**A Broad Introductory to the Herpes Simplex Lineage and Epidemiology** Author: William L. Colton  
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**Key Words:**

Herpes Simplex Virus, HSV1, HSV2, Retrovirus, Ganglia

**Abstract**

**The herpes virus family is a well know and culturally significant clade of virological origin. HSV1 and HSV2 especially are both common and incurable diseases afflicting society. “Human herpes simplex virus infections: Epidemiology, pathogenesis, symptomatology, diagnosis, and management” is a broad spectrum article providing both past and recent information representative of the herpes simplex family to better educate readers on the related viruses. This article provides an exemplary array of portrayals to elaborate to the reader how, what, when, and why the simplex virus family should be better known. All known herpes simplex infections are incurable and are one of the most wide spread infections among adults with 60-90% of adults being infected (Fatahzadeh, 2007).). They tend to lay latent within the host with reoccurring outbreaks and can spread from the site of infection. The range of presentation with herpes infections can go from latent, meaning no visible or obvious symptoms, to a full blown infection and destruction of localized tissues. Infected patients can control their symptoms with antiretroviral drugs that prevent viral outbreaks, but these drugs never completely clear the body of its infection. As the medical society discovers more concerning the herpes simplex virus, we can further educate the public on the ailments it may cause and prevent further spread of infection.**

**Introduction and Background**

**The family of eight herpes simplex viruses consists of morphologically similar viral pathogens approximately 200nm in diameter (Fatahzadeh, 2007). They have double stranded DNA, much like our own, that is approximately 150 Kbp within the “head”, a protein capsid that stores their DNA (Fatahzadeh, 2007). HSV1 and HSV2 are considered less of a threat compared to the rest of the herpes simplex virus family due to observations made by researchers using a viral cytopathy experiment. Members of the herpes simplex virus family tend to be antigenically different from one another due to variations in their protein capsid coating. Herpes simplex viruses tend to infect epithelial cells during primary exposure, but travel through nervous to the localized ganglia of the host nervous system. These viruses persist in a dormant state in an infected host as a survival mechanism to evade the host immune cells. Reoccurrence or Outbreaks of the virus in an infected host is due to a reactivation of the dormant virus in infected cells. During an outbreak the virus will lyse the infected cell and travel from the ganglial locus through nerves into the mucosal regions where is can proceed to become an contagion. The mucosal site is where the small bumps, or lesions, tend to appear that are so readily associated with the herpes simplex virus. Asymptomatic shedding is a variation of reoccurrence where the infected host has the activated virus present in their mucosa but they present no form of clinical disease related to infection. The reactivation of the virus in an infected host can be triggered by many factors including stress and sexual exposure that induce viral replication and migration (Fatahzadeh, 2007).**

**The human demographical impact of the herpes simplex virus family is astounding, HSV1 being the most prevent of the family and is to have thought to be present in approximately 45% to 98% of the world population with 40% to 63% of individuals within the United States testing positive for the virus (Fatahzadeh, 2007). Socioeconomic status also shows a correlation with herpes simplex virus prevalence as lower income populations have a higher rate of infection when compared to higher income communities. Within the United States, in poorer socioeconomic populations the prevalence of HSV1 is 33% in children under the age of 5, while in wealthier communities the prevalence is 20% in children before the age of 5 test positive for HSV1 (Fatahzadeh, 2007). Herpes simplex virus is the leading cause of genital lesions globally (Fatahzadeh, 2007). The primary cause of infection with herpes simplex virus is through the exchange of contaminated bodily fluids across a mucosal membrane, usually through contact with an exposed broken lesion. HSV1 can also be transferred laterally in an infected patient from one region of the body to another, leading to a systematic infection of the host. Primary herpes simplex virus infections, where a patient is infected without any previous exposure to a herpes simplex virus or any other strain being present in their body, tend to be more taxing on the patient and more symptomatic as compared to non-primary infections, where the individual has been exposed to another member of the herpes simplex virus or has viral antibodies present in their serum. Non-primary and primary can be differentiated based on the presence of either high levels of IgM antibodies or IgG antibodies after exposure, high levels of IgM antibodies coinciding with a primary infection and higher levels of IgG antibodies coinciding with non-primary infections. Recovery from a primary infection tends to take 10 to 14 days after exposure for an immunocompetent individual and is non-life threatening (Fatahzadeh, 2007).**

**Discussion and Conclusion**

**The article presents a broad and informative approach with a focus on the most prevalent strains of the herpes simplex virus family, HSV1 and HSV2, and with this being a informative article it does fulfill the intended purpose. The article aptly describes symptoms in both immunocompetent and immunocompromised patients as well as localities where symptoms present themselves, such as the inside of the lip or surrounding genital area. HSV1 and HSV2 are commonly referred to as oral and genital herpes due to the locality they are most commonly present in, this article also addresses lateral transmission throughout the patient where infection can move to different parts of the body, which I applaud for its clarity to the reader, But as stated earlier these may be the most prevalent of the herpes simplex virus clade but they are also the most tame of the bunch. The other 6 members of the family are left in obscurity in this article with little to no reference to pathology, epidemiology, and diagnosis. This might be due to the fact that this articles main goal seems to be to inform the reader of what they are most likely to either experience or associate with, yet this leaves a disparity in information with the more lethal strains. The article also provides elaborate variations of imagery that show the common clinical symptoms of the infected in both immunocompetent and immunocompromised individuals. The article is also adept at refereeing to the many various forms of infection form orofacial herpes to herpes gladiatorum. This gives the reader a broad understanding beyond basic common knowledge of just how infectious this pathogen can be, but yet again most for the infections are either caused by HSV1 or HSV2. Therapy for herpes simplex virus infections is also discussed near the end of the article, as the herpes simplex infection has no known cure the best way of aiding the patient is through managing both the virus internally and the symptoms externally, which the article discusses in a professional manor. I also personally appreciate the attention to the neonatal implications associated with the herpes simplex virus as the disease can be passed from mother to child during natural child birth. This sort of awareness is definitely needed as the number of individuals infected skyrockets and the risk of death to neonatal that are infected is tremendous. This article is wonderfully informative and is fairly easy to read and interpret as compared to other published scientific articles. The background given also unveils the mode of transfer of the disease and makes aware the reader of how the herpes simplex virus can lie dormant and even be infectious without symptoms being present. As a summary their article is informative and practical for the reader but forgets about the other 6 remaining members of the herpes simplex family apart from a quick mention in the abstract.**

**References**

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