Pituitary Tumor Pathogenesis

- Methylation and gene expression patterns in adamantinomatous craniopharyngioma highlight a panel of genes associated with disease progression-free survival
- A Bridge Too Far? Towards Medical Therapy for Clinically Nonfunctioning Pituitary Tumors
- Sample Preparation and Sequencing Efficiency of microRNA Libraries from Pituitary Adenoma Tissue and Blood Plasma of Patients with Acromegaly for the Illumina Platform
- Multimodal Model for Non-Invasive Detection of DRD2, SSTR2 and ESR1 Receptor Profiling in Pituitary Neuroendocrine Tumors: A Retrospective Study
- Baseline testosterone levels as a predictor of hypogonadism resolution in male patients with isolated hyperprolactinemia
- Genetic profiling of synchronous pituitary corticotroph adenomas
- Secondary Pituitary Abscess Inside a Macroadenoma Complicated by Postoperative Hemorrhage and Reinfection: A Case Report
- Hypersomatotropism and Hypercortisolism Caused by a Plurihormonal Pituitary Adenoma in a Dog

The pathogenesis of pituitary tumors (PT) is unclear. Deregulation of apoptosis is one of the factors involving tumor growth.

In a study, pituitary tumor (PT) samples were collected from 20 patients after surgery. The expression of Histone deacetylases (HDAC) and p53 was analyzed in the PT samples. PT cell line, AtT-20 cells, were cultured to test the role of HDAC in the regulation of apoptosis in PT cells. The results showed that high levels of HDAC11 and lower levels of p53 were detected in PT. A negative correlation was detected between the data of HDAC11 and p53. A complex of HDAC11 and HEY1, the gene transcription factor of p53, was detected in the PT cells. Less acetylated HEY1 was found in the PT cells. In addition, lower levels of HEY1 and gene transcription activities were detected at the PT53 promoter locus. This phenomenon was mimicked by overexpression of HDAC11 in AtT-20 cells. Knockdown of HDAC11 enhanced the p53 expression in AtT-20 cells. In conclusion, HDAC11 interferes with p53 expression in PT cells. The fact suggests that inhibition of HDAC11 has therapeutic potential in the treatment of PT [1)].

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Wang W, Fu L, Li S, Xu Z, Li X. Histone deacetylase 11 suppresses p53 expression in pituitary tumor cells. Cell Biol Int. 2017 Aug 7. doi: 10.1002/cbin.10834. [Epub ahead of print] PubMed PMID: 28782861.