

Anti-Scn1a Antibody Picoband®

Catalog Number: PB9887

About SCN1A

Nav1.1, also known as the sodium channel, voltage-gated, type I, alpha subunit (SCN1A), is a protein which in humans is encoded by the SCN1A gene. Voltage-dependent sodium channels are heteromeric complexes that regulate sodium exchange between intracellular and extracellular spaces and are essential for the generation and propagation of action potentials in muscle cells and neurons. Each sodium channel is composed of a large pore-forming, glycosylated alpha subunit and two smaller beta subunits. This gene encodes a sodium channel alpha subunit, which has four homologous domains, each of which contains six transmembrane regions. Allelic variants of this gene are associated with generalized epilepsy with febrile seizures and epileptic encephalopathy. Alternative splicing results in multiple transcript variants. The RefSeq Project has decided to create four representative RefSeq records. Three of the transcript variants are supported by experimental evidence and the fourth contains alternate 5' untranslated exons, the exact combination of which have not been experimentally confirmed for the full-length transcript.

Overview

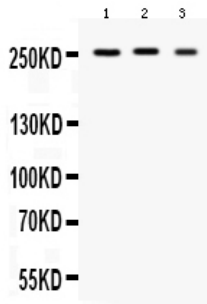
Product Name	Anti-Scn1a Antibody Picoband®
Reactive Species	Human, Mouse, Rat
Description	Boster Bio Anti-Scn1a Antibody Picoband® catalog # PB9887. Tested in WB applications. This antibody reacts with Human, Mouse, Rat. The brand Picoband indicates this is a premium antibody that guarantees superior quality, high affinity, and strong signals with minimal background in Western blot applications. Only our best-performing antibodies are designated as Picoband, ensuring unmatched performance.
Application	WB
Clonality	Polyclonal
Formulation	Each vial contains 4mg Trehalose, 0.9mg NaCl and 0.2mg Na ₂ HPO ₄ .
Storage Instructions	Store at -20°C for one year from date of receipt. After reconstitution, at 4°C for one month. It can also be aliquotted and stored frozen at -20°C for six months. Avoid repeated freeze-thaw cycles.
Host	Rabbit
Uniprot ID	P35498

Technical Details

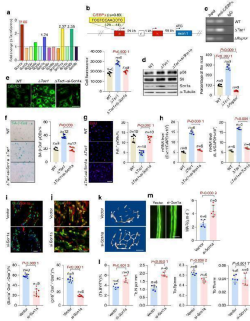
Immunogen	A synthetic peptide corresponding to a sequence at the C-terminus of human Scn1a, identical to the related rat sequence.
Recommended Detection Systems	Boster recommends Enhanced Chemiluminescent Kit with anti-Rabbit IgG (EK1002) for Western blot.
Cross Reactivity	No cross-reactivity with other proteins.

Isotype	Rabbit IgG
Form	Lyophilized
Concentration	Adding 0.2 ml of distilled water will yield a concentration of 500 ug/ml.
Purification	Immunogen affinity purified.
Suggested Dilutions	Western blot, 0.1-0.5ug/ml, Human, Mouse, Rat

Anti-Scn1a Antibody Picoband® (PB9887) Images

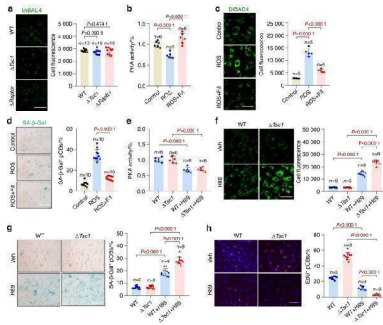


Western blot analysis of Scn1a using anti-Scn1a antibody (PB9887). Electrophoresis was performed on a 5-20% SDS-PAGE gel at 70V (Stacking gel) / 90V (Resolving gel) for 2-3 hours. The sample well of each lane was loaded with 30 ug of sample under reducing conditions. Lane 1: rat brain tissue lysates, Lane 2: mouse brain tissue lysates, Lane 3: U87 whole cell lysates. After electrophoresis, proteins were transferred to a nitrocellulose membrane at 150 mA for 50-90 minutes. Blocked the membrane with 5% non-fat milk/TBS for 1.5 hour at RT. The membrane was incubated with rabbit anti-Scn1a antigen affinity purified polyclonal antibody (Catalog # PB9887) at 0.5 ug/mL overnight at 4°C, then washed with TBS-0.1%Tween 3 times with 5 minutes each and probed with a goat anti-rabbit IgG-HRP secondary antibody at a dilution of 1:5000 for 1.5 hour at RT. The signal is developed using an Enhanced Chemiluminescent detection (ECL) kit (Catalog # EK1002) with Tanon 5200 system. A specific band was detected for Scn1a at approximately 250 kDa. The expected band size for Scn1a is at 229 kDa.

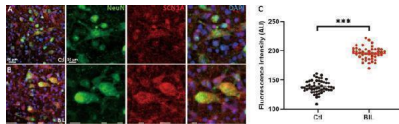


The sodium channel Scn1a mediates plasma membrane depolarization in senescent preosteoblasts. a mRNA expression of voltage-sensitive sodium channels in the Δ Tsc1 and control calvarial preosteoblasts indicated by our previous global mRNA expression profile (GSE74781). b Binding site of C/EBPalpha in the 5'-UE of Scn1a (yellow box) predicted by the JASPAR database (). rs: JASPAR relative scores, which are defined as 1 for the maximum-likelihood sequence. Boxes represent exons: blue coding exons, red noncoding exons conserved between humans and mice, white noncoding exons identified in either human or mouse transcripts. Noncoding exons are named alphabetically, and the first coding exon (exon 1) of the Scn1a gene is indicated. Genomic distances between exons are indicated. c Binding of C/EBPalpha to the 5'-UE sequence of Scn1a in vivo, determined by a ChIP assay using the Δ Tsc1 and Δ Raptor cells and anti-C/EBPalpha antibody or IgG. The ChIP samples were then subjected to qPCR with the Scn1a 5'-UE primers. The percentage of the input of the sample by using the anti-C/EBPalpha antibody in the wild-type cells was normalized to 100. The Δ Tsc1 osteoblasts were transfected with Scn1a siRNA and subjected to (d) Scn1a detection with western blotting, (e) measurement of relative plasma membrane potential with DiBAC4 dye, (f) SA-beta-gal staining and quantification of the proportion of SA-beta-gal-positive cells, (g) immunostaining of EdU and quantitative analysis of EdU + cells relative to total cells, and (h) qPCR analysis of IL-6 and Cxcl1 mRNA. Double immunostaining of Osx plus Scn1a (i) and Osx plus p16 (j) in the tibias of the 18-month-old Δ Tsc1 mice injected with adenovirus encoding si-Scn1a for 1 month. Double positively stained cells were quantified. Representative uCT images (k) and quantification of trabecular bone (l) in the mice. (m) Representative

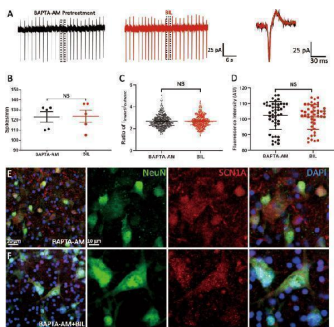
images of calcein labels and quantification of the mineral apposition rate (MAR) in femurs from the 18-month-old Δ Tsc1 mice receiving si-Scn1a. Scale bars: 50 μ m in e, g, m; 100 μ m in i, j; and 500 μ m in k. Data are shown as the mean \pm SD. The numbers of samples (n) are indicated in each figure panel. P values were determined with two-tailed Student's t test for single comparisons Index in PubMed under a CC BY license. PMID: 35256591



Prosenescent stress activates Scn1a by inhibiting PKA. a Replicative wild-type (WT), Δ Tsc1 and Δ Raptor calvarial osteoblasts were incubated with the fluorescent DiBAC4 dye and photographed under a confocal microscope. Scale bar, 50 μ m. Relative plasma membrane potentials were measured. ROS-induced senescent wild-type preosteoblasts were treated with F/I (forskolin + IBMx, PKA activator) or left untreated. The cells were subjected to (b) measurement of total cellular PKA activities, (c) DiBAC4 staining and measurement of relative plasma membrane potentials. Scale bar, 50 μ m. d SA-beta-gal staining of cells in b and quantification of the proportion of SA-beta-gal-positive cells in each population. Scale bar, 100 μ m. Replicative WT and Δ Tsc1 osteoblasts were treated with H-89 (PKA inhibitor) and were subjected to (e) measurement of total cellular PKA activities, (f) DiBAC4 staining and measurement of relative plasma membrane potentials. Scale bar, 50 μ m. g SA-beta-gal staining of cells in e and quantification of the proportion of SA-beta-gal-positive cells in each population. Scale bar, 100 μ m. h Immunostaining of EdU in cells in e and quantitative analysis of EdU + cells relative to total cells. Scale bar, 100 μ m. Data are shown as the mean \pm SD. The numbers of samples (n) are indicated in each figure panel. P values were determined by two-tailed Student's t test for single comparisons Index in PubMed under a CC BY license. PMID: 35256591

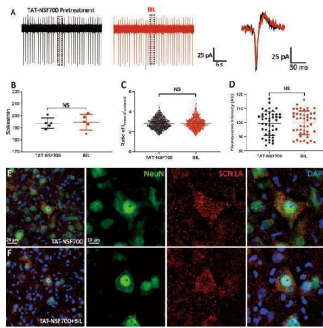


BIL elevated the expression level of SCN1A in MVN neurons. a, b Immunofluorescence labeling images of nuclei (blue), NeuN (green), and SCN1A (red) of control (a) and BIL group (b). Neurons with co-labeling of NeuN and SCN1A were chosen as AOI, and the mean fluorescence intensities of SCN1A from AOI were quantified in artificial units (AUs). c Statistical comparison showing larger MISCN1A in the BIL group, implicating elevated expression level of Nav1.1 after BIL treatment. *** p



Ca²⁺ was required for BIL-induced upregulation of activity and level of VGSCs. a An example recording showing that neither the frequency of spontaneous firings nor the spike waveform of a MVN neuron was altered in slices preincubated in BAPTA-AM (40 μ M, 30 min). b, c Statistical results showing no significant change in spike frequency, ratio of I_{inward} / I_{outward}. d - f MI SCN1A was not statistically different between control and BIL groups (d) as exemplified by the immunofluorescence staining of nuclei (blue), NeuN (green), and SCN1A (red) in control (e) and BIL groups (f). NS no statistical difference, independent-

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PMID: 31601780



Blocking exocytosis with TAT-NSF700 precluded the effects of BIL on MVN neurons. a - c Example recording traces (a) showing that when the slice was preincubated with TAT-NSF700 (5 uM, 30 min), a permeable thrombin-induced exocytosis inhibitor, neither the discharge frequency of MVN neuron (b) nor the ratio of I inward / I outward (c) was altered by BIL (3 uM, 3 min). d-f MI SCN1A was not statistically different between control and BIL groups (d) in slices pretreated with TAT-NSF700, as illustrated by the immunofluorescence images comparing staining of nuclei (blue), NeuN (green), and SCN1A (red) in control (e) and BIL group (f). NS no statistical difference, independent-samples t- test. Index in PubMed under a CC BY license.
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Anti-Scn1a Antibody

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