

ULK2 Blocking Peptide (N-term) Synthetic peptide Catalog # BP8105a

Specification

ULK2 Blocking Peptide (N-term) - Product Information

Primary Accession Other Accession <u>Q8IYT8</u> Q9QY01

ULK2 Blocking Peptide (N-term) - Additional Information

Gene ID 9706

Other Names Serine/threonine-protein kinase ULK2, Unc-51-like kinase 2, ULK2, KIAA0623

Target/Specificity The synthetic peptide sequence is selected from aa 250-264 of HUMAN ULK2

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.

ULK2 Blocking Peptide (N-term) - Protein Information

Name ULK2

Synonyms KIAA0623

Function

Serine/threonine-protein kinase involved in autophagy in response to starvation. Acts upstream of phosphatidylinositol 3-kinase PIK3C3 to regulate the formation of autophagophores, the precursors of autophagosomes. Part of regulatory feedback loops in autophagy: acts both as a downstream effector and a negative regulator of mammalian target of rapamycin complex 1 (mTORC1) via interaction with RPTOR. Activated via phosphorylation by AMPK, also acts as a negative regulator of AMPK through phosphorylation of the AMPK subunits PRKAA1, PRKAB2 and PRKAG1. May phosphorylate ATG13/KIAA0652, FRS2, FRS3 and RPTOR; however such data need additional evidences. Not involved in ammonia-induced autophagy or in autophagic response of cerebellar granule neurons (CGN) to low potassium concentration. Plays a role early in neuronal differentiation and is required for granule cell axon formation: may govern axon formation via Ras-like GTPase signaling and through regulation of the Rab5-mediated endocytic pathways within developing axons.



Cellular Location Cytoplasmic vesicle membrane; Peripheral membrane protein. Note=Localizes to pre-autophagosomal membrane

ULK2 Blocking Peptide (N-term) - Protocols

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

<u>Blocking Peptides</u>

ULK2 Blocking Peptide (N-term) - Images

ULK2 Blocking Peptide (N-term) - Background

Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes which enclose the cytoplasmic constituent targeted for degradation in a membrane bound structure, which then fuse with the lysosome (or vacuole) releasing a single-membrane bound autophagic bodies which are then degraded within the lysosome (or vacuole). Two human homologs of the yeast autophagy-specific kinase exist: ULK1(APG1) and ULK2. APG1 plays a critical role in regulating key elements of the autophagy pathway. APG1 stimulates autophagy, leading to autophagy-dependent restriction of cell growth and ultimately cell apoptosis at high levels of activity, and is a negative regulator of mTOR signaling.

ULK2 Blocking Peptide (N-term) - References

Yan, J., et al., Oncogene 18(43):5850-5859 (1999).