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APG4B Antibody (E135) Blocking Peptide

Synthetic peptide
Catalog # BP1809e

Specification

APG4B Antibody (E135) Blocking Peptide - Product Information

Primary Accession Q9Y4P1

APG4B Antibody (E135) Blocking Peptide - Additional Information

Gene ID 23192

Other Names
Cysteine protease ATG4B, 3422-, AUT-like 1 cysteine endopeptidase, Autophagin-1, Autophagy-related cysteine endopeptidase 1, Autophagy-related protein 4 homolog B, hAPG4B, ATG4B, APG4B, AUTL1, KIAA0943

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP1809e was selected from the E135 region of human Autophagy APG4B. 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.

APG4B Antibody (E135) Blocking Peptide - Protein Information

Name ATG4B {ECO:0000303|PubMed:15187094, ECO:0000312|HGNC:HGNC:20790}

Function
Cysteine protease that plays a key role in autophagy by mediating both proteolytic activation and delipidation of ATG8 family proteins (PubMed:15169837, PubMed:15187094, PubMed:17347651, PubMed:19322194, PubMed:21177865, PubMed:26378241, PubMed:29232556, PubMed:28821708, PubMed:<a

href="http://www.uniprot.org/citations/30443548" target="_blank">30443548, PubMed:30661429, PubMed:22302004, PubMed:27527864, PubMed:28633005, PubMed:30076329). Required for canonical autophagy (macroautophagy), non-canonical autophagy as well as for mitophagy (PubMed:33773106, PubMed:33909989). The protease activity is required for proteolytic activation of ATG8 family proteins: cleaves the C-terminal amino acid of ATG8 proteins MAP1LC3A, MAP1LC3B, MAP1LC3C, GABARAPL1, GABARAPL2 and GABARAP, to reveal a C- terminal glycine (PubMed:15169837, PubMed:15187094, PubMed:17347651, PubMed:20818167, PubMed:19322194, PubMed:21177865, PubMed:22302004, PubMed:27527864, PubMed:28633005, PubMed:29458288, PubMed:30661429, PubMed:28287329). Exposure of the glycine at the C-terminus is essential for ATG8 proteins conjugation to phosphatidylethanolamine (PE) and insertion to membranes, which is necessary for autophagy (PubMed:15169837, PubMed:15187094, PubMed:17347651, PubMed:19322194, PubMed:21177865, PubMed:22302004). Protease activity is also required to counteract formation of high-molecular weight conjugates of ATG8 proteins (ATG8ylation): acts as a deubiquitinating-like enzyme that removes ATG8 conjugated to other proteins, such as ATG3 (PubMed:31315929, PubMed:33773106). In addition to the protease activity, also mediates delipidation of ATG8 family proteins (PubMed:15187094, PubMed:28633005, PubMed:29458288, PubMed:32686895, PubMed:33909989, PubMed:19322194). Catalyzes delipidation of PE- conjugated forms of ATG8 proteins during macroautophagy (PubMed:15187094, PubMed:29458288, PubMed:32686895, PubMed:33909989, PubMed:19322194). Also involved in non-canonical autophagy, a parallel pathway involving conjugation of ATG8 proteins to single membranes at endolysosomal compartments, by catalyzing delipidation of ATG8 proteins conjugated to phosphatidylserine (PS) (PubMed:33909989). Compared to other members of the family (ATG4A, ATG4C or ATG4C), constitutes the major protein for proteolytic activation of ATG8 proteins, while it displays weaker delipidation activity than other ATG4 paralogs (PubMed:<a href="http://www.uniprot.org/citations/29458288"

target="_blank">29458288, PubMed:30661429). Involved in phagophore growth during mitophagy independently of its protease activity and of ATG8 proteins: acts by regulating ATG9A trafficking to mitochondria and promoting phagophore-endoplasmic reticulum contacts during the lipid transfer phase of mitophagy (PubMed:33773106).

Cellular Location
Cytoplasm. Cytoplasm, cytosol. Cytoplasmic vesicle, autophagosome. Endoplasmic reticulum. Mitochondrion. Note=Mainly localizes to the cytoplasm, including cytosol (PubMed:29165041). A samll potion localizes to mitochondria; phosphorylation at Ser-34 promotes localization to mitochondria (PubMed:29165041).

APG4B Antibody (E135) Blocking Peptide - Protocols

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

- Blocking Peptides

APG4B Antibody (E135) Blocking Peptide - Images

APG4B Antibody (E135) 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). APG4 is a cysteine protease required for autophagy, which cleaves the C-terminal part of either MAP1LC3, GABARAPL2 or GABARAP, allowing the liberation of form I. A subpopulation of form I is subsequently converted to a smaller form (form II). Form II, with a revealed C-terminal glycine, is considered to be the phosphatidylethanolamine (PE)-conjugated form, and has the capacity for the binding to autophagosomes.

APG4B Antibody (E135) 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)