

APG4B Antibody (Center G254)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP19349c**Specification**

APG4B Antibody (Center G254) - Product Information

Application	WB,E
Primary Accession	Q9Y4P1
Other Accession	Q8BGE6 , Q6DG88 , Q6PZ02 , Q6PZ03 , NP_037457 , A0A0G2QC33
Reactivity	Human
Predicted	Bovine, Chicken, Zebrafish, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	44294
Antigen Region	239-265

APG4B Antibody (Center G254) - 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 APG4B antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 239-265 amino acids from the Central region of human APG4B.

Dilution

WB~~1:1000

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

APG4B Antibody (Center G254) is for research use only and not for use in diagnostic or therapeutic procedures.

APG4B Antibody (Center G254) - Protein Information

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

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, GABARAP1, GABARAP2 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

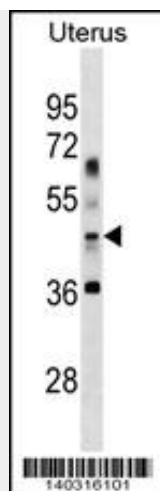
Cytoplasm. Cytoplasm, cytosol. Cytoplasmic vesicle, autophagosome. Endoplasmic reticulum. Mitochondrion. Note=Mainly localizes to the cytoplasm, including cytosol (PubMed:29165041). A sam1l1 protein localizes to mitochondria; phosphorylation at Ser-34 promotes localization to mitochondria (PubMed:29165041).

APG4B Antibody (Center G254) - 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](#)

APG4B Antibody (Center G254) - Images



APG4B Antibody (Center G254)(Cat. #AP19349c) western blot analysis in Uterus tissue lysates (35ug/lane). This demonstrates the APG4B antibody detected the APG4B protein (arrow).

APG4B Antibody (Center G254) - 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 different isoforms, have been characterized. [provided by RefSeq].

APG4B Antibody (Center G254) - References

Satoo, K., et al. EMBO J. 28(9):1341-1350(2009) Sugiyama, N., et al. Mol. Cell Proteomics 6(6):1103-1109(2007) Ewing, R.M., et al. Mol. Syst. Biol. 3, 89 (2007) : Olsen, J.V., et al. Cell 127(3):635-648(2006) Olsen, J.V., et al. Cell 127(3):635-648(2006)