

**Anti-Atg4A Monoclonal Antibody**  
**Catalog # ABO14469****Specification****Anti-Atg4A Monoclonal Antibody - Product Information**

|                   |                        |
|-------------------|------------------------|
| Application       | WB, IHC, IP            |
| Primary Accession | <a href="#">Q8WYN0</a> |
| Host              | Rabbit                 |
| Isotype           | Rabbit IgG             |
| Reactivity        | Rat, Human, Mouse      |
| Clonality         | Monoclonal             |
| Format            | Liquid                 |

**Description**

Anti-Atg4A Monoclonal Antibody . Tested in WB, IHC, IP applications. This antibody reacts with Human, Mouse, Rat.

**Anti-Atg4A Monoclonal Antibody - Additional Information**

**Gene ID** 115201

**Other Names**

Cysteine protease ATG4A, 3.4.22.-, AUT-like 2 cysteine endopeptidase, Autophagy-related cysteine endopeptidase 2, Autophagin-2, Autophagy-related protein 4 homolog A, HsAPG4A, hAPG4A, ATG4A {ECO:0000303|Ref.20, ECO:0000312|HGNC:HGNC:16489}

**Application Details**

WB 1:500-1:2000<br>IHC 1:50-1:200<br>IP 1:50

**Contents**

Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol, 0.4-0.5mg/ml BSA.

**Immunogen**

A synthesized peptide derived from human Atg4A The cysteine protease Atg4 is pivotal to autophagosome membrane generation and regulation. Atg4 primes the Atg8 homologue for lipidation by cleaving its carboxy terminus and exposing its glycine residue for E1-like enzyme Atg7. The Atg8 homologue is transferred to the E2-like enzyme Atg3 before forming the Atg8-PE conjugate. During later stages of autophagy, Atg4 can reverse this lipidation event by cleaving PE, thereby recycling the Atg8 homologue.

**Purification**

Affinity-chromatography

**Storage**

**Store at -20°C for one year. For short term storage and frequent use, store at 4°C for up to one month. Avoid repeated freeze-thaw cycles.**

**Anti-Atg4A Monoclonal Antibody - Protein Information**

## Function

[12473658](http://www.uniprot.org/citations/12473658)</a>, PubMed:<a href="http://www.uniprot.org/citations/15169837" target="\_blank">15169837</a>, PubMed:<a href="http://www.uniprot.org/citations/17347651" target="\_blank">17347651</a>, PubMed:<a href="http://www.uniprot.org/citations/21177865" target="\_blank">21177865</a>, PubMed:<a href="http://www.uniprot.org/citations/21245471" target="\_blank">21245471</a>, PubMed:<a href="http://www.uniprot.org/citations/22302004" target="\_blank">22302004</a>, PubMed:<a href="http://www.uniprot.org/citations/32732290" target="\_blank">32732290</a>). The protease

<http://www.uniprot.org/citations/12473658> target="\_blank">12473658</a>, PubMed:<a href="http://www.uniprot.org/citations/15169837">http://www.uniprot.org/citations/15169837 target="\_blank">15169837</a>, PubMed:<a href="http://www.uniprot.org/citations/17347651">http://www.uniprot.org/citations/17347651 target="\_blank">17347651</a>, PubMed:<a href="http://www.uniprot.org/citations/21177865">http://www.uniprot.org/citations/21177865 target="\_blank">21177865</a>, PubMed:<a href="http://www.uniprot.org/citations/21245471">http://www.uniprot.org/citations/21245471 target="\_blank">21245471</a>, PubMed:<a

phosphatidylethanolamine (PE) and insertion to membranes, which is necessary for autophagy (PubMed:<a href="http://www.uniprot.org/citations/12473658" target=" blank">12473658</a>).

PubMed: <a href="http://www.uniprot.org/citations/15169837" target="\_blank">15169837</a>  
PubMed: <a href="http://www.uniprot.org/citations/17347651" target="\_blank">17347651</a>

PubMed: [21177865](http://www.uniprot.org/citations/21177865),  
PubMed: [21245471](http://www.uniprot.org/citations/21245471),

PubMed:<a href="http://www.uniprot.org/citations/22302004" target="\_blank">22302004</a>). Preferred substrate is GABARAPL2 followed by MAP1LC3A and GABARAP (PubMed:<a href="http://www.uniprot.org/citations/22302004" target="\_blank">22302004</a>).

[12473658](http://www.uniprot.org/citations/12473658)</a>, PubMed:<a href="http://www.uniprot.org/citations/15169837" target="\_blank">15169837</a>, PubMed:<a href="http://www.uniprot.org/citations/15169837" target="\_blank">15169837</a>

href="http://www.uniprot.org/citations/17347651" target="\_blank">17347651</a>, PubMed:<a href="http://www.uniprot.org/citations/21177865" target="\_blank">21177865</a>, PubMed:<a

href="http://www.uniprot.org/citations/21245471" target="\_blank">21245471</a>, PubMed:<a href="http://www.uniprot.org/citations/22302004" target="\_blank">22302004</a>). Protease

activity is also required to counteract formation of high-molecular weight conjugates of ATG8 proteins (ATG8ylation): acts as a deubiquitinating- like enzyme that removes ATG8 conjugated to

other proteins, such as ATG3 (PubMed:<a href="http://www.uniprot.org/citations/31315929" target="\_blank">31315929</a>, PubMed:<a href="http://www.uniprot.org/citations/33773106"

target=" \_blank">33773106</a>). In addition to the protease activity, also mediates delipidation of ATG8 family proteins (PubMed:<a href="http://www.uniprot.org/citations/29458288"

target="\_blank">29458288</a>, PubMed:<a href="http://www.uniprot.org/citations/33909989" target="\_blank">33909989</a>). Catalyzes delipidation of PE- conjugated forms of ATG8 proteins

during macroautophagy (PubMed:<a href="http://www.uniprot.org/citations/29458288" target="\_blank">29458288</a>, PubMed:<a href="http://www.uniprot.org/citations/33909989" target="\_blank">33909989</a>). G-proteins are involved in the regulation of the autophagy pathway, and the G-protein-coupled receptor (GPCR) is a major player in this process. The GPCR is a transmembrane protein that is activated by a variety of ligands, including hormones, neurotransmitters, and growth factors. Upon activation, the GPCR activates a G-protein, which in turn activates a variety of downstream signaling pathways, including the autophagy pathway. The GPCR is a key component of the autophagy pathway, and its activation is essential for the regulation of autophagy.

target=" \_blank">33909989</a>). Compared to ATG4B, the major protein for proteolytic activation of ATG8 proteins, shows weaker ability to cleave the C-terminal amino acid of ATG8 proteins, while it displays strong autolysis activity (PubMed, [33909989](#)).

It displays stronger delipidation activity (PubMed:<a href="http://www.uniprot.org/citations/29458288" target="\_blank">29458288</a>). Involved in phagosome growth during *Leishmania* independently of its *phagosome* activity and of ATG9 protein

phagophore growth during mitophagy independently of its protease activity and of A1 G8 proteins acts by regulating ATG9A trafficking to mitochondria and promoting phagophore-endoplasmic reticulum contacts during the lipid transfer phase of mitophagy (DittMed 2016).

reticulum contacts during the lipid transfer phase of mitophagy (PubMed:<a href="http://www.uniprot.org/citations/33773106" target="\_blank">33773106</a>).

Cytoplasm {ECO:0000250|UniProtKB:Q8BGE6}.

## Anti-Atg4A Monoclonal Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## Anti-Atg4A Monoclonal Antibody - Images

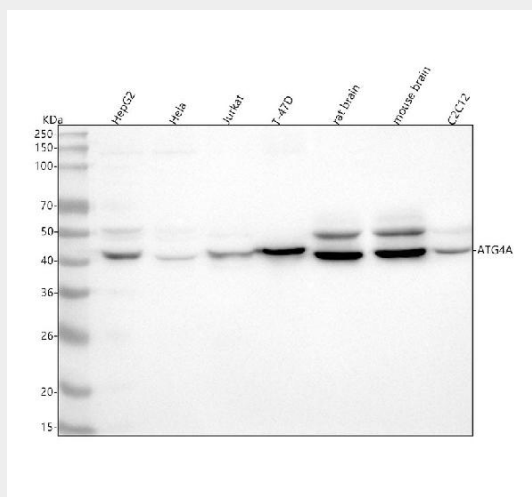


Figure 1. Western blot analysis of Atg4A using anti-Atg4A antibody (M06539).

Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions.

Lane 1: human HepG2 whole cell lysates,

Lane 2: human Hela whole cell lysates,

Lane 3: human Jurkat whole cell lysates,

Lane 4: human T-47D whole cell lysates,

Lane 5: rat brain tissue lysates,

Lane 6: mouse brain tissue lysates,

Lane 7: mouse C2C12 whole cell 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-Atg4A antigen affinity purified monoclonal antibody (Catalog # M06539) at 1:500 overnight at 4°C, 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 1:5000 for 1.5 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for Atg4A at approximately 45 kDa. The expected band size for Atg4A is at 45 kDa.