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Endogenous tenascin-C enhances glioblastoma invasion with reactive change of surrounding brain tissue.

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Tenascin-C is an extracellular matrix glycoprotein implicated in embryogenesis, wound healing and tumor progression. We previously revealed that tenascin-C expression is correlated with the prognosis of patients with glioblastoma. However, the exact role of endogenous tenascin-C in regulation of glioblastoma proliferation and invasion remains to be established. We show here that endogenous tenascin-C facilitates glioblastoma invasion, followed by reactive change of the surrounding brain tissue. Although shRNA-mediated knockdown of endogenous tenascin-C does not affect proliferation of glioblastoma cells, it abolishes cell migration on a two-dimensional substrate and tumor invasion with brain tissue changes in a xenograft model. The tyrosine phosphorylation of focal adhesion kinase, a cytoplasmic tyrosine kinase that associates with integrins, was decreased in tenascin-C-knockdown cells. In the analysis of clinical samples, tenascin-C expression correlates with the volume of peritumoral reactive change detected by magnetic resonance imaging. Interestingly, glioblastoma cells with high tenascin-C expression infiltrate brain tissue in an autocrine manner. Our results suggest that endogenous tenascin-C contributes the invasive nature of glioblastoma and the compositional change of brain tissue, which renders tenascin-C as a prime candidate for anti-invasion therapy for glioblastoma. (Cancer Sci 2009).

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