abcam

Product datasheet

Anti-Scn1a antibody - N-terminal ab140984

1 Image

Overview

Product name	Anti-Scn1a antibody - N-terminal
Description	Rabbit polyclonal to Scn1a - N-terminal
Host species	Rabbit
Specificity	BLAST analysis of the peptide immunogen showed no homology with other Human proteins, except Scn2a (100%) and Scn3a (94%).
Tested applications	**Suitable for:** IHC-P
Species reactivity	**Reacts with:** Human **Predicted to work with:** Mouse, Rat, Rabbit, Horse, Chicken, Hamster, Cow, Dog, Turkey, Pig, Xenopus laevis, Monkey, Zebrafish, Gorilla, Opossum, Common marmoset, Bat, Platypus, Elephant
Immunogen	Synthetic 16 amino acid peptide from the N terminus of Human Scn1a.
Positive control	Human brain, cerebellum 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.
Storage buffer	Preservative: 0.1% Sodium azide Constituent: 99% PBS
Purity	Immunogen affinity purified
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee Our Abpromise guarantee covers the use of ab140984 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 µg/ml. Perform heat mediated antigen retrieval before commencing with IHC staining protocol.

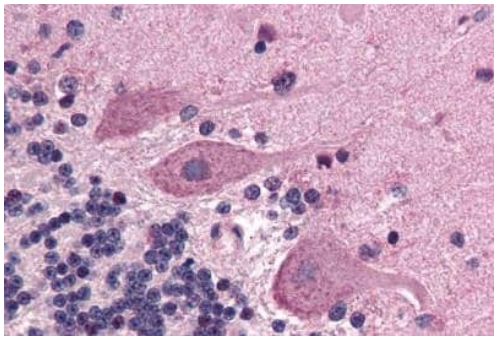
Target

Function 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.

Involvement in disease Defects in SCN1A are the cause of generalized epilepsy with febrile seizures plus type 2 (GEFS+2) [MIM:604233]. Generalized epilepsy with febrile seizures-plus refers to a rare autosomal dominant, familial condition with incomplete penetrance and large intrafamilial variability. Patients display febrile seizures persisting sometimes beyond the age of 6 years and/or a variety of afebrile seizure types. GEFS+ is a disease combining febrile seizures, generalized seizures often precipitated by fever at age 6 years or more, and partial seizures, with a variable degree of severity.
Defects in SCN1A are a cause of severe myoclonic epilepsy in infancy (SMEI) [MIM:607208]; also called Dravet syndrome. SMEI is a rare disorder characterized by generalized tonic, clonic, and tonic-clonic seizures that are initially induced by fever and begin during the first year of life. Later, patients also manifest other seizure types, including absence, myoclonic, and simple and complex partial seizures. Psychomotor development delay is observed around the second year of life. SMEI is considered to be the most severe phenotype within the spectrum of generalized epilepsies with febrile seizures-plus.
Defects in SCN1A are a cause of intractable childhood epilepsy with generalized tonic-clonic seizures (ICEGTC) [MIM:607208]. ICEGTC is a disorder characterized by generalized tonic-clonic seizures beginning usually in infancy and induced by fever. Seizures are associated with subsequent mental decline, as well as ataxia or hypotonia. ICEGTC is similar to SMEI, except for the absence of myoclonic seizures.
Defects in SCN1A are the cause of migraine familial hemiplegic type 3 (FHM3) [MIM:609634]. FHM3 is an autosomal dominant severe subtype of migraine with aura characterized by some degree of hemiparesis during the attacks. The episodes are associated with variable features of nausea, vomiting, photophobia, and phonophobia. Age at onset ranges from 6 to 15 years. FHM is occasionally associated with other neurologic symptoms such as cerebellar ataxia or epileptic seizures. A unique eye phenotype of elicited repetitive daily blindness has also been reported to be cosegregating with FHM in a single Swiss family.
Defects in SCN1A are the cause of familial febrile convulsions type 3A (FEB3A) [MIM:604403]; also known as familial febrile seizures 3. Febrile convulsions are seizures associated with febrile episodes in childhood without any evidence of intracranial infection or defined pathologic or traumatic cause. It is a common condition, affecting 2-5% of children aged 3 months to 5 years. The majority are simple febrile seizures (generally defined as generalized onset, single seizures with a duration of less than 30 minutes). Complex febrile seizures are characterized by focal onset, duration greater than 30 minutes, and/or more than one seizure in a 24 hour period. The likelihood of developing epilepsy following simple febrile seizures is low. Complex febrile seizures

	are associated with a moderately increased incidence of epilepsy.
Sequence similarities	Belongs to the sodium channel (TC 1.A.1.10) family. Nav1.1/SCN1A 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.
Cellular localization	Membrane.

Images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Scn1a antibody - N-terminal (ab140984)

Immunohistochemical analysis of formalin-fixed, paraffin-embedded Human brain cerebellum tissue labeling Scn1a with ab140984 at 5 µg/ml.

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