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D6 is an atypical chemokine receptor acting as a decoy and scavenger for inflammatory CC chemokines expressed in lymphatic endothelial cells. Here, we report that D6 is expressed in Kaposi sarcoma (KS), a tumor ontogenetically related to the lymphatic endothelium. Both in human tumors and in an experimental model, D6 expression levels were inversely correlated with tumor aggressiveness and increased infiltration of proangiogenic macrophages. Inhibition of monocyte recruitment reduced the growth of tumors, while adoptive transfer of wild-type, but not CCR2(-/-) macrophages, increased the growth rate of D6-competent neoplasms. In the KS model with the B-Raf V600E-activating mutation, inhibition of B-Raf or the downstream ERK pathway induced D6 expression; in progressing human KS tumors, the activation of ERK correlates with reduced levels of D6 expression. These results indicate that activation of the K-Ras-B-Raf-ERK pathway during KS progression downregulates D6 expression, which unleashes chemokine-mediated macrophage recruitment and their acquisition of an M2-like phenotype supporting angiogenesis and tumor growth. Combined targeting of CCR2 and the ERK pathway should be considered as a therapeutic option for patients with KS.