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REVIEW

Autocrine Factors That Sustain Glioma Invasion and Paracrine Biology in the Brain Microenvironment

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Invasion is a defining hallmark of glioblastoma multiforme, just as metastasis characterizes other high-grade tumors. Glial tumors invariably recur due to the regrowth of invasive cells, which are unaffected by standard treatment modalities. Drivers of glioma invasion include autocrine signals propagated by secreted factors that signal through receptors on the tumor. These secreted factors are able to diffuse through the peritumoral stroma, thereby influencing parenchymal cells that surround the tumor mass. Here we describe various autocrine motility factors that are expressed by invasive glioma cells and explore the effects that they may have on normal cells present in the path of invasion. Conversely, normal brain parenchymal cells secrete ligands that can stimulate receptors on invasive glioma cells and potentially facilitate glioma invasion or create a permissive microenvironment for malignant progression. Parallel observations have been made for solid tumors of epithelial origin, in which parenchymal and stromal cells either support or suppress tumor invasion. Most autocrine and paracrine interactions involved in glioma invasion constitute known signaling systems in stages of central nervous system development that involve the migration of precursor cells that populate the developing brain. Key paracrine interactions between glioma cells and the brain microenvironment can influence glioma pathobiology and therefore contribute to its poor prognosis. Current therapies

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for glioma that could have an impact on paracrine communication between tumors and normal cells are discussed. We suggest that cells in the normal brain parenchyma be considered as potential targets for adjuvant therapies to control glioma growth because such cells are less likely to develop resistance than glioma cells.

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