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# **Product Information**

Anti-Sodium Channel, Brain Type I (α subunit) produced in rabbit, affinity isolated antibody

Catalog Number S4936

Synonyms: Anti-B<sub>1</sub>; Anti- Scna1; Anti- SP11<sub>1</sub>

# **Product Description**

Anti-Sodium Channel, Brain Type I ( $\alpha$  subunit) is produced in rabbit using a synthetic peptide corresponding to amino acids 465-481 of the  $\alpha$  subunit of rat type I voltage-gated sodium channel (VGSC, SP11<sub>I</sub>) (with an additional N-terminal lysine and tyrosine), conjugated to KLH, as immunogen. The epitope corresponds to the sequence in the intracellular loop between the I and II domains of type I VGSC  $\alpha$  subunit. The antibody is affinity isolated using peptide-agarose.

Anti-Sodium Channel, Brain Type I ( $\alpha$  subunit) recognizes type I  $\alpha$  subunit of VGSC in rat and mouse by immunoblotting. The antibody may also be used in immunohistochemistry.<sup>3,4</sup>

Chloride channels have several functions including: regulating cell volume; membrane potential stabilization; signal transduction; and 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. CLC-2 channels exhibit differential brain distribution and are implicated in regulating and maintaining the chloride gradient in cells that exhibit primarily inhibitory GABAA responses. CLC-3 channels are important in cardiac function and their volume sensitivity may be due to PKC/PKA modulated phosphorylation.

Voltage-gated sodium channels (VGSC) are present in most excitable cells. In neuronal tissue, they are responsible for generating and propagating action potentials. Brain VGSC are heteromers of  $\alpha\beta1\beta2$  Subunits. Of these, the  $\alpha$  subunit forms the channel pore. Twelve  $\alpha$  subunit genes have been identified. VGSC have been implicated in numerous neurological and cardiac disorders. Further, VGSC are important in mediating many therapeutic drug effects (including the actions of anesthetics, antiarrhythmics and antiepileptics).  $^{9,10}$ 

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 whereas, the other family includes: voltagesensing (KV); big conductance, calcium activated (BK<sub>CA</sub>); and small conductance, calcium activated (SK) potassium channels. In neuronal tissue, BK and SK channels modulate the action potential duration, the speed of repolarization and the after hyperpolarization. 11,12 These channels are implicated both in therapeutic drug effects and also in disease. 11-13 KV channels have been implicated in activitydependent, plastic changes in neuronal tissue. 14,15 HERG (human 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. 16

Many subunits that form the ion channels have been cloned and expressed. With the combination of molecular biology and electrophysiology, although much has been learned about the structure and function of the ion channels, much remains to be determined about the *in vivo* physiological roles of the ion channel subtypes and also in their roles in mediating therapeutic drug effects.

Monovalent ion channels are being associated with a growing number of diseases. 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

Supplied lyophilized from phosphate buffered saline, pH 7.4, containing 1% bovine serum albumin and 0.05% sodium azide.

#### **Precautions and Disclaimer**

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

# **Preparation Instructions**

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

 $\alpha$  Subunits of voltage-gated Na $^{+}$  channels are highly sensitive to proteases. All procedures that are going to receive a full-length protein should be performed at 2-8 °C with a protease inhibitor mixture (1  $\mu g/ml$  pepstatin A, 1  $\mu g/ml$  leupeptin, 1  $\mu g/ml$  aprotinin, 0.2 mM 4-(2-aminoethyl)-benzenesulfonyl fluoride, 0.1mg/ml benzamidine, 8  $\mu g/ml$  each calpain inhibitors I and II).

# Storage/Stability

Prior to reconstitution, store at -20 °C. After reconstitution, the stock antibody solution may be stored at 2-8 °C for up to one month. For extended storage, freeze in working aliquots at -20 °C. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

### **Product Profile**

<u>Immunoblotting</u>: The recommended working antibody dilution is 1:200 using rat brain membranes.

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

## References

- 1. Noda, M., et al., *Nature*, **322**, 826 (1986).
- Gordon, D., et al., Proc. Natl. Acad. Sci. USA, 87, 8682 (1987).
- 3. Westenbroek, R.E., et al., Neuron, 3, 695 (1989).
- 4. Westenbroek, R.E., et al., *J. Neurosci.*, **12**, 2259 (1992).
- 5. Jentsch, T.J. et al., *J. Physiol.*, **482**, 19S, (1995).
- Staley, K. et al., Neuron, 17, 543, (1996).
- 7. Nagasaki M. et al., *J. Physiol.*, **523**, 705 (2000).
- 8. Jeong, S.Y. et al., *Biochem. Biophys. Res. Commun.*, **267**, 262 (2000).
- 9. Catterall, W.A., Adv. Neurol., 79, 441 (1999).
- 10. Vincent, G.M. et al., Cardiol. Rev., 7, 44 (1999).
- 11. Scholtz, A. et al., J. Physiol., **513**, 55 (1998).
- 12. Dreixler, J.C. et al., Anesth. Analg., 90, 727 (2000).
- 13. Bond, C.T. et al., *Ann. N.Y. Acad. Sci.*, **868**, 370 (1999).
- 14. Grosse, G. et al., J. Neurosci., 20, 1869 (2000).
- 15. McFarlane, S. and Pollock, N.S., *J. Neurosci.*, **20**, 1020 (2000).
- 16. Teschemacher, A.G. et al., *Br. J. Pharmacol.*, **128**, 479 (1999).
- 17. Lehmann-Horn, F. and Jurkat-Rott, K., *Physiol. Rev.*, **79**, 1317 (1999).

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