3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Anti-Atg13
produced in rabbit, affinity isolated antibody

Product Number **SAB4200100**

Product Description

Anti-Atg13 is produced in rabbit using as the immunogen a synthetic peptide corresponding to a fragment of human Atg13 (GeneID: 9776), conjugated to KLH. The corresponding sequence is identical in mouse and rat Atg13. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Atg13 recognizes human, mouse, and rat Atg13. The antibody can be used in several immunochemical techniques including immunoblotting (~65 kDa). 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 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 phosphatidyl-ethanolamine (PE). The complex Atg8-PE is also deconjugated by Atg4, leading to the release of Atg8 from membranes.[7,8]

Atg13 is 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

Store at –20 °C. For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots at –20 °C. 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 dilutions should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working antibody concentration of 1-2 μg/mL is recommended using whole extracts of human HeLa cells and 0.5-1 μg/mL using whole extracts of mouse 3T3 or rat PC12 cells.

Note: In order to obtain best results in various techniques and preparations, it is recommended to determine 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).

VS,ST,TD,KAA,PHC,MAM 05/19-1