



The Duality of Epidermal Growth Factor Receptor (EGFR) Signaling and Neural Stem Cell Phenotype: Cell Enhancer or Cell Transformer?

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Recruitment of neural stem cells (NSCs) represents an elegant strategy for replacing adult central nervous system (CNS) cells lost to injury or disease. However, except in the rostral migratory stream to the olfactory bulb, the adult CNS harbors a relatively non permissive environment for motility of neural stem cells. This opens the possibility of therapeutic enhancement of NSC motility towards sites of CNS injury or disease. The Epidermal Growth Factor Receptor (EGFR) is involved in the activation of a number of downstream pathways that regulate the phenotype of progenitor cells. Activated EGFR tyrosine kinase activity enhances NSC migration, proliferation, and survival. However, EGFR signaling is also known to play a role in the most malignant and highly invasive of human tumors, *glioblastoma multiforme (GBM)*. Recent evidence supports the theory that GBM derives from a 'cancer stem cell' and that EGFR signals are commonly altered in these precursor cells. This article will review the role of EGFR signaling as it relates to neural stem cell motility and invasion. The duality of altered EGFR signaling in neural progenitor cells is discussed and opportunities for enhancing the recruitment of adult progenitors, and consequences of altering EGFR signaling in progenitor cells will be highlighted.