Revision 1

Store at -20C

#54831

PhosphoPlus® Beclin-1 (Ser30) Antibody Duet

Cell Signaling TECHNOLOGY®

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.

UniProt ID:	Entrez-Gene Id:
#Q14457	8678

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-Beclin-1 (Ser30) (E1C4X) Rabbit mAb	35955	100 µl	60 kDa	Rabbit IgG
Beclin-1 (D40C5) Rabbit mAb	3495	100 µl	60 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 proteins activated in response to nutrient deprivation and in neurodegenerative conditions (1). One of the proteins critical to this process is Beclin-1, the mammalian orthologue of the yeast autophagy protein Apg6/Vps30 (2). Beclin-1 can complement defects in yeast autophagy caused by loss of Apg6 and can also stimulate autophagy when overexpressed in mammalian cells (3). Mammalian Beclin-1 was originally isolated in a yeast two-hybrid screen for Bcl-2 interacting proteins and has been shown to interact with Bcl-2 and Bcl-xL, but not with Bax or Bak (4). While Beclin-1 is generally ubiquitously expressed, research studies have shown it is monoallelically deleted in 40-75% of sporadic human breast and ovarian cancers (5). Beclin-1 is localized within cytoplasmic structures including the mitochondria, although overexpression of Beclin-1 reveals some nuclear staining and CRM1-dependent nuclear export (6). Investigators have demonstrated that Beclin-1$^{-/-}$ mice die early in embryogenesis and Beclin-1$^{-/+}$ mice have a high incidence of spontaneous tumors. Stem cells from the null mice demonstrate an altered autophagic response, although responses to apoptosis appeared normal (7). Researchers have also found that overexpression of Beclin-1 in virally infected neurons *in vivo* resulted in significant protection against Sindbis virus-induced disease and neuronal apoptosis (4).
ULK1 phosphorylates Beclin-1 at Ser30 to induce autophagy in response to amino acid starvation, mTORC1 inhibition, and hypoxia (8).

Background References

1. Reggiori, F. and Klionsky, D.J. (2002) *Eukaryot Cell* 1, 11-21.
2. Kametaka, S. et al. (1998) *J Biol Chem* 273, 22284-91.
3. Liang, X.H. et al. (1999) *Nature* 402, 672-6.
4. Liang, X.H. et al. (1998) *J Virol* 72, 8586-96.
5. Aita, V.M. et al. (1999) *Genomics* 59, 59-65.
6. Liang, X.H. et al. (2001) *Cancer Res* 61, 3443-9.
7. Yue, Z. et al. (2003) *Proc Natl Acad Sci USA* 100, 15077-82.
8. Park, J.M. et al. (2018) *Autophagy* 14, 584-597.

Trademarks and Patents

Limited Uses

force or effect.

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