Revision 1

Store at -20C

#14573

Nav1.7 Antibody

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	M R	Endogenous	230-250	Rabbit	#Q15858	6335

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Nav1.7 Antibody recognizes endogenous levels of total Nav1.7 protein.

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human Nav1.7 protein. Antibodies are purified by protein A and peptide affinity chromatography.

Background

Voltage gated sodium channels are composed of a large alpha subunit and auxiliary beta subunits. The alpha subunit has 4 homologous domains, with each domain containing 6 transmembrane segments. These segments function as the voltage sensor and sodium permeable pore. Upon change of membrane potential, the sodium channel is activated, which allows sodium ions to flow through (1,2). When associated with beta subunits or other accessory proteins, the alpha subunit is regulated at the level of cell surface expression, kinetics, and voltage dependence (3,4).

There are 9 mammalian alpha subunits, named Nav1.1-Nav1.9 (5). These alpha subunits differ in tissue specificity and biophysical functions (6,7). Seven of these subunits are essential for the initiation and propagation of action potentials in the central and peripheral nervous system while Nav1.4 and Nav1.5 are mainly expressed in skeletal muscle and cardiac muscle (8,9). Mutations in these alpha channel subunits have been identified in patients with epilepsy, seizure, ataxia, sensitivity to pain, and cardiomyopathy (reviewed in 10).
The Nav1.7 alpha subunit (Nav1.7, SCN9A) plays an important role in nociception signaling and is essential for acute, inflammatory, and neuropathic pain perception (11,12). Mutations in the corresponding *SCN9A* gene are associated with primary erythermalgia, autosomal recessive congenital indifference to pain, and paroxysmal extreme pain disorder (13-15). Mutations in *SCN9A* cause the GEFSP7 form of generalized epilepsy with febrile seizures, and are implicated in many cases of Dravet syndrome, a severe form of pediatric epileptic encephalopathy (16).

Background References

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Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

M: Mouse **R:** Rat

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