Revision 1

Store at -20C

#36105

# PhosphoPlus® Atg13 (Ser355) Antibody Duet

Cell Signaling TECHNOLOGY®

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

| UniProt ID: | Entrez-Gene Id: |
|---|---|
| #O75143 | 9776 |

| Product Includes | Product # | Quantity | Mol. Wt | Isotype/Source |
|---|---|---|---|---|
| Phospho-Atg13 (Ser355) (E4D3T) Rabbit mAb | 46329 | 100 µl | 72 kDa | Rabbit IgG |
| Atg13 (E1Y9V) Rabbit mAb | 13468 | 100 µl | 72 kDa | Rabbit IgG |

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

## Description

PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

## 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 antibodies.*

## 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

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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.
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10. Alers, S. et al. (2011) *Autophagy* 7, 1423-33.
11. Joo, J.H. et al. (2011) *Mol Cell* 43, 572-85.

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