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ATG7 Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP1813a

Specification

ATG7 Antibody (N-term) Blocking peptide - Product Information

Primary Accession O95352

ATG7 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 10533

Other Names
Ubiquitin-like modifier-activating enzyme ATG7, ATG12-activating enzyme E1 ATG7, Autophagy-related protein 7, APG7-like, hAGP7, Ubiquitin-activating enzyme E1-like protein, ATG7, APG7L

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP1813a was selected from the N-term region of human Autophagy APG7L. 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.

ATG7 Antibody (N-term) Blocking peptide - Protein Information

Name ATG7 (HGNC:16935)

Synonyms APG7L

Function
E1-like activating enzyme involved in the 2 ubiquitin-like systems required for cytoplasm to vacuole transport (Cvt) and autophagy. Activates ATG12 for its conjugation with ATG5 as well as the ATG8 family proteins for their conjugation with phosphatidylethanolamine. Both systems are needed for the ATG8 association to Cvt vesicles and autophagosomes membranes. Required for autophagic death induced by caspase-8 inhibition. Facilitates LC3-I lipidation with phosphatidylethanolamine to form LC3-II which is found on autophagosomal membranes (PubMed:34161705). Required for mitophagy which contributes to regulate mitochondrial quantity and quality by

eliminating the mitochondria to a basal level to fulfill cellular energy requirements and preventing excess ROS production. Modulates p53/TP53 activity to regulate cell cycle and survival during metabolic stress. Also plays a key role in the maintenance of axonal homeostasis, the prevention of axonal degeneration, the maintenance of hematopoietic stem cells, the formation of Paneth cell granules, as well as in adipose differentiation. Plays a role in regulating the liver clock and glucose metabolism by mediating the autophagic degradation of CRY1 (clock repressor) in a time-dependent manner (By similarity).

Cellular Location
Cytoplasm. Preautophagosomal structure. Note=Localizes also to discrete punctae along the ciliary axoneme and to the base of the ciliary axoneme

Tissue Location
Widely expressed, especially in kidney, liver, lymph nodes and bone marrow.

ATG7 Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

ATG7 Antibody (N-term) Blocking peptide - Images

ATG7 Antibody (N-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). APG7 functions as an E1 enzyme essential for multisubstrates such as GABARAPL1 and ATG12. APG3L is an E2-like conjugating enzyme facilitating covalent binding of APG8 (MAP1LC3) to phosphatidylethanolamine (PE). APG7 (an E1-like enzyme) facilitates this reaction by forming an E1-E2 complex with APG3. Formation of the PE conjugate is essential for autophagy.

ATG7 Antibody (N-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)Tanida I., et al. Biochem. Biophys. Res. Commun. 292:256-262(2002) Tanida I., et al. J. Biol. Chem. 277:13739-13744(2002)