ECT2

Epithelial cell transforming sequence 2

The protein encoded by this gene is a transforming protein that is related to Rho-specific exchange factors and yeast cell cycle regulators. The expression of this gene is elevated with the onset of DNA synthesis and remains elevated during G2 and M phases. In situ hybridization analysis showed that expression is at a high level in cells undergoing mitosis in regenerating liver. Thus, this protein is expressed in a cell cycle-dependent manner during liver regeneration, and is thought to have an important role in the regulation of cytokinesis.

It is up-regulated in glioma and promotes glioma cell proliferation. A preliminary experiment showed a positive correlation between ECT2 and Pituitary tumor-transforming 1 (PTTG1). The aim of this study was to explore how ECT2 affects PTTG1 to influence the proliferation of glioma cells.

The expression of ECT2 in glioma was detected by western blotting and Reverse Transcription-Polymerase Chain Reaction (RT-PCR). The effect of ECT2 on glioma proliferation was examined using cell proliferation-related assays and In vivo experiments. The effect of ECT2 on the stability of E2F1 and the expression of PTTG1 were examined by western blotting, co-immunoprecipitation and in vivo ubiquitination assays.

ECT2 was up-regulated in gliomas and was negatively correlated with prognosis; its downregulation inhibited the glioma cell proliferation. Furthermore, ECT2 regulated PTTG1 expression by affecting the stability of E2F1, thereby affecting the glioma cell proliferation. In addition, the deubiquitinating enzyme PSMD14 affected the degradation of E2F1 and regulated the stability of E2F1. Interestingly, ECT2 regulated the expression of PSMD14.

In this study, we clarify a new mechanism by which ECT2 regulates the expression of PTTG1 and thus affects the proliferation of glioma cells: ECT2 influences the stability of E2F1 by regulating the expression of the deubiquitinating enzyme PSMD14, thereby affecting the expression of PTTG1. Intensive and extensive understanding of the mechanism of ECT2 in glioma proliferation may provide an opportunity for the development of new molecular therapeutic targets for glioma treatment.