

Anti-Potassium Channel K_{Ca}3.1

(Intermediate-conductance Ca²⁺-activated Potassium Channel 4; SK4; IK_{Ca}1; KCNN4)

Developed in Rabbit, Affinity Isolated Antibody

Product Number **P4997**

Product Description

Anti-Potassium Channel K_{Ca}3.1 was developed in rabbit using a synthetic peptide RQVRLKHRKLREQV(C), corresponding to amino acid residues 350-363 of rat K_{Ca}3.1 as the immunogen. This sequence has 100% homology with human, mouse and pig. The antibody was affinity isolated on immobilized immunogen.

Anti-Potassium Channel K_{Ca}3.1 recognizes K_{Ca}3.1 from human and rat samples by Western blotting.

The action of potassium (K⁺) channels is regulated by voltage, calcium and a variety of neurotransmitters. Each subfamily generally consists of a primary pore forming α subunit that is associated with several regulatory subunits.¹ To date, some 70 different genes that encode the α subunits of K⁺ channels have been identified. Recently, the crystal structure of the K⁺ channels has been identified.²

The vast family of K⁺ channels has been subdivided into the three main subfamilies: the 2 TM, 4 TM and 6 TM K⁺ channels.³ The 6 TM family of K⁺ channels includes the voltage-gated K⁺ (K_v) channels, the KCNQ, the EAG, the calcium-activated K⁺ (Slo) subfamily (which is actually a 7 TM not a 6 TM type of channel) and the calcium-activated SK subfamily.

Structurally, the calcium-activated K⁺ channels can be divided into two groups: the small or intermediate conductance potassium channels (SK/IK), and the high conductance potassium channels (BK). BK channels are expressed in virtually all cell types where they cause hyperpolarization and help to connect between intracellular Ca²⁺ signaling pathways and membrane excitability. K_{Ca}1.1 channels have a crucial role in smooth muscle contractility, neuronal spike shaping and neurotransmitter release.^{4,5}

Small or intermediate conductance calcium-activated K⁺ channels (SK/IK) are responsible for the slow after-hyperpolarization following an action potential. These channels are distinguished from BK channels by their high sensitivity to intracellular calcium, low conductivity and weak or negligible voltage-dependence.⁶ Recently, four SK channels (SK1-4) have been cloned.⁷⁻⁹ SK1 and SK2 channels have highest densities in subregions of the hippocampus and neocortex, while SK3 channels are distributed more diffusely in these brain regions and predominantly expressed in phylogenetically older brain regions.¹⁰ The SK3 channels appear to be implicated in the regulation of electrical excitability and neurotransmitter release of monoaminergic neurons and have been implicated in schizophrenia, ataxia and anorexia nervosa.¹¹ The SK4 (also called IK_{Ca}1) possess intermediate conductivity and are expressed in many tissue types including red blood cells and T lymphocytes.¹²

Reagent

The antibody is supplied as lyophilized powder from phosphate buffered saline containing 1% bovine serum albumin and 0.05% sodium azide as preservative.

Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices

Preparation Instructions

Reconstitute the lyophilized vial with either 0.05 ml or 0.2 ml deionized water, depending on the package size. Further dilutions should be made using a carrier protein such as BSA (1%).

Product Information

sigma-aldrich.com

3050 Spruce Street, Saint Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com *sigma-aldrich.com*

Storage/Stability

Store at –20 °C. For extended storage, freeze in working aliquots. Avoid repeated freezing and thawing. Storage in “frost-free” freezers is not recommended. Centrifuge before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

The recommended working dilution is 1:200 for immunoblotting.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

1. Alexander, S.P., et al., Br. J. Pharmacol., **141**, Suppl 1:S1-S126 (2004).
2. MacKinnon, R., FEBS Letters, **555**, 62-65 (2003).
3. Gutman, G.A., et al., Pharmacol. Rev., **55**, 583-586 (2003).
4. Knaus, H.G., et al., J. Neurosci., **16**, 955-963 (1996).
5. Heppner, T.J., et al., Am. J. Physiol., **273**, C110-C117 (1997).
6. Vergara, C., et al., Curr. Opin. Neurobiol., **8**, 321-329 (1998).
7. Chandy, K.G., et al., Mol. Psychiatry, **3**, 32-37 (1998).
8. Joiner, W.J., et al., Proc. Natl. Acad. Sci. USA, **94**, 11013-11018 (1997).
9. Kohler, M., et al., *Science*, **273**, 1709-1714 (1996).
10. Sailer C.A., et al., J. Neurosci., **22**, 9698-9707 (2002).
11. Tomita, H., et al., Mol. Psychiatry, **8**, 524-535 (2003).
12. Khanna, R., et al., J. Biol. Chem., **274**, 14838-14849 (1999).

MCT, PHC, CY11/21-1