

ATG16L1 Antibody

Purified Mouse Monoclonal Antibody (Mab) Catalog # AM1817b

Product Information

Application WB, IHC-P, E **Primary Accession** Q676U5 Reactivity Human Host Mouse Clonality Monoclonal Isotype IgG1,k **Clone Names** 54CT27.2.6 Calculated MW 68265

Additional Information

Gene ID 55054

Other Names Autophagy-related protein 16-1, APG16-like 1, ATG16L1, APG16L

Target/Specificity Purified His-tagged ATG16L1 protein(Fragment) was used to produced this

monoclonal antibody.

Dilution WB~~1:1000 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.

Format Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide.

This antibody is purified through a protein G column, 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 ATG16L1 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:<u>23376921</u>, PubMed:<u>23392225</u>, PubMed:<u>24553140</u>,

PubMed:<u>24954904</u>, PubMed:<u>27273576</u>, PubMed:<u>29317426</u>,

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: <u>27273576</u>). Thereby, controls the elongation of the nascent autophagosomal membrane (PubMed:23376921, PubMed:23392225, PubMed:<u>24553140</u>, PubMed:<u>24954904</u>, PubMed:<u>27273576</u>). 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:32960676}

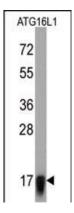
Background

The protein encoded by this gene is part of a large protein complex that is necessary for autophagy, the major process by which intracellular components are targeted to lysosomes for degradation. Defects in this gene are a cause of susceptibility to inflammatory bowel disease type 10 (IBD10). Several transcript variants encoding different isoforms have been found for this gene.

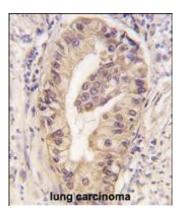
References

Age at onset in Huntington's disease is modified by the autophagy pathway: implication of the V471A polymorphism in Atg7. Metzger S, et al. Hum Genet, 2010 Oct. PMID 20697744. Plasma membrane contributes to the formation of pre-autophagosomal structures. Ravikumar B, et al. Nat Cell Biol, 2010 Aug. PMID 20639872. Replication and meta-analysis of 13,000 cases defines the risk for interleukin-23 receptor and autophagy-related 16-like 1 variants in Crohn's disease. Cotterill L, et al. Can J Gastroenterol, 2010 May. PMID 20485703. Is there a role for Crohn's disease-associated autophagy genes ATG16L1 and IRGM in formation of granulomas? Wolfkamp SC, et al. Eur J Gastroenterol Hepatol, 2010 Aug. PMID 20395867. NOD2/CARD15, ATG16L1 and IL23R gene polymorphisms and childhood-onset of Crohn's disease. Gazouli M, et al. World J Gastroenterol, 2010 Apr 14. PMID 20380008.

Images



Western blot analysis of anti-ATG16L1 Monoclonal Antibody (Cat.#AM1817b) by ATG16L1 recombinant protein (Fragment). ATG16L1 (Fragment) protein (arrow) was detected using the purified Mab. (1:2000)



Formalin-fixed and paraffin-embedded human lung carcinoma tissue reacted with ATG16L1 Monoclonal Antibody (Cat.#AM1817b), 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.

Citations

- <u>Deficient tRNA posttranscription modification dysregulated the mitochondrial quality controls and apoptosis</u>
- Autophagy in cancer associated fibroblasts promotes tumor cell survival: Role of hypoxia, HIF1 induction and NFkB activation in the tumor stromal microenvironment.

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