

PrecisION® hNav1.8/β1 Recombinant Stable Cell Line

Catalog Number CYL3025 Lot Number See Vial

Contents 2 Vials, 2 x 10⁶ to 4 x 10⁶ in 1 mL

Background Information

hNav1.8 is a voltage-gated sodium channel alpha subunit. It is expressed in mammalian adult dorsal root ganglion (DRG) neurons, predominantly those with small diameter cell bodies that give rise to the unmyelinated (C-type) fibres. These fibres form synapses in the dorsal horn of the spinal cord and are mainly involved in pain signaling. Additional information can be found on page 2.

Product Information

Description Recombinant HEK 293 cell line expressing the human Nav1.8 (tetrodotoxin-resistant voltage-gated

sodium channel type X, SCN10A) and the human sodium channel beta 1 subunit (SCN1B)

Family Sodium, Voltage-Gated

HEK 293

Target Nav1.8/β1

	Target Protein	Accession Number
1	Nav1.8	AF117907
2	β1	NM_001037
3	N/A	N/A
4	N/A	N/A

Species Human

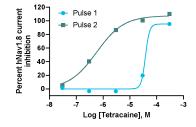
Host Cell Type

Application Electrophysiology assay (conventional and automated patch clamp platforms)

Storage Vials are to be stored in vapor phase of liquid nitrogen

Functional Performance

HEK293 cells expressing hNav1.8/ β 1 were characterized in terms of their pharmacological and biophysical properties using whole-cell patch clamp techniques.



Electrophysiology Method QPatch

Reference Agonist

Reference Antagonist Tetracaine

Antagonist IC₅₀ (μ M) 0.57

1



Passage Stability

This cell line has been confirmed to be stable through at least 12 passages with no significant drop in assay window or change in pharmacology.

Mycoplasma Testing

This lot was tested and found to be free of mycoplasma contamination. Data available upon request.

Notes

Additional functional (pharmacological and electrophysiological) validation on multiple platforms is available upon request.

Additional Ligand Information

Control Compound Tetracaine

Vendor Name: Sigma-Aldrich

Vendor Catalog No. T7383

Additional Background Information

This channel conducts the characteristic slow, tetrodotoxin-resistant (TTX-r) currents involved in action potential initiation and transmission in these neurons and is therefore a potential target for analgesic agents. Altered expression of Nav1.8 is observed in pain models and human pain states - channel protein can be seen to re-localise from cell bodies to the site of insult or injury. There is also good evidence from antisense oligonucleotide studies for a role in development of hyperexciteable states in pain models. However KO mice show surprisingly limited pain phenotypes, possibly due to compensatory up-regulation of other Nav subtypes. Native sodium channels are multi-subunit complexes, composed of not only the pore forming α subunit, but also auxiliary β subunits. The $\beta1$ subunit is known to increase peak current amplitude as well as increasing sodium ion channel expression at the cell surface (Vijayaragavan et al., 2004).

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Generated on June 29, 2020