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LC3B Antibody

Rabbit mAb
Catalog # AP90990

Product Information

Application	WB, IHC, IF, ICC, IP, IHF
Primary Accession	Q9GZQ8
Reactivity	Rat, Human, Mouse
Clonality	Monoclonal
Other Names	ATG8F; LC3B; MAP1LC3B; MLP3B; MAP1LC3B;
Isotype	Rabbit IgG
Host	Rabbit
Calculated MW	14688

Additional Information

Dilution	WB 1:500~1:2000 ICC/IF 1:50~1:200 IP 1:50
Purification	Affinity-chromatography
Immunogen	A synthesized peptide derived from human LC3B
Description	Ubiquitin-like modifier involved in formation of autophagosomal vacuoles (autophagosomes). Plays a role in mitophagy which contributes to regulate mitochondrial quantity and quality by eliminating the mitochondria to a basal level to fulfill cellular energy requirements and preventing excess ROS production.
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	MAP1LC3B (HGNC:13352)
Synonyms	MAP1ALC3
Function	Ubiquitin-like modifier involved in formation of autophagosomal vacuoles (autophagosomes) (PubMed:20418806, PubMed:23209295, PubMed:28017329). Plays a role in mitophagy which contributes to regulate mitochondrial quantity and quality by eliminating the mitochondria to a basal level to fulfill cellular energy requirements and preventing excess ROS production (PubMed:23209295, PubMed:28017329). In response to cellular stress and upon mitochondria fission, binds C-18 ceramides and anchors autophagolysosomes to outer mitochondrial membranes to eliminate damaged mitochondria (PubMed:22922758). While LC3s are involved in elongation of the phagophore membrane, the GABARAP/GATE-16 subfamily is essential for a later stage in autophagosome maturation (PubMed:20418806, PubMed:23209295, PubMed:28017329). Promotes primary ciliogenesis by

removing OFD1 from centriolar satellites via the autophagic pathway (PubMed:24089205). Through its interaction with the reticulophagy receptor TEX264, participates in the remodeling of subdomains of the endoplasmic reticulum into autophagosomes upon nutrient stress, which then fuse with lysosomes for endoplasmic reticulum turnover (PubMed:31006537, PubMed:31006538). Upon nutrient stress, directly recruits cofactor JMY to the phagophore membrane surfaces and promotes JMY's actin nucleation activity and autophagosome biogenesis during autophagy (PubMed:30420355).

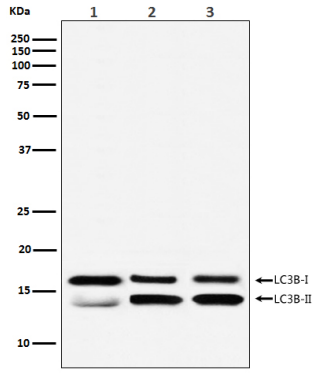
Cellular Location

Cytoplasmic vesicle, autophagosome membrane; Lipid-anchor Endomembrane system; Lipid-anchor Mitochondrion membrane; Lipid-anchor. Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:Q9CQV6}. Cytoplasmic vesicle. Note=LC3-II binds to the autophagic membranes. LC3-II localizes with the mitochondrial inner membrane during Parkin-mediated mitophagy (PubMed:28017329). Also localizes to discrete punctae along the ciliary axoneme

Tissue Location

Most abundant in heart, brain, skeletal muscle and testis. Little expression observed in liver

Images



Western blot analysis of LC3B expression in (1) Human brain lysate; (2) RAW 264.7 cell lysate; (3) C6 cell lysate.

Image not found : 202311/AP90990-IF.jpg

Immunofluorescent analysis of Hela cells treated with choroquine, using LC3B Antibody.

Image not found : 202311/AP90990-IHC.jpg

Prostaglandin E1 Inhibited Diabetes-Induced Phenotypic Switching of Vascular Smooth Muscle Cells Through Activating Autophagy. -Cellular Physiology and Biochemistry

Image not found : 202311/AP90990-wb5.jpg

SIRT 5-mediated deacetylation of LDHB promotes autophagy and tumorigenesis in colorectal cancer. -Molecular Oncology

Image not found : 202311/AP90990-wb6.jpg

Prostaglandin E1 Inhibited Diabetes-Induced Phenotypic Switching of Vascular Smooth Muscle Cells Through Activating Autophagy. -Cellular Physiology and Biochemistry

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