

**Atg2A Antibody**

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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	Endogenous	213	Rabbit	#Q2TAZ0	23130

**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 and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

**Specificity/Sensitivity**

Atg2A Antibody recognizes endogenous levels of total Atg2A protein.

**Source / Purification**

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Glu750 of human Atg2A protein. Antibodies are purified by protein A and peptide affinity chromatography.

**Background**

Autophagy is a catabolic process that results in the degradation of bulk cytoplasmic contents within autophagosomes and lysosomes. The control of autophagy involves proteins encoded by a set of autophagy-related genes (Atg) that were originally characterized in yeast (1). Research studies in yeast indicate that Atg2 is essential for autophagy and the retrograde transport of Atg9 through an interaction with Atg18 (2-6). Two human Atg2 homologs (Atg2A, Atg2B) are critical for autophagosome formation as silencing of both results in the accumulation of unclosed autophagic structures (7). Starvation-induced autophagy targets Atg2A to the initiation site of autophagosome biogenesis, where it associates with DFCP1, WIPI-1, and other autophagy-related proteins (8). Atg2 proteins also function in lipid droplet metabolism as depletion of both Atg2A and AtgB results in changes in the size, number, and distribution of lipid droplets (7,8). These morphological changes in lipid droplets are not observed in Atg5-depleted cells, suggesting that this function is independent of the role of Atg2 in autophagy (7). Starvation-induced autophagy directs Atg2A (along with Atg14L) to localize to early autophagosomal membranes enriched in PI3P, while another subpopulation of Atg2A and Atg14L localizes to the lipid droplets independent of autophagic status (8). An increase in Atg2A expression during etoposide- and doxorubicin-induced apoptosis suggests that Atg2A may be a useful indicator of topoisomerase II inhibitor-mediated apoptosis (9). Mutations in the corresponding *Atg2B* gene are associated with gastric and colorectal carcinomas with high microsatellite instability (10).

**Background References**

1. Reggiori, F. and Klionsky, D.J. (2002) *Eukaryot Cell* 1, 11-21.
2. Shintani, T. et al. (2001) *J Biol Chem* 276, 30452-60.
3. Wang, C.W. et al. (2001) *J Biol Chem* 276, 30442-51.
4. Suzuki, K. et al. (2001) *EMBO J* 20, 5971-81.
5. Obara, K. et al. (2008) *J Biol Chem* 283, 23972-80.
6. Reggiori, F. et al. (2004) *Dev Cell* 6, 79-90.
7. Velikkakath, A.K. et al. (2012) *Mol Biol Cell* 23, 896-909.
8. Pfisterer, S.G. et al. (2014) *J Lipid Res* 55, 1267-78.
9. Kusama, Y. et al. (2009) *Apoptosis* 14, 1165-75.
10. Kang, M.R. et al. (2009) *J Pathol* 217, 702-6.

**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 nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

**Applications Key**

**W:** Western Blotting

**Cross-Reactivity Key**

**H:** Human

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