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**Volume 39, Issue 3, Pages 193 - 206**

Published Online: 11 Jun 2002  
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## Original Article

### Human cortical glial tumors contain neural stem-like cells expressing astroglial and neuronal markers in vitro

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**Funded by:**

- NIH/NINDS; Grant Number: NS37556
- McKnight Brain Institute
- Department of Neurosurgery and Shands Cancer Center

**Keywords**  
neural stem cell • glial tumors • neural cell diversity • serum and anchorage withdrawal • pleiotropic growth factors • *nestin* • *Survivin* • *Delta* • *Jagged*

**Abstract**  
Neural stem cells from neurogenic regions of mammalian CNS are clonogenic in an in vitro culture system exploiting serum and anchorage withdrawal in medium supplemented with methyl cellulose and the pleiotropic growth factors EGF, FGF2, and insulin. The aim of this study was to test whether cortical glial tumors contain stem-like cells capable, under this culture system, of forming clones showing intraclonal heterogeneity in the expression of neural lineage-specific proteins. The high frequencies of clone-forming cells (about 0.1-10 × 10<sup>-3</sup>) in clinical tumor specimens with mutated p53, and in neurogenic regions of normal human CNS, suggest that the ability to form clones in this culture system is induced epigenetically. RT-PCR analyses of populations of normal brain- and tumor-derived sister clones revealed transcripts for nestin, neuron-specific enolase, and glial fibrillary acidic protein (GFAP). However, the tumor-derived clones were different from clones derived from neurogenic regions of normal brain in the expression of transcripts specific for genes associated with neural cell fate determination via the *Notch*-signaling pathway (*Delta* and *Jagged*), and cell survival at G2 or mitotic phases (*Survivin*). Moreover, the individual glioma-derived clones contain cells immunopositive separately for GFAP or neuronal β-III tubulin, as well as single cells coexpressing both glial and neuronal markers. The data suggest that the latent critical stem cell characteristics can be epigenetically induced by growth conditions not only in cells from neurogenic regions of normal CNS but also in cells from cortical glial tumors. Moreover, tumor stem-like cells with genetically defective responses to epigenetic stimuli may contribute to gliomagenesis and the developmental pathological heterogeneity of glial tumors. GLIA 39:193-206, 2002. © 2002 Wiley-Liss, Inc.

Received: 18 December 2001; Accepted: 8 April 2002

Digital Object Identifier (DOI)

10.1002/glia.10094 [About DOI](#)

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