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ATG16L Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1817b

Product Information

Application	WB, IHC-P, IF, E
Primary Accession	Q676U5
Reactivity	Human, Mouse
Predicted	Rat, Bovine, Canine, Rabbit, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	68265
Antigen Region	161-190

Additional Information

Gene ID	55054
Other Names	Autophagy-related protein 16-1, APG16-like 1, ATG16L1, APG16L
Target/Specificity	This ATG16L antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 161-190 amino acids from human ATG16L.
Dilution	WB~~1:1000 IHC-P~~1:100~500 IF~~1:25 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	ATG16L Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	ATG16L1 {ECO:0000303|PubMed:17200669, ECO:0000312|HGNC:HGNC:21498}
Function	Plays an essential role in both canonical and non-canonical autophagy: interacts with ATG12-ATG5 to mediate the lipidation to ATG8 family proteins (MAP1LC3A, MAP1LC3B, MAP1LC3C, GABARAPL1, GABARAPL2 and GABARAP)

(PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904, PubMed:27273576, PubMed:29317426, PubMed:30778222, PubMed:33909989). Acts as a molecular hub, coordinating autophagy pathways via distinct domains that support either canonical or non- canonical signaling (PubMed:29317426, PubMed:30778222). During canonical autophagy, interacts with ATG12-ATG5 to mediate the conjugation of phosphatidylethanolamine (PE) to ATG8 proteins, to produce a membrane-bound activated form of ATG8 (PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904, PubMed:27273576). Thereby, controls the elongation of the nascent autophagosomal membrane (PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904, PubMed:27273576). As part of the ATG8 conjugation system with ATG5 and ATG12, required for recruitment of LRRK2 to stressed lysosomes and induction of LRRK2 kinase activity in response to lysosomal stress (By similarity). Also involved in non-canonical autophagy, a parallel pathway involving conjugation of ATG8 proteins to single membranes at endolysosomal compartments, probably by catalyzing conjugation of phosphatidylserine (PS) to ATG8 (PubMed:33909989). Non-canonical autophagy plays a key role in epithelial cells to limit lethal infection by influenza A (IAV) virus (By similarity). Regulates mitochondrial antiviral signaling (MAVS)-dependent type I interferon (IFN-I) production (PubMed:22749352, PubMed:25645662). Negatively regulates NOD1- and NOD2-driven inflammatory cytokine response (PubMed:24238340). Instead, promotes an autophagy-dependent antibacterial pathway together with NOD1 or NOD2 (PubMed:20637199). Plays a role in regulating morphology and function of Paneth cell (PubMed:18849966).

Cellular Location

Cytoplasm. Preautophagosomal structure membrane; Peripheral membrane protein. Endosome membrane; Peripheral membrane protein. Lysosome membrane; Peripheral membrane protein. Note=Recruited to omegasomes membranes by WIPI2 (By similarity). Omegasomes are endoplasmic reticulum connected strutures at the origin of preautophagosomal structures (By similarity). Localized to preautophagosomal structure (PAS) where it is involved in the membrane targeting of ATG5 (By similarity). Also localizes to discrete punctae along the ciliary axoneme (By similarity). Upon activation of non-canonical autophagy, recruited to single-membrane endolysosomal compartments (PubMed:29317426). Under starved conditions, the ATG12-ATG5-ATG16L1 complex is translocated to phagophores driven by RAB33B (PubMed:32960676). {ECO:0000250|UniProtKB:Q8C0J2, ECO:0000269|PubMed:29317426, ECO:0000269|PubMed:32960676}

Background

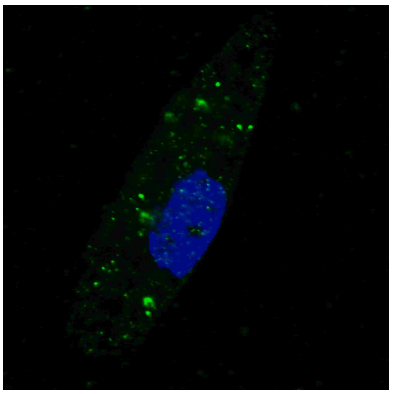
Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes 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). The APG12-APG5-APG16L complex is esential for the elongation of autophagic isolation membranes. This complex initially associates in uniform distribution with small vesicle membranes. During membrane elongation, the complex partitions, with a great concentration building on the outer side of the isolation membrane. Upon completion of the formation of the autophagosome, the APG12-APG5-APG16L dissociates from the membrane.

References

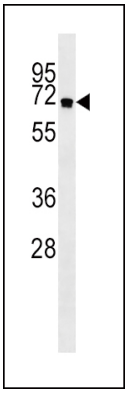
References for protein:
1.Baehrecke EH. Nat Rev Mol Cell Biol. 6(6):505-10. (2005)

2.Lum JJ, et al. Nat Rev Mol Cell Biol. 6(6):439-48. (2005)
3.Greenberg JT. Dev Cell. 8(6):799-801. (2005)
4.Levine B. Cell. 120(2):159-62. (2005)
5.Shintani T and Klionsky DJ. Science. 306(5698):990-5. (2004)
References for U251 cell line:
1. Westermark B.; Pontén J.; Hugosson R. (1973).” Determinants for the establishment of permanent tissue culture lines from human gliomas”. Acta Pathol Microbiol Scand A. 81:791-805. [PMID: 4359449].
2. Pontén, J.,Westermark B. (1978).” Properties of Human Malignant Glioma Cells in Vitro”. Medical Biology 56: 184-193.[PMID: 359950].
3. Geng Y.;Kohli L.; Klocke B.J.; Roth K.A.(2010). “Chloroquine-induced autophagic vacuole accumulation and cell death in glioma cells is p53 independent”. Neuro Oncol. 12(5): 473–481.[PMID: 20406898].

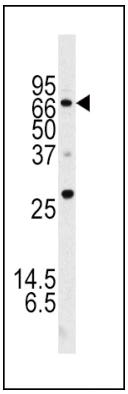
Images



Fluorescent image of U251 cells stained with ATG16L antibody. U251 cells were treated with Chloroquine (50 µM,16h), then fixed with 4% PFA (20 min), permeabilized with Triton X-100 (0.2%, 30 min). Cells were then incubated with AP1817b ATG16L primary antibody (1:200, 2 h at room temperature). For secondary antibody, Alexa Fluor® 488 conjugated donkey anti-rabbit antibody (green) was used (1:1000, 1h). Nuclei were counterstained with Hoechst 33342 (blue) (10 µg/ml, 5 min). ATG16L immunoreactivity is localized to autophagic vacuoles in the cytoplasm of U251 cells, supported by Human Protein Atlas Data (http://www.proteinatlas.org/ENSG00000085978).

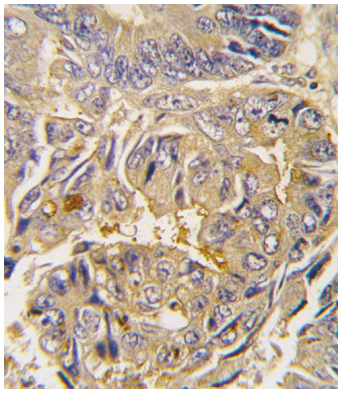


APG16L Antibody (Cat. #AP1817b) western blot analysis in NCI-H460 cell line lysates (35ug/lane).This demonstrates the APG16L antibody detected the APG16L protein (arrow).

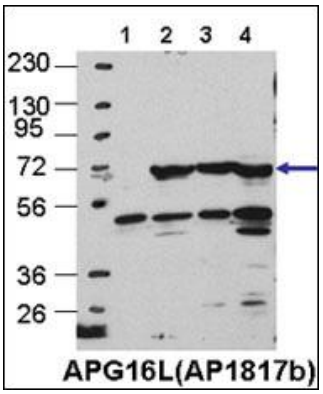


Western blot of APG16L (L92) Pab (Cat. #AP1817b) in mouse brain tissue lysate.

Formalin-fixed and paraffin-embedded human colon carcinoma tissue reacted with Autophagy APG16L antibody (L176), 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.



Cos7, HEK293, MEF, and Hela cells, left to right, respectively. Data courtesy of Drs. Jiefei Geng and Dan Klionsky, University of Michigan.

Citations

- A non-canonical autophagy-dependent role of the ATG16L1 variant in urothelial vesicular trafficking and uropathogenic Escherichia coli persistence
- Autophagy induction during stem cell activation plays a key role in salivary gland self-renewal
- Integrative analysis of Paneth cell proteomic and transcriptomic data from intestinal organoids reveals functional processes dependent on autophagy.
- RNA binding protein HuR promotes autophagosome formation by regulating expressions of autophagy-related protein 5, 12, and 16 in human hepatocellular carcinoma cells.
- Overexpression of the autophagic beclin-1 protein clears mutant ataxin-3 and alleviates Machado-Joseph disease.
- Nod1 and Nod2 direct autophagy by recruiting ATG16L1 to the plasma membrane at the site of bacterial entry.
- Overexpression and altered subcellular localization of autophagy-related 16-like 1 in human oral squamous-cell carcinoma: correlation with lymphovascular invasion and lymph-node metastasis.
- Intestinal epithelial vitamin D receptor deletion leads to defective autophagy in colitis.
- Atg16L1 deficiency confers protection from uropathogenic Escherichia coli infection in vivo.
- Elevated p62/SQSTM1 determines the fate of autophagy-deficient neural stem cells by increasing superoxide.
- TRIM16 controls assembly and degradation of protein aggregates by modulating the p62-NRF2 axis and autophagy.
- 1,25(OH)2 D3 attenuates hepatic steatosis by inducing autophagy in mice.
- Intraflagellar transport protein IFT20 is essential for male fertility and spermiogenesis in mice.

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