

**APG1 (ULK1) Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP8104b****Specification**

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**APG1 (ULK1) Antibody (Center) Blocking peptide - Product Information**Primary Accession [O75385](#)**APG1 (ULK1) Antibody (Center) Blocking peptide - Additional Information****Gene ID** 8408**Other Names**

Serine/threonine-protein kinase ULK1, Autophagy-related protein 1 homolog, ATG1, hATG1, Unc-51-like kinase 1, ULK1, KIAA0722

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8104b](/product/products/AP8104b) was selected from the ULK1 region of human Autophagy APG1 (ULK1). A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**APG1 (ULK1) Antibody (Center) Blocking peptide - Protein Information****Name** ULK1 {ECO:0000303|PubMed:9693035, ECO:0000312|HGNC:HGNC:12558}**Function**

Serine/threonine-protein kinase involved in autophagy in response to starvation (PubMed:[18936157](http://www.uniprot.org/citations/18936157), PubMed:[21460634](http://www.uniprot.org/citations/21460634), PubMed:[21795849](http://www.uniprot.org/citations/21795849), PubMed:[23524951](http://www.uniprot.org/citations/23524951), PubMed:[25040165](http://www.uniprot.org/citations/25040165), PubMed:[29487085](http://www.uniprot.org/citations/29487085), PubMed:[31123703](http://www.uniprot.org/citations/31123703)). Acts upstream of phosphatidylinositol 3-kinase PIK3C3 to regulate the formation of autophagophores, the precursors of autophagosomes (PubMed:[18936157](http://www.uniprot.org/citations/18936157), PubMed:[21460634](http://www.uniprot.org/citations/21460634)

target="\_blank">21460634</a>, PubMed:<a href="http://www.uniprot.org/citations/21795849" target="\_blank">21795849</a>, PubMed:<a href="http://www.uniprot.org/citations/25040165" target="\_blank">25040165</a>). Part of regulatory feedback loops in autophagy: acts both as a downstream effector and negative regulator of mammalian target of rapamycin complex 1 (mTORC1) via interaction with RPTOR (PubMed:<a href="http://www.uniprot.org/citations/21795849" target="\_blank">21795849</a>). Activated via phosphorylation by AMPK and also acts as a regulator of AMPK by mediating phosphorylation of AMPK subunits PRKAA1, PRKAB2 and PRKAG1, leading to negatively regulate AMPK activity (PubMed:<a href="http://www.uniprot.org/citations/21460634" target="\_blank">21460634</a>). May phosphorylate ATG13/KIAA0652 and RPTOR; however such data need additional evidences (PubMed:<a href="http://www.uniprot.org/citations/18936157" target="\_blank">18936157</a>). Plays a role early in neuronal differentiation and is required for granule cell axon formation (PubMed:<a href="http://www.uniprot.org/citations/11146101" target="\_blank">11146101</a>). Also phosphorylates SESN2 and SQSTM1 to regulate autophagy (PubMed:<a href="http://www.uniprot.org/citations/25040165" target="\_blank">25040165</a>, PubMed:<a href="http://www.uniprot.org/citations/37306101" target="\_blank">37306101</a>). Phosphorylates FLCN, promoting autophagy (PubMed:<a href="http://www.uniprot.org/citations/25126726" target="\_blank">25126726</a>). Phosphorylates AMBRA1 in response to autophagy induction, releasing AMBRA1 from the cytoskeletal docking site to induce autophagosome nucleation (PubMed:<a href="http://www.uniprot.org/citations/20921139" target="\_blank">20921139</a>). Phosphorylates ATG4B, leading to inhibit autophagy by decreasing both proteolytic activation and delipidation activities of ATG4B (PubMed:<a href="http://www.uniprot.org/citations/28821708" target="\_blank">28821708</a>).

#### **Cellular Location**

Cytoplasm, cytosol. Preautophagosomal structure. Note=Under starvation conditions, is localized to punctate structures primarily representing the isolation membrane that sequesters a portion of the cytoplasm resulting in the formation of an autophagosome.

#### **Tissue Location**

Ubiquitously expressed. Detected in the following adult tissues: skeletal muscle, heart, pancreas, brain, placenta, liver, kidney, and lung

### **APG1 (ULK1) Antibody (Center) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

### **APG1 (ULK1) Antibody (Center) Blocking peptide - Images**

### **APG1 (ULK1) Antibody (Center) Blocking peptide - Background**

Macroautophagy is the major inducible pathway for the general turnover of cytoplasmic constituents in eukaryotic cells, it is also responsible for the degradation of active cytoplasmic enzymes and organelles during nutrient starvation. Macroautophagy involves the formation of double-membrane bound autophagosomes which enclose the cytoplasmic constituent targeted for degradation in a membrane bound structure, which then fuse with the lysosome (or vacuole) releasing a single-membrane bound autophagic bodies which are then degraded within the lysosome (or vacuole). APG1, an autophagy-specific protein kinase, plays a critical role in regulating key elements of the pathway. APG1 stimulates autophagy, leading to autophagy-dependent restriction of cell growth and ultimately cell apoptosis at high levels of activity, and is a negative regulator of mTOR signaling.

### **APG1 (ULK1) Antibody (Center) Blocking peptide - References**

Scott, R., et al., Current Biology 17: 1-11 (2007). Kuroyanagi, H., et al., Genomics 51(1):76-85 (1998).