10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

AMPKα Antibody
Rabbit Polyclonal Antibody
Catalog # ABV10089

Specification

AMPKα Antibody - Product Information

Application	**WB, IHC, E**
Primary Accession	Q13131
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**64009**

AMPKα Antibody - Additional Information

Gene ID 5562

Application & Usage — **The antibody can be used for ELISA (0.25 µg/ml), Western blotting (2.5 – 5.0 µg/ml) and Immunohistochemistry (5 -25 µg/ml).**

Other Names
Protein kinase AMP-activated alpha-1 catalytic subunit, AMPK alpha-1 chain,

Target/Specificity
AMPKα

Antibody Form
Liquid

Appearance
Colorless liquid

Formulation
100 µg (0.25 mg/ml) purified rabbit Ig polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide.

Handling
The antibody solution should be gently mixed before use.

Reconstitution & Storage
-20 °C

Background Descriptions

Precautions
AMPKα Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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AMPKα Antibody - Protein Information

Name PRKAA1

Synonyms AMPK1

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, PubMed:24563466, PubMed:37821951). 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 (By similarity). 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). 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:11554766, PubMed:11518699, 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:18439900, 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 also activates WDR45/WIPI4 (PubMed:28561066). Phosphorylates CASP6, thereby preventing its autoprocessing and subsequent activation (PubMed:32029622). In response to nutrient limitation, phosphorylates transcription factor FOXO3 promoting FOXO3 mitochondrial import (By similarity). Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin (PubMed:17486097). 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 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 (By similarity). Also phosphorylates CFTR, EEF2K, KLC1, NOS3 and SLC12A1 (PubMed:20074060, PubMed:12519745). Regulates hepatic lipogenesis. Activated via SIRT3, represses sterol regulatory element- binding protein (SREBP) transcriptional activities and ATP-consuming lipogenesis to restore cellular energy balance.

Cellular Location
Cytoplasm. Nucleus Note=In response to stress, recruited by p53/TP53 to specific promoters.

AMPKα Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

AMPKα Antibody - Images

AMPKα Antibody - Background

AMPKα (PRKAA1) 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 thro μgh phosphorylation. It protects cells from stresses that cause ATP depletion by switching off ATP-consuming biosynthetic pathways.