

Atg9A Antibody

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

Applications: W, IP	Reactivity: H	Sensitivity: Endogenous	MW (kDa): 100-110	Source/Isotype: Rabbit	UniProt ID: #Q7Z3C6	Entrez-Gene Id: 79065
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Product Usage Information**Application**

Western Blotting
Immunoprecipitation

Dilution

1:1000
1:50

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

Atg9A Antibody recognizes endogenous levels of total Atg9A protein.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Gly780 of human Atg9A protein. 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). It is generally activated by conditions of nutrient deprivation but is also 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 is directed by a number of autophagy-related (Atg) genes (4). Atg9, one of the Atg proteins identified in yeast, is essential for autophagosome formation (5). There are two human functional orthologues based on the yeast homolog Atg9p: Atg9A, which has also been identified as Atg9L1 and mAtg9, and Atg9L2, which was first reported as nitric-oxide synthase 3 antisense (NOS3AS) (6,7). Atg9A is an integral membrane protein that is required for both the initiation and the expansion of the autophagosome (6,7). Recruitment of Atg9A to the autophagosomal membrane is dynamic and transient as Atg9A also cycles between autophagy-related structures known as omegasomes, the trans-Golgi network (TGN), and endosomes, and at no point becomes a stable component of the autophagosomal membrane (6,8). The precise regulation of Atg9A trafficking is not fully clarified, yet it is suggested to involve p38 mitogen-activated protein kinase (MAPK)-binding protein p38IP and the Beclin-1-binding protein Bif-1 (9,10).

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. Klionsky, D.J. et al. (2003) *Dev Cell* 5, 539-45.
5. Noda, T. et al. (2000) *J Cell Biol* 148, 465-80.
6. Young, A.R. et al. (2006) *J Cell Sci* 119, 3888-900.
7. Yamada, T. et al. (2005) *J Biol Chem* 280, 18283-90.
8. Orsi, A. et al. (2012) *Mol Biol Cell* 23, 1860-73.
9. Webber, J.L. and Tooze, S.A. (2010) *EMBO J* 29, 27-40.
10. Takahashi, Y. et al. (2011) *Autophagy* 7, 61-73.

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

Cross-Reactivity Key

H: Human

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