GPM6a

- Brain-derived neurotrophic factor (BDNF), an autocrine/paracrine regulator of basic ovarian functions and antagonist of kisspeptin
- Human sensory-like neuron surfaceome analysis
- Mechanisms of GPM6A in Malignant Tumors
- Non-synonymous single nucleotide polymorphisms (nsSNPs) within the extracellular domains of the GPM6A gene impair hippocampal neuron development
- Combined additive effects of neuronal membrane glycoprotein GPM6a and the intercellular cell adhesion molecule ICAM5 on neuronal morphogenesis
- Zdhhc1- and Zdhhc2-mediated Gpm6a palmitoylation is essential for maintenance of mammary stem cell activity
- Low- and High-Grade Glioma-Associated Vascular Cells Differentially Regulate Tumor Growth
- Epigenome-wide methylation and progression to high-grade cervical intraepithelial neoplasia (CIN2+): a prospective cohort study in the United States

GPM6a (Glycoprotein M6A) is a neuronal membrane glycoprotein that plays several important roles in the nervous system. Here are some key points about GPM6a:

Structure: GPM6a is a glycoprotein, meaning it has carbohydrate groups attached to it, which can affect its function and interactions with other molecules.

Function: GPM6a is involved in various cellular processes, including:

Cell adhesion: It helps neurons to adhere to one another and to the extracellular matrix. Neuronal development: It may play a role in the growth and differentiation of neurons during development. Synaptic function: GPM6a is thought to be involved in synaptic plasticity, which is essential for learning and memory. Pathological Implications: Alterations in GPM6a expression or function have been implicated in various neurological disorders, including Alzheimer's disease and schizophrenia. Its role in cell signaling and neuronal interactions suggests that it may be a target for therapeutic interventions.

Research: Studies are ongoing to better understand the specific mechanisms by which GPM6a functions in the brain, its interactions with other proteins, and its potential role in disease.

The mechanisms underlying neuronal development and synaptic formation in the brain depend on intricate cellular and molecular processes. The neuronal membrane glycoprotein GPM6a promotes neurite elongation, filopodia/spine formation, and synapse development, yet its molecular mechanisms remain unknown. Since the extracellular domains of GPM6a (ECs) command its function, Gutiérrez Fuster et al. investigated the interaction between ICAM5, the neuronal member of the intercellular adhesion molecule (ICAM) family, and GPM6a's ECs.

A study aimed to explore the functional relationship between GPM6a and ICAM5 in hippocampal culture neurons and cell lines. Immunostaining of 15 days in vitro (DIV) neurons revealed significant co-localization between endogenous GPM6a clusters and ICAM5 clusters in the dendritic shaft. These results were further corroborated by overexpressing GPM6a and ICAM5 in N2a cells and hippocampal neurons at 5 DIV. Moreover, results from the co-immunoprecipitations and cell aggregation assays prove the cis and trans interaction between both proteins in GPM6a/ICAM5 overexpressing HEK293

cells. Additionally, GPM6a and ICAM5 overexpression additively enhanced neurite length, the number of neurites in N2a cells, and filopodia formation in 5 DIV neurons, indicating their cooperative role. These findings highlight the dynamic association between GPM6a and ICAM5 during neuronal development, offering insights into their contributions to neurite outgrowth, filopodia formation, and cell-cell interactions [1)]

1)
Gutiérrez Fuster R, León A, Aparicio GI, Brizuela Sotelo F, Scorticati C. Combined additive effects of neuronal membrane glycoprotein GPM6a and the intercellular cell adhesion molecule ICAM5 on neuronal morphogenesis. J Neurochem. 2024 Oct 1. doi: 10.1111/jnc.16231. Epub ahead of print. PMID: 39352694.