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Beclin 1 Antibody Blocking peptide

Synthetic peptide
Catalog # BP1818a

Specification

Beclin 1 Antibody Blocking peptide - Product Information

Primary Accession Q14457

Beclin 1 Antibody Blocking peptide - Additional Information

Gene ID 8678

Other Names
Beclin-1, Coiled-coil myosin-like BCL2-interacting protein, Protein GT197, BECN1, GT197

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP1818a was selected from the Q196 region of human Autophagy BECN1. 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.

Beclin 1 Antibody Blocking peptide - Protein Information

Name BECN1

Synonyms GT197

Function
Plays a central role in autophagy (PubMed:18570871, PubMed:21358617, PubMed:23184933, PubMed:23974797, PubMed:28445460, PubMed:25484083, PubMed:37776275). Acts as a core subunit of the PI3K complex that mediates formation of phosphatidylinositol 3-phosphate; different complex forms are believed to play a role in multiple membrane trafficking pathways: PI3KC3-C1 is involved in initiation of autophagosomes

and PI3KC3-C2 in maturation of autophagosomes and endocytosis. Involved in regulation of degradative endocytic trafficking and required for the abcission step in cytokinesis, probably in the context of PI3KC3-C2 (PubMed:20643123, PubMed:20208530, PubMed:23974797, PubMed:26783301). Essential for the formation of PI3KC3-C2 but not PI3KC3-C1 PI3K complex forms. Involved in endocytosis (PubMed:25275521). Protects against infection by a neurovirulent strain of Sindbis virus (PubMed:9765397). May play a role in antiviral host defense.

Cellular Location
Cytoplasm. Golgi apparatus, trans-Golgi network membrane; Peripheral membrane protein. Endosome membrane; Peripheral membrane protein. Endoplasmic reticulum membrane; Peripheral membrane protein. Mitochondrion membrane; Peripheral membrane protein. Endosome {ECO:0000250|UniProtKB:O88597} Cytoplasmic vesicle, autophagosome. Note=Interaction with ATG14 promotes translocation to autophagosomes. Expressed in dendrites and cell bodies of cerebellar Purkinje cells (By similarity) {ECO:0000250|UniProtKB:O88597, ECO:0000269|PubMed:19050071} [Beclin-1-C 37 kDa]: Mitochondrion {ECO:0000250|UniProtKB:O88597}

Tissue Location
Ubiquitous.

Beclin 1 Antibody Blocking peptide - Protocols

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

- Blocking Peptides

Beclin 1 Antibody Blocking peptide - Images

Beclin 1 Antibody Blocking peptide - 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). Beclin 1 plays a role in two fundamentally important cell biological pathways: autophagy and apoptosis. Beclin 1 is thought to function as a VPS and autophagy protein as part of a complex with Class III PI3 kinase, Vps34.

Beclin 1 Antibody Blocking peptide - References

Baehrecke EH. Nat Rev Mol Cell Biol. 6(6):505-10. (2005) Lum JJ, et al. Nat Rev Mol Cell Biol. 6(6):439-48. (2005) Greenberg JT. Dev Cell. 8(6):799-801. (2005) Levine B. Cell. 120(2):159-62. (2005) Shintani T and Klionsky DJ. Science. 306(5698):990-5. (2004)Liang,X.H., et al. J. Virol. 72 (11), 8586-8596 (1998) Aita V.M., et al. Genomics 59:59-65(1999).