



## Polyclonal Anti- Sodium channel, voltage-gated, beta 1, SCN1B (Sephacrose Bead Conjugate)

**Catalogue No.** PA1244-S

**Lot No.** 09G01

**Ig type:** rabbit IgG

**Size:** 100µg/vial

### Specificity

Human, rat, mouse.  
No cross reactivity  
with other proteins.

### Recommended application

(Immunoprecipitation(IP))

### Immunogen

A synthetic peptide corresponding to a sequence at the N-terminal of human SCN1B, identical to the related rat and mouse sequence.

### Purification

Immunogen affinity purified.

### Formulation

50% slurry in PBS pH 7.2 with 0.01mg NaN<sub>3</sub> preservative.

### Storage

Store at 4°C for frequent use.

### Description:

This Antagene antibody is immobilized via covalent binding of primary amino groups to N-hydroxysuccinimide (NHS)-activated sepharose beads. It is useful for immunoprecipitation assays

## BACKGROUND

Voltage-gated sodium (Na<sup>+</sup>) channels are essential for the generation and propagation of action potentials in striated muscle and neuronal tissues. The complete coding region of Sodium channel, voltage-gated, beta 1(SCN1B) is found in approximately 9.0 kb of genomic DNA and consists of five exons (72 to 749 bp) and four introns (90 bp to 5.5 kb). It is mapped to 19q13.1 and can act as a candidate gene for hereditary disorders affecting membrane excitability.<sup>1</sup> Sodium channel beta1 subunits play important roles in the regulation of sodium channel density and localization and are involved in axo-glial communication at nodes of Ranvier.<sup>2</sup>

## REFERENCE

1. Makita, N.; Sloan-Brown, K.; Weghuis, D. O.; Ropers, H. H.; George, A. L., Jr. : Genomic organization and chromosomal assignment of the human voltage-gated Na(+) channel beta-1 subunit gene (SCN1B). *Genomics* 23: 628-634, 1994.
2. Chen, C.; Westenbroek, R. E.; Xu, X.; Edwards, C. A.; Sorenson, D. R.; Chen, Y.; McEwen, D. P.; O'Malley, H. A.; Bharucha, V.; Meadows, L. S.; Knudsen, G. A.; Vilaythong, A.; Noebels, J. L.; Saunders, T. L.; Scheuer, T.; Shrager, P.; Catterall, W. A.; Isom, L. L. : Mice lacking sodium channel beta-1 subunits display defects in neuronal excitability, sodium channel expression, and nodal architecture. *J. Neurosci.* 24: 4030-4042, 2004.

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