LINC00511

Tumour invasion is closely related to the prognosis and glioblastoma recurrence and partially attributes to epithelial-mesenchymal transition. Long intergenic non-coding RNA 00511 (LINC00511) plays a pivotal role in the tumour; however, the role of LINC00511 in GBM, especially in the epigenetic molecular regulation mechanism of EMT, is still unclear. They found that LINC00511 was up-regulated in GBM tissues and relatively high LINC00511 expression predicted poorer prognosis. Moreover, ectopic LINC00511 enhanced GBM cell proliferation, EMT, migration and invasion, whereas LINC00511 knockdown had the opposite effects. Mechanistically, we confirmed that ZEB1 acted as a transcription factor for LINC00511 in GBM cells. Subsequently, Du et al. found that LINC00511 served as a competing endogenous RNA that sponged miR-524-5p to indirectly regulate YB1, whereas, up-regulated YB1 promoted ZEB1 expression, which inversely facilitated LINC00511 expression. Finally, orthotopic xenograft models were performed to further demonstrate the LINC00511 on GBM tumorigenesis. This study demonstrates that a LINC00511/miR-524-5p/YB1/ZEB1 positive feedback loop provides potential therapeutic targets for GBM progression [1)].

1)
Du X, Tu Y, Liu S, Zhao P, Bao Z, Li C, Li J, Pan M, Ji J. LINC00511 contributes to glioblastoma tumorigenesis and epithelial-mesenchymal transition via LINC00511/miR-524-5p/YB1/ZEB1 positive feedback loop. J Cell Mol Med. 2019 Dec 19. doi: 10.1111/jcmm.14829. [Epub ahead of print] PubMed PMID: 31856394.