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The extracellular matrix molecule tenascin-C promotes metastasis by multiple mechanisms

Gertraud Orend
Université de Strasbourg, France

Today, an orchestrating role of the tumor microenvironment is widely accepted where especially stromal cells and soluble factors are recognized as active players. Yet, the extracellular matrix, although highly abundant, is often considered as passive bystander. A better understanding of the functions of the extracellular matrix in cancer is largely hampered by the lack of relevant models. This applies also to the extracellular matrix molecule tenascin-C, which is a marker of the cancer specific tumor microenvironment. We used a comprehensive approach comprising novel immune competent mouse models (with engineered tenascin-C levels) and demonstrated that tenascin-C plays multiple roles in cancer. Tenascin-C is dangerous as soon as it is expressed out of control. Through assembly into "Tumor Matrix Tracks", tenascin-C impacts tumor and stromal cells including immune cells thereby regulating tumor immunity. Tenascin-C also enhances formation of new but leaky blood vessels. Direct interactions with tenascin-C causes endothelial cell rounding and death or survival upon induction of an insulating pericellular fibronectin coat. Tenascin-C enhances the angiogenic switch and upregulates a proangiogenic secretome. Finally, tenascin-C is an important component of metastatic vascular invasions by promoting endothelialization and cellular plasticity thereby increasing lung metastasis.

e: gertraud.orend@inserm.fr