

SMAGP

Small trans-membrane and glycosylated protein (SMAGP), a novel small [transmembrane glycoprotein](#), is reported to be upregulated in multiple [cancers](#) and involved in tumor development. However, little is known about its role in the development of glioblastoma (Glioblastoma). [GEPIA](#) database was used to analyze SMAGP expression and evaluate the prognostic value of SMAGP in Glioblastoma. GO and [KEGG](#) pathway enrichment analyses were used to predict the biological functions and pathways of SMAGP and 948 SMAGP-correlated genes using [DAVID](#) database. Cell viability, colony formation ability, apoptosis, and invasion were evaluated by MTT, colony formation assay, flow cytometry analysis, and Transwell invasion assay, respectively. Western blot was applied to detect the expression of SMAGP, matrix metalloproteinase (MMP)-2, and MMP-9 and analyze the changes of phosphoinositide 3-kinase (PI3K)/protein kinase B (Akt) signaling. Results showed that SMAGP was upregulated and correlated with poor prognosis in Glioblastoma. Functional annotation analysis revealed that SMAGP and 948 SMAGP-correlated genes were primarily associated with cell adhesion and PI3K/Akt pathway. SMAGP interference inhibited cell viability and colony formation ability and promoted apoptosis in Glioblastoma cells. Moreover, SMAGP interference inhibited Glioblastoma cell invasion and suppressed MMP-2 and MMP-9 expression. Additionally, SMAGP silencing inhibited the PI3K/Akt pathway in Glioblastoma cells. Overexpression of Akt abolished the effects of SMAGP knockdown on the malignant phenotypes of [Glioblastoma](#) cells. In conclusion, SMAGP silencing inhibited the malignant phenotypes of Glioblastoma cells by inactivating the [PI3K/Akt pathway](#) ¹⁾.

¹⁾

Ni H, Ji D, Huang Z, Li J. SMAGP knockdown inhibits the malignant phenotypes of glioblastoma cells by inactivating the PI3K/Akt pathway. Arch Biochem Biophys. 2020 Oct 10;108628. doi: 10.1016/j.abb.2020.108628. Epub ahead of print. PMID: 33049294.

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