

Phospho-Atg13 (Ser355) Antibody



Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H	Transfected Only	72	Rabbit	#O75143	9776

Product Usage Information

Application

Western Blotting

Dilution

1:1000

Storage

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

Specificity/Sensitivity

Phospho-Atg13 (Ser355) Antibody recognizes transfected levels of human Atg13 protein only when phosphorylated at Ser355 (or Ser354 for mouse Atg13).

Species predicted to react based on 100% sequence homology

Mouse, Rat

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser355 of human Atg13 protein (Ser318 of isoform 2 of Atg13). Antibodies are purified by protein A and peptide affinity chromatography.

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). ULK1-dependent phosphorylation of Atg13 at Ser355, which corresponds to Ser318 of isoform 2 of Atg13, leads to the recruitment of Atg13 to damaged mitochondria, enabling efficient mitophagy (11).

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.
11. Joo, J.H. et al. (2011) *Mol Cell* 43, 572-85.

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 BSA, 1X TBS, 0.1% Tween@ 20 at 4°C with gentle shaking, overnight.

Applications Key**W:** Western Blotting**Cross-Reactivity Key****H:** Human**Trademarks and Patents**

Cell Signaling Technology is a trademark of Cell Signaling Technology, Inc.

All other trademarks are the property of their respective owners. Visit cellsignal.com/trademarks for more information.**Limited Uses**

Except as otherwise expressly agreed in a writing signed by a legally authorized representative of CST, the following terms apply to Products provided by CST, its affiliates or its distributors. Any Customer's terms and conditions that are in addition to, or different from, those contained herein, unless separately accepted in writing by a legally authorized representative of CST, are rejected and are of no force or effect.

Products are labeled with For Research Use Only or a similar labeling statement and have not been approved, cleared, or licensed by the FDA or other regulatory foreign or domestic entity, for any purpose. Customer shall not use any Product for any diagnostic or therapeutic purpose, or otherwise in any manner that conflicts with its labeling statement. Products sold or licensed by CST are provided for Customer as the end-user and solely for research and development uses. Any use of Product for diagnostic, prophylactic or therapeutic purposes, or any purchase of Product for resale (alone or as a component) or other commercial purpose, requires a separate license from CST. Customer shall (a) not sell, license, loan, donate or otherwise transfer or make available any Product to any third party, whether alone or in combination with other materials, or use the Products to manufacture any commercial products, (b) not copy, modify, reverse engineer, decompile, disassemble or otherwise attempt to discover the underlying structure or technology of the Products, or use the Products for the purpose of developing any products or services that would compete with CST products or services, (c) not alter or remove from the Products any trademarks, trade names, logos, patent or copyright notices or markings, (d) use the Products solely in accordance with CST Product Terms of Sale and any applicable documentation, and (e) comply with any license, terms of service or similar agreement with respect to any third party products or services used by Customer in connection with the Products.