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Kaposi's sarcoma-associated herpes virus disrupts adherens junctions and increases endothelial permeability by inducing degradation of VE-cadherin

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Kaposi's sarcoma (KS) is a vascular tumor of proliferative endothelial cells caused by infection of Kaposi's sarcoma-associated herpesvirus (KSHV). Aberrant vascular permeability is a hallmark of KS manifested as multifocal edematous skin and visceral lesions with dysregulated angiogenesis and vast inflammatory infiltrations. In this study, we showed that KSHV infection increased the permeability of confluent endothelial monolayers to serum albumin, blood-derived cells, KSHV-infected cells and KSHV virions. KSHV-induced permeability was associated with the disruption of adherens junctions and degradation of vascular endothelial (VE)-cadherin protein. Ultraviolet irradiation that inactivated KSHV virions and cycloheximide that blocked *de novo* protein synthesis failed to reverse KSHV-induced disruption of adherens junctions. However, soluble heparin that blocked KSHV entry into cells completely inhibited KSHV-induced permeability. Furthermore, KSHV-induced degradation of VE-cadherin was dose-dependent on the internalized virus particles. Together, these results indicate that KSHV infection induces vascular permeability by inducing VE-cadherin degradation during virus entry into cells. KSHV-induced aberrant vascular permeability could facilitate virus spread, promote inflammation and angiogenesis, and contribute to the pathogenesis of KSHV-induced malignancies.