

Responses of vascular endothelial cells to angiogenic signaling are important for tumor cell survival. ^[1]

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Abstract

Neoplastic cells overexpress several angiogenic cytokines, which stimulate neovascularization. Whether the responses of the host endothelial cells to these signaling molecules affect tumor cells during early tumorigenesis has not been investigated. We investigated pre-angiogenic tumor cell survival and angiogenesis initiation by two murine tumor lines (4T1 mammary carcinoma and B16 melanoma), which constitutively expressed GFP, in dorsal skin-fold window chambers of mice treated with extracellular domain of Tie-2 (ExTek) or bFGF. ExTek reduced tumor cell survival, retarded tumor growth, and inhibited angiogenesis onset compared with controls. bFGF increased tumor cell survival and promoted earlier angiogenesis and tumor growth. Neither bFGF nor ExTek affected cell proliferation in vitro. RT-PCR showed mRNA expression of bFGF receptor 2 (FGFR2) IIIb, which does not bind bFGF efficiently, by 4T1 cells and B16 cells express FGFR1 but not FGFR2. B16 cells expressed angiopoietin (Ang) 2, but neither cell line expresses Ang1. Both tumor lines express VEGF. These findings suggested that effects of bFGF and ExTek on tumor cell survival and angiogenesis were not due to direct action but were instead a result of paracrine factors secreted by endothelial cells. These subsequent signals from endothelial cells promote early survival and proliferation of disseminated tumor cells before onset of angiogenesis.

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