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cells and the disruption of the surrounding microvasculature. Although there is considerable evidence to suggest the role of inflammatory and immune cells in the modulation of renal tumor growth, cytokines are also essential mediators in renal tumor biology. Notably, it has recently been shown that the stromal cell-derived factor-1-CXC chemokine receptor 4/fractalkine, which leads to the recruitment of CXCL8-producing tumor infiltrating dendritic cells to the tumor site, is involved in tumor progression. In turn, cytokines can be secreted by the tumor cells themselves. High levels of fibroblast growth factor, platelet-derived growth factor and vascular endothelial growth factor, as well as their specific receptors, have been detected in renal carcinoma. In addition, angiogenesis inhibitors have been evaluated in the management of renal cell carcinoma. Sustained antitumor activities have been observed in patients with metastatic renal cell carcinoma treated with the monoclonal antibody bevacizumab. Efficacy has been documented with a combination of interleukin-10 and bevacizumab. The exact mechanisms whereby these cytokines exert their antitumor activities remain to be clarified. In addition, the possibility that these cytokines act as angiogenic factors has been demonstrated. All these data are consistent with the concept that the cytokine network is a key event in renal carcinoma.

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