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phospho-AMPK alpha (Thr172) Recombinant Rabbit mAb

Catalog # AP96647

Product Information

Application	WB
Reactivity	Human
Predicted	Rat, Mouse
Host	Rabbit
Clonality	Recombinant
Calculated MW	64 KDa
Physical State	Liquid
Immunogen	A synthesized peptide derived from human AMPK alpha 1 around the phosphorylation site of T183
Epitope Specificity	LR-pT-SC
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS(pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the protein kinase superfamily. CAMK Ser/Thr protein kinase family. SNF1 subfamily. Contains 1 protein kinase domain.
SUBUNIT	AMPK is a heterotrimer of an alpha catalytic subunit (PRKAA1 or PRKAA2), a beta (PRKAB1 or PRKAB2) and a gamma non-catalytic subunits (PRKAG1, PRKAG2 or PRKAG3). Interacts with FNIP1 and FNIP2.
Post-translational modifications	Ubiquitinated. Phosphorylated at Thr-183 by STK11/LKB1 in complex with STE20-related adapter-alpha (STRADA) pseudo kinase and CAB39. Also phosphorylated at Thr-183 by CAMKK2; triggered by a rise in intracellular calcium ions, without detectable changes in the AMP/ATP ratio. CAMKK1 can also phosphorylate Thr-183, but at much lower lvel. Dephosphorylated by protein phosphatase 2A and 2C (PP2A and PP2C). Phosphorylated by ULK1 and ULK2; leading to negatively regulate AMPK activity and suggesting the existence of a regulatory feedback loop between ULK1, ULK2 and AMPK.
DISEASE	Defects in CRYAB are the cause of myofibrillar alpha-B crystallin-related (MFM-CRYAB) [MIM:608810]. A neuromuscular disorder that results in weakness of the proximal and distal limb muscles, weakness of the neck, velopharynx and trunk muscles, hypetrophic cardiomyopathy, and cataract in a subset of patients.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	The protein encoded by this gene belongs to the ser/thr protein kinase family. It is the catalytic subunit of the 5'-prime-AMP-activated protein kinase (AMPK). AMPK is a cellular energy sensor conserved in all eukaryotic cells. The kinase activity of AMPK is activated by the stimuli that increase the cellular AMP/ATP ratio. AMPK regulates the activities of a number of key metabolic enzymes through phosphorylation. It protects cells from stresses that cause ATP depletion by switching off ATP-consuming biosynthetic pathways. Alternatively spliced transcript variants encoding distinct isoforms have been observed. [provided by RefSeq, Jul 2008]

Additional Information

Gene ID	5562
Other Names	AMPKalpha1; C130083N04Rik; AAPK1_MOUSE; Prkaa1; AMPK subunit alpha-1; Acetyl-CoA carboxylase kinase (ACACA kinase); Hydroxymethylglutaryl-CoA reductase kinase (HMGCR kinase); Tau-protein kinase PRKAA1; 2.7.11.1; AAPK1_RAT; Ampk1; AMPK; AMPK alpha 1; AMPKa1; protein kinase AMP-activated catalytic subunit alpha 1; protein kinase, AMP-activated, alpha 1 catalytic subunit; AMPK, alpha, 1; PRKAA1 | AMPK alpha-1 (phospho-T172); p-AMPK alpha-1; phospho-AMPK alpha-1; p-AMPK1; PRKAA1 | AMPK alpha-1 (phospho-Thr172)
Dilution	WB=1:500-2000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Name	phospho-AMPK alpha (Thr172) Recombinant Rabbit mAb
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. 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: 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 ULK1. 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 has tau-protein kinase activity: in response to amyloid beta A4 protein (APP) exposure, activated by CAMKK2, leading to phosphorylation of MAPT/TAU; however the relevance of such data remains unclear in vivo. Also phosphorylates CFTR, EEF2K, KLC1, NOS3 and SLC12A1.

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