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O-sulfated bacterial polysaccharides with low anticoagulant activity inhibit metastasis.

Borgenström M[1], Wärri A, Hiilesvuo K, Käkönen R, Käkönen S, Nissinen L, Pihlavisto M, Marjamäki A, Vlodavsky I, Naggi A, Torri G, Casu B, Veromaa T, Salmivirta M, Elenius K.

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Abstract

Heparin-like **polysaccharides** possess the capacity to **inhibit** cancer cell proliferation, angiogenesis, heparanase-mediated cancer cell invasion, and cancer cell adhesion to vascular endothelia via adhesion receptors, such as selectins. The clinical applicability of the antitumor effect of such **polysaccharides**, however, is compromised by their **anticoagulant activity**. We have compared the potential of chemically **O-sulfated** and N,**O-sulfated bacterial** polysaccharide (capsular polysaccharide from E. COLI K5 [K5PS]) species to **inhibit metastasis** of mouse B16-BL6 melanoma cells and human MDA-MB-231 breast cancer cells in two in vivo models. We demonstrate that in both settings, **O-sulfated** K5PS was a potent inhibitor of **metastasis**. Reducing the molecular weight of the polysaccharide, however, resulted in lower antimetastatic capacity. Furthermore, we show that **O-sulfated** K5PS efficiently inhibited the invasion of B16-BL6 cells through Matrigel and also inhibited the in vitro **activity** of heparanase. Moreover, treatment with **O-sulfated** K5PS lowered the ability of B16-BL6 cells to adhere to endothelial cells, intercellular adhesion molecule-1, and P-selectin, but not to E-selectin. Importantly, **O-sulfated** K5PSs were largely devoid of **anticoagulant activity**. These findings indicate that **O-sulfated** K5PS polysaccharide should be considered as a potential antimetastatic agent.

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