

Biology Colloquium

How to Enter B and Epithelial Cells: Lessons from Epstein-Barr Virus

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Epstein-Barr virus (EBV) causes infectious mononucleosis in adolescents and is known to play an etiological role in human malignancies such as endemic Burkitt's lymphoma and nasopharyngeal carcinoma. EBV is also a factor in a variety of other human malignancies including some T-cell lymphomas, Hodgkin's Disease, and gastric carcinoma. The pathologies associated with EBV suggest a limited tissue tropism for EBV in vivo – being primarily for lymphocytes and epithelial cells. In vitro, the cells that are most susceptible to EBV infection and permissive for viral replication are of B cell origin. The major viral envelope glycoprotein 350 (gp350) binds to the complement receptor type two (CD21) that is abundantly expressed on B cells. Fusion of the virion membrane with the cell membrane minimally requires a complex of viral proteins that includes gB, gH/gL, and gp42. gp42 has been specifically found to bind to human leukocyte antigen (HLA) class II and this interaction is required for EBV entry into B lymphocytes. To date, little is known about the mechanism that EBV uses to bind and penetrate B cells and epithelial cells. Using both structural and functional studies, the mechanism of EBV into B cells and epithelial cells is binding investigated with a focus on key glycoproteins known to be important for B cell fusion (gp42, gB, and gH/gL) and epithelial fusion (gB and gH/gL).