Transmembrane Glycoprotein

A **transmembrane glycoprotein** is a type of membrane protein that:

- Spans the lipid bilayer of the cell membrane (**transmembrane**),
- Contains **carbohydrate chains** covalently attached to amino acid side chains (**glycoprotein**).

Structure

- Composed of:
  - **Extracellular domain** (often glycosylated)
  - **Transmembrane domain** (hydrophobic region embedded in the membrane)
  - **Intracellular domain** (involved in signal transduction)

Functions

- **Receptor activity** (e.g., EGFR, insulin receptor)
- **Cell adhesion**
- **Signal transduction**
- **Immune recognition**
- **Transport of molecules**

Glycosylation Role

- Stabilizes protein folding
- Protects against proteolysis
- Modulates **cell-cell** and **cell-matrix interactions**
- Influences receptor-ligand binding and signaling specificity

Examples

- EGFR (Epidermal Growth Factor Receptor)
- NCAM (Neural Cell Adhesion Molecule)
- CD4 and CD8 in immune cells

Related Terms

- Integral Membrane Protein
- Protein Glycosylation
- Lipid Bilayer

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.