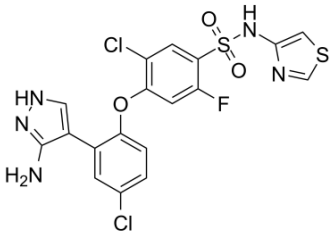
PF 05089771

Cat. No.:	HY-12883
CAS No.:	1235403-62-9
Molecular Formula:	$C_{18}H_{12}Cl_2FN_5O_3S_2$
Molecular Weight:	500.35
Target:	Sodium Channel
Pathway:	Membrane Transporter/Ion Channel
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 34 mg/mL (67.95 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9986 mL	9.9930 mL	19.9860 mL
5 mM	0.3997 mL	1.9986 mL	3.9972 mL
10 mM	0.1999 mL	0.9993 mL	1.9986 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.25 mg/mL (4.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.25 mg/mL (4.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.25 mg/mL (4.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PF 05089771 is a potent, orally active and selective arylsulfonamide $Na_V1.7$ inhibitor, with IC_{50} values of 11 nM, 12 nM, 13 nM, 171 nM and 8 nM for $hNa_V1.7$, $cynNa_V1.7$, $dogNa_V1.7$, $ratNa_V1.7$, and $musNa_V1.7$, respectively. PF 05089771 is under the study for pain and diabetic neuropathy[1][2].

IC_{50} & Target

IC50: 11 nM ($hNa_V1.7$), 12 nM ($cynNa_V1.7$), 13 nM ($dogNa_V1.7$), 171 nM ($ratNa_V1.7$), 8 nM ($musNa_V1.7$)[1][2].

In Vitro	PF-05089771 is determined to be more than 1000-fold selective over tetrodotoxin-resistant (TTX-R) $Na_v1.5$ and $Na_v1.8$ channels (IC_{50}s >10 μM) and exhibited a range of selectivity over TTX-sensitive (TTX-S) channels (10-fold for $Na_v1.2$ to 900-fold for $Na_v1.3$ and $Na_v1.4$)[1]. PF-05089771 (30 nM) blocks the majority of TTX-S current (75.5 ± 10.5%, n = 5, Fig 5D) whilst 100 nM resulted in complete block[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Alexandrou AJ, et al. Subtype-Selective Small Molecule Inhibitors Reveal a Fundamental Role for Nav1.7 in Nociceptor Electrogenesis, Axonal Conduction and Presynaptic Release. PLoS One. 2016 Apr 6;11(4):e0152405.

[2]. Theile JW, et al. The Selective Nav1.7 Inhibitor, PF-05089771, Interacts Equivalently with Fast and Slow Inactivated Nav1.7 Channels. Mol Pharmacol. 2016 Nov;90(5):540-548.

Caution: Product has not been fully validated for medical applications. For research use only.

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