

Poster presentation

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Suppression of galectin-3 in KSHV infected DMVEC cells and Kaposi's sarcoma: implications for tumorigenesis

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The Galectins are a family of proteins that share an affinity for Beta-galactoside containing glycoconjugates. Galectin-3 has been implicated in tumor progression and metastasis. It is also known that in prostate, ovarian and breast cancer, down regulation of Galectin-3 is associated with malignancy. Kaposi's sarcoma (KS) is characterized as an angioproliferative tumor of vascular endothelial cells and produces rare B cell lymphoproliferative diseases in the form of primary effusion lymphomas (PEL) and some forms of Multicentric Castleman's Disease (MDC). Kaposi's Sarcoma-Associated Herpesvirus also known as KSHV or Human Herpesvirus type 8 (HHV8) is the etiological agent of KS. We have observed suppression of galectin-3 expression in Kaposi's sarcoma (KS). Here we demonstrate that galectin-3 protein expression is down-regulated 20-fold in KSHV infected dermal microvascular endothelial cells (DMVEC) cells. We show loss of Galectin-3 staining by dual labeled immunofluorescence in latency associated nuclear antigen (LANA) positive spindle cells. We also find reduced levels of galectin-3 expression in LANA positive spindle cell regions in archival KS tissue. There is also transcriptional suppression of Galectin-3 message in KSHV infected DMVEC cells compared to mock infected controls. We demonstrate that KSHV vFLIP is the likely viral gene that targets galectin-3 downregulation in HeLa cells. In pleural effusion lymphoma cell lines (PEL), we observe different levels of galectin-3 expression associated with varying levels of KSHV replication. The search for novel host cell factors that may contribute to

the overall pathogenesis of KS is essential for early detection of KS and development of innovative therapies for treatment.