

Phospho-ATG16L(S287) Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP3366a

Product Information

Application WB, DB, E **Primary Accession Q676U5** Reactivity Human Host Rabbit Clonality Polyclonal Isotype Rabbit IgG **Clone Names** RB11878 Calculated MW 68265

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 phosphopeptide corresponding to amino acid residues

surrounding S287 of human ATG16L.

Dilution WB~~1:1000 DB~~1:500 E~~Use at an assay dependent concentration.

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

This antibody is purified through a protein A column, followed by peptide

affinity purification.

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 Phospho-ATG16L(S287) 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: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}

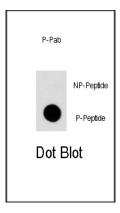
Background

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 essential 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

Zheng H., DNA Seq. 15:303-305(2004). Clark H.F., Genome Res. 13:2265-2270(2003). Ota T., Nat. Genet. 36:40-45(2004).

Images



Dot blot analysis of Phospho-APG16L-S287 Antibody (Cat.#AP3366a) on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

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