

APG4C Antibody (S166) Blocking Peptide
Synthetic peptide
Catalog # BP1810e**Specification**

APG4C Antibody (S166) Blocking Peptide - Product InformationPrimary Accession [Q96DT6](#)**APG4C Antibody (S166) Blocking Peptide - Additional Information**

Gene ID 84938

Other Names

Cysteine protease ATG4C, 3422-, AUT-like 3 cysteine endopeptidase, Autophagin-3, Autophagy-related cysteine endopeptidase 3, Autophagy-related protein 4 homolog C, ATG4C, APG4C, AUTL1, AUTL3

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1810e](/products/AP1810e) was selected from the S166 region of human APG4C. 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.

APG4C Antibody (S166) Blocking Peptide - Protein Information**Name** ATG4C {ECO:0000303|PubMed:21177865, ECO:0000312|HGNC:HGNC:16040}**Function**

Cysteine protease that plays a key role in autophagy by mediating both proteolytic activation and delipidation of ATG8 family proteins (PubMed:[21177865](http://www.uniprot.org/citations/21177865), PubMed:[29458288](http://www.uniprot.org/citations/29458288), PubMed:[30661429](http://www.uniprot.org/citations/30661429)). The protease activity is required for proteolytic activation of ATG8 family proteins: cleaves the C-terminal amino acid of ATG8 proteins MAP1LC3 and GABARAPL2, to reveal a C-terminal glycine (PubMed:[21177865](http://www.uniprot.org/citations/21177865)). 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 (By

similarity). 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). In contrast to other members of the family, weakly or not involved in phagophore growth during mitophagy (PubMed:33773106).

Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:Q8BGE6}.

APG4C Antibody (S166) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

APG4C Antibody (S166) Blocking Peptide - Images

APG4C Antibody (S166) Blocking Peptide - Background

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. 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).

APG4C Antibody (S166) 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)