BACE2

Gliomas are the most common primary malignant tumours of the central nervous system, and new molecular biomarkers are urgently needed for diagnosis and targeted therapy. Here, we report that increased Beta-Site APP-Cleaving Enzyme 2 (BACE2) expression is associated with increases in the grade of human glioma, the incidence of the mesenchymal molecular glioblastoma multiforme (GBM) subtype and the likelihood of poor prognoses for patients. BACE2 knockdown suppressed cell invasion, cell migration and tumour growth both in vitro and in vivo, while BACE2 overexpression promoted the mesenchymal transition and cell proliferation. Furthermore, TGFβ1 stimulated BACE2 expression through Smad-dependent signalling, which modulated TNF-α-induced NF-κB activity through the PP1A/IKK pathway to promote tumorigenesis in both U87MG and U251 cells. Our study indicated that BACE2 plays a significant role in glioma development. Therefore, BACE2 is a potential therapeutic target for human gliomas due to its function and ability to be regulated [1)].

1)
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