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# Telomerase activity-independent function of TERT allows glioma cells to attain cancer stem cell characteristics by inducing EGFR expression.

Beck S, Jin X, Sohn YW, Kim JK, Kim SH, Yin J, Pian X, Kim SC, Nam DH, Choi YJ, Kim H.

National Research Laboratory of Animal Cell Biotechnology, School of Agricultural Biotechnology, Seoul National University, Seoul, 152-742, Korea.

### Abstract

Telomerase reverse transcriptase (TERT), the catalytic subunit of the enzyme telomerase, is robustly expressed in cancer cells. TERT enables cells to avoid chromosome shortening during repeated replication by maintaining telomere length. However, several lines of evidence indicate that many cancer cells exhibit shorter telomere length than normal tissues, implying an additional function of TERT in tumor formation and progression. Here, we report a telomerase activity-independent function of TERT that induces cancer stemness in glioma cells. Overexpression of TERT712, a telomerase activity-deficient form of TERT, in U87MG cells promoted cell self-renewal in vitro, and induced EGFR expression and formation of gliomas exhibiting cellular heterogeneity in vivo. In patients with glioblastoma multiforme, TERT expression showed a high correlation with EGFR expression, which is closely linked to the stemness gene signature. Induction of differentiation and TERT-knockdown in glioma stem cells led to a marked reduction in EGFR expression, cancer stemness, and anticancer drug resistance. Together, our findings indicate that TERT plays a crucial role in tumor progression by promoting cancer stemness through expression of EGFR.

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