

AMPK alpha2 (PRKAA2) Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7411b**Specification**

AMPK alpha2 (PRKAA2) Antibody (C-term) - Product Information

Application	IHC-P, WB,E
Primary Accession	P54646
Other Accession	Q09137 , Q8BRK8
Reactivity	Human
Predicted	Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	62320
Antigen Region	453-483

AMPK alpha2 (PRKAA2) Antibody (C-term) - 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

This AMPK alpha2 (PRKAA2) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 453-483 amino acids from the C-terminal region of human AMPK alpha2 (PRKAA2).

Dilution

IHC-P~~1:10~50

WB~~1:1000

E~~Use at an assay dependent concentration.

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

AMPK alpha2 (PRKAA2) Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

AMPK alpha2 (PRKAA2) Antibody (C-term) - 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:[17307971](#), PubMed:[17712357](#)). 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:[17307971](#), PubMed:[17712357](#)). AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators (PubMed:[17307971](#), PubMed:[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](#)). Promotes lipolysis of lipid droplets by mediating phosphorylation of isoform 1 of CHKA (CHKalpha2) (PubMed:[34077757](#)). Regulates insulin-signaling and glycolysis by phosphorylating IRS1, PFKFB2 and PFKFB3 (By similarity). Involved in insulin receptor/INSR internalization (PubMed:[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](#), PubMed:[11554766](#), PubMed:[15866171](#), PubMed:[17711846](#), PubMed:[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](#), PubMed:[20160076](#), PubMed:[21205641](#)). Also phosphorylates and inhibits GATOR2 subunit WDR24 in response to nutrient limitation, leading to suppress glucose-mediated mTORC1 activation (PubMed:[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](#)). In response to nutrient limitation, promotes autophagy by phosphorylating and activating ATG1/ULK1 (PubMed:[21205641](#)). In that process, it also activates WDR45/WIPI4 (PubMed:[28561066](#)). Phosphorylates CASP6, thereby preventing its autoprocessing and subsequent activation (PubMed:[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](#)). Also phosphorylates CFTR, EEF2K, KLC1, NOS3 and SLC12A1 (PubMed:[12519745](#), PubMed:[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](#)). 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:[28552616](#)). Upon stress, regulates mitochondrial fragmentation through phosphorylation of MTFR1L (PubMed:[36367943](#)).

Cellular Location

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

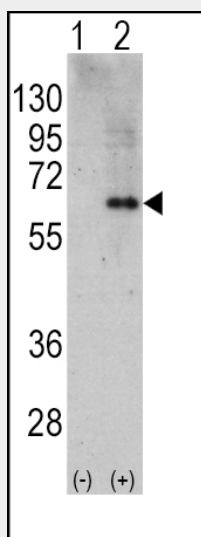
p53/TP53 to specific promoters.

AMPK alpha2 (PRKAA2) Antibody (C-term) - Protocols

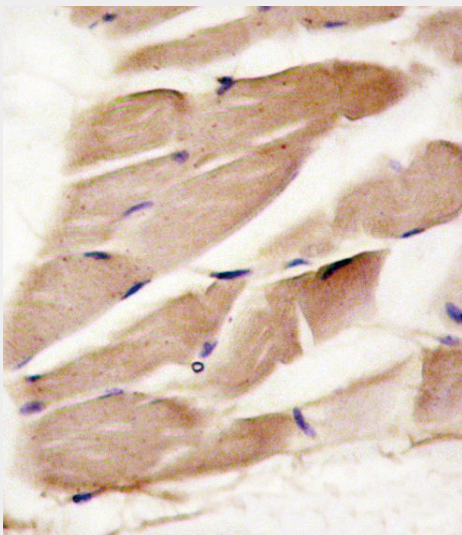
Provided below are standard protocols that you may find useful for product applications.

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

AMPK alpha2 (PRKAA2) Antibody (C-term) - Images



Western blot analysis of PRKAA2 (arrow) using rabbit polyclonal PRKAA2 Antibody (C-term) (RB11657). 293 cell lysates (2 ug/lane) either nontransfected (Lane 1) or transiently transfected with the PRKAA2 gene (Lane 2) (Origene Technologies).



Formalin-fixed and paraffin-embedded human skeletal muscle reacted with PRKAA2 antibody (C-term)(Cat.#AP7411b), which was peroxidase-conjugated to the secondary antibody, followed by DAB staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated.

AMPK alpha2 (PRKAA2) Antibody (C-term) - Background

The protein encoded by this gene is a catalytic subunit of the AMP-activated protein kinase (AMPK). AMPK is a heterotrimer consisting of an alpha catalytic subunit, and non-catalytic beta and gamma subunits. AMPK is an important energy-sensing enzyme that monitors cellular energy status. In response to cellular metabolic stresses, AMPK is activated, and thus phosphorylates and inactivates acetyl-CoA carboxylase (ACC) and beta-hydroxy beta-methylglutaryl-CoA reductase (HMGCR), key enzymes involved in regulating de novo biosynthesis of fatty acid and cholesterol. Studies of the mouse counterpart suggest that this catalytic subunit may control whole-body insulin sensitivity and is necessary for maintaining myocardial energy homeostasis during ischemia.

AMPK alpha2 (PRKAA2) Antibody (C-term) - References

Wyatt,C.N., J. Biol. Chem. 282 (11), 8092-8098 (2007)
Cheung,S.T., Neoplasia 8 (9), 696-701 (2006)
Lee-Young,R.S., Am. J. Physiol. Endocrinol. Metab. 291 (3), E566-E573 (2006)
Gregory,S.G., Nature 441 (7091), 315-321 (2006)