

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide
Synthetic peptide
Catalog # BP10648a**Specification**

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - Product Information

Primary Accession [O9GZQ8](#)
Other Accession [NP_073729.1](#)

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - 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

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - Protein Information

Name MAP1LC3B ([HGNC:13352](#))

Synonyms MAP1ALC3

Function

Ubiquitin-like modifier involved in formation of autophagosomal vacuoles (autophagosomes) (PubMed: [20418806](http://www.uniprot.org/citations/20418806)), PubMed: [23209295](http://www.uniprot.org/citations/23209295), PubMed: [28017329](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/23209295), PubMed: [28017329](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/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

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - Images

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - Background

The product of this gene is a subunit of neuronal microtubule-associated MAP1A and MAP1B proteins, which are involved in microtubule assembly and important for neurogenesis. Studies on the rat homolog implicate a role for this gene in autophagy, a process that involves the bulk degradation of cytoplasmic component.

APG8a/b(MAP1LC3A/B) Antibody Blocking peptide - References

Rouschop, K.M., et al. J. Clin. Invest. 120(1):127-141(2010) Kirkin, V., et al. Mol. Cell 33(4):505-516(2009) Othman, E.Q., et al. J. Clin. Lab. Anal. 23(4):249-258(2009) Liu, Q., et al. Ai Zheng 27(1):25-29(2008) Komatsu, M., et al. Cell 131(6):1149-1163(2007)