

ATG4B Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP1809d

Product Information

Application	WB, IHC-P, E
Primary Accession	Q9Y4P1
Other Accession	Q8BGE6 , Q6DG88 , Q6PZ02 , Q6PZ03
Reactivity	Human
Predicted	Bovine, Chicken, Zebrafish, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB11843
Calculated MW	44294
Antigen Region	258-284

Additional Information

Gene ID	23192
Other Names	Cysteine protease ATG4B, 3422-, AUT-like 1 cysteine endopeptidase, Autophagin-1, Autophagy-related cysteine endopeptidase 1, Autophagy-related protein 4 homolog B, hAPG4B, ATG4B, APG4B, AUTL1, KIAA0943
Target/Specificity	This ATG4B antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 258-284 amino acids from human ATG4B.
Dilution	WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
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	ATG4B Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	ATG4B {ECO:0000303 PubMed:15187094, ECO:0000312 HGNC:HGNC:20790}
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Function

Cysteine protease that plays a key role in autophagy by mediating both proteolytic activation and delipidation of ATG8 family proteins (PubMed:[15169837](#), PubMed:[15187094](#), PubMed:[17347651](#), PubMed:[19322194](#), PubMed:[21177865](#), PubMed:[22302004](#), PubMed:[26378241](#), PubMed:[27527864](#), PubMed:[28633005](#), PubMed:[28821708](#), PubMed:[29232556](#), PubMed:[30076329](#), PubMed:[30443548](#), PubMed:[30661429](#)). Required for canonical autophagy (macroautophagy), non-canonical autophagy as well as for mitophagy (PubMed:[33773106](#), PubMed:[33909989](#)). The protease activity is required for proteolytic activation of ATG8 family proteins: cleaves the C-terminal amino acid of ATG8 proteins MAP1LC3A, MAP1LC3B, MAP1LC3C, GABARAPL1, GABARAPL2 and GABARAP, to reveal a C- terminal glycine (PubMed:[15169837](#), PubMed:[15187094](#), PubMed:[17347651](#), PubMed:[19322194](#), PubMed:[20818167](#), PubMed:[21177865](#), PubMed:[22302004](#), PubMed:[27527864](#), PubMed:[28287329](#), PubMed:[28633005](#), PubMed:[29458288](#), PubMed:[30661429](#)). 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:[15169837](#), PubMed:[15187094](#), PubMed:[17347651](#), PubMed:[19322194](#), PubMed:[21177865](#), 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:[15187094](#), PubMed:[19322194](#), PubMed:[28633005](#), PubMed:[29458288](#), PubMed:[32686895](#), PubMed:[33909989](#)). Catalyzes delipidation of PE- conjugated forms of ATG8 proteins during macroautophagy (PubMed:[15187094](#), PubMed:[19322194](#), PubMed:[29458288](#), PubMed:[32686895](#), PubMed:[33909989](#)). Also involved in non-canonical autophagy, a parallel pathway involving conjugation of ATG8 proteins to single membranes at endolysosomal compartments, by catalyzing delipidation of ATG8 proteins conjugated to phosphatidylserine (PS) (PubMed:[33909989](#)). Compared to other members of the family (ATG4A, ATG4C or ATG4D), constitutes the major protein for proteolytic activation of ATG8 proteins, while it displays weaker delipidation activity than other ATG4 paralogs (PubMed:[29458288](#), PubMed:[30661429](#)). 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. Cytoplasm, cytosol. Cytoplasmic vesicle, autophagosome. Endoplasmic reticulum. Mitochondrion. Note=Mainly localizes to the cytoplasm, including cytosol (PubMed:29165041). A small portion localizes to mitochondria; phosphorylation at Ser-34 promotes localization to mitochondria (PubMed:29165041).

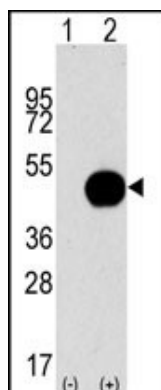
Background

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). ATG4, a cysteine protease required for autophagy, cleaves the C-terminal part of MAP1LC3 to form the activated molecule LC3-I. LC3-I is subsequently conjugated with phosphatidylethanolamine at the C-terminal glycine to form LC3-II, a marker for autophagy via its capacity to bind to autophagosomes.

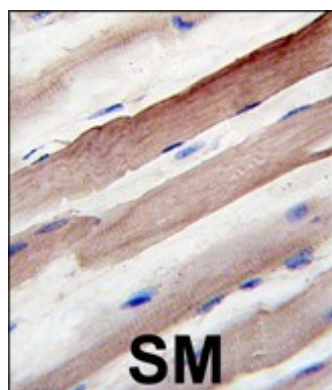
References

Tanida, I., et al., J. Biol. Chem. 279(35):36268-36276 (2004).
Marino, G., et al., J. Biol. Chem. 278(6):3671-3678 (2003).
Kabeya, Y., et al., J. Cell. Sci. 117 (Pt 13), 2805-2812 (2004) (): ().

Images



Western blot analysis of APG4B (arrow) using purified Pab (Cat.#AP1809d). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the APG4B gene (Lane 2) (Origene Technologies).



Formalin-fixed and paraffin-embedded human skeletal muscle reacted with Autophagy APG4B Antibody (E273)(Cat.#AP1809d), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

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