PTTG1

The encoded protein is a homolog of yeast securin proteins, which prevent separins from promoting sister chromatid separation. It is an anaphase-promoting complex (APC) substrate that associates with a separin until activation of the APC. The gene product has transforming activity in vitro and tumorigenic activity in vivo, and the gene is highly expressed in various tumors. The gene product contains 2 PXXP motifs, which are required for its transforming and tumorigenic activities, as well as for its stimulation of basic fibroblast growth factor expression. It also contains a destruction box (D box) that is required for its degradation by the APC. The acidic C-terminal region of the encoded protein can act as a transactivation domain. The gene product is mainly a cytosolic protein, although it partially localizes in the nucleus.

ECT2 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 a study of Zhi et al., 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, they 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 [1].

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Zhi T, Jiang K, Xu X, Yu T, Zhou F, Wang Y, Liu N, Zhang J. ECT2/PSMD14/PTTG1 axis promotes the proliferation of glioma through stabilizing E2F1. Neuro Oncol. 2018 Dec 24. doi: 10.1093/neuonc/noy207. [Epub ahead of print] PubMed PMID: 30590814.