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Atg4A Antibody

Rabbit mAb
Catalog # AP91481

Product Information

Application	WB, IHC, IP
Primary Accession	Q8WYN0
Reactivity	Rat, Human, Mouse
Clonality	Monoclonal
Other Names	Apg4a; ATG4A; Atg4al; AUT like 2 cysteine endopeptidase; Autl2; Autophagin 2; Autophagin-2; Autophagy related 4A cysteine peptidase;
Isotype	Rabbit IgG
Host	Rabbit
Calculated MW	45378

Additional Information

Dilution	WB 1:500~1:2000 IHC 1:50~1:200 IP 1:50
Purification	Affinity-chromatography
Immunogen	A synthesized peptide derived from human Atg4A
Description	The cysteine protease Atg4 is pivotal to autophagosome membrane generation and regulation. Atg4 primes the Atg8 homologue for lipidation by cleaving its carboxy terminus and exposing its glycine residue for E1-like enzyme Atg7. The Atg8 homologue is transferred to the E2-like enzyme Atg3 before forming the Atg8-PE conjugate. During later stages of autophagy, Atg4 can reverse this lipidation event by cleaving PE, thereby recycling the Atg8 homologue.
Storage Condition and Buffer	Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle.

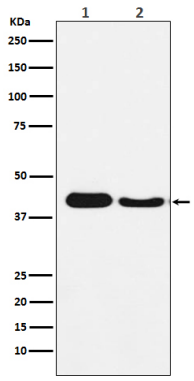
Protein Information

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}.

Images



Western blot analysis of Atg4A expression in (1) HepG2 cell lysate; (2) Mouse brain lysate.

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