abcam

Product datasheet

Anti-Nav1.5/SCN5A antibody ab116706

1 Image

Overview

Product name	Anti-Nav1.5/SCN5A antibody
Description	Goat polyclonal to Nav1.5/SCN5A
Host species	Goat
Tested applications	**Suitable for:** IHC-P
Species reactivity	**Reacts with:** Human **Predicted to work with:** Mouse, Rat, Dog, Pig
Immunogen	Synthetic peptide corresponding to Human Nav1.5/SCN5A aa 1500-1600 (internal sequence) (Cysteine residue). Database link: **NP_000326.2** **Run BLAST with** **Run BLAST with**
Positive control	Human Brain Cortex tissue.
General notes	The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing. If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.
Storage buffer	pH: 7.3 Preservative: 0.02% Sodium azide Constituents: 99% Tris buffered saline, 0.5% BSA
Purity	Protein G purified
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab116706 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
IHC-P		Use a concentration of 5 - 10 µg/ml. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.

Target

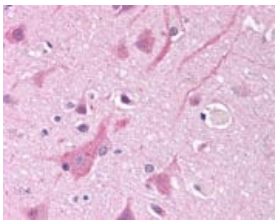
Function This protein mediates the voltage-dependent sodium ion permeability of excitable membranes. Assuming opened or closed conformations in response to the voltage difference across the membrane, the protein forms a sodium-selective channel through which Na(+) ions may pass in accordance with their electrochemical gradient. It is a tetrodotoxin-resistant Na(+) channel isoform. This channel is responsible for the initial upstroke of the action potential.

Tissue specificity Found in jejunal circular smooth muscle cells (at protein level). Expressed in human atrial and ventricular cardiac muscle but not in adult skeletal muscle, brain, myometrium, liver, or spleen. Isoform 4 is expressed in brain.

Involvement in disease Defects in SCN5A are a cause of progressive familial heart block type 1A (PFHB1A) [MIM:113900]; also known as Lenegre-Lev disease or progressive cardiac conduction defect (PCCD). PFHB1A is an autosomal dominant cardiac bundle branch disorder that may progress to complete heart block. PFHB1A is characterized by progressive alteration of cardiac conduction through the His-Purkinje system with right or left bundle branch block and widening of QRS complexes, leading to complete atrio-ventricular block and causing syncope and sudden death.
Defects in SCN5A are the cause of long QT syndrome type 3 (LQT3) [MIM:603830]. Long QT syndromes are heart disorders characterized by a prolonged QT interval on the ECG and polymorphic ventricular arrhythmias. They cause syncope and sudden death in response to exercise or emotional stress. LQT3 inheritance is an autosomal dominant.
Defects in SCN5A are the cause of Brugada syndrome type 1 (BRS1) [MIM:601144]. BRS1 is an autosomal dominant tachyarrhythmia characterized by right bundle branch block and ST segment elevation on an electrocardiogram (ECG). It can cause the ventricles to beat so fast that the blood is prevented from circulating efficiently in the body. When this situation occurs (called ventricular fibrillation), the individual will faint and may die in a few minutes if the heart is not reset.
Defects in SCN5A are the cause of sick sinus syndrome type 1 (SSS1) [MIM:608567]. The term 'sick sinus syndrome' encompasses a variety of conditions caused by sinus node dysfunction. The most common clinical manifestations are syncope, presyncope, dizziness, and fatigue. Electrocardiogram typically shows sinus bradycardia, sinus arrest, and/or sinoatrial block. Episodes of atrial tachycardias coexisting with sinus bradycardia ('tachycardia-bradycardia syndrome') are also common in this disorder. SSS occurs most often in the elderly associated with underlying heart disease or previous cardiac surgery, but can also occur in the fetus, infant, or child without heart disease or other contributing factors, in which case it is considered to be a congenital disorder.
Defects in SCN5A are the cause of ventricular fibrillation paroxysmal familial type 1 (VF1) [MIM:603829]. A cardiac arrhythmia marked by fibrillary contractions of the ventricular muscle due to rapid repetitive excitation of myocardial fibers without coordinated contraction of the ventricle and by absence of atrial activity.

	Defects in SCN5A can be a cause of sudden infant death syndrome (SIDS) [MIM:272120]. SIDS is the sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of clinical history. Pathophysiologic mechanisms for SIDS may include respiratory dysfunction, cardiac dysrhythmias, cardiorespiratory instability, and inborn errors of metabolism, but definitive pathogenic mechanisms precipitating an infant sudden death remain elusive. Long QT syndromes-associated mutations can be responsible for some of SIDS cases. Defects in SCN5A may be a cause of familial atrial standstill (FAS) [MIM:108770]. Atrial standstill is an extremely rare arrhythmia, characterized by the absence of electrical and mechanical activity in the atria. Electrocardiographically, it is characterized by bradycardia, the absence of P waves, and a junctional narrow complex escape rhythm. Defects in SCN5A are the cause of cardiomyopathy dilated type 1E (CMD1E) [MIM:601154]; also known as dilated cardiomyopathy with conduction disorder and arrhythmia or dilated cardiomyopathy with conduction defect 2. Dilated cardiomyopathy is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death.
Sequence similarities	Belongs to the sodium channel (TC 1.A.1.10) family. Nav1.5/SCN5A subfamily. Contains 1 IQ domain.
Domain	The sequence contains 4 internal repeats, each with 5 hydrophobic segments (S1,S2,S3,S5,S6) and one positively charged segment (S4). Segments S4 are probably the voltage-sensors and are characterized by a series of positively charged amino acids at every third position.
Post-translational modifications	Ubiquitinated by NEDD4L; which promotes its endocytosis. Does not seem to be ubiquitinated by NEDD4 or WWP2.
Cellular localization	Membrane.

Images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Nav1.5/SCN5A antibody (ab116706)

ab116706, at 5-10 µg/ml, staining Nav1.5/SCN5A in Formalin-fixed, Paraffin-embedded Human Brain cortex tissue by Immunohistochemistry followed by biotinylated secondary antibody, alkaline phosphatase-streptavidin and chromogen.

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