

HERPES B VIRUS

[Herpesvirus simiae, Simian B Disease]

SPECIES: Old World Macaques

AGENT: Double-stranded DNA Virus. Direct zoonosis. For each Herpesvirus there exists a host for which the virus is almost uniformly fatal and reservoir hosts in which the virus exists in subclinical or latent infection. a. H. suis latent in swine, fatal in cattle. b. H. simplex I latent in man, fatal in Aotus, Gibbon, Marmoset (especially). c. H. saimiri I latent in Saimiri sciureus, fatal in Marmosets, Aotus (also known as Herpes T, Herpes M, Herpes Tamarinus). d. H. saimiri II latent in Saimiri sciureus, fatal in Marmosets, Aotus (this is an oncogenic virus, causing Malignant Melanoma of Reticulum Cell Type). e. H. simiae latent in rhesus and other macaques, fatal in man (also known as Herpes B Virus). f. Liverpool Vervet Monkey Virus: reservoir host ???, fatal in Cercopithecus aethiops. The Herpesviruses are serologically distinct but do share some antigenic properties. Herpes B Virus has been shown to have some antigenic relationship to Herpesvirus simplex by serum neutralization tests. However, antibody from Herpesvirus simplex does not confer immunity to Herpes B Virus.

RESERVOIR AND INCIDENCE: **Incidence of infection of the reservoir host is normally high but you rarely see clinical disease.** Clinical disease is usually confined to the very young or to the immunologically compromised. On occasion lethal infection occurs in the reservoir host: in humans, generalized Herpes simplex is often fatal in infants; in swine, Herpes suis is fatal in piglets. **Natural infection does not result in elimination of the virus or in immunity but produces a latent infection or carrier, which always makes vaccination of reservoirs impractical.** Herpesvirus B was first described in 1934 by Sabin/Wright. The virus was recovered from a laboratory worker who had been bitten 18 days previously by an apparently healthy rhesus. Since 1934, there have been 24 reported cases, all fatal, except four. Of the four, only one is reasonably free of severe neurological deficits. (Patients have remained in coma for as long as 40 months prior to succumbing to the disease.) Incubation is considered to be 10-20 days from exposure to the virus; however, in the Pensacola cases, clinical disease occurred within 4-5 days of exposure. Contact with macaques does not constitute exposure. The susceptibility of man to clinical B infection is low although mortality is high.

TRANSMISSION: direct contact, including sexual transmission, aerosols, and fomites.

THE DISEASE IN NHP: **The reservoir hosts for Herpesvirus simiae are monkeys of the genus Macaca.** Monkeys incriminated include M. mulatta (rhesus), M. fascicularis (cyno), M. fuscata (Japanese macaque), and M. arctoides (Stump-tail macaque). B virus may produce mild cold-sore type lesions, primarily at mucocutaneous junctions, mucous membranes, and tongue. But, it has been demonstrated that infection is not confined to the mouth but can also be found in the genitalia. These lesions are similar to those caused by Herpesvirus simplex I in man. Two factors have been associated with antibody increases with age; 80-100% of imported adult rhesus may have antibody compared to

20% imported juveniles. The second factor was type of caging. Animals housed together had significantly higher titers than individually-caged animals. Clinical disease may develop at the time of primary infection but it is not known if lesions invariably follow infection. However, **periodic shedding of the virus may occur without the presence of visible lesions. Once a monkey is infected with B Virus, it should be considered infective for life.**

THE DISEASE IN MAN: Human infection is characterized by encephalitis with diplopia; nystagmus, patch paresthesia of head, neck and upper extremities. Acute abdominal pain, fever and diarrhea have also been observed prior to neurologic symptoms. Patients may also have a vesicular rash and/or keratoconjunctivitis. The histopathological changes resemble those of fatal, systemic Herpesvirus simplex in infants: encephalitis, myelitis and foci of necrosis in liver, spleen, lymph nodes and adrenals. The case fatality rate is 70%.

DIAGNOSIS: ELISA and viral isolation.

TREATMENT: acyclovir for life

PREVENTION/CONTROL: Control includes personal hygiene, protective clothing and common sense in handling monkeys. The virus is susceptible to oxidizing agents, soap, and water. Guidelines for prevention and treatment have been developed. Immediately available bite kits are critical to initial treatment for potential exposure. The reader is referred to the reference by Holmes, GP, et al for further details. Emphasis can not be too strong concerning the use of protective clothing when entering a room with macaques. A perfectly healthy monkey may be lethal to you.

BIOSAFETY LEVEL: Experimental at BL-3

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