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Glioma-produced extracellular matrix influences brain tumor tropism of human neural stem cells.

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Abstract

A major obstacle in the treatment of gliomas is the invasive capacity of the tumor cells. Previous studies have demonstrated the capability of neural stem cells (NSCs) to target these disseminated tumor cells and to serve as therapeutic delivery vehicles. Less is known about the factors involved in brain tumor tropism of NSCs and their interactions within the tumor environment. As gliomas progress and invade, an extensive modulation of the extracellular matrix (ECM) occurs. Tumor-ECM derived from six glioblastoma cell lines, ECM produced by normal human astrocytes and purified ECM compounds known to be upregulated in the glioma environment were analyzed for their effects on NSCs motility in vitro. We found that tumor-produced ECM was highly permissive for NSC migration. Laminin was the most permissive substrate for human NSC migration, and tenascin-C the strongest inducer of a directed human NSC migration (haptotaxis). A positive correlation between the degree of adhesion and migration of NSCs on different ECM compounds exists, as for glioma cells. Our in vitro data suggest that the ECM of malignant gliomas is a modulator of NSC migration. ECM proteins preferentially expressed in areas of glioma cell invasion may provide a permissive environment for NSC tropism to disseminated tumor cells.

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