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PRKAA1 Antibody (Ascites)

Mouse Monoclonal Antibody (Mab)
Catalog # AM1858a

Product Information

Application	IHC-P, WB, E
Primary Accession	Q13131
Other Accession	P54645, NP_006242.5
Reactivity	Human
Predicted	Rat
Host	Mouse
Clonality	Monoclonal
Isotype	IgG1,K
Clone Names	167CT22.1.6
Calculated MW	64009

Additional Information

Gene ID	5562
Other Names	5'-AMP-activated protein kinase catalytic subunit alpha-1, AMPK subunit alpha-1, Acetyl-CoA carboxylase kinase, ACACA kinase, Hydroxymethylglutaryl-CoA reductase kinase, HMGCR kinase, Tau-protein kinase PRKAA1, PRKAA1, AMPK1
Target/Specificity	This PRKAA1 Monoclonal antibody was raised using purified His-tagged recombinant human PRKAA1.
Dilution	IHC-P~~1:100~500 WB~~1:8000 E~~Use at an assay dependent concentration.
Format	Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.
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	PRKAA1 Antibody (Ascites) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PRKAA1 (HGNC:9376)
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:31492851, PubMed:37821951, PubMed:40233740). 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: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: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, it 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:12519745, PubMed:20074060). Regulates hepatic lipogenesis. Activated via SIRT3, represses sterol regulatory element- binding protein (SREBP) transcriptional activities and ATP-consuming lipogenesis to restore cellular energy balance. Upon stress, regulates mitochondrial fragmentation through phosphorylation of MTFR1L (PubMed:36367943). Phosphorylates ALDH7A1 in response to cellular stress,

such as hypoxia or ferroptotic stress, promoting ALDH7A1 recruitment to membranes (PubMed:31492851, PubMed:40233740).

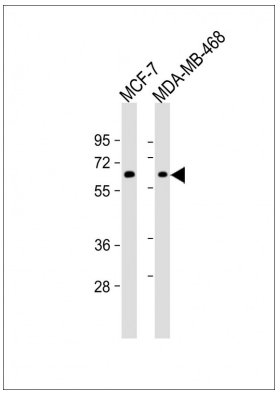
Cellular Location

Cytoplasm. Nucleus Late endosome {ECO:0000250|UniProtKB:Q5EG47}. Note=In response to stress, recruited by p53/TP53 to specific promoters. Translocates to late endosomes in response to calorie restriction (By similarity) {ECO:0000250|UniProtKB:Q5EG47, ECO:0000269|PubMed:15866171}

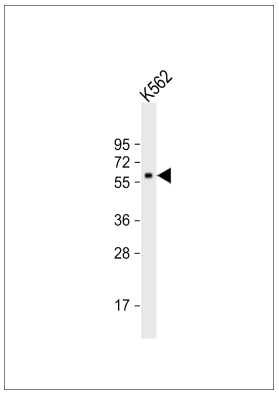
Background

Responsible for the regulation of fatty acid synthesis by phosphorylation of acetyl-CoA carboxylase. It also regulates cholesterol synthesis via phosphorylation and inactivation of hormone-sensitive lipase and hydroxymethylglutaryl-CoA reductase. Appears to act as a metabolic stress-sensing protein kinase switching off biosynthetic pathways when cellular ATP levels are depleted and when 5'-AMP rises in response to fuel limitation and/or hypoxia. This is a catalytic subunit.

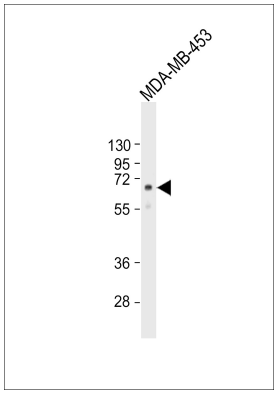
Images



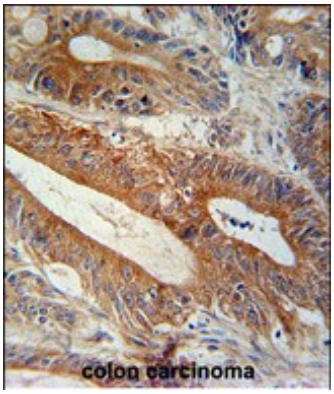
All lanes : Anti-PRKAA1 Antibody at 1:1000 dilution Lane 1: MCF-7 whole cell lysate Lane 2: MDA-MB-468 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 64 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Anti-PRKAA1 Antibody (Ascites) at 1:8000 dilution + K562 whole cell lysate Secondary Goat Anti-mouse IgG, (H+L),Peroxidase conjugated at 1/10000 dilution. Predicted band size : 64009 Da Blocking/Dilution buffer: 5% NFDM/TBST.



Anti-PRKAA1 Antibody (Ascites) at 1:4000 dilution + MDA-MB-453 whole cell lysate Secondary Goat Anti-mouse IgG, (H+L),Peroxidase conjugated at 1/10000 dilution. Predicted band size : 64009 Da Blocking/Dilution buffer: 5% NFDM/TBST.



PRKAA1 Monoclonal(Ascites) (Cat. #AM1858a) immunohistochemistry analysis in formalin fixed and paraffin embedded human colon carcinoma followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the PRKAA1 Monoclonal(Ascites) for immunohistochemistry. Clinical relevance has not been evaluated.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.