

Product Information

Anti-Potassium Channel KCNQ2 (K_v7.2, Voltage-gated potassium channel QKT subfamily member 2)

Developed in Rabbit
Affinity Isolated Antibody

Product Number **P 4371**

Product Description

Anti-Potassium Channel KCNQ2 (K_v7.2) is developed in rabbit using a highly purified peptide RGPTITDKDR-TKGPAAE, corresponding to amino acid residues 578-593 of rat KCNQ2 with an additional N-terminal cysteine as the immunogen. The epitope is highly conserved in human (18/19 residues identical) antigens. The antibody was affinity isolated on immobilized immunogen.

Anti-Potassium Channel KCNQ2 specifically recognizes KCNQ2 protein in KCNQ2 transfected HEK-293 cells by immunoblotting. It does not cross react with KCNQ3 or other QKT proteins. There are at least 9 recognized splice variants of rat KCNQ2. This antibody recognizes all except splice variants B and G.

Chloride channels have several functions including: (1) regulation of cell volume; (2) membrane potential stabilization; (3) signal transduction; and (4) transepithelial transport. The CLC chloride channel family (which includes voltage-gated chloride channels) represents one of the structural families of chloride channels. Mammals have at least nine different members.¹ CLC-2 channels exhibit differential brain distribution and are implicated in regulating and maintaining the chloride gradient in cells that exhibit primarily inhibitory GABA_A responses.² CLC-3 channels are important in cardiac function and their volume sensitivity may be due to PKC/PKA modulated phosphorylation.³

Voltage-gated sodium channels (VGSCs) are present in most excitable cells. In neuronal tissue, they are responsible for generating and propagating action potentials. Brain VGSCs are heteromers of $\alpha\beta_1\beta_2$ subunits. Of these, the α subunit forms the channel pore. Twelve α subunit genes have been identified.⁴ VGSCs have been implicated in numerous neurological and cardiac disorders. Further, VGSCs are important in mediating many therapeutic drug effects (including the actions of anesthetics, antiarrhythmics and antiepileptics).^{5,6}

Potassium channels contribute to maintaining cell volume, membrane potential, neuronal excitability and the secretion of transmitters, salt and hormones. Two families of potassium channels have been identified. One family includes the inwardly rectifying potassium channels. The other family includes: voltage sensing (KV); big conductance, calcium activated (BK_{CA}); and small conductance, calcium activated (SK) potassium channels. In neuronal tissue, BK and SK channels modulate the action potential duration, speed of repolarization and the after hyperpolarization.^{7,8} These channels are implicated both in therapeutic drug effects and also in disease.^{7,8,9} KV channels have been implicated in activity-dependent, plastic changes in neuronal tissue.^{10,11} The KCNQ family of voltage-gated potassium channels includes 5 known members: KCNQ1 to KCNQ5.¹² KCNQ2/KCNQ3 heteromultimers are believed to be the molecular correlates of the so-called M current.¹³ ERG or EAG (ether-a-go-go-related gene) is similar to the delayed rectifier channel and is important in cardiac function and may also play a role in certain cardiac arrhythmias.¹⁴ The role of EAG channels in the brain is still largely unknown.¹⁵

Many subunits that form the ion channels have been cloned and expressed. Although much has been learned about the structure and function of the ion channels, much more remains to be determined about their physiological roles and also their roles in mediating therapeutic drug effects.

Monovalent ion channels are being associated with a growing number of diseases.^{6,16} Thus, further research is required to determine the physiological function and role of Cl, K and Na channel subtypes as well as the ion channels themselves in the hopes of discovering new treatments for these pathologies.

Reagents

Anti-Potassium Channel KCNQ2 is supplied lyophilized from phosphate buffered saline, pH 7.4, with 1% bovine serum albumin and 0.025 % sodium azide as preservative.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling.

Preparation Instructions

Reconstitute the lyophilized vial with 0.05 ml or 0.2 ml deionized water, depending on the package size purchased. Antibody dilutions should be made in buffer containing 1-3 % bovine serum albumin.

Storage/Stability

Lyophilized powder can be stored intact at room temperature for several weeks. For extended storage, it should be stored at -20°C or below. The reconstituted solution can be stored at 4°C for up to 2 weeks. For longer storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in "frost-free" freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Centrifuge all antibody preparations before use ($10000 \times g$ 5 min). Working dilution samples should be discarded if not used within 12 hours.

Product Profile

The recommended working dilution is 3.5 $\mu\text{g/ml}$ (1:200) for immunoblotting.

Note: In order to obtain best results and assay sensitivities of different techniques and preparations, we recommend determining optimal working dilutions by titration test.

References

1. Jentsch, T.J. et al., *J. Physiol.*, **482**, 19S (1995).
2. Staley, K. et al., *Neuron*, **17**, 543 (1996).
3. Nagasaki M. et al., *J.Physiol.*, **523**, 705 (2000).
4. Jeong, S.Y. et al., *Biochem. Biophys. Res.Commun.*, **267**, 262 (2000).
5. Catterall, W.A., *Adv. Neurol.*, **79**, 441 (1999).
6. Vincent, G.M. et al., *Cardiol. Rev.*, **7**, 44 (1999).
7. Scholtz, A. et al., *J. Physiol.*, **513**, 55 (1998).
8. Dreixler, J.C. et al., *Anesth. Analg.*, **90**, 727 (2000).
9. Bond, C.T. et al., *Ann. N.Y. Acad. Sci.*, **868**, 370 (1999)
10. Grosse, G. et al., *J. Neurosci.*, **20**, 1869 (2000).
11. McFarlane, S. and Pollock, N.S., *J Neurosci.* **20**, 1020 (2000).
12. Robbins, J., *Pharmacol. Ther.*, **90**, 1 (2001).
13. Wang, H.S., et al., *Science*, **282**, 1890 (1998).
14. Teschemacher, A.G. et al., *Br. J. Pharmacol.*, **128**, 479 (1999).
15. Schonerr, R., et al., *FEBS Lett.*, **514**, 204 (2002).
16. .Lehmann-Horn, F. and Jurkat-Rott, K., *Physiol. Rev.*, **79**, 1317 (1999).

mct/jk 4/2004