

Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide
Synthetic peptide
Catalog # BP3531a**Specification**

Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide - Product InformationPrimary Accession [Q9BXW4](#)**Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide - Additional Information****Gene ID** 440738**Other Names**

Microtubule-associated proteins 1A/1B light chain 3C, Autophagy-related protein LC3 C, Autophagy-related ubiquitin-like modifier LC3 C, MAP1 light chain 3-like protein 3, MAP1A/MAP1B light chain 3 C, MAP1A/MAP1B LC3 C, Microtubule-associated protein 1 light chain 3 gamma, MAP1LC3C

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP3531a](#) was selected from the APG8c region of human Phospho-LC3 (APG8c) - S137/138. 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.

Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide - Protein Information**Name** MAP1LC3C**Function**

Ubiquitin-like modifier that plays a crucial role in antibacterial autophagy (xenophagy) through the selective binding of CALCOCO2 (PubMed: <http://www.uniprot.org/citations/23022382> target="_blank">23022382). Recruits all ATG8 family members to infecting bacteria such as *S.typhimurium* (PubMed: <http://www.uniprot.org/citations/23022382> target="_blank">23022382). May also play a role in aggrephagy, the macroautophagic degradation of ubiquitinated and aggregated proteins (PubMed: <http://www.uniprot.org/citations/28404643> target="_blank">28404643).

Cellular Location

Cytoplasmic vesicle, autophagosome membrane; Lipid-anchor. Endomembrane system;

Lipid-anchor. Cytoplasm, cytoskeleton. Note=LC3-II binds to the autophagic membranes.

Tissue Location

Most abundant in placenta, lung and ovary.

Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide - Protocols

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

- [Blocking Peptides](#)

Bi-Phospho-LC3C(S137/138) Antibody Blocking peptide - Images**Bi-Phospho-LC3C(S137/138) Antibody 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. MAP1LC3c 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.

Bi-Phospho-LC3C(S137/138) 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)