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ATG4A Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1808b

Product Information

Application	WB, IHC-P, E
Primary Accession	Q8WYN0
Reactivity	Human
Predicted	Mouse, Rat, Bovine, Canine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	45378
Antigen Region	82-111

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	This ATG4A antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 82-111 amino acids from human ATG4A.
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 prepared by Saturated Ammonium Sulfate (SAS) precipitation 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	ATG4A Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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, PubMed:15169837, PubMed:17347651,

PubMed:21177865, PubMed:21245471, PubMed:22302004, PubMed: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}.

Background

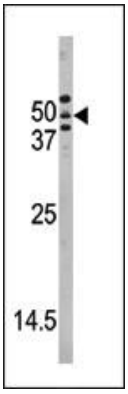
Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes 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.

References

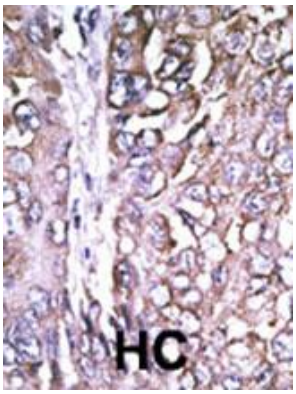
Baehrecke EH. Nat Rev Mol Cell Biol. 6(6):505-10. (2005)
Lum JJ, et al. Nat Rev Mol Cell Biol. 6(6):439-48. (2005)
Greenberg JT. Dev Cell. 8(6):799-801. (2005)
Levine B. Cell. 120(2):159-62. (2005)
Shintani T and Klionsky DJ. Science. 306(5698):990-5. (2004)

Images

Western blot analysis of anti-APG4A Pab (Cat. #AP1808b) in HepG2 cell line lysate. APG4A(arrow) was detected



using the purified Pab.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Citations

- Kinetics comparisons of mammalian Atg4 homologues indicate selective preferences toward diverse Atg8 substrates.
- MicroRNA-144-3p inhibits autophagy activation and enhances Bacillus Calmette-Guérin infection by targeting ATG4a in RAW264.7 macrophage cells.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.