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ATG16L Antibody Blocking peptide

Synthetic peptide
Catalog # BP1817b

Specification

ATG16L Antibody Blocking peptide - Product Information

Primary Accession Q676U5

ATG16L Antibody Blocking peptide - Additional Information

Gene ID 55054

Other Names
Autophagy-related protein 16-1, APG16-like 1, ATG16L1, APG16L

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP1817b was selected from the L92 region of human Autophagy APG16L. 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.

ATG16L Antibody Blocking peptide - Protein Information

Name ATG16L1 {ECO:0000303|PubMed:17200669, ECO:0000312|HGNC:HGNC:21498}

Function
Plays an essential role in both canonical and non-canonical autophagy: interacts with ATG12-ATG5 to mediate the lipidation to ATG8 family proteins (MAP1LC3A, MAP1LC3B, MAP1LC3C, GABARAPL1, GABARAPL2 and GABARAP) (PubMed:23376921, PubMed:23392225, PubMed:29317426, PubMed:30778222, PubMed:33909989, PubMed:24553140, PubMed:24954904, PubMed:27273576). Acts as a molecular hub, coordinating autophagy pathways via distinct domains that support either canonical or non- canonical signaling (PubMed:<a

href="http://www.uniprot.org/citations/29317426" target="_blank">29317426, PubMed:30778222). During canonical autophagy, interacts with ATG12-ATG5 to mediate the conjugation of phosphatidylethanolamine (PE) to ATG8 proteins, to produce a membrane-bound activated form of ATG8 (PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904, PubMed:27273576). Thereby, controls the elongation of the nascent autophagosomal membrane (PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904, PubMed:27273576). Also involved in non-canonical autophagy, a parallel pathway involving conjugation of ATG8 proteins to single membranes at endolysosomal compartments, probably by catalyzing conjugation of phosphatidylserine (PS) to ATG8 (PubMed:33909989). Non-canonical autophagy plays a key role in epithelial cells to limit lethal infection by influenza A (IAV) virus (By similarity). Regulates mitochondrial antiviral signaling (MAVS)-dependent type I interferon (IFN-I) production (PubMed:22749352, PubMed:25645662). Negatively regulates NOD1- and NOD2-driven inflammatory cytokine response (PubMed:24238340). Instead, promotes an autophagy-dependent antibacterial pathway together with NOD1 or NOD2 (PubMed:20637199). Plays a role in regulating morphology and function of Paneth cell (PubMed:18849966).

Cellular Location
Cytoplasm. Preautophagosomal structure membrane; Peripheral membrane protein. Endosome membrane; Peripheral membrane protein. Lysosome membrane; Peripheral membrane protein. Note=Recruited to omegasomes membranes by WIPI2 (By similarity). Omegasomes are endoplasmic reticulum connected strutures at the origin of preautophagosomal structures (By similarity) Localized to preautophagosomal structure (PAS) where it is involved in the membrane targeting of ATG5 (By similarity). Localizes also to discrete punctae along the ciliary axoneme (By similarity). Upon activation of non-canonical autophagy, recruited to single-membrane endolysosomal compartments (PubMed:29317426) {ECO:0000250|UniProtKB:Q8C0J2, ECO:0000269|PubMed:29317426}

ATG16L Antibody Blocking peptide - Protocols

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

- Blocking Peptides

ATG16L Antibody Blocking peptide - Images

ATG16L 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). The APG12-APG5-APG16L complex is esential for the elongation of autophagic isolation membranes. This complex initially associates in uniform distribution with small vesicle membranes. During membrane elongation, the complex partitions, with a great concentration building on the outer side of the isolation membrane. Upon completion of the formation of the autophagosome, the APG12-APG5-APG16L dissociates from the membrane.

ATG16L 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)