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A novel cellular crosstalk promoting tumor progression and metastasis

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Treatment of cancer patients with advanced or recurrent disease presents a considerable challenge. The reasons behind disease progression and spread are not fully understood, but their elucidation will lead to new targets for cancer therapy. Our goals are to define what drives metastatic progression and to develop novel therapies that would eliminate the mortality associated with metastatic disease. Emerging data support a key role of the tumor microenvironment (TME) in cancer progression, development of metastases, and response to therapy. Research indicates that tumor cells are involved in a complex and dynamic crosstalk with various cellular components of TME. Our team investigates the interaction of tumor cells with tumor-associated fibroblasts in breast carcinoma models. We have recently reported that the interaction of tumor cells with fibroblasts alters production and signaling by pro-inflammatory cytokines, e.g. TNF and IL1, and anti-inflammatory TGF-β cytokines. Our findings revealed that these cytokines are engaged in a dynamic crosstalk exerting synergistic and cytokine-specific responses in paracrine and autocrine fashions. The overall force of these complex interactions promotes formation of the tumor vasculature through a process of angiogenesis, changes the fibrotic material, and alters immune cell populations within the TME. The lecture will present updates on the molecular details underlying the biological effects of these cellular and cytokine interactions. We will also discuss the translational implications of our research and potential new venues for therapeutic intervention into cancer progression.

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