Cross-talk between tumor microenvironment and the immune system

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The tumor microenvironment affects engagement by interfering with signaling pathways required for vascular construction. The absence of normal vasculature causes a physical constraint on the microenvironment. Tumors recruit endothelial cells, fibroblasts, inflammatory cells, and pericytes with components of the ECM contribute to the microenvironment composition. Stromal cells generate both tumor enhancing and suppressing signals. MMPs and TIMPs are known to shift the balance from pro-angiogenic to an inhibitory phenotype.

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TIMPs are endogenous inhibitors of MMP's and tumor growth through a paracrine manner. TGF-β causes activation of fibroblasts while PDGF recruits fibroblasts and expression of SDF-1. Tumor cells also express CXCR4, the receptor for SDF-1 and stromal SDF-1 can stimulate tumor growth factors and cytokines that produce oncogenic signals. Activated fibroblasts promote angiogenesis via proteoglycan.

Activated lymphocytes secrete cytokines that orchestrate the vascular network. The absence of normal vasculature causes a physical constraint on the microenvironment. The tumor microenvironment affects angiogenesis by interfering with signaling pathways required for vascular construction. The absence of normal vasculature causes a physical constraint on the microenvironment. Tumors recruit endothelial cells, fibroblasts, inflammatory cells, and pericytes with components of the ECM contribute to the microenvironment composition. Stromal cells generate both tumor enhancing and suppressing signals. MMPs and TIMPs are known to shift the balance from pro-angiogenic to an inhibitory phenotype.

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