

PrecisION[®] hNav1.2 Recombinant Stable Cell Line

Catalog Number CYL3023

Lot Number

See Vial

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

Background Information

The voltage gated sodium channel isoform Nav1.2 is involved with axon potential initiation and conduction. Nav1.2 is the target of antiepileptic drugs and the probable target causing side effects of centrally active local anaesthetics. It is detected in relative abundance on myelin-deficient areas on axons in the central and peripheral nervous systems. Demyelinated axons expressing Nav1.2 are much less susceptible to axonal degeneration. Additional information can be found on page 2.

Product Information

Description Recombinant CHO-K1 cell line expressing the human Nav1.2 (type II voltage-gated sodium channel alpha subunit, SCN2A)

Family Sodium, Voltage-Gated

Target Nav1.2

	Target Protein	Accession Number
1	Nav1.2	NM_021007
2	N/A	N/A
3	N/A	N/A
4	N/A	N/A

Species Human

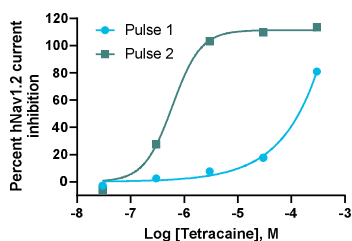
Host Cell Type CHO-K1

Application Electrophysiology assay (conventional and automated patch clamp platforms)

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

Functional Performance

CHO cells expressing hNav1.2 were characterized in terms of their pharmacological and biophysical properties using whole-cell patch clamp techniques.



Electrophysiology Method QPatch

Reference Agonist ATX-II

Reference Antagonist Tetracaine

Antagonist IC₅₀ (μM) 0.60

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

The action of a drug on Nav1.2 sodium channels may interfere with the fine balance between axon growth and nerve impulse transmission.

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