Product Informatiion Sheet

Polyclonal Anti- Sodium channel, voltage-gated, beta 1, **SCN1B (Magnetic Bead Conjugate)**

Catalogue No. PA1244-M

Lot No. 09G01

Ig type: rabbit IgG1

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

Contents

Each vial contains 1mg/ml Magnetic Bead in PBS, pH 7.2, 0.05mg NaN_3.

Storage

Store at 4˚C for frequent use.

Description:

This Antagene antibody is immobilized by the covalent reaction of hydrazinonicotinamide-modified antibody with formylbenzamide-modified magnetic beads. It is useful for immunoprecipitation

BACKGROUND

Voltage-gated sodium (Na+) 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.[1] 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.[2]

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.