Retrovirology

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Oral presentation

The HHV-8 chemokine receptor vGPCR triggers autonomous proliferation of endothelial cells

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We have used a novel conditional transgenic system to study the mechanisms of angioproliferation induced by vGPCR, the constitutively active chemokine receptor encoded by HHV-8 (also known as KSHV). Using this system we were able to control temporal expression of vGPCR and to monitor its expression in situ via the use of the surrogate marker LacZ. Upon treatment with doxycycline (DOX), cells expressing vGPCR and LacZ (vGPCR/LacZ+ cells) progressively accumulated in areas where angioproliferation was observed. Sorted vGPCR/LacZ+ cells from angiogenic lesions expressed markers characteristic of endothelial progenitor cells, produced angiogenic factors, and proliferated in vitro. Prolonged treatment of the transgenic mice with DOX led to development of tumors in the skin of ears, tail, nose and paws. vGPCR/LacZ+ cells were frequent in early lesions but scarce within these tumors. Finally, transfer of vGPCR/LacZ+ cells into RAG1-/- mice treated with DOX led to angioproliferation and with time to development of tumors containing both vGPCR/LacZ+ and vGPCR/LacZ- cells. Taken together these results indicate that vGPCR triggers angioproliferation directly and suggest a novel role for this molecule in the pathogenesis of KS.