

APG4A Antibody (Center) Blocking Peptide
Synthetic peptide
Catalog # BP1808b**Specification**

APG4A Antibody (Center) Blocking Peptide - Product InformationPrimary Accession [Q8WYN0](#)**APG4A Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 115201**Other Names**

Cysteine protease ATG4A, 3422-, AUT-like 2 cysteine endopeptidase, Autophagin-2, Autophagy-related cysteine endopeptidase 2, Autophagy-related protein 4 homolog A, hAPG4A, ATG4A, APG4A, AUTL2

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1808b](/product/products/AP1808b) was selected from the Center region of human Autophagy APG4A. 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.

APG4A Antibody (Center) Blocking Peptide - Protein Information**Name** ATG4A {ECO:0000303|Ref.20, ECO:0000312|HGNC:HGNC:16489}**Function**

Cysteine protease that plays a key role in autophagy by mediating both proteolytic activation and delipidation of ATG8 family proteins (PubMed:[12473658](http://www.uniprot.org/citations/12473658), PubMed:[15169837](http://www.uniprot.org/citations/15169837), PubMed:[17347651](http://www.uniprot.org/citations/17347651), PubMed:[21177865](http://www.uniprot.org/citations/21177865), PubMed:[21245471](http://www.uniprot.org/citations/21245471), PubMed:[22302004](http://www.uniprot.org/citations/22302004), PubMed:[32732290](http://www.uniprot.org/citations/32732290)). The protease activity is required for proteolytic activation of ATG8 family proteins: cleaves the C-terminal amino

acid of ATG8 proteins to reveal a C-terminal glycine (PubMed:12473658, PubMed:15169837, PubMed:17347651, PubMed:21177865, PubMed:21245471, PubMed:22302004). 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:12473658, PubMed:15169837, PubMed:17347651, PubMed:21177865, PubMed:21245471, PubMed:22302004). Preferred substrate is GABARAPL2 followed by MAP1LC3A and GABARAP (PubMed:12473658, PubMed:15169837, PubMed:17347651, PubMed:21177865, PubMed:21245471, 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:29458288, PubMed:33909989). Catalyzes delipidation of PE- conjugated forms of ATG8 proteins during macroautophagy (PubMed:29458288, PubMed:33909989). Compared to ATG4B, the major protein for proteolytic activation of ATG8 proteins, shows weaker ability to cleave the C-terminal amino acid of ATG8 proteins, while it displays stronger delipidation activity (PubMed:29458288). 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 {ECO:0000250|UniProtKB:Q8BGE6}.

APG4A Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

APG4A Antibody (Center) Blocking Peptide - Images

APG4A Antibody (Center) 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). APG4A 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. Preferred substrate is GABARAPL2 followed by MAP1LC3A and GABARAP.

APG4A Antibody (Center) 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)