

# **KCNMB1** Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP14747c

### **Specification**

# KCNMB1 Antibody (Center) - Product Information

**Application** WB.E **Primary Accession** 016558 NP 004128.1 Other Accession Reactivity Human Host **Rabbit** Clonality **Polyclonal** Isotype Rabbit IgG Calculated MW 21797 Antigen Region 43-72

### KCNMB1 Antibody (Center) - Additional Information

#### **Gene ID 3779**

#### **Other Names**

Calcium-activated potassium channel subunit beta-1, BK channel subunit beta-1, BKbeta, BKbeta1, Hbeta1, Calcium-activated potassium channel, subfamily M subunit beta-1, Calcium-activated potassium channel subunit beta, Charybdotoxin receptor subunit beta-1, K(VCA)beta-1, Maxi K channel subunit beta-1, Slo-beta-1, Slo-beta, KCNMB1

### Target/Specificity

This KCNMB1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 43-72 amino acids from the Central region of human KCNMB1.

# **Dilution**

WB~~1:1000

E~~Use at an assay dependent concentration.

#### **Format**

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

#### 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**

KCNMB1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

#### KCNMB1 Antibody (Center) - Protein Information



#### Name KCNMB1

Function Regulatory subunit of the calcium activated potassium KCNMA1 (maxiK) channel. Modulates the calcium sensitivity and gating kinetics of KCNMA1, thereby contributing to KCNMA1 channel diversity. Increases the apparent Ca(2+)/voltage sensitivity of the KCNMA1 channel. It also modifies KCNMA1 channel kinetics and alters its pharmacological properties. It slows down the activation and the deactivation kinetics of the channel. Acts as a negative regulator of smooth muscle contraction by enhancing the calcium sensitivity to KCNMA1. Its presence is also a requirement for internal binding of the KCNMA1 channel opener dehydrosoyasaponin I (DHS-1) triterpene glycoside and for external binding of the agonist hormone 17-beta-estradiol (E2). Increases the binding activity of charybdotoxin (CTX) toxin to KCNMA1 peptide blocker by increasing the CTX association rate and decreasing the dissociation rate.

#### **Cellular Location**

Membrane; Multi-pass membrane protein.

#### **Tissue Location**

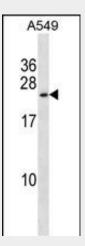
Abundantly expressed in smooth muscle. Low levels of expression in most other tissues. Within the brain, relatively high levels found in hippocampus and corpus callosum

# KCNMB1 Antibody (Center) - Protocols

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

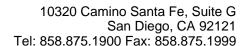
- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

# KCNMB1 Antibody (Center) - Images



KCNMB1 Antibody (Center) (Cat. #AP14747c) western blot analysis in A549 cell line lysates (35ug/lane). This demonstrates the KCNMB1 antibody detected the KCNMB1 protein (arrow).

## KCNMB1 Antibody (Center) - Background





MaxiK channels are large conductance, voltage and calcium-sensitive potassium channels which are fundamental to the control of smooth muscle tone and neuronal excitability. MaxiK channels can be formed by 2 subunits: the pore-forming alpha subunit and the product of this gene, the modulatory beta subunit. Intracellular calcium regulates the physical association between the alpha and beta subunits.

# KCNMB1 Antibody (Center) - References

Bailey, S.D., et al. Diabetes Care (2010) In press: Xie, M.J., et al. Am. J. Physiol., Cell Physiol. 298 (6), C1489-C1500 (2010): Yokoyama, K., et al. Nephron Clin Pract 115 (4), C237-C243 (2010): Long, X., et al. J. Biol. Chem. 284(48):33671-33682(2009) Talmud, P.J., et al. Am. J. Hum. Genet. 85(5):628-642(2009)