

LC3 Antibody (APG8A) Blocking peptide
Synthetic peptide
Catalog # BP1800a**Specification**

LC3 Antibody (APG8A) Blocking peptide - Product InformationPrimary Accession [Q9H492](#)**LC3 Antibody (APG8A) Blocking peptide - Additional Information****Gene ID** 84557**Other Names**

Microtubule-associated proteins 1A/1B light chain 3A, Autophagy-related protein LC3 A, Autophagy-related ubiquitin-like modifier LC3 A, MAP1 light chain 3-like protein 1, MAP1A/MAP1B light chain 3 A, MAP1A/MAP1B LC3 A, Microtubule-associated protein 1 light chain 3 alpha, MAP1LC3A

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1800a](/product/products/AP1800a) was selected from the APG8a region of human MAP1LC3a. 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.

LC3 Antibody (APG8A) Blocking peptide - Protein Information**Name** MAP1LC3A ([HGNC:6838](#))**Function**

Ubiquitin-like modifier involved in formation of autophagosomal vacuoles (autophagosomes) (PubMed: [20713600](http://www.uniprot.org/citations/20713600), PubMed: [24290141](http://www.uniprot.org/citations/24290141)). 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: [20713600](http://www.uniprot.org/citations/20713600)). 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: [20713600](#)).

href="http://www.uniprot.org/citations/31006538" target="_blank">31006538, PubMed:31006537).

Cellular Location

Cytoplasmic vesicle, autophagosome membrane; Lipid-anchor. Endomembrane system; Lipid-anchor. Cytoplasm, cytoskeleton {ECO:0000250|UniProtKB:Q91VR7}. Note=LC3-II binds to the autophagic membranes.

Tissue Location

Most abundant in heart, brain, liver, skeletal muscle and testis but absent in thymus and peripheral blood leukocytes

LC3 Antibody (APG8A) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

LC3 Antibody (APG8A) Blocking peptide - Images**LC3 Antibody (APG8A) Blocking peptide - 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. MAP1LC3a 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.

LC3 Antibody (APG8A) Blocking peptide - References

Tanida, I., et al., J. Biol. Chem. 279(35):36268-36276 (2004).He, H., et al., J. Biol. Chem. 278(31):29278-29287 (2003).Mann, S.S., et al., J. Neurosci. Res. 43(5):535-544 (1996).