

The role and mechanism of [collagen](#) type VI alpha 6 (COL6A6) on tumor growth and [metastasis](#) in - [pituitary adenoma](#) (PA) was determined. COL6A6 was downregulated in PA tissues and cell lines, which was negatively associated with the expression of prolyl-4-hydroxylase alpha polypeptide III ([P4HA3](#)) in the progression of PA. Overexpression of COL6A6 significantly suppressed tumor growth and metastasis capacity in PA. In addition, P4HA3 worked as the upstream of the [PI3K-Akt](#) pathway to alleviate the antitumor activity of COL6A6 on the growth and metastasis of both [AtT-20](#) and [HP75](#) cells. Furthermore, the inhibitory effect of COL6A6 on cell proliferation, migration and invasion, and epithelial-mesenchymal transition (EMT) was reversed by [P4HA3](#) overexpression or activation of the PI3K-Akt pathway induced by IGF-1 addition, which provided a new biomarker for clinical PA treatment <sup>1)</sup>.

<sup>1)</sup>

Long R, Liu Z, Li J, Yu H. COL6A6 interacted with P4HA3 to suppress the growth and metastasis of pituitary adenoma via blocking PI3K-Akt pathway. Aging (Albany NY). 2019 Oct 17;11. doi: 10.18632/aging.102300. [Epub ahead of print] PubMed PMID: 31627190.

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