

ATG4C Antibody
Mouse Monoclonal Antibody (Mab)
Catalog # AM1933b**Specification**

ATG4C Antibody - Product Information

Application	WB,E
Primary Accession	Q96DT6
Other Accession	NP_116241.2 , NP_835739.1
Reactivity	Mouse
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1,k
Calculated MW	52497

ATG4C Antibody - 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

This ATG4C monoclonal antibody is generated from mouse immunized with ATG4C recombinant protein.

Dilution

WB~~~1:100

E~~~Use at an assay dependent concentration.

Format

Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

ATG4C Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

ATG4C Antibody - 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](#), PubMed:[29458288](#), PubMed:[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](#)). 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

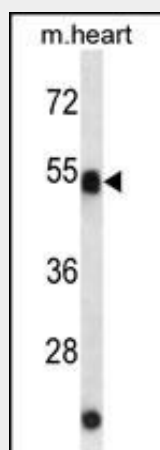
Cytoplasm {ECO:0000250|UniProtKB:Q8BGE6}.

ATG4C Antibody - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

ATG4C Antibody - Images



ATG4C Antibody (Cat. #AM1933b) western blot analysis in mouse heart tissue lysates (35µg/lane). This demonstrates the ATG4C antibody detected the ATG4C protein (arrow).

ATG4C Antibody - Background

Autophagy is the process by which endogenous proteins and damaged organelles are destroyed intracellularly. Autophagy is postulated to be essential for cell homeostasis and cell remodeling during differentiation, metamorphosis, non-apoptotic cell death,

and aging. Reduced levels of autophagy have been described in some malignant tumors, and a role for autophagy in controlling the unregulated cell growth linked to cancer has been proposed. This gene encodes a member of the autophagin protein family. The encoded protein is also designated as a member of the C-54 family of cysteine proteases. Alternate transcriptional splice variants, encoding the same protein, have been characterized. [provided by RefSeq].

ATG4C Antibody - References

Kathiresan, S., et al. Nat. Genet. 40(2):189-197(2008)
Marino, G., et al. J. Biol. Chem. 282(25):18573-18583(2007)
Levy, D., et al. BMC Med. Genet. 8 SUPPL 1, S3 (2007) :
Marino, G., et al. J. Biol. Chem. 278(6):3671-3678(2003)