

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP7048c**Specification**

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Product Information

Primary Accession [P54619](#)
Other Accession [NP_997626](#)

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Additional Information

Gene ID 5571

Other Names

5'-AMP-activated protein kinase subunit gamma-1, AMPK gamma1, AMPK subunit gamma-1, AMPKg, PRKAG1

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP7048c](/product/products/AP7048c) was selected from the Center region of human PRKAG1. 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.

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Protein Information

Name PRKAG1

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: [21680840](http://www.uniprot.org/citations/21680840), PubMed: [24563466](http://www.uniprot.org/citations/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: [21680840](http://www.uniprot.org/citations/21680840), PubMed: [24563466](http://www.uniprot.org/citations/24563466)). AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators (PubMed: [24563466](#)).

href="http://www.uniprot.org/citations/21680840" target="_blank">21680840, PubMed:24563466). Also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin (PubMed:21680840, 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:21680840, PubMed:24563466). ADP also stimulates phosphorylation, without stimulating already phosphorylated catalytic subunit (PubMed:21680840, PubMed:24563466). ATP promotes dephosphorylation of catalytic subunit, rendering the AMPK enzyme inactive (PubMed:21680840, PubMed:24563466).

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Images

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - Background

PRKAG1 is a regulatory subunit of the AMP-activated protein kinase (AMPK). AMPK is a heterotrimer consisting of an alpha catalytic subunit, and non-catalytic beta and gamma subunits. AMPK is an important energy-sensing enzyme that monitors cellular energy status. In response to cellular metabolic stresses, AMPK is activated, and thus phosphorylates and inactivates acetyl-CoA carboxylase (ACC) and beta-hydroxy beta-methylglutaryl-CoA reductase (HMGCR), key enzymes involved in regulating de novo biosynthesis of fatty acid and cholesterol. This subunit is one of the gamma regulatory subunits of AMPK.

AMPK gamma (PRKAG1) Antibody (Center) Blocking peptide - References

Minokoshi, Y., et al., Nature 428(6982):569-574 (2004). Hamilton, S.R., et al., FEBS Lett. 500(3):163-168 (2001). Zidovetzki, R., et al., AIDS Res. Hum. Retroviruses 14(10):825-833 (1998). Reinton, N., et al., Genomics 49(2):290-297 (1998). Stapleton, D., et al., FEBS Lett. 409(3):452-456 (1997).