

## BACKGROUND

Voltage-gated sodium channels (NavCh) are glycoprotein complexes responsible for initiation and propagation of action potentials in excitable cells such as central and peripheral neurons, cardiac and skeletal muscle myocytes, and neuroendocrine cells. Mammalian sodium channels are heterotrimers and consist of a single large (approximately 260 kDa) pore-forming  $\alpha$  subunit complexed with 1 or 2 smaller accessory beta subunits. Nine genes (SCN1A, SCN2A, etc.) encoding distinct  $\alpha$  subunit isoforms and 4 beta subunit genes (SCN1B, SCN2B, etc.) have been identified in the human genome. Many isoforms are expressed in the central and peripheral nervous system, while skeletal muscle and cardiac muscle express more restricted NavCh repertoires. Mutations in alpha subunit genes have been linked to paroxysmal disorders such as epilepsy, long QT syndrome, and hyperkalemic periodic paralysis in humans, and motor endplate disease and cerebellar ataxia in mice. A mutation in the beta 1 subunit gene has been linked to generalized epilepsy with febrile seizures plus type 1 (GEFS + 1) in a human family with this disease.<sup>1</sup> Sodium channel beta subunits are multifunctional. Sodium channel beta1 subunit was encoded by SCN1B. Sodium channel beta1 subunits modulate channel voltage-dependence and gating as well as channel cell surface expression.<sup>2</sup> Beta1 also participates in cell-cell and cell-matrix adhesion. beta1-mediated homophilic cell adhesion *in vitro* results in cellular aggregation, ankyrin recruitment, and neurite outgrowth. A sodium channel signaling complex is proposed that involves beta subunits as channel modulators as well as cell adhesion molecules, other cell adhesion molecules such as neurofascin and contactin, RPTP beta, and extracellular matrix molecules such as tenascin.<sup>3</sup> *In vivo*, the loss of beta1 results in spontaneous seizures, ataxia, and aberrant neuronal pathfinding and fasciculation. SCN1B gives rise to at least two splice variants, beta1 and beta1B, that differ in their carboxyl-terminal domains.<sup>4</sup>

### References:

1. Catterall, W.A.: Adv Neurol. 79:441-56, 1999
2. Isom, L.L.: Neuroscientist. 7:42-54, 2001
3. Isom, L.L.: Front Biosci. 7:12-23, 2002
4. Plummer, N.W. & Meisler, M.H.: Genomics 57: 323-331, 1999

## TECHNICAL INFORMATION

**Source:** Anti-SCN1B is a rabbit polyclonal antibody raised against a synthetic peptide corresponding to a sequence at the middle region of rat and mouse SCN1B, different from the related human sequence by single amino acid.

**Specificity and Sensitivity:** Anti-SCN1B reacts specifically with SCN1B of human, rabbit, mouse & rat origin in immunostaining and western blotting, no cross-reactivity with other members of the family.

**Storage Buffer:** PBS and 30% glycerol.

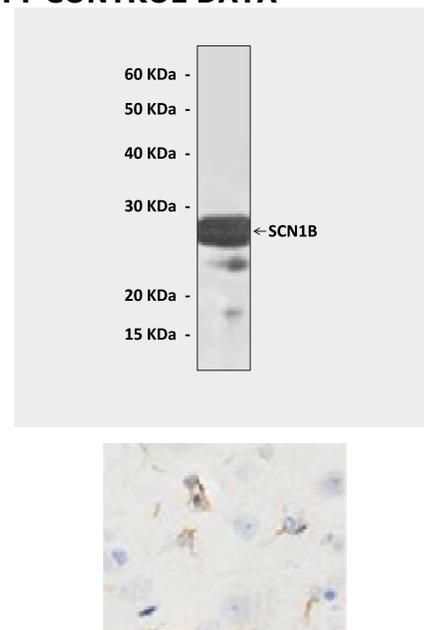
**Storage:** Store at -20°C for at least one year. Store at 4°C for frequent use. Avoid repeated freeze-thaw cycles.

## APPLICATIONS

Application:	*Dilution:
WB	1:500 – 1:1000
IP	n/d
IHC	1:50 – 1:200
ICC	n/d
FACS	n/d

*\*Optimal dilutions must be determined by end user.*

## QUALITY CONTROL DATA



**Top:** Detection of SCN1B from rat brain tissue lysate in Western blot assay, using Anti-SCN1B. **Bottom:** Immunohistochemical staining of paraffin-embedded rat brain tissue, using Anti-SCN1B.

