6. LncRNA PLAC2 down-regulates RPL36 expression and blocks cell cycle progression in glioma through a mechanism involving STAT1.
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文章简介

研究表明长链非编码 RNA 在肿瘤发生发展中起重要调节作用，能够影响肿瘤细胞的增殖和凋亡等过程。本研究发现 lncRNA PLAC2 通过靶向胶质瘤中的核糖体蛋白（RPL36）诱导细胞周期停滞。RPL36 可以促进细胞增殖和 G1 / S 细胞周期进展。ChIRP-MS 结合生物信息学分析发现转录激活因子 1（STAT1）与 lncRNA PLAC2 和 RPL36 启动子相互作用，lncRNA PLAC2 抑制 STAT1 核转移，从而降低 RPL36 表达，起到抑制细胞增殖并诱导细胞周期停滞的作用。研究结果发现胶质瘤中细胞周期调节新通路，有望作为神经胶质瘤治疗的新靶标。

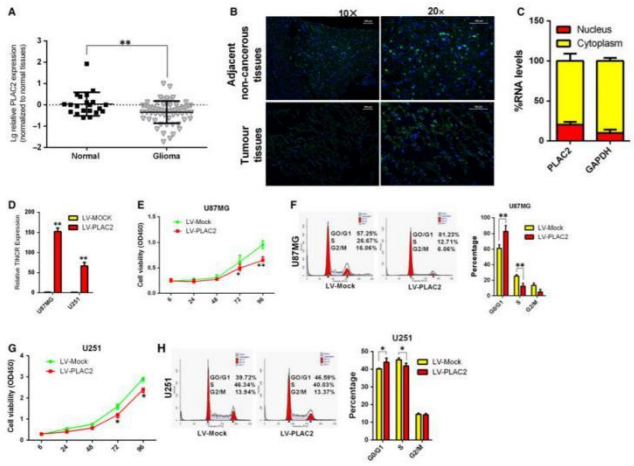

Fig. LncRNA PLAC2 is strongly down-regulated in glioma tissues and inhibits glioma cell proliferation, induces cell cycle arrest. (A) PLAC2 was down-regulated in glioma as compared to normal brain tissues. Log10 transformation was applied to expression levels. *P < 0.05, **P < 0.01. (B) Representative images of PLAC2 expression in glioma (Tumour) and adjacent non-cancerous tissue by FISH (n = 6). (C) Percentage of nuclear and cytoplasmic RNA levels of PLAC2 and GAPDH measured by qRT-PCR after cell fractionation in U87MG cells. The graph shows the average of three independent experiments. (D) Stable overexpression of PLAC2 in U87MG and U251 cells after lentiviral infection with LV-PLAC2. Control cells were infected with the empty lentiviral vector LV-Mock. Experiments were performed in triplicate. **P < 0.01. (E–H) U87MG and U251 cells were treated with LV-Mock or LV-PLAC2 and cell viability and cell cycle phase were evaluated with the CCK-8 assay (E and G) and by FACS (F and H), respectively. PLAC2 overexpression in U87MG and U251 cells decreased cell proliferation and increased G1/S arrest relative to control cells. *P < 0.05, **P < 0.01.