

ATG5 Antibody (C-term) Blocking peptide
Synthetic peptide
Catalog # BP1812b**Specification**

ATG5 Antibody (C-term) Blocking peptide - Product InformationPrimary Accession [Q9H1Y0](#)**ATG5 Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 9474**Other Names**

Autophagy protein 5, APG5-like, Apoptosis-specific protein, ATG5, APG5L, ASP

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1812b](/product/products/AP1812b) was selected from the C-term region of human Autophagy APG5L. 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.

ATG5 Antibody (C-term) Blocking peptide - Protein Information**Name** ATG5 ([HGNC:589](#))**Synonyms** APG5L, ASP**Function**

Involved in autophagic vesicle formation. Conjugation with ATG12, through a ubiquitin-like conjugating system involving ATG7 as an E1-like activating enzyme and ATG10 as an E2-like conjugating enzyme, is essential for its function. The ATG12-ATG5 conjugate acts as an E3-like enzyme which is required for lipidation of ATG8 family proteins and their association to the vesicle membranes. Involved in mitochondrial quality control after oxidative damage, and in subsequent cellular longevity. Plays a critical role in multiple aspects of lymphocyte development and is essential for both B and T lymphocyte survival and proliferation. Required for optimal processing and presentation of antigens for MHC II. Involved in the maintenance of axon morphology and membrane structures, as well as in normal adipocyte differentiation. Promotes primary ciliogenesis through removal of OFD1 from centriolar satellites and degradation of IFT20 via the autophagic

pathway.

Cellular Location

Cytoplasm. Preautophagosomal structure membrane; Peripheral membrane protein
Note=Colocalizes with nonmuscle actin. The conjugate detaches from the membrane immediately before or after autophagosome formation is completed (By similarity). Localizes also to discrete punctae along the ciliary axoneme and to the base of the ciliary axoneme.

Tissue Location

Ubiquitous. The mRNA is present at similar levels in viable and apoptotic cells, whereas the protein is dramatically highly expressed in apoptotic cells

ATG5 Antibody (C-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

ATG5 Antibody (C-term) Blocking peptide - Images

ATG5 Antibody (C-term) 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). APG5, required for autophagy, conjugates to ATG12 and associates with an isolation membrane to form a cup-shaped isolation membrane and autophagosome. The conjugate detaches from the membrane immediately before or after autophagosome formation is completed. APG5 may also play an important role in the apoptotic process, possibly within the modified cytoskeleton. Its expression is a relatively late event in the apoptotic process, occurring downstream of caspase activity.

ATG5 Antibody (C-term) 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)Hammond E.M., et al. FEBS Lett. 425:391-395(1998) Strausberg R.L., et al. PNAS 99:16899-16903(2002)Grand R.J.A., et al. Exp. Cell Res. 218:439-451(1995)Mizushima N., et al. J. Biol. Chem. 273:33889-33892(1998)Mizushima N., et al. J. Cell Biol. 152:657-668(2001)