

Phospho-Beclin-1 (Ser15) (D4B7R) Rabbit mAb

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H M	Transfected Only	60	Rabbit IgG	#Q14457	8678

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, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Phospho-Beclin-1 (Ser15) (D4B7R) Rabbit mAb recognizes transfected levels of Beclin-1 protein only when phosphorylated at Ser15 (which corresponds to Ser14 in mouse).

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser15 of human Beclin-1 protein.

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). Autophagy inducers, including amino-acid starvation and mTOR inhibition, lead to phosphorylation of mouse Beclin-1 at Ser14 (equivalent to human Ser15) by the serine/threonine kinase ULK1. This results in an increase in VPS34 lipid kinase activity and leads to the induction of autophagy (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. Russell, R.C. et al. (2013) *Nat Cell Biol* 15, 741-50.

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 **M:** Mouse

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