Revision 4

Store at -20C

#13273

Atg13 (D4P1K) Rabbit mAb

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H M R	Endogenous	72	Rabbit IgG	#O75143	9776

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

For a carrier free (BSA and azide free) version of this product see product #41634.

Specificity/Sensitivity

Atg13 (D4P1K) Rabbit mAb recognizes endogenous levels of total Atg13 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Asp462 of human Atg13 protein.

Background

Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents (1,2). Autophagy is generally activated by conditions of nutrient deprivation but has also been associated with a number of physiological processes including development, differentiation, neurodegeneration, infection, and cancer (3). The molecular machinery of autophagy was largely discovered in yeast and referred to as autophagy-related (Atg) genes.

Atg13/Apg13 was originally identified in yeast as a constitutively expressed protein that was genetically linked to Atg1/Apg1, a protein kinase required for autophagy (4). Overexpression of Atg1 suppresses the defects in autophagy observed in Atg13 mutants (4). Autophagy requires a direct association between Atg1 and Atg13, and is inhibited by TOR-dependent phosphorylation of Atg13 under high-nutrient conditions (5). Similarly, mammalian Atg13 forms a complex with the Atg1 homologues ULK1/2, along with FIP200, which localizes to autophagic isolation membranes and regulates autophagosome biogenesis (6-8). mTOR phosphorylates both Atg13 and ULK1, suppressing ULK1 kinase activity and autophagy (7-9). ULK1 can directly phosphorylate Atg13 at a yet unidentified site, presumably to promote autophagy (7,8). Additional studies suggest that Atg13 and FIP200 can function independently of ULK1 and ULK2 to induce autophagy through an unknown mechanism (10).

Background References

1. Reggiori, F. and Klionsky, D.J. (2002) *Eukaryot Cell* 1, 11-21.
2. Codogno, P. and Meijer, A.J. (2005) *Cell Death Differ* 12 Suppl 2, 1509-18.
3. Levine, B. and Yuan, J. (2005) *J Clin Invest* 115, 2679-88.
4. Funakoshi, T. et al. (1997) *Gene* 192, 207-13.
5. Kamada, Y. et al. (2000) *J Cell Biol* 150, 1507-13.
6. Ganley, I.G. et al. (2009) *J Biol Chem* 284, 12297-305.
7. Hosokawa, N. et al. (2009) *Mol Biol Cell* 20, 1981-91.
8. Jung, C.H. et al. (2009) *Mol Biol Cell* 20, 1992-2003.
9. Kim, J. et al. (2011) *Nat Cell Biol* 13, 132-41.
10. Alers, S. et al. (2011) *Autophagy* 7, 1423-33.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat

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