

Atg16L1 Antibody

Rabbit mAb Catalog # AP91339

Product Information

Application WB, IHC Primary Accession Q676U5

Reactivity Rat, Human, Mouse

Clonality Monoclonal

Other Names APG16 like 1; APG16L; APG16L beta; ATG16A; ATG16L; Atg16l1; IBD10; WD

repeat domain 30; WDR30;

IsotypeRabbit IgGHostRabbitCalculated MW68265

Additional Information

Dilution WB 1:500~1:2000 IHC 1:50~1:200

Purification Affinity-chromatography

Immunogen A synthesized peptide derived from human Atg16L1

Description Plays an essential role in autophagy: interacts with ATG12-ATG5 to mediate

the conjugation of phosphatidylethanolamine (PE) to LC3 (MAP1LC3A, MAP1LC3B or MAP1LC3C), to produce a membrane-bound activated form of

LC3 named LC3-II. Thereby, controls the elongation of the nascent

autophagosomal membrane.

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 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

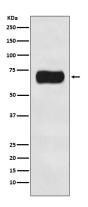
of phosphatidylethanolamine (PE) to ATG8 proteins, to produce a membrane-bound activated form of ATG8 (PubMed:23376921, PubMed:23392225, PubMed:24553140, PubMed:24954904,

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

Images



Western blot analysis of Atg16L1 expression in HeLa cell lysate.

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