Flotillin-1

- FLOT1, stabilized by WTAP/IGF2BP2 mediated N6-methyladenosine modification, predicts poor prognosis and promotes growth and invasion in gliomas
- Flotillin-1 is a prognostic biomarker for glioblastoma and promotes cancer development through enhancing invasion and altering tumour microenvironment
- Neural stem cell-derived exosomes transfer miR-124-3p into cells to inhibit glioma growth by targeting FLOT2
- FLOT2 upregulation promotes growth and invasion by interacting and stabilizing EphA2 in gliomas
- Extracellular alpha-synuclein enters dopaminergic cells by modulating flotillin-1-assisted dopamine transporter endocytosis
- Flot2 targeted by miR-449 acts as a prognostic biomarker in glioma
- Treatment with methyl-β-cyclodextrin prevents mechanical allodynia in resiniferatoxin neuropathy in a mouse model
- PLCD3, a flotillin2-interacting protein, is involved in proliferation, migration and invasion of nasopharyngeal carcinoma cells

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Flotillin-1 (FLOT1), is a plasma membrane protein involved in endocytosis.

Flotillin-1(FLOT1) has long been recognized as a tumor promoter gene in several types of cancer. However, the expression and function of FLOT1 in glioblastomas (GBM) has not been elucidated.

Wang et al. find that the expression level of FLOT1 in GBM tissue was much higher than that in normal brain, and the expression was even higher in the more aggressive subtypes and IDH status of glioma. Kaplan-Meier survival revealed that high FLOT1 expression is closely associated with poor outcome in GBM patients. FLOT1 knockdown markedly reduced the proliferation, migration and invasiveness of GBM cells, while FLOT1 overexpression significantly increases GBM cell proliferation, migration and invasiveness. Mechanistically, FLOT1 expression may play a potential role in the microenvironment of GBM. Therefore, FLOT1 promotes GBM proliferation and invasion in vitro and in vivo and may serve as a biomarker of prognosis and therapeutic potential in the fight against GBM [1].

It was identified as a binding partner of ALK. RNAi-mediated attenuation of FLOT1 expression in neuroblastoma cells caused ALK dissociation from endosomes along with membrane accumulation of ALK, thereby triggering activation of ALK and downstream effector signals. These features enhanced the malignant properties of neuroblastoma cells in vitro and in vivo. Conversely, oncogenic ALK mutants showed less binding affinity to FLOT1 than wild-type ALK. Clinically, lower expression levels of FLOT1 were documented in highly malignant subgroups of human neuroblastoma specimens. Taken together, our findings suggest that attenuation of FLOT1-ALK binding drives malignant phenotypes of neuroblastoma by activating ALK signaling [2].

1)

Wang R, Chen Z, Zhang Y, Xiao S, Zhang W, Hu X, Xiao Q, Liu Q, Wang X. Flotillin-1 is a prognostic

biomarker for glioblastoma and promotes cancer development through enhancing invasion and altering tumour microenvironment. J Cell Mol Med. 2023 Jan 17. doi: 10.1111/jcmm.17660. Epub ahead of print. PMID: 36647700.
2)
Tomiyama A, Uekita T, Kamata R, Sasaki K, Takita J, Ohira M, Nakagawara A, Kitanaka C, Mori K, Yamaguchi H, Sakai R. Flotillin-1 regulates oncogenic signaling in neuroblastoma cells by regulating ALK membrane association. Cancer Res. 2014 Jul 15;74(14):3790-801. doi: 10.1158/0008-5472.CAN-14-0241. Epub 2014 May 15. PubMed PMID: 24830726.