SANTA CRUZ BIOTECHNOLOGY, INC.

MAP LC3β (N-20): sc-16755

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Microtubule-associated proteins (MAPs) regulate microtubule stability and play critical roles in neuronal development and in maintaining the balance between neuronal plasticity and rigidity. MAP-light chain 3β (MAP LC3β) and MAP-light chain 3α (MAP LC3α) are subunits of both MAP1A and MAP1B. MAP LC3β, a homolog of Apg8p, is essential for autophagy and associated to the autophagosome membranes after processing. Two forms of LC3β, the cytosolic LC3-I and the membrane-bound LC3-II, are produced posttranslationally. LC3-I is formed by the removal of the C-terminal 22 amino acids from newly synthesized LC3β, followed by the conversion of a fraction of LC3-I into LC3-II. LC3 enhances Fibronectin mRNA translation in ductus arteriosus cells through association with 60S ribosomes and binding to an AU-rich element in the 3' untranslated region of Fibronectin mRNA. This facilitates sorting of Fibronectin mRNA onto rough endoplasmic reticulum and translation. MAP LC3β may also be involved in formation of autophagosomal vacuoles. It is expressed primarily in heart, testis, brain and skeletal muscle.

REFERENCES

1. Fink, J.K., et al. 1996. Human microtubule-associated protein 1a (MAP1A) gene: genomic organization, cDNA sequence, and developmental and tissue-specific expression. Genomics 35: 577-585.
2. Mann, S.S., et al. 1996. Gene localization and developmental expression of light chain 3: a common subunit of microtubule-associated protein 1A (MAP1A) and MAP1B. J. Neurosci. Res. 43: 535-544.

CHROMOSOMAL LOCATION

Genetic locus: MAP1LC3B (human) mapping to 16q24.2, MAP1LC3B2 (human) mapping to 12q24.22; Map1lc3b (mouse) mapping to 8 E1.

SOURCE

MAP LC3β (N-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of MAP LC3β of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-16755 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

APPLICATIONS

MAP LC3β (N-20) is recommended for detection of MAP LC3β and MAP LC3β2 of human origin and MAP LC3β of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

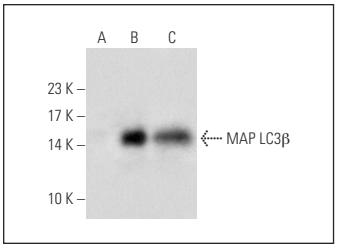
MAP LC3β (N-20) is also recommended for detection of MAP LC3β and MAP LC3β2 in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for MAP LC3β siRNA (m): sc-43391, MAP LC3β shRNA Plasmid (m): sc-43391-SH and MAP LC3β shRNA (m) Lentiviral Particles: sc-43391-V.

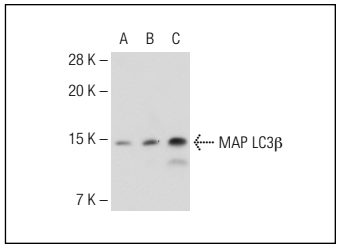
Molecular Weight of MAP LC3β: 15 kDa.

Positive Controls: MAP LC3β (h): 293T Lysate: sc-129100, mouse brain extract: sc-2253 or IMR-32 cell lysate: sc-2409.

DATA



MAP LC3β (N-20): sc-16755. Western blot analysis of MAP LC3β expression in non-transfected 293T: sc-117752 (**A**), human MAP LC3β transfected 293T: sc-129100 (**B**) and IMR-32 (**C**) whole cell lysates.



MAP LC3β (N-20): sc-16755. Western blot analysis of MAP LC3β expression in non-transfected: sc-117752 (**A**) and human MAP LC3β transfected: sc-173201 (**B**) 293T whole cell lysates and mouse brain tissue extract (**C**).

SELECT PRODUCT CITATIONS

1. Le Fourn, V., et al. 2009. Basal autophagy is involved in the degradation of the ERAD component EDEM1. Cell. Mol. Life Sci. 66: 1434-1445.
2. Cotán, D., et al. 2011. Secondary coenzyme Q10 deficiency triggers mitochondria degradation by mitophagy in MELAS fibroblasts. FASEB J. 25: 2669-2687.
3. Sanchez-Varo, R., et al. 2012. Abnormal accumulation of autophagic vesicles correlates with axonal and synaptic pathology in young Alzheimer's mice hippocampus. Acta Neuropathol. 123: 53-70.
4. De la Mata, M., et al. 2012. Recovery of MERRF fibroblasts and cybrids pathophysiology by Coenzyme Q_{10}. Neurotherapeutics 9: 446-463.
5. Bullon, P., et al. 2012. Autophagy in periodontitis patients and gingival fibroblasts: unraveling the link between chronic diseases and inflammation. BMC Med. 10: 122.
6. Hsueh, Y.S., et al. 2013. Autophagy is involved in endogenous and NVP-AUY922-induced KIT degradation in gastrointestinal stromal tumors. Autophagy 9: 220-233.