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SRGN interact with TGFβ2 and endow triple-negative breast cancer with higher metastasis properties

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Patients with triple-negative breast cancers (TNBC) are at high risk for recurrent or metastatic disease despite standard treatment, underscoring the need for novel therapeutic targets and strategies. Several studies have been reported that the proteoglycan serglycin (SRGN) play an important role in tumor metastasis. However, the function of SRGN in breast cancer is not yet clear. Here, we report the SRGN as a functionally significant regulator of metastasis in TNBC. Our results show that SRGN expression levels were significantly higher in TNBC patient tissues and cell lines than those in non-TNBC. The shRNA-mediated inhibition of SRGN expression blocked serglycin secretion and the invasive motility of TNBC cell line (MDA-MB-231), reducing metastatic capacity in vivo by suppresses transforming growth factor-β2 (TGFβ2) secretion and epithelial-to-mesenchymal transition. Conversely, SRGN overexpression in poorly metastatic non-TNBC cell line (MCF-7) increased their motile behavior and metastatic capacity in vivo by promotes TGFβ2 secretion and epithelial-to-mesenchymal transition. Interestingly, TGFβ2 can also increase SRGN mRNA expression by activating Smad3 to target SRGN relative promoter domain in TNBC cells. Our findings establish that SRGN interact with TGFβ2 regulates TNBC metastasis via autocrine and paracrine routes, and that it serves as a potential drug targets to prevent metastatic disease of breast cancer.

Biography

Zhimin Zhang is the Director of cancer research Institute of Guangzhou Medical University. He has published more than 50 papers in reputed journals and has been serving as an Editorial Board Member of repute.

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