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BIFL1 is a G protein-coupled receptor encoded by human EBV. It signals constitutively through G_alpha_i and is an orphan receptor known to down regulate MHCI expression. BILF1 also engage in dimerization with several chemokine receptors and it induced the activity of NF-kappa beta and inhibits the phosphorylation of PKR. My research on BILF1 has contributed to increase the knowledge of BILF1 signaling and has identified BILF1 as a potential oncogene. During the proteomics study NFAT was identified as being up-regulated in BILF1 expressing cells eventually revealing an increased NFAT activity induced by BILF1 G_alpha_i signaling. Statmin was also identified as being up-regulated in both BILF1 and ORF74 expressing cells. Following verification experiemnts, ORF74 and US28 was shown to increase the phosphorylation on serine 38 in Statmin; this was however not verified for BILF1. During the investigation of BILF1's oncogenic potential a clear correlation between G_alpha_i signaling and (a) focus formation, (b) NFAT activity, (c) VEGF secretion and (d) tumor onset an foramation was revealed. Interestingly also a silenced receptor, EAT-BILF1 induced tumors in vivo although to a lesser extent than the fully active receptor. This suggests the existence of an alternative signaling pathway activated by BILF1. The rhesus EBV BILF1 also signals constitutive via G_alpha_i and induced foci formation of 3T3 cells but it was not able to induce tumor in mice or induce secretion of VEGF to the same extent as BILF1. In this thesis BILF1 is revealed as a potential oncogene, inducing transformation of NIH 3T3 cells and tumors in vivo, and this ability is clearly correlated to the constitutive activity through G_alpha_i.

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