10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Cleaved LC3B Antibody

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP1806a

Product Information

Application	IF, ICC, E
Primary Accession	Q9GZQ8
Other Accession	O41515
Reactivity	Human, Mouse
Predicted	Bovine, Rat, Canine, Rabbit, Chicken
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	14688
Antigen Region	89-122

Additional Information

Gene ID	81631
Other Names	Microtubule-associated proteins 1A/1B light chain 3B, Autophagy-related protein LC3 B, Autophagy-related ubiquitin-like modifier LC3 B, MAP1 light chain 3-like protein 2, MAP1A/MAP1B light chain 3 B, MAP1A/MAP1B LC3 B, Microtubule-associated protein 1 light chain 3 beta, MAP1LC3B, MAP1ALC3
Target/Specificity	This Cleaved LC3B antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 89-122 amino acids from human Cleaved LC3B.
Dilution	IF~~1:10~50 ICC~~1:10~50 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	Cleaved LC3B Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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

Background

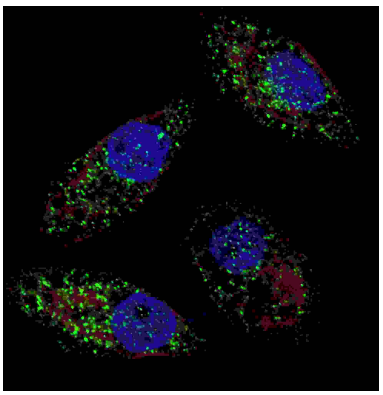
Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes 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). MAP1A and MAP1B are microtubule-associated proteins which mediate the physical interactions between microtubules and components of the cytoskeleton. These proteins are involved in formation of autophagosomal vacuoles (autophagosomes). MAP1A and MAP1B each consist of a heavy chain subunit and multiple light chain subunits. MAP1LC3b is one of the light chain subunits and can associate with either MAP1A or MAP1B. The precursor molecule is cleaved by APG4B/ATG4B to form the cytosolic form, LC3-I. This is activated by APG7L/ATG7, transferred to ATG3 and conjugated to phospholipid to form the membrane-bound form, LC3-II.

References

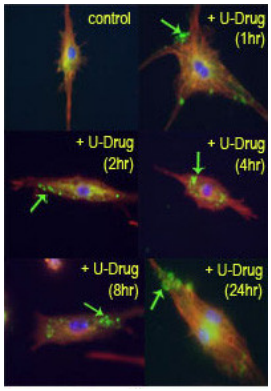
References for protein:
1.Baehrecke EH. Nat Rev Mol Cell Biol. 6(6):505-10. (2005)
2.Lum JJ, et al. Nat Rev Mol Cell Biol. 6(6):439-48. (2005)
3.Greenberg JT. Dev Cell. 8(6):799-801. (2005)
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5.Shintani T and Klionsky DJ. Science. 306(5698):990-5. (2004)
6.Tanida I., et al. Int. J. Biochem. Cell Biol. 36:2503-2518(2004)

7.He H., et al. J. Biol. Chem. 278:29278-29287(2003)
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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].

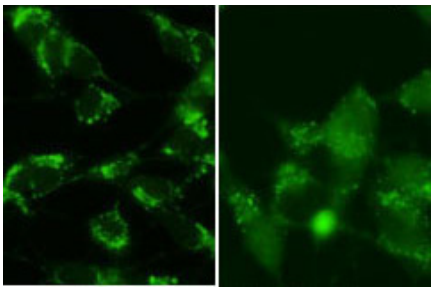
Images



Fluorescent image of U251 cells stained with cleaved LC3B 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 AP1806a cleaved LC3B primary antibody (1:100, 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). LC3 immunoreactivity is localized to autophagic vacuoles in the cytoplasm of U251 cells.



Time course study of mouse leukaemic monocyte macrophage cells treated with U18666A, a drug that causes cholesterol and lipid storage in cells, thereby blocking fusion between late endosomes and lysosomes. Cleaved-LC3 (APG8b) antibody (Cat AP1806a) detected punctuate staining indicative of autophagic vacuole or phagosome structures. Data courtesy of Dr. Barry Boland, Department of Pharmacology, Oxford University.



SY5Y cells were pretreated with 5nM bafilomycin for 24hr and fixed in methanol (left panel) or 4% of paraformaldehyde (right panel). Treatment with Cat# AP1806a antibody at dilution 1:100. Data courtesy of Jianhui Zhu, MD, PhD & Charleen T. Chu, MD, PhD, University of Pittsburgh School of Medicine.

Citations

- Lithium chloride ameliorated spatial cognitive impairment through activating mTOR phosphorylation and inhibiting excessive autophagy in the repeated cerebral ischemia-reperfusion mouse model
- The gluttonous side of malignant melanoma: basic and clinical implications of macroautophagy.
- Regulation of the autophagy protein LC3 by phosphorylation.
- ISG20L1 is a p53 family target gene that modulates genotoxic stress-induced autophagy.
- Autophagy enhances the presentation of endogenous viral antigens on MHC class I molecules during HSV-1 infection.
- Inhibition of the host translation shutoff response by herpes simplex virus 1 triggers nuclear envelope-derived

autophagy.

- EGFR overexpressing cells and tumors are dependent on autophagy for growth and survival.
- Prodigiosin stimulates endoplasmic reticulum stress and induces autophagic cell death in glioblastoma cells.
- L-3-n-Butylphthalide Activates Akt/mTOR Signaling, Inhibits Neuronal Apoptosis and Autophagy and Improves Cognitive Impairment in Mice with Repeated Cerebral Ischemia-Reperfusion Injury.
- Mitigated NSAID-induced apoptotic and autophagic cell death with Smad7 overexpression.
- Genetic ablation or pharmacologic inhibition of autophagy mitigated NSAID-associated gastric damages.
- Regulation of the autophagic machinery in human neutrophils.
- TSC2 Deficiency Unmasks a Novel Necrosis Pathway That Is Suppressed by the RIP1/RIP3/MLKL Signaling Cascade.
- FoxG1/BNIP3 axis promotes mitophagy and blunts cisplatin resistance in osteosarcoma

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