

Product Data Sheet

Product Name: PRE-084 hydrochloride

Cat. No.: GC14960

Chemical Properties

Cas. No. 75136-54-8

Chemical Name 2-morpholinoethyl 1-phenylcyclohexanecarboxylate hydrochloride

SMILES O=C(C1(C2=CC=CC=C2)CCCCC1)OCCN3CCOCC3.Cl

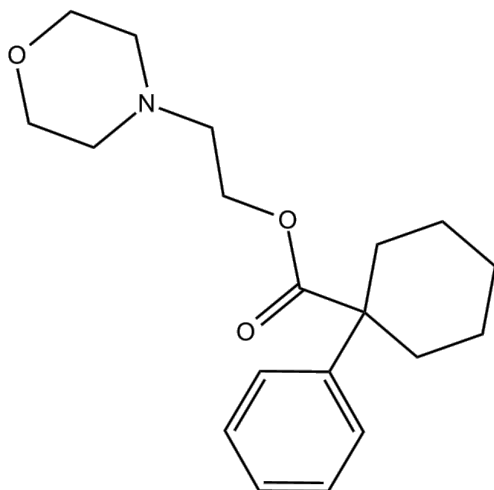
Formula $C_{19}H_{27}NO_3 \cdot HCl$ M.Wt 353.89

Solubility $\geq 17.3\text{mg/mL}$ in DMSO with gentle warming Storage Store at RT

General tips For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.

Structure HCl



Protocol

Cell experiment [1]:

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

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Cell lines	Rat dorsal root ganglion (DRG) neurons
Preparation Method	DRG neurons were collected from six sham rats and six rats after three days of CFA injection. One portion was used as control, and the other was treated with PRE-084 hydrochloride (10μM) for 30min before further processing.
Reaction Conditions	10μM; 30min
Applications	Activation of Sig-1R through PRE-084 hydrochloride led to a decrease in the fast sodium current in isolated DRG neurons from CFA-treated rats, which was associated with a diminished action potential (AP) peak and maximum depolarizing rate (MDR), as well as an increased rheobase. PRE-084 hydrochloride was also observed to enhance the slow component of the sodium current, resulting in hyperpolarization of the threshold potential and an increase in AP firing frequency.
Animal experiment [2]:	
Animal models	Sprague-Dawley rat
Preparation Method	The rats were injected intraperitoneally with PRE-084 hydrochloride or saline (1mg/kg). One hour later, ischemia was induced by ligation at the left anterior descending coronary artery. Rats were subjected to 30min of myocardial ischemia and 24h of reperfusion.
Dosage form	1mg/kg; i.p; 1h before myocardial ischemia operation
Applications	PRE-084 hydrochloride preserved cardiac function and reduced myocardial apoptosis through the activation of Akt and eNOS.

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References:

- [1] Song Y L, Xu Z F, Zhang L P, et al. Sigma-1 Receptor Modulates CFA-Induced Inflammatory Pain via Sodium Channels in Small DRG Neurons. *Biomolecules*. 2025 Jan 6;15(1):73.
- [2] Gao Q J, Yang B, Chen J, et al. Sigma-1 Receptor Stimulation with PRE-084 Ameliorates Myocardial Ischemia-Reperfusion Injury in Rats. *Chin Med J (Engl)*. 2018 Mar 5;131(5):539-543.

Background

PRE-084 hydrochloride is a highly selective sigma-1 receptor (S1R) agonist with an IC₅₀ value of 44nM^[1]. PRE-084 hydrochloride has demonstrated broad neuroprotective and anti-inflammatory effects across multiple models of neurodegeneration and neuroinflammation such as Parkinson's disease and amyotrophic lateral sclerosis^{[3][4][5]}.

In vitro, PRE-084 hydrochloride (10μM) treatment on dorsal root ganglia (DRG) neurons isolated from CFA-treated rats for 30min led to a decrease in the fast sodium current, which was associated with a diminished action potential (AP) peak and maximum depolarizing rate (MDR), as well as an increased rheobase^[2]. PRE-084 hydrochloride (0.1–20μM) and β-amyloid fragment 25–35 (25μM) were co-applied to rat cortical neuronal cultures for 24h. PRE-084 hydrochloride reduced neuronal toxicity in a bell shaped-manner with maximally neuroprotective effect at 10μM and blocked the β-amyloid-induced rise in the pro-apoptotic protein Bax^[3]. PRE-084 hydrochloride (2.5μg/mL) applied on BV-2 cells when cells challenged with *S. pneumoniae* at 20 MOI for 6h exerted anti-inflammatory effect by reducing the expression of phosphorylated STAT1 (p-STAT1) and NLRP3^[4].

In vivo, PRE-084 hydrochloride (0.25μg/g) was administered intraperitoneally into the wobbler (wr) mouse model of spontaneous motor-neuron degeneration three times per week from the 4th to the 12th week of age. PRE-084 hydrochloride treatment increased the number of surviving motor neurons, raised cervical spinal-cord BDNF levels, while attenuating reactive astrogliosis and shifting microglia/macrophages toward a CD68⁺ and CD206⁺ tissue-repair phenotype in wr mice^[5]. PRE-084 hydrochloride (3mg/kg) administrated intraperitoneally into streptozotocin induced diabetic rats once a day for a week enhanced vasoconstrictor and platelet aggregator and reduced vasodilator and anti-aggregator cyclooxygenase product formation^[6]. PRE-084 hydrochloride (1mg/kg) were administered into myocardial ischemia/reperfusion model rats 1h before operation. PRE-084

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hydrochloride preserved cardiac function and reduced myocardial apoptosis through the activation of Akt and eNOS^[7].

References:

- [1] Su T P, Wu X Z, Cone E J, et al. Sigma compounds derived from phencyclidine: identification of PRE-084, a new, selective sigma ligand. J Pharmacol Exp Ther. 1991 Nov;259(2):543-50.
- [2] Song Y L, Xu Z F, Zhang L P, et al. Sigma-1 Receptor Modulates CFA-Induced Inflammatory Pain via Sodium Channels in Small DRG Neurons. Biomolecules. 2025 Jan 6;15(1):73.
- [3] Marrazzo A, Caraci F, Salinaro E T, et al. Neuroprotective effects of sigma-1 receptor agonists against beta-amyloid-induced toxicity. Neuroreport. 2005 Aug 1;16(11):1223-6.
- [4] Gao Z F, Xie S, Wang L Y, et al. Hypidone hydrochloride (YL-0919) protects mice from meningitis via Sigma1R-STAT1-NLRP3-GSDMD pathway. Int Immunopharmacol. 2024 Feb 15;128:111524.
- [5] Peviani M, Salvaneschi E, Bontempi L, et al. Neuroprotective effects of the Sigma-1 receptor (S1R) agonist PRE-084, in a mouse model of motor neuron disease not linked to SOD1 mutation. Neurobiol Dis. 2014 Feb;62:218-32.
- [6] Vácz S, Barna L, Laczi K, et al. Effects of sub-chronic, in vivo administration of sigma-1 receptor ligands on platelet and aortic arachidonate cascade in streptozotocin-induced diabetic rats. PLoS One. 2022 Nov 17;17(11):e0265854.
- [7] Gao Q J, Yang B, Chen J, et al. Sigma-1 Receptor Stimulation with PRE-084 Ameliorates Myocardial Ischemia-Reperfusion Injury in Rats. Chin Med J (Engl). 2018 Mar 5;131(5):539-543.

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