands. The viral DNA polymerase complex, assisted by helicase-primase, is essential for efficient replication (Iheukwumere *et al*., 2025e).

5. Assembly and Maturation

Capsid assembly occurs in the nucleus, where viral DNA concatemers are cleaved into unit-length genomes and

packaged into preformed capsids. Tegument proteins associate with the nucleocapsid, and the virus acquires its envelope by budding through the nuclear membrane and Golgi-derived vesicles containing viral glycoproteins (Iheukwumere *et al*., 2025f).

6. Egress
Mature virions are transported via vesicles to the cell surface and released by exocytosis. This completes the lytic replication cycle. Importantly, HSV can also enter latency in sensory neurons, during which viral DNA persists as an episome with minimal gene expression. Reactivation under stress or immunosuppression results in renewed lytic replication and recurrent infections (Iheukwumere *et al*., 2025g).

Mode of Transmission
Herpes simplex virus is mainly transmitted by oral to oral contact to cause oral- herpes infection through contact with sores, saliva, during kissing and mouth to genital contact (oral sex). It is can be sexually transmitted during period of asymptomatic shedding. The greatest risk of transmission is when there are active sores. Mothers with genital HSV-1 infection rarely transmit it to the child during delivery. HSV-2 subclinical shedding may account for most of the transmission. It is primary a sexually transmitted infection. Both viruses are transmitted vertically during childbirth although the real risk is very low. Herpes simplex virus can affect areas of skin exposed to contact with someone that has herpetic whitlow which is a herpes infection on the fingers (Schiffer *et al.,* 2014).

Pathogenesis of Herpes Simplex Virus (HSV)
The pathogenesis of HSV involves a dynamic interplay between viral replication, latency, reactivation, and host immune responses. HSV primarily causes mucocutaneous lesions but can also lead to severe complications such as encephalitis, neonatal infections, and systemic disease in immunocompromised individuals.

1. Initial Infection and Local Replication
Pathogenesis begins when HSV gains entry through mucosal surfaces or skin abrasions. The virus attaches to epithelial cells via glycoproteins (gB, gC, gD, gH, and gL), which bind to receptors such as nectin-1 and heparan sulfate proteoglycans. Following fusion, viral replication occurs within epithelial cells, leading to localized cytopathic effects characterized by vesicle formation, ulceration, and inflammation (Iheukwumere *et al*., 2024a).

2. Neuroinvasion and Latency Establishment
A hallmark of HSV pathogenesis is its ability to establish lifelong latency. After initial replication in epithelial cells, HSV invades sensory nerve endings and is retrogradely transported to the neuronal cell bodies in sensory ganglia (e.g., trigeminal or sacral ganglia). Within neurons, the viral genome persists as a circular episome. Most viral genes are transcriptionally silent, except for latency-associated transcripts (LATs), which play a crucial role in maintaining latency and inhibiting apoptosis of infected neurons (Iheukwumere *et al*., 2024b).

3. Reactivation and Recurrence
Reactivation occurs under conditions of stress, immunosuppression, fever, or UV exposure. During reactivation, HSV resumes lytic replication in neurons, and viral particles are transported anterogradely to peripheral tissues, causing recurrent lesions at or near the site of primary infection. This explains the periodic cold sores (HSV-1) and genital herpes outbreaks (HSV-2) (Iheukwumere *et al*., 2024c).

4. Host Immune Response and Immune Evasion
The immune system plays a critical role in controlling HSV infection. Innate responses involve type I interferons, natural killer (NK) cells, and dendritic cells, which limit viral spread. Adaptive immunity, especially HSV-specific CD8+ T cells, is essential for long-term control. However, HSV employs multiple immune evasion strategies, such as blocking antigen presentation by downregulating MHC class I molecules, inhibiting complement activation, and producing proteins that counteract interferon responses (Iheukwumere *et al*., 2024d).

5. Severe and Complicated Infections
- **HSV encephalitis:** HSV-1 is the most common cause of sporadic viral encephalitis, leading to inflammation and necrosis in the temporal lobe.
- **Neonatal herpes:** Perinatal transmission of HSV-2 can cause disseminated disease in newborns, with high morbidity and mortality.
- **Immunocompromised hosts:** In patients with weakened immunity (e.g., HIV-positive or transplant recipients), HSV can cause extensive mucocutaneous lesions and systemic involvement (Iheukwumere *et al*., 2024e).

Clinical Manifestation of the Virus
- Blistering sores (in the mouth or on the genitals)
- Pain during urination (genital herpes)
- Itching and lesions

You may experience symptoms that are similar to the flu. These symptoms can include;
- Fever
- Swollen lymph nodes
- Headache
- Tiredness
- Loss of appetite

- Blisters

Herpes simplex virus can also spread to the eyes causing a condition called herpes keratitis. This can cause symptoms such as eye pain, discharge, and a gritty feeling in the eye.

Diseases of Herpes Simplex Virus

- Mucocutaneous HSV infection (most common): lesions appear on the skin or mucosa but mostly on the mouth or lips (perioral infection) (Fig. 3), genitals, conjunctiva and cornea. Clusters of small, tense vesicles appear on an erythematosus base. Clusters vary in size from 0.5 to 1.5 cm but may coalesce. Lesions on the nose, ears, fingers or genitals may be particularly painful. Vesicles typically persist for a few days, then rupture and dry, forming a thin, yellowish crust. Lesions usually heal completely, but recurrent lesions at the same site may cause atrophy and scaring (Ayoade and Staut, 2017).

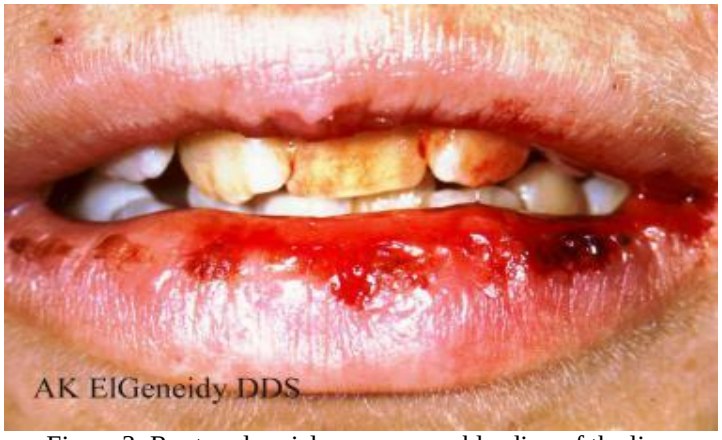

Figure 3: Ruptured vesicles appears as bleeding of the lips.
Source: Ayoade and Stuart (2017).

- Ocular Infection (herpes keratitis): Herpes simplex keratitis (HSV infection of the cornea epithelium) (Fig. 4). It causes pain, tearing, photophobia, and corneal ulcers that often have a branching pattern (Harrison, 2017).

Figure 4: Herpetic blepharitis
Source: Harrison (2017)

- CNS infection: herpes encephalitis occurs sporadically and may be severe (Fig. 5). Viral meningitides may result from HSV-2. It is usually self-limited and may involve lumbosacrae myeloraduculitis, which may cause urinary retention or obstruction. Signs include; fever, headache, altered mental status, often accompanied by seizures or focal neurologic deficits (Patei *et al.*, 2015).

Figure 5: Herpes encephalitis
Source: Patei *et al (*2015)

- Neonatal Herpes: Neonatal herpes simplex virus infection (Fig. 6) usually involves HSV-2 and its transmitted during delivery, through an infected maternal genital tract. A typical sign is vesicular eruption which may be accompanied by or progress to dissemination disease. More serious disease frequently follows within 7 to 10 days if left untreated (Caserta, 2015).

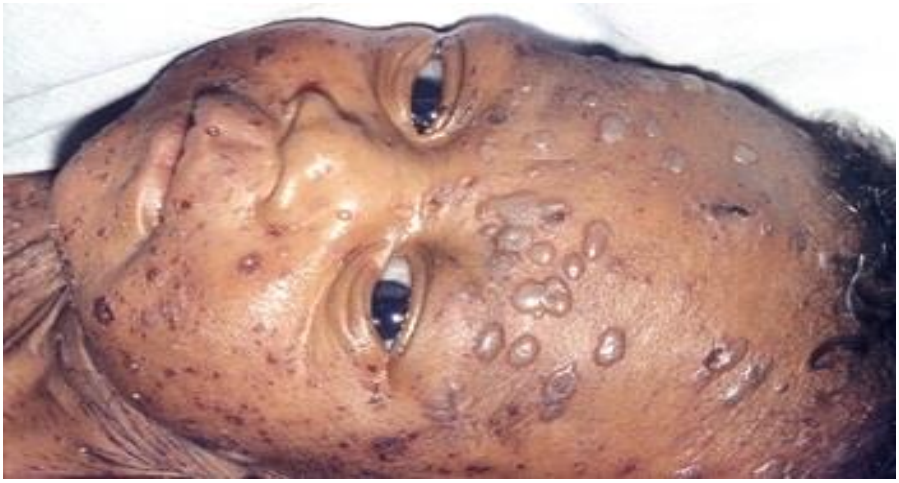

Figure 6: Neonatal herpes
Source: Caserta (2015)

Distribution of the Disease

- Age: 140 million people aged 15-49 years are infected with genital HSV-1 infection primarily in the Americas, Europe and western pacific.
- Life style: Fewer people in high-income countries are becoming infected with HSV-1 as children, likely due to better hygiene and living conditions, and are instead at risk of contracting it genetically through oral sex after they become sexually active. People with weak immune system (those with HIV) HSV-1 have more severe symptoms and more frequent recurrent. People who sexually promiscuous. People who experience malnutrition can get HSV-1.(Smith *et al.,* 1962).
- Most genital HSV-1 infections are estimated to occur in the Americas, Europe and Western Pacific, where HSV-1 continues to be acquired well into adulthood. In other regions, for example in Africa, most HSV-1 infections are acquired in childhood, before the age of sexual debut.

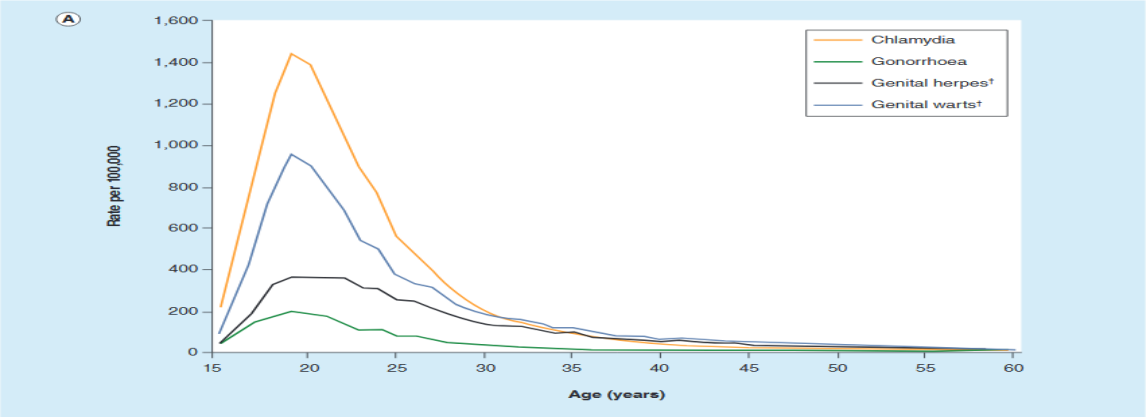

Figure 7: Distribution of the virus.
Source: Smith *et al.* (1962)

Physical Observations
The dev of herpes virus in human lung fibroblasts was studied and the production of sickly recognizable particles preceded infectious virus parphicle production by about 5-6h, suggesting the occurrence of a maturation process during formation (Smith,1963).

Aggregation of particles in clusters and chains where observed in many cases. One of the mechanisms for this aggregation was the connection of particles by DNA strands. These strands appeared to connect some particles in a way that suggests a structural continuity between their cores.

Filamentous structures of this kind have been seen in both tissue culture herpes virus preparations and have been associated with particles taken directly from vesicular lesions of the skin and mucous membranes. These strands often connect particles where there are several microns apart, thus making large networks of loosely aggregates virus particles (smith and melnick, 1929)

During the growth of the virus on tissue culture, there was development of methods; for preparing and staining, morphological study of the view in preparation, method for counting negatively stained herpes particles, the particulate forming unit (PFU), ratio determinants and growth curve data in which both particle counts and infectivity measurements. Al these are methods of studying the physical changes of herpes virus (Watson, 1962).

Immumological Test/ Kit
Serological tests for herpes simplex virus (HSV) that can accurately distinguish between HSV-1 and HSV-2 are now commercially available. These tests detect antibodies to HSV glycoproteins G-1 and G-2, which evoke a type-specific antibody response. Focus Technologies produces the HerpeSelect-1 and HerpeSelect-2 enzyme-linked immunosorbent assay tests and the HSV-1 and HSV-2 HerpeSelect1/2 Immunoblotting. Diagnology has marketed POCkit-HSV-2, a point-of-care test for HSV-2 that allows blood from a finger stick to be tested in a clinic. These tests can be used to confirm a genital herpes diagnosis, establish diagnosis of HSV infection in patients with atypical complaints, identify asymptomatic carriers, and identify persons at risk for acquiring HSV. Potential settings for use of these tests include sexually transmitted disease clinics, prenatal clinics, and clinics that care for patients with human immunodeficiency virus. Patient interest in HSV serological tests appears high (Iheukwumere *et al*., 2024f).

Isolation, identification and inoculation of the virus
- The virus can be isolated from fibroblast & epithelial cell lines from animals or humans.
- Cytopathic effects usually occur in only 2-3 days.
- Culture fluid is identical by neutralization test and immunofluorescence test.
- Typing of HSV isolates may be done using a monoclonal antibody or by restriction endonucleases analysis of viral DNA.
- Sodium-iodide density gradient centrifugation of lysates of BSC cells infected with herpes simplex virus type 2 was carried out in the presence of ethidium bromide. Two DNA bands could be visualized. The lower DNA band was further identified as herpes simplex virus type 2 DNA by its plaque-forming capacity and sedimentation behavior (Iheukwumere *et al*., 2025h).

Molecular detection
The technical and diagnostic impact of PCR for evaluation of HSV CNS disease has been evident in clinical practice. In the past, physicians were limited to assessment of clinical symptomatology of the patient, neurodiadiagnostic imaging information, and culture of

brain biopsy specimens for acute diagnosis of HSV CNS disease (Iheukwumere *et al*., 2025i). Generally, only those with typical and severe CNS infections with this virus were identified; most often, patients were comatose and denied the efficacy of effective antiviral treatment because of the invasive procedures associated with laboratory diagnosis in the early stages of this disease. In contrast, today clinicians can submit CSF specimens rather than a brain biopsy specimen for the detection of HSV DNA by PCR when CNS infection is initially suspected. By this practice, the spectrum of CNS disease caused by this virus has been rapidly expanded to recognition of mild forms and atypical forms of meningitis and encephalitis. This technology provides us the facility for investigating the pathogenesis of HSV CNS disease. Analogous to the findings of the β-chemokine receptor CCR-5 regarding the control of entry of HIV into cells, molecular studies of genes coding for IFN-γR and IL-12 receptor and the MBL and the α134.5 genes can now be expanded to include their role in neuroinvasion by HSV .Detection of HSV DNA in CSF is rapid, sensitive, and specific and is clearly the gold standard for the laboratory diagnosis of CNS disease caused by this virus (Iheukwumere *et al*., 2025j).

Treatment

The management of Herpes Simplex Virus (HSV) infections primarily focuses on reducing symptom severity, minimizing recurrence, and preventing transmission. Oral antiviral agents such as Acyclovir, Famciclovir, Penciclovir, and Valaciclovir are the mainstay of treatment (Robert, 2009). Suppressive therapy with Valaciclovir (500 mg once daily) has been shown to significantly decrease viral shedding and interrupt HSV transmission. These medications inhibit viral DNA synthesis, thereby limiting replication and promoting faster healing of lesions.

Prevention

There should be safer sex behavior, including the use of condoms, to prevent genital herpes transmission (Coreyl.*et al* 2004).

Personal Measures

- Avoid contact with the blisters or contact with the saliva of a person shedding the virus.
- Avoid kissing on the mouth
- Avoid sharing eating utensils with infected patient.
- Avoid sharing toothpaste brushes, bottles, pacifiers and toys that have been mouthed, can spread it.
- Infants and children should not be kissed on the mouth.
- One should be practicing good hand washing.

Vaccination

- Use of oral antiviral drugs like Acyclovir, Famiclovir, and Peniclovir and valaciclovir therapy(500mg, once daily) to interrupt transmition of HSV(Robert, 2009).
- The gD deletion virus as a vaccine,(the researchers grew the virus in a cell line that expresses the HSV-1 version of gD. The HSV-2 virus, with gD deleted from its genome, grabbed the available HSV-1 gD proteins from the cell. When introduced to a mouse, HSV-2 was able to use the HSV-1 gD to enter the mouse's cells. Once inside, HSV-2 replicated abundantly, but because it could not produce gD, future progeny were unable to infect new cells). Herold (2004) states that infected cells became "little factories for making viral proteins" that spurred the immune system to produce antibodies to HSV-2.

The next step for the researchers in producing a herpes vaccine for use in humans is demonstrating its efficacy and safety in an FDA-approved cell line. The researchers are also looking for an industry partner to help make large quantities of the vaccine for future clinical tests.

Conclusion

Effective management of HSV infections requires a combination of antiviral therapy, behavioral prevention, and ongoing vaccine research. While current antiviral drugs successfully reduce disease severity and transmission, the persistent nature of HSV underscores the need for continued efforts in vaccine development and public health awareness to achieve long-term control of the virus.

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