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VEGF-B promotes cancer metastasis through a VEGF-A– independent mechanism and serves as a marker of poor prognosis for cancer patients

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The biological functions of VEGF-B in cancer progression remain poorly understood. Here, we report that VEGF-B promotes cancer metastasis through the remodeling of tumor microvasculature. Knockdown of VEGF-B in tumors resulted in increased perivascular cell coverage and impaired pulmonary metastasis of human melanomas. In contrast, the gain of VEGF-B function in tumors led to pseudonormalized tumor vasculatures that were highly leaky and poorly perfused. Tumors expressing high levels of VEGF-B were more metastatic, although primary tumor growth was largely impaired. Similarly, VEGF-B in a VEGF-A–null tumor resulted in attenuated primary tumor growth but substantial pulmonary metastases. VEGF-B also led to highly metastatic phenotypes in *Vegfr1 tk−/−* mice and mice treated with anti–VEGF-A. These data indicate that VEGF-B promotes cancer metastasis through a VEGF-A–independent mechanism. High expression levels of VEGF-B in two large-cohort studies of human patients with lung squamous cell carcinoma and melanoma correlated with poor survival. Taken together, our findings demonstrate that VEGF-B is a vascular remodeling factor promoting cancer metastasis and that targeting VEGF-B may be an important therapeutic approach for cancer metastasis.

Biography:

Patrik Andersson received his B.Sc. at the age of 21 within biomedical sciences and is now pursuing his Ph.D. studies on the topic of tumor lymph- and heme angiogenesis in promoting metastasis. He is receiving his training at the Karolinska Institutet, Sweden under the supervision of Professor Yihai Cao. He was invited as a speaker at the prestigious Gordon Conference in angiogenesis (2013) and at the International Cancer and Prevention methods conference (2015). He has obtained several research and travel grants, and has co-authored ten peer-reviewed articles either published or under current revision.