Product Information

Anti-Atg13 Antibody, Mouse Monoclonal

Clone ATG13-8, Purified from Hybridoma Cell Culture

SAB4200376

Product Description

Monoclonal Anti-ATG13 (mouse IgG2a isotype) is derived from the hybridoma ATG13-8 produced by the fusion of mouse myeloma cells and splenocytes from BALB/c mice immunized with a synthetic peptide corresponding to the C-terminal region of human ATG13 (GeneID: 9776), conjugated to KLH. The corresponding sequence is identical in mouse and rat Atg13. The isotype is determined by ELISA using Mouse Monoclonal Antibody Isotyping Reagents (Cat. No. ISO2). The antibody is purified from culture supernatant of hybridoma cells grown in a bioreactor.

Monoclonal Anti-ATG13 recognizes human and mouse Atg13. The antibody may be used in various immunochemical techniques including immunoblotting (~ 65 kDa) and immunoprecipitation. Detection of the Atg13 band by immunoblotting is specifically inhibited by the immunizing peptide.

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, the 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 is required for normal turnover of cellular components during starvation. It plays an essential role in cellular differentiation, cell death and aging. Defective autophagy may contribute to certain human diseases such as cancer, neurodegenerative diseases, muscular disorders and pathogen infections.[4,5] Autophagy is an evolutionary conserved pathway seen in all eukaryotic cells.[1]

At least 16 ATG genes 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]
Two ubiquitin-like conjugation systems are involved in autophagosome formation: Atg12 and Atg8 conjugation systems. Atg8 is synthesized as a precursor protein, which is cleaved after a Gly residue by the cysteine proteinase Atg4. The modified Atg8 is activated by Atg7, an E1-like enzyme, and then transferred to Atg3, an E2-like enzyme, followed by conjugation to membrane- bound phosphatidylethanolamine (PE). The complex Atg8-PE is also deconjugated by Atg4, leading to the release of Atg8 from membranes.[7,8]

Atg13 is also essential for autophagosome formation in mammalian cells. Atg13 forms a stable complex with ULK1 and FIP200. mTOR interacts with this complex in a nutrient dependent manner and phosphorylates Atg13 and ULK1, suggesting that mTOR regulates autophagy through the ULK1-Atg13-FIP200 complex.[9,10]

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 at −20 °C in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, 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 1-2 µg/mL is recommended using whole extracts of HEK-293T cells over expressing human Atg13. recommended using lysates of mouse LA-4 cells.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining optimal working dilutions by titration.

References

1. Klionsky, D.J., and Emr, S.D., *Science*, **290**: 1717-1721 (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., *Eukaryotic Cell*, **1**: 11-21 (2002).
5. Shintani, T., and Klionsky, D.J., *Science*, **306**: 990-995 (2004).
6. Klionsky, D.J., et al., *Develop. Cell*, **5**: 539-545 (2003).
7. Kirisako, T., et al., *J. Cell Biol.*, **151:** 263-276 (2000).
8. Ichimura, Y., et al., *Nature*, **408:** 488-492 (2000).
9. Hosokawa, N., et al., *Mol. Biol. Cell*, **20**: 1981- 1991 (2009).
10. Jung, C.H., et al., *Mol. Biol. Cell*, **20**: 1992-2003 (2009).

Notice

We provide information and advice to our customers on application technologies and regulatory matters to the best of our knowledge and ability, but without obligation or liability. Existing laws and regulations are to be observed in all cases by our customers. This also applies in respect to any rights of third parties. Our information and advice do not relieve our customers of their own responsibility for checking the suitability of our products for the envisaged purpose.

The information in this document is subject to change without notice and should not be construed as a commitment by the manufacturing or selling entity, or an affiliate. We assume no responsibility for any errors that may appear in this document.

Technical Assistance

Visit the tech service page at SigmaAldrich.com/techservice.

Standard Warranty

The applicable warranty for the products listed in this publication may be found at SigmaAldrich.com/terms.

Contact Information

For the location of the office nearest you, go to SigmaAldrich.com/offices.

SAB4200376dat Rev 08/21