Spotlight

By Anne Forde

Nestin's role in brain tumor carcinogenesis

Lu *et al*.
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The neural stem cell marker nestin has shown to be strongly upregulated in glioblastoma multiforme – the most common and aggressive adult brain tumor. To date, nestin is associated with high-grade tumors and disease malignancy. In this paper, Lu and coauthors examine whether nestin also has a role in glioblastoma carcinogenesis.

The authors subcloned rat glioblastoma cells expressing high and low levels of nestin. The proliferation rates of the subclones were not significantly different *in vitro*. Mice injected either with cells expressing low or high nestin levels both formed palpable tumors but those tumors derived from low-expressing clones were significantly smaller ($p<0.05$). Interestingly, once the tumors developed to a palpable size, those derived from both kinds of cells started to grow at similar rates. To confirm the importance of nestin in the development of the tumors, the authors conducted tumor formation assays in nude mice using human glioblastoma cells that were negative for nestin. Tumors derived from these cells *in vivo* actually re-expressed nestin as observed by histology and western blot . Taken together, these findings indicate that nestin plays a role in *in vivo* carcinogenesis.

Lu and colleagues then created a nestin knockdown model using a nestin shRNA adenovirus system. Tumor-bearing animals treated with this knockdown vector developed tumors at significantly slower rates than controls.

These results suggest that nestin is expressed heterogenously in glioblastoma but nonetheless is important in tumor development and could be a target for treatment of this aggressive disease.

Tumor volume derived from 4 rat C6 subclones that expressed high (H1 and H2) and low (L 1 and L2) levels of nestin.