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The role of KSHV vGPCR in Kaposi's Sarcomagenesis

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My laboratory is focused on the molecular mechanisms involved in the development of Kaposi's Sarcoma, a multifocal vascular neoplasm invariably associated with infection with the KS-associated human herpesvirus (KSHV/HHV8). In particular, we are interested in the role of the KSHV G protein-coupled receptor (vGPCR) in KS initiation, progression and maintenance. In this effort, we have identified the Akt/TSC/mTOR pathway as an essential intracellular route whereby vGPCR triggers KS. Our results have suggested the valuable use of mTOR inhibitors (rapalogs) as a mechanism-based therapeutic approach for KS patients. Indeed, rapamycin has already been successfully used in the management of patients with iatrogenic and classic KS, and clinical trials are already ongoing for AIDS-related KS. We have further identified novel PI3K/mTOR inhibitors that could serve as alternative therapeutic drugs for KS treatment, and that may be especially effective in the most aggressive manifestations of this tumor. More recently, our lab has focused on identifying novel angiogenic and inflammatory molecules (e.g. cytokines, chemokines and growth factors) that contribute to vGPCR paracrine neoplasia. Of interest, we have identified a novel angiogenic factor, Angiopoietin-like 4, that promote both angiogenesis and vascular permeability in KS, exposing a novel target for the development of mechanism-based treatments for KS. As KS serves as a model of tumor-induced angiogenesis, this work may have a broad impact on the treatment of numerous other solid tumors.