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PRKAG2 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP7049a

Specification

PRKAG2 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession Q9UGJ0

PRKAG2 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 51422

Other Names
5'-AMP-activated protein kinase subunit gamma-2, AMPK gamma2, AMPK subunit gamma-2, H91620p, PRKAG2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7049a was selected from the N-term region of human PRKAG2 . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

PRKAG2 Antibody (N-term) Blocking Peptide - 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:<a

href="http://www.uniprot.org/citations/24563466" target="_blank">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

PRKAG2 Antibody (N-term) Blocking Peptide - Protocols

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

- Blocking Peptides

PRKAG2 Antibody (N-term) Blocking Peptide - Images

PRKAG2 Antibody (N-term) Blocking Peptide - Background

AMP-activated protein kinase (AMPK) is a heterotrimeric protein composed of a catalytic alpha subunit, a noncatalytic beta subunit, and a noncatalytic regulatory gamma subunit. Various forms of each of these subunits exist, encoded by different genes. AMPK is an important energy-sensing enzyme that monitors cellular energy status and functions by inactivating key enzymes involved in regulating de novo biosynthesis of fatty acid and cholesterol. This gene is a member of the AMPK gamma subunit family and encodes a protein with four cystathionine beta-synthase domains. Mutations in this gene have been associated with ventricular pre-excitation (Wolff-Parkinson-White syndrome), progressive conduction system disease and cardiac hypertrophy. Alternate transcriptional splice variants, encoding different isoforms, have been characterized.

PRKAG2 Antibody (N-term) Blocking Peptide - References

Vaughan, C.J., et al., J. Cardiovasc. Electrophysiol. 14(3):263-268 (2003).Daniel, T., et al., J. Biol. Chem. 277(52):51017-51024 (2002).Gollob, M.H., et al., Curr Opin Cardiol 17(3):229-234 (2002).Gollob, M.H., et al., Circulation 104(25):3030-3033 (2001).Lang, T., et al., Genomics 70(2):258-263 (2000).