

**PRKAG3**  
**Purified Mouse Monoclonal Antibody**  
**Catalog # AO2518a****Specification**

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**PRKAG3 - Product Information**

Application	<b>E, WB</b>
Primary Accession	<a href="#">O9UGI9</a>
Reactivity	<b>Human</b>
Host	<b>Mouse</b>
Clonality	<b>Monoclonal</b>
Isotype	<b>Mouse IgG1</b>
Calculated MW	<b>54.3kDa KDa</b>

**Immunogen**

Purified recombinant fragment of human PRKAG3 (AA: 9-151) expressed in E. Coli.

**Formulation**

Purified antibody in PBS with 0.05% sodium azide

**PRKAG3 - Additional Information**

**Gene ID** 53632

**Other Names**

AMPKG3

**Dilution**

E~~ 1/10000

WB~~ 1/500 - 1/2000

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

PRKAG3 is for research use only and not for use in diagnostic or therapeutic procedures.

**PRKAG3 - Protein Information**

**Name** PRKAG3

**Synonyms** AMPKG3

**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:<a href="http://www.uniprot.org/citations/14722619" target="\_blank">14722619</a>, PubMed:<a

href="http://www.uniprot.org/citations/17878938" target="\_blank">17878938</a>, PubMed:<a href="http://www.uniprot.org/citations/24563466" target="\_blank">24563466</a>). 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. AMPK acts via direct phosphorylation of metabolic enzymes, and by longer-term effects via phosphorylation of transcription regulators. AMPK also acts as a regulator of cellular polarity by remodeling the actin cytoskeleton; probably by indirectly activating myosin. The AMPK gamma3 subunit is a non-catalytic subunit with a regulatory role in muscle energy metabolism (PubMed:<a href="http://www.uniprot.org/citations/17878938" target="\_blank">17878938</a>). It mediates binding to AMP, ADP and ATP, leading to AMPK activation or inhibition: AMP-binding results in allosteric activation of alpha catalytic subunit (PRKAA1 or PRKAA2) both by inducing phosphorylation and preventing dephosphorylation of catalytic subunits. ADP also stimulates phosphorylation, without stimulating already phosphorylated catalytic subunit. ATP promotes dephosphorylation of catalytic subunit, rendering the AMPK enzyme inactive.

**Tissue Location**

Skeletal muscle, with weak expression in heart and pancreas

**PRKAG3 - Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**PRKAG3 - Images**

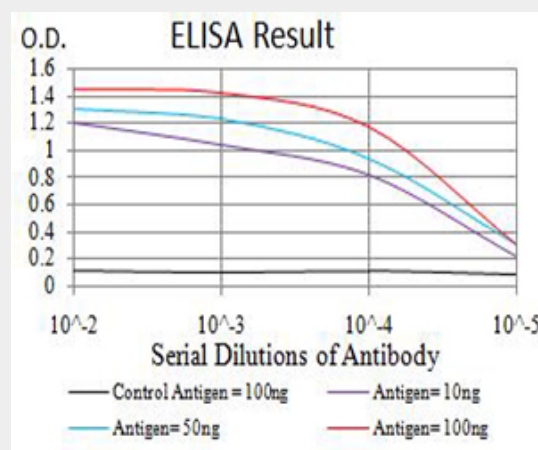


Figure 1: Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line: Antigen (100 ng)

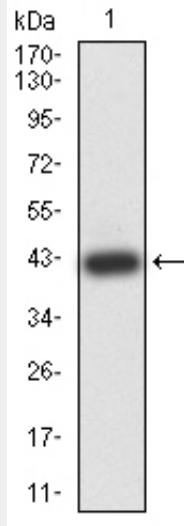


Figure 2:Western blot analysis using PRKAG3 mAb against human PRKAG3 (AA: 9-151) recombinant protein. (Expected MW is 41.1 kDa)

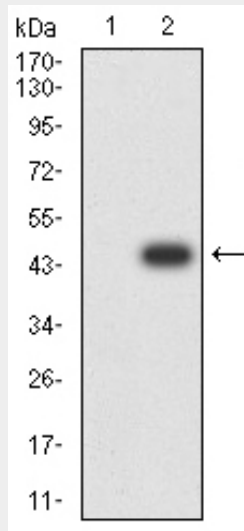


Figure 3:Western blot analysis using PRKAG3 mAb against HEK293 (1) and PRKAG3 (AA: 9-151)-hlgGfc transfected HEK293 (2) cell lysate.

**PRKAG3 - References**

- 1.Diabetologia. 2010 Sep;53(9):1986-97.2.PLoS One. 2007 Sep 19;2(9):e903.