

BCL3

B-cell CLL/lymphoma 3 (BCL3) was previously found to be a putative proto-oncogene in human cancers and the decoy receptor DcR1 is induced in a p50/Bcl3-dependent manner and attenuates the efficacy of [temozolomide](#) in [glioblastoma](#) cells. However, its expression status, clinical significance and biological functions in glioma remain largely unknown.

In a study, the levels of BCL3 were overexpressed in glioma compared to normal brain tissues. Furthermore, high expression of BCL3 protein was confirmed by immunoblotting in glioma cells as compared with normal human astrocyte cell line. The positive expression of BCL3 was correlated with adverse prognostic features and reduced overall survival rate of glioma patients. BCL3 silencing resulted in prominent decreased proliferation, cell cycle arrest in G1 phase and increased apoptosis in U251 cells. In contrast, BCL3 overexpression in U87 cells remarkably facilitated proliferative ability and cell cycle progression and induced apoptosis. In vivo studies showed that BCL3 knockdown inhibited the tumor growth of U251 cells in a mouse xenograft model. Mechanistically, BCL3 positively regulated the abundance of [STAT3](#), p-STAT3 and the downstream targets of STAT3 pathway including BCL2, MCL-1 and cyclin D1 in glioma cells. Furthermore, a positive correlation between BCL3 and STAT3 expression was observed in glioma specimens. Notably, we confirmed that STAT3 knockdown abolished the oncogenic roles of BCL3 in glioma. In conclusion, we suggest that BCL3 serves as an oncogene in glioma by modulating proliferation, cell cycle progression and apoptosis, and its oncogenic effects are mediated by the STAT3 signaling pathway ¹⁾.

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Wu J, Li L, Jiang G, Zhan H, Wang N. B-cell CLL/lymphoma 3 promotes glioma cell proliferation and inhibits apoptosis through the oncogenic STAT3 pathway. *Int J Oncol*. 2016 Oct 12. doi: 10.3892/ijo.2016.3729. PubMed PMID: 27748795.

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