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PRKAG2 Rabbit pAb

Catalog # AP99223

Product Information

Application	IHC-P, IHC-F, IF
Primary Accession	Q9UGJ0
Other Accession	Q9UGJ0
Reactivity	Rat
Predicted	Pig, Human, Mouse, Chicken, Dog, Horse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	63066
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human PRKAG2
Epitope Specificity	265-370/569
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 5'-AMP-activated protein kinase gamma subunit family.Contains 4 CBS domains.
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	Phosphorylated by ULK1; leading to negatively regulate AMPK activity and suggesting the existence of a regulatory feedback loop between ULK1 and AMPK. Phosphorylated upon DNA damage, probably by ATM or ATR.
DISEASE	Defects in PRKAG2 are the cause of cardiomyopathy, familial hypertrophic type 6 (CMH6) [MIM:600858]. A hereditary heart disorder characterized by ventricular hypertrophy, which is usually asymmetric and often involves the interventricular septum. The symptoms include dyspnea, syncope, collapse, palpitations, and chest pain. They can be readily provoked by exercise. The disorder has inter- and intrafamilial variability ranging from benign to malignant forms with high risk of cardiac failure and sudden cardiac death. CMH6 patients present Wolff-Parkinson-White ventricular preexcitation, enlarged myocytes without myofiber disarray, and glycogen-containing cytosolic vacuoles within cardiomyocytes. Defects in PRKAG2 are a cause of glycogen storage disease of heart lethal congenital (GSDH) [MIM:261740]; also known as phosphorylase kinase deficiency of heart or congenital nonlysosomal cardiac glycogenosis. GSDH is a rare disease which leads to death within a few weeks to a few months after birth, through heart failure and respiratory compromise.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	AMPK is a heterotrimeric complex comprising a catalytic a subunit and regulatory b and g subunits. It protects cells from stresses that cause ATP depletion by switching off ATP-consuming biosynthetic pathways. AMPK is activated by high AMP and low ATP through a mechanism involving allosteric regulation, promotion of phosphorylation by an upstream protein kinase known as AMPK kinase and inhibition of dephosphorylation. Activated AMPK

can phosphorylate and regulate in vivo hydroxy-methylglutaryl-CoA reductase and acetyl-CoA carboxylase, which are key regulatory enzymes of sterol synthesis and fatty acid synthesis, respectively. The human AMPKa1 and AMPKa2 genes encode 548 amino acid and 552 amino acid proteins, respectively. Human AMPKb1 encodes a 271 amino acid protein and human AMPKb2 encodes a 272 amino acid protein. The human AMPKg1 gene encodes a 331 amino acid protein. Human AMPKg2 and AMPKg3, which are 569 and 492 amino acid proteins, respectively, contain unique N-terminal domains and may participate directly in the binding of AMP within the AMPK complex.

Additional Information

Gene ID	51422
Other Names	AAKG; AAKG2; CMH6; H91620p; WPWS; 2410051C13Rik; AAKG2_HUMAN; PRKAG2; AMPK gamma2; AMPK subunit gamma-2; AAKG2_MOUSE; protein kinase AMP-activated non-catalytic subunit gamma 2; protein kinase, AMP-activated, gamma 2 non-catalytic subunit
Dilution	IHC-P=1:100-500,IHC-F=1:100-500,IF=1:100-500
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.

Protein Information

Name	PRKAG2
Function	AMP/ATP-binding subunit of AMP-activated protein kinase (AMPK), an energy sensor protein kinase that plays a key role in regulating cellular energy metabolism (PubMed:14722619, PubMed:24563466). 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:14722619, PubMed:24563466). AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators (PubMed:14722619, PubMed:24563466). Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin (PubMed:14722619, PubMed:24563466). Gamma non-catalytic subunit mediates binding to AMP, ADP and ATP, leading to activate or inhibit AMPK: AMP-binding results in allosteric activation of alpha catalytic subunit (PRKAA1 or PRKAA2) both by inducing phosphorylation and preventing dephosphorylation of catalytic subunits (PubMed:14722619, PubMed:24563466). ADP also stimulates phosphorylation, without stimulating already phosphorylated catalytic subunit (PubMed:14722619, PubMed:24563466). ATP promotes dephosphorylation of catalytic subunit, rendering the AMPK enzyme inactive (PubMed:14722619, PubMed:24563466).
Tissue Location	Isoform B is ubiquitously expressed except in liver and thymus. The highest level is detected in heart with abundant expression in placenta and testis

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