

FLCN Antibody (Center) Blocking Peptide Synthetic peptide Catalog # BP8658c

Specification

FLCN Antibody (Center) Blocking Peptide - Product Information

Primary Accession

<u>Q8NFG4</u>

FLCN Antibody (Center) Blocking Peptide - Additional Information

Gene ID 201163

Other Names Folliculin, BHD skin lesion fibrofolliculoma protein, Birt-Hogg-Dube syndrome protein, FLCN, BHD

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP8658c was selected from the Center region of human FLCN. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

FLCN Antibody (Center) Blocking Peptide - Protein Information

Name FLCN {ECO:0000303|PubMed:15657874, ECO:0000312|HGNC:HGNC:27310}

Function

Multi-functional protein, involved in both the cellular response to amino acid availability and in the regulation of glycolysis (PubMed:17028174, PubMed:18663353, PubMed:18663353, PubMed:21209915, PubMed:24081491, PubMed:24095279, PubMed:31672913, PubMed:31704029, PubMed:33704029, PubMed:34381247, PubMed:34381247, PubMed:36103527, PubMed:36103527, PubMed:<a href="http://www.uniprot.org/citations/37079666"



target="_blank">37079666). GTPase-activating protein that plays a key role in the cellular response to amino acid availability through regulation of the non-canonical mTORC1 signaling cascade controlling the MiT/TFE factors TFEB and TFE3 (PubMed:17028174, PubMed:18663353, PubMed:21209915, PubMed:24081491, PubMed:24095279, PubMed:24448649, PubMed:31672913, PubMed:31704029, PubMed:32612235, PubMed:36103527, PubMed:37079666). Activates mTORC1 by acting as a GTPase-activating protein: specifically stimulates GTP hydrolysis by RagC/RRAGC or RagD/RRAGD, promoting the conversion to the GDP-bound state of RagC/RRAGC or RagD/RRAGD, and thereby activating the kinase activity of mTORC1 (PubMed:24095279, PubMed:31672913, PubMed:31704029, PubMed:32612235, PubMed:37079666). The GTPase-activating activity is inhibited during starvation and activated in presence of nutrients (PubMed:31672913, PubMed:32612235). Acts as a key component for non- canonical mTORC1-dependent control of the MiT/TFE factors TFEB and TFE3, while it is not involved in mTORC1-dependent phosphorylation of canonical RPS6KB1/S6K1 and EIF4EBP1/4E-BP1 (PubMed:21209915, PubMed:24081491, PubMed:31672913, PubMed:32612235). In low-amino acid conditions, the lysosomal folliculin complex (LFC) is formed on the membrane of lysosomes, which inhibits the GTPase-activating activity of FLCN, inactivates mTORC1 and maximizes nuclear translocation of TFEB and TFE3 (PubMed: 31672913). Upon amino acid restimulation, RagA/RRAGA (or RagB/RRAGB) nucleotide exchange promotes disassembly of the LFC complex and liberates the GTPase-activating activity of FLCN, leading to activation of mTORC1 and subsequent cytoplasmic retention of TFEB and TFE3 (PubMed:31672913). Indirectly acts as a positive regulator of Wnt signaling by promoting mTOR-dependent cytoplasmic retention of MiT/TFE factor TFE3 (PubMed:31272105). Required for the exit of hematopoietic stem cell from pluripotency by promoting mTOR-dependent cytoplasmic retention of TFE3, thereby increasing Wnt signaling (PubMed: 30733432). Acts as an inhibitor of browning of adipose tissue by regulating mTOR-dependent cytoplasmic retention of TFE3 (By similarity). Involved in the control of embryonic stem cells differentiation; together with LAMTOR1 it is necessary to recruit and activate RagC/RRAGC and RagD/RRAGD at the lysosomes, and to induce exit of embryonic stem cells from pluripotency via non-canonical, mTORindependent TFE3 inactivation (By similarity). In response to flow stress, regulates STK11/LKB1 accumulation and mTORC1 activation through primary cilia: may act by recruiting STK11/LKB1 to primary cilia for activation of AMPK resided at basal bodies, causing mTORC1 down- regulation (PubMed:27072130). Together with FNIP1 and/or FNIP2, regulates autophagy: following phosphorylation by ULK1, interacts with GABARAP and promotes autophagy (PubMed: 25126726). Required for

href="http://www.uniprot.org/citations/25126726" target="_blank">25126726). Required for starvation-induced perinuclear clustering of lysosomes by promoting association of RILP with its



effector RAB34 (PubMed:27113757). Regulates glycolysis by binding to lactate dehydrogenase LDHA, acting as an uncompetitive inhibitor (PubMed:34381247).

Cellular Location

Lysosome membrane. Cytoplasm, cytosol. Cell projection, cilium. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome. Cytoplasm, cytoskeleton, spindle. Nucleus Note=Localizes to lysosome membrane in amino acid-depleted conditions and relocalizes to the cytosol upon refeeding (PubMed:24095279, PubMed:29848618, PubMed:31672913). Colocalizes with FNIP1 and FNIP2 in the cytoplasm (PubMed:17028174, PubMed:18663353). Also localizes to motile and non-motile cilia, centrosomes and the mitotic spindle (PubMed:23784378).

Tissue Location

Expressed in most tissues tested, including skin, lung, kidney, heart, testis and stomach.

FLCN Antibody (Center) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

Blocking Peptides

FLCN Antibody (Center) Blocking Peptide - Images

FLCN Antibody (Center) Blocking Peptide - Background

FLCN may play a role in the pathogenesis of an uncommon form of kidney cancer through its association with an inherited disorder of the hair follicle (fibrofolliculomas). FLCN may be a tumor suppressor. May be involved in colorectal tumorigenesis. It may be involved in energy and/or nutrient sensing through the AMPK and mTOR signaling pathways.

FLCN Antibody (Center) Blocking Peptide - References

Khoo,S.K., et.al., J. Med. Genet. 39 (12), 906-912 (2002)Shin,J.H., et.al., J. Med. Genet. 40 (5), 364-367 (2003)