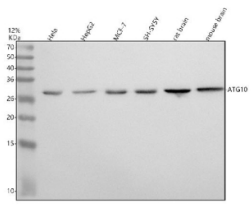
ATG10 Antibody / Autophagy-related protein 10 [clone ABBD-1] (FY12636)

Catalog No.	Formulation	Size
FY12636	Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol, 0.4-0.5mg/ml BSA	100 ul

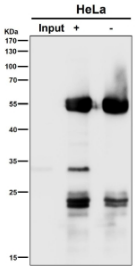
Recombinant RABBIT MONOCLONAL

Bulk quote request

Availability	2-3 weeks
Species Reactivity	Human, Mouse, Rat
Format	Liquid
Host	Rabbit
Clonality	Recombinant Rabbit Monoclonal
Isotype	Rabbit IgG
Clone Name	ABBD-1
Purity	Affinity-chromatography
Buffer	Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol, 0.4-0.5mg/ml BSA.
UniProt	Q9H0Y0
Applications	Western Blot: 0.25-0.5ug/ml Immunoprecipitation: 2-4ug/500ug of lysate
Limitations	This ATG10 antibody is available for research use only.



Western blot analysis of ATG10 using anti-ATG10 antibody. Lane 1: human Hela whole cell lysates, Lane 2: human HepG2 whole cell lysates, Lane 3: human MCF-7 whole cell lysates, Lane 4: human SH-SY5Y whole cell lysates, Lane 5: rat brain tissue lysates, Lane 6: mouse brain tissue lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-ATG10 antibody at 1:1000 overnight at 4oC, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:500 for 1.5 hour at RT. The signal was developed using enhanced chemiluminescent. The expected molecular weight of ATG10 is ~25 kDa.



Immunoprecipitation analysis using the ATG10 antibody at 1:50 dilution. Western blot at 1:3000 dilution, the expected molecular weight of ATG10 is ~25 kDa.

Description

ATG10 antibody detects Autophagy-related protein 10, an essential E2-like enzyme that catalyzes conjugation of ATG12 to ATG5, a key step in autophagosome formation. ATG10 plays a central role in the autophagy machinery by facilitating complex assembly required for membrane elongation and vesicle maturation. The ATG10 antibody is widely used in cell biology and metabolism research to study autophagy regulation, stress adaptation, and cellular degradation pathways.

ATG10 is encoded by the ATG10 gene located on human chromosome 5q14.1. The protein is approximately 220 amino acids long and resides in the cytoplasm, where it transiently associates with autophagic membranes. Functionally, ATG10 acts as an E2-like enzyme, transferring the ATG12 conjugate to ATG5 under the direction of E1-like enzyme ATG7. This conjugation system forms the ATG12-ATG5-ATG16L1 complex that defines the site of autophagosome formation.

The ATG10 antibody detects a 25 kilodalton band by western blot and shows diffuse cytoplasmic staining under confocal microscopy. ATG10 expression increases under nutrient deprivation and oxidative stress, linking metabolic signals to autophagy induction. Inhibition or depletion of ATG10 results in defective autophagosome biogenesis, accumulation of damaged organelles, and increased susceptibility to stress-induced apoptosis.

ATG10 also participates in noncanonical autophagy and immune signaling by modulating antigen presentation and cytokine secretion. Variations in ATG10 have been linked to cancers, inflammatory disorders, and aging-related diseases. Its regulation is tightly controlled through transcriptional activation by FOXO and ATF4, and by post-translational modifications that modulate enzyme activity.

Because of its indispensable role in the autophagy conjugation system, ATG10 provides a molecular link between stress sensing and degradative homeostasis. NSJ Bioreagents provides a validated ATG10 antibody optimized for its applications, supporting research into cellular degradation, metabolism, and survival mechanisms.

Application Notes

Optimal dilution of the ATG10 antibody should be determined by the researcher.

Immunogen

A synthesized peptide derived from human Apg10 (Atg10) was used as the immunogen for the ATG10 antibody.

Storage

Store the ATG10 antibody at -20oC.

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