

Introduction

Description

Selectin Ligands and Related Structures on the Surface of the Infected Cell.

Human herpesviruses are usually acquired early in life and are widely distributed in the population. A common feature of all human herpesviruses is that they persist in the host after the primary infection. Thus, the host immune system resolves the acute stage of the infection but these viruses have evolved means to remain in a state of latency in some cells from which they occasionally reactivate into a state of replication. A functional immune system will clear these episodes and the clinical manifestations are therefore usually mild or absent. On the other hand, when the immune system is dysfunctional the herpesviruses pose a serious threat. Especially cytomegalovirus (CMV) and Epstein-Barr virus (EBV) are associated with severe infections in transplant patients and other immunosuppressed patients, where infiltration of virusinfected leukocytes into organ tissue can give rise to pneumonia, hepatitis and renal failure.

The mechanism behind organ colonization of herpesvirus-infected leukocytes is not clear. However, the normal pathway for leukocyte transmigration over the endothelial wall is well characterized and involves interaction between carbohydrate binding proteins, selectins, and selectin ligands, including the Lewis antigen sialyl Lewis X (sLeX). The selectin ligands are therefore potential targets in viral pathogenesis and we have previously demonstrated that several herpesviruses can in fact activate the cellular pathway for synthesis of sLeX and related structures.