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Bovine leukemia virus gp30 transmembrane (TM) protein is not tyrosine phosphorylated:
Examining potential interactions with host tyrosine-mediated signaling

Abstract: Leukemia of B lymphocyte origin is a common cancer of humans, affecting both young and old. Current treatment options rely primarily on chemotherapy, which hopefully kills neoplastic cells prior to normal ones. If we could better understand the normal growth and development in B cells, investigators could design treatments to stop or prevent growth of the abnormal cells which leads to neoplasia. Some viruses have evolved ways to alter cellular signal pathways. By studying these viral alterations, we may be able to identify crucially important pathways of B cells. Bovine leukemia virus (BLV) is a retrovirus which causes a preneoplastic, polyclonal expansion of B cells, and may lead to leukemia or lymphoma. The cytoplasmic portion of BLV transmembrane protein gp30 (TM) has homology to B cell signaling proteins, including two immunoreceptor tyrosine-based activation motifs (ITAMs), which are homologous to B cell receptor (BCR) ITAMs. We hypothesized in the BLV TM, like in the BCR, ITAM tyrosines are phosphorylated and interact with host cell tyrosine kinase signal pathways, altering the normal signals and resulting in the preneoplastic expansion. Phosphorylation was tested in four cell populations or lines including cultured peripheral blood mononuclear cells from infected cows, BLV-expressing fetal lamb kidney and bat lung cell lines, and DT40 B cells transfected with a fusion of mouse extracellular CD8 α and cytoplasmic TM. No phosphorylation of TM in any cell type or under any conditions of stimulation was detected either by immunoblotting or by radiolabeling. Although BLV TM interacts with host cell signaling proteins, phosphotyrosine-mediated signal transduction via host nonreceptor protein tyrosine kinases or phosphatases is unlikely.