Revision 6

Store at -20C

#8089

Atg16L1 (D6D5) 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, IP, IF-IC	H M R	Endogenous	66, 68	Rabbit IgG	#Q676U5	55054

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:100
Immunofluorescence (Immunocytochemistry)	1:50 - 1:200

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

Atg16L1 (D6D5) Rabbit mAb recognizes endogenous levels of total Atg16L1 protein. A background band is detected at 40 kDa in some cell lines.

Species predicted to react based on 100% sequence homology

Monkey

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val51 of human Atg16L1 protein.

Background

Autophagy is a catabolic process for the autophagosomic-lysosomal degradation of bulk cytoplasmic contents. Control of autophagy was largely discovered in yeast and involves proteins encoded by a set of autophagy-related genes (Atg) (1). Formation of autophagic vesicles requires a pair of essential ubiquitin-like conjugation systems, Atg12-Atg5 and Atg8 (LC3)-phosphatidylethanolamine (LC3-PE), which are widely conserved in eukaryotes (2).Mammalian Atg16L1, containing an amino-terminal coiled-coil domain and carboxyl-terminal WD-repeats, has multiple isoforms produced by alternative splicing (3,4). Atg16L1 provides a functional link between the two crucial ubiquitin-like conjugation systems of autophagy. Atg16L1 binds Atg5 of the Atg12-Atg5 conjugate forming an 800 kDa multimeric complex (3). The Atg12-Atg-5-Atg16L1 complex localizes to pre-autophagosomal membranes, where it determines the site of LC3 lipidation and catalyzes the reaction required for the formation of mature autophagosomes (3,5). Genome-wide association scanning revealed variations in the *Atg16L1* gene associated with Crohn's disease (6,7). Mice lacking the coiled-coil domain of Atg16L1 have impaired autophagosome formation and elevated inflammatory cytokines, consistent with its role in inflammatory disease pathogenesis (8). Hypomorphic Atg16L1 mice also show defects in autophagy and abnormalities in intestinal Paneth cell function similar to that found in Crohn's disease (9).

Background References

1. Reggiori, F. and Klionsky, D.J. (2002) *Eukaryot Cell* 1, 11-21.
2. Ohsumi, Y. (2001) *Nat Rev Mol Cell Biol* 2, 211-6.
3. Mizushima, N. et al. (2003) *J Cell Sci* 116, 1679-88.
4. Zheng, H. et al. (2004) *DNA Seq* 15, 303-5.
5. Fujita, N. et al. (2008) *Mol Biol Cell* 19, 2092-100.
6. Hampe, J. et al. (2007) *Nat Genet* 39, 207-11.
7. Rioux, J.D. et al. (2007) *Nat Genet* 39, 596-604.
8. Saitoh, T. et al. (2008) *Nature* 456, 264-8.
9. Cadwell, K. et al. (2008) *Nature* 456, 259-63.

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 **IP:** Immunoprecipitation **IF-IC:** Immunofluorescence (Immunocytochemistry)

Cross-Reactivity Key

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

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