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Titel des Beitrags: Inflammatory chemokines and metastasis--tracing the accessory.

Abstract: The tumor microenvironment consists of stromal cells and leukocytes that contribute to cancer progression. Cross-talk between tumor cells and their microenvironment is facilitated by a variety of soluble factors, including growth factors and cytokines such as chemokines. Due to a wide expression of chemokine receptors on cells in the tumor microenvironment, including tumor cells, chemokines affect various processes such as leukocyte recruitment, angiogenesis, tumor cell survival, tumor cell adhesion, proliferation, vascular permeability, immune suppression, invasion and metastasis. Inflammatory chemokines are instrumental players in cancer-related inflammation and significantly contribute to numerous steps during metastasis. Recruitment of myeloid-derived cells to metastatic sites is mainly mediated by the inflammatory chemokines CCL2 and CCL5. Tumor cell homing and extravasation from the circulation to distant organs are also regulated by inflammatory chemokines. Recent experimental evidence demonstrated that besides leukocyte recruitment, tumor cell-derived CCL2 directly activated endothelial cells and together with monocytes facilitated tumor cell extravasation, in a CCL2- and CCL5-dependent manner. Furthermore, CX3CL1 expression in the bone facilitated metastasis of CX3CR1 expressing tumor cells to this site. Current findings in preclinical models strongly suggest that inflammatory chemokines have an important role during metastasis and
targeting of the chemokine axis might have a therapeutic potential.