

**Phospho-LC3B-T93 Antibody Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP3645a****Specification**

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**Phospho-LC3B-T93 Antibody Blocking peptide - Product Information**Primary Accession [O9GZQ8](#)**Phospho-LC3B-T93 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

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP3645a](/products/AP3645a) was selected from the MAP1LC3B-T93 region of human Phospho-APG8b (MAP1LC3B)-T93. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**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.

**Phospho-LC3B-T93 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))

target="\_blank">23209295</a>, PubMed:<a href="http://www.uniprot.org/citations/28017329" target="\_blank">28017329</a>). 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:<a href="http://www.uniprot.org/citations/22922758" target="\_blank">22922758</a>). 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:<a href="http://www.uniprot.org/citations/20418806" target="\_blank">20418806</a>, PubMed:<a href="http://www.uniprot.org/citations/23209295" target="\_blank">23209295</a>, PubMed:<a href="http://www.uniprot.org/citations/28017329" target="\_blank">28017329</a>). Promotes primary ciliogenesis by removing OFD1 from centriolar satellites via the autophagic pathway (PubMed:<a href="http://www.uniprot.org/citations/24089205" target="\_blank">24089205</a>). 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:<a href="http://www.uniprot.org/citations/31006537" target="\_blank">31006537</a>, PubMed:<a href="http://www.uniprot.org/citations/31006538" target="\_blank">31006538</a>). 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:<a href="http://www.uniprot.org/citations/30420355" target="\_blank">30420355</a>).

### 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). Localizes also to discrete punctae along the ciliary axoneme

### Tissue Location

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

## Phospho-LC3B-T93 Antibody Blocking peptide - Protocols

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

- [Blocking Peptides](#)

## Phospho-LC3B-T93 Antibody Blocking peptide - Images

## Phospho-LC3B-T93 Antibody Blocking peptide - Background

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. 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).

## Phospho-LC3B-T93 Antibody Blocking peptide - References

Baehrecke EH. Nat Rev Mol Cell Biol. 6(6):505-10. (2005) Lum JJ, et al. Nat Rev Mol Cell Biol. 6(6):439-48. (2005) Greenberg JT. Dev Cell. 8(6):799-801. (2005) Levine B. Cell. 120(2):159-62. (2005) Shintani T and Klionsky DJ. Science. 306(5698):990-5. (2004) Tanida I., et al. Int. J. Biochem. Cell Biol. 36:2503-2518(2004) He H., et al. J. Biol. Chem. 278:29278-29287(2003) Tanida I., et al. J. Biol. Chem. 279:36268-36276(2004)