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AMPK alpha2 (PRKAA2) Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP7411b

Specification

AMPK alpha2 (PRKAA2) Blocking Peptide (C-term) - Product Information

Primary Accession	P54646
Other Accession	O09137, Q8BRK8

AMPK alpha2 (PRKAA2) Blocking Peptide (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
The synthetic peptide sequence is selected from aa 468-483 of HUMAN PRKAA2

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 (PRKAA2) Blocking Peptide (C-term) - Protein Information

Name PRKAA2

Synonyms AMPK, AMPK2

AMPK alpha2 (PRKAA2) Blocking Peptide (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) Blocking Peptide (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)

Function

Catalytic subunit of AMP-activated protein kinase (AMPK), an energy sensor protein kinase that plays a key role in regulating cellular energy metabolism. 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. AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators. Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin. 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. Regulates insulin-signaling and glycolysis by phosphorylating IRS1, PFKFB2 and PFKFB3. Involved in insulin receptor/INSR internalization (PubMed:<a hr ef="http://www.uniprot.org/citations/25687 571" target="_blank">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. 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. Acts as a key regulator of glucose homeostasis in liver by phosphorylating CRTC2/TORC2, leading to CRTC2/TORC2 sequestration in the cytoplasm. In response to stress, phosphorylates 'Ser-36' of histone H2B (H2BS36ph), leading to promote transcription. Acts as a key regulator of cell growth and proliferation by phosphorylating TSC2, RPTOR 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. In response to nutrient limitation, promotes autophagy by phosphorylating

and activating ATG1/ULK1. In that process also activates WDR45 (PubMed:28561066). AMPK also acts as a regulator of circadian rhythm by mediating phosphorylation of CRY1, leading to destabilize it. May regulate the Wnt signaling pathway by phosphorylating CTNNB1, leading to stabilize it. Also phosphorylates CFTR, EEF2K, KLC1, NOS3 and SLC12A1. 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. Can inhibit the non-autophagy complex by phosphorylating PIK3C3 and can activate the pro-autophagy complex by phosphorylating BECN1 (By similarity).

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

AMPK alpha2 (PRKAA2) Blocking Peptide (C-term) - Protocols

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

- Blocking Peptides