

**AMPK alpha2 Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP7203a****Specification**

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**AMPK alpha2 Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [P54646](#)**AMPK alpha2 Antibody (C-term) Blocking Peptide - Additional Information**

Gene ID 5563

**Other Names**

5'-AMP-activated protein kinase catalytic subunit alpha-2, AMPK subunit alpha-2, Acetyl-CoA carboxylase kinase, ACACA kinase, Hydroxymethylglutaryl-CoA reductase kinase, HMGCR kinase, PRKAA2, AMPK, AMPK2

**Target/Specificity**

The synthetic peptide sequence is selected from aa 490~508 of human AMPK alpha2.

**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.

**AMPK alpha2 Antibody (C-term) Blocking Peptide - Protein Information**Name PRKAA2 ([HGNC:9377](#))

Synonyms AMPK, AMPK2

**Function**

Catalytic subunit of AMP-activated protein kinase (AMPK), an energy sensor protein kinase that plays a key role in regulating cellular energy metabolism (PubMed:<a href="http://www.uniprot.org/citations/17307971" target="\_blank">17307971</a>, PubMed:<a href="http://www.uniprot.org/citations/17712357" target="\_blank">17712357</a>). In response to reduction of intracellular ATP levels, AMPK activates energy-producing pathways and inhibits energy-consuming processes: inhibits protein, carbohydrate and lipid biosynthesis, as well as cell growth and proliferation (PubMed:<a href="http://www.uniprot.org/citations/17307971" target="\_blank">17307971</a>, PubMed:<a href="http://www.uniprot.org/citations/17712357" target="\_blank">17712357</a>). AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators (PubMed:<a href="http://www.uniprot.org/citations/17307971" target="\_blank">17307971</a>, PubMed:<a href="http://www.uniprot.org/citations/17712357" target="\_blank">17712357</a>).

[17712357](http://www.uniprot.org/citations/17712357)). Regulates lipid synthesis by phosphorylating and inactivating lipid metabolic enzymes such as ACACA, ACACB, GYS1, HMGCR and LIPE; regulates fatty acid and cholesterol synthesis by phosphorylating acetyl-CoA carboxylase (ACACA and ACACB) and hormone-sensitive lipase (LIPE) enzymes, respectively (PubMed: [7959015](http://www.uniprot.org/citations/7959015)). Promotes lipolysis of lipid droplets by mediating phosphorylation of isoform 1 of CHKA (CHKalpha2) (PubMed: [34077757](http://www.uniprot.org/citations/34077757)). Regulates insulin-signaling and glycolysis by phosphorylating IRS1, PFKFB2 and PFKFB3 (By similarity). Involved in insulin receptor/INSR internalization (PubMed: [25687571](http://www.uniprot.org/citations/25687571)). AMPK stimulates glucose uptake in muscle by increasing the translocation of the glucose transporter SLC2A4/GLUT4 to the plasma membrane, possibly by mediating phosphorylation of TBC1D4/AS160 (By similarity). Regulates transcription and chromatin structure by phosphorylating transcription regulators involved in energy metabolism such as CRTC2/TORC2, FOXO3, histone H2B, HDAC5, MEF2C, MLXIPL/ChREBP, EP300, HNF4A, p53/TP53, SREBF1, SREBF2 and PPARGC1A (PubMed: [11518699](http://www.uniprot.org/citations/11518699), PubMed: [11554766](http://www.uniprot.org/citations/11554766), PubMed: [15866171](http://www.uniprot.org/citations/15866171), PubMed: [17711846](http://www.uniprot.org/citations/17711846), PubMed: [18184930](http://www.uniprot.org/citations/18184930)). Acts as a key regulator of glucose homeostasis in liver by phosphorylating CRTC2/TORC2, leading to CRTC2/TORC2 sequestration in the cytoplasm (By similarity). In response to stress, phosphorylates 'Ser-36' of histone H2B (H2BS36ph), leading to promote transcription (By similarity). Acts as a key regulator of cell growth and proliferation by phosphorylating FNIP1, TSC2, RPTOR, WDR24 and ATG1/ULK1: in response to nutrient limitation, negatively regulates the mTORC1 complex by phosphorylating RPTOR component of the mTORC1 complex and by phosphorylating and activating TSC2 (PubMed: [14651849](http://www.uniprot.org/citations/14651849), PubMed: [20160076](http://www.uniprot.org/citations/20160076), PubMed: [21205641](http://www.uniprot.org/citations/21205641)). Also phosphorylates and inhibits GATOR2 subunit WDR24 in response to nutrient limitation, leading to suppress glucose-mediated mTORC1 activation (PubMed: [36732624](http://www.uniprot.org/citations/36732624)). In response to energetic stress, phosphorylates FNIP1, inactivating the non-canonical mTORC1 signaling, thereby promoting nuclear translocation of TFEB and TFE3, and inducing transcription of lysosomal or autophagy genes (PubMed: [37079666](http://www.uniprot.org/citations/37079666)). In response to nutrient limitation, promotes autophagy by phosphorylating and activating ATG1/ULK1 (PubMed: [21205641](http://www.uniprot.org/citations/21205641)). In that process, it also activates WDR45/WIPI4 (PubMed: [28561066](http://www.uniprot.org/citations/28561066)). Phosphorylates CASP6, thereby preventing its autoprocessing and subsequent activation (PubMed: [32029622](http://www.uniprot.org/citations/32029622)). AMPK also acts as a regulator of circadian rhythm by mediating phosphorylation of CRY1, leading to destabilize it (By similarity). May regulate the Wnt signaling pathway by phosphorylating CTNNB1, leading to stabilize it (By similarity). Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin (PubMed: [17486097](http://www.uniprot.org/citations/17486097)). Also phosphorylates CFTR, EEF2K, KLC1, NOS3 and SLC12A1 (PubMed: [12519745](http://www.uniprot.org/citations/12519745), PubMed: [20074060](http://www.uniprot.org/citations/20074060)). Plays an important role in the differential regulation of pro-autophagy (composed of PIK3C3, BECN1, PIK3R4 and UVRAG or ATG14) and non-autophagy (composed of PIK3C3, BECN1 and PIK3R4) complexes, in response to glucose starvation (By similarity). Can inhibit the non-autophagy complex by phosphorylating PIK3C3 and can activate the pro-autophagy complex by phosphorylating BECN1 (By similarity). Upon glucose starvation, promotes ARF6 activation in a kinase-independent manner leading to cell migration (PubMed: [36017701](http://www.uniprot.org/citations/36017701)

target="\_blank">36017701</a>). Upon glucose deprivation mediates the phosphorylation of ACSS2 at 'Ser- 659', which exposes the nuclear localization signal of ACSS2, required for its interaction with KPNA1 and nuclear translocation (PubMed:<a href="http://www.uniprot.org/citations/28552616" target="\_blank">28552616</a>). Upon stress, regulates mitochondrial fragmentation through phosphorylation of MTFR1L (PubMed:<a href="http://www.uniprot.org/citations/36367943" target="\_blank">36367943</a>).

#### **Cellular Location**

Cytoplasm {ECO:0000250|UniProtKB:Q8BRK8}. Nucleus. Note=In response to stress, recruited by p53/TP53 to specific promoters.

### **AMPK alpha2 Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

### **AMPK alpha2 Antibody (C-term) Blocking Peptide - Images**

### **AMPK alpha2 Antibody (C-term) Blocking Peptide - Background**

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the gamma phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains.

### **AMPK alpha2 Antibody (C-term) Blocking Peptide - References**

Blume-Jensen P, et al. Nature 2001. 411: 355.Cantrell D, J. Cell Sci. 2001. 114: 1439.Jiang S Oncogene 2000. 19: 5590.Manning G, et al. Science 2002. 298: 1912.Moller, D, et al. Am. J. Physiol. 1994. 266: C351-C359.Robertson, S. et al. Trends Genet. 2000. 16: 368.Robinson D, et al. Oncogene 2000. 19: 5548.Van der Ven, P, et al. Hum. Molec. Genet. 1993. 2: 1889.Vanhaesebroeck, B, et al. Biochem. J. 2000. 346: 561.Van Weering D, et al. Recent Results Cancer Res. 1998. 154: 271.