

Product Information

Anti-Pro LC3C antibody

Mouse monoclonal, Clone BTY-1
purified from hybridoma cell culture
affinity isolated antibody

Product Number **SAB4200840**

Product Description

Monoclonal Anti-Pro LC3C antibody (mouse IgG1 isotype) is derived from the BTY-1 hybridoma, produced by the fusion of mouse myeloma cells and splenocytes from a mouse immunized with a synthetic peptide corresponding to the C-terminal region of human LC3C (GeneID: 440738), conjugated to KLH as immunogen. The isotype is determined by ELISA using Mouse Monoclonal Antibody Isotyping Reagents (Product Number ISO2). The antibody is affinity isolated antibody, purified from the culture supernatant of hybridoma cells.

Monoclonal Anti-Pro LC3C antibody specifically recognizes an epitope at the C-terminal pro-peptide domain of human LC3C-I and/or LC3C-II, and does not cross react with human LC3A or LC3B. The antibody may be used in various immunochemical techniques including immunoblotting and immunofluorescence. Detection of the LC3C band by immunoblotting is specifically inhibited by the immunogen.

Macroautophagy, usually referred to as autophagy, is a major pathway for bulk degradation of cytoplasmic constituents and organelles. In this process, portions of the cytoplasm are sequestered into double membrane vesicles, called autophagosomes, and subsequently delivered to the lysosome for degradation and recycling.^{1,2} Although autophagy is a constitutive cellular event, it is enhanced under certain conditions such as starvation, hormonal stimulation, and drug treatments.³ Autophagy plays an essential role in cellular differentiation, cell death and aging, and is required for normal turnover of cellular components during starvation. Defects in the autophagy process may contribute to certain human diseases such as cancer, neurodegenerative diseases, muscular disorders, and pathogen infections.^{4,5} Autophagy pathway is evolutionary conserved in all eukaryotic cells.¹

At least 16 genes encoding for autophagy (ATG) related proteins that are required for autophagosome formation were identified in yeast by genetic screens. For many of these genes, related homologs have been identified in mammals.⁶ The microtubule-associated protein light chain 3 (MAP1LC3), the mammalian homolog of yeast Atg8/Apg8/Aut7, is essential for the formation of autophagosomes.⁷ In addition, three human orthologs of yeast Atg8, named LC3A (MAP1LC3A), LC3B (MAP1LC3B), and LC3C (MAP1LC3C), were identified. These three human isoforms show different expression patterns in human tissues.⁸

The LC3 protein exists in two forms, cytoplasmic LC3-I (18 kDa) and autophagosome membrane associated LC3-II (16 kDa). LC3-II is the result of LC3-I cleavage at the C-terminal glycine by the cysteine protease Atg4, which is further modified by the mammalian E1- and E2-like enzymes: Atg7 and Atg3.⁷ The levels of LC3-II protein correlates with the extent of autophagosome formation.³ Thus, LC3C can be used as an autophagosomal and mitophagy marker.^{3,7,9-11}

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody Concentration: ~1.0 mg/mL

Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working concentration of 0.5-1 µg/mL is recommended using whole extracts of HEK-293T cells overexpressing human LC3C.

Note: In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration test.

References

1. Klionsky, D.J., and Emr, S.D., *Science*, **290**, 1717-21 (2000).
2. Kuma, A., et al., *Nature*, **432**, 1032-1036 (2004).
3. Kabeya, Y., et al., *EMBO J.*, **19**, 5720-5728 (2000).
4. Reggiori, F., and Klionsky, D.J., *Eukaryot Cell*, **1**, 11-21 (2002).
5. Shintani, T., and Klionsky, D.J., *Science*, **306**, 990-5 (2004).
6. Klionsky, D.J., et al., *Dev. Cell*, **5**, 539-545 (2003).
7. Tanida, I., et al., *J. Biol. Chem.*, **279**, 47704-10 (2004).
8. He, H., et al., *J. Biol. Chem.*, **278**, 29278-87 (2003).
9. Maier, H.J., et al., *Autophagy*, **9**, 496-509 (2013).
10. Le Guerroué, F., et al., *Mol. Cell*, **68**, 786-796 (2017).
11. Stolz, A., et al., *EMBO J.*, **36**, 549-564 (2017).

VS,DR,OKF,MAM 07/19-1