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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

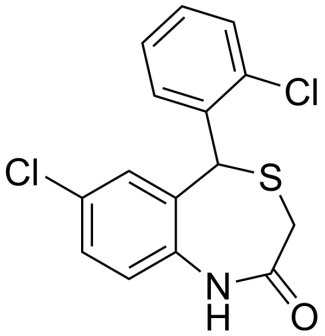
F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

CGP37157



Cat. No.: HY-15754

CAS No.: 75450-34-9

Molecular Formula: $C_{15}H_{11}Cl_2NOS$

Molecular Weight: 324.22

Target: Na+/Ca2+ Exchanger

Pathway: Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 125 mg/mL (385.54 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0843 mL	15.4216 mL	30.8433 mL
5 mM	0.6169 mL	3.0843 mL	6.1687 mL
10 mM	0.3084 mL	1.5422 mL	3.0843 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: ≥ 2.5 mg/mL (7.71 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CGP37157 is a potent, selective inhibitor of Na^+/Ca^{2+} exchanger, inhibiting the Na^+-induced Ca^{2+}-release from guinea-pig heart mitochondria, with an IC_{50} of 0.8 μM.

IC_{50} & Target

IC50: 0.8 μM (Na^+/Ca^{2+} exchanger)[1]

In Vitro

CGP37157 (Compound XVI) is a potent, selective inhibitor of Na^+/Ca^{2+} exchanger, inhibiting the Na^+-induced Ca^{2+}-release from guinea-pig heart mitochondria, with an IC_{50} of 0.8 μM[1].

CGP37157 (10 μM) shows inhibitory effect on mitochondrial Na^+/Ca^{2+} exchanger in cortical neurons, modulates intracellular Ca^{2+} levels via suppresssing voltage-gated calcium channels, and reduces NMDA-induced cytosolic and mitochondrial Ca^{2+}

overloads. CGP37157 (10 μM) also reduces NMDA-induced excitotoxicity, and such an effect is via attenuating mitochondrial damage and calpain activity in neurons[2].
CGP37157 (10 μM) in combination with salinomycin significantly attenuates cell viability and increases apoptosis of FaDu and HLaC79 cells. Moreover, CGP37157 has no inhibitory effect on salinomycin tumor toxicity[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]

Cell toxicity assays are performed. Neurons are exposed to NMDA in HBSS (free of Ca^{2+} and Mg^{2+}) containing 2.6 mM $CaCl_2$, 10 mM glucose and 10 μM glycine for 10 or 30 min at 37°C, depending on the experiment. CGP37157 is present before and during the excitotoxic insult and cell viability is assessed 24 h later using Citotox 96 colorimetric assay. All experiments are performed in quadruplicate and the values provided are the normalized mean ± S.E.M. of at least three independent experiments[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2022 Apr 22.
- J Cell Physiol. 2021 Mar 11.

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REFERENCES

[1]. Chiesi M, et al. Structural dependency of the inhibitory action of benzodiazepines and related compounds on the mitochondrial Na+-Ca2+ exchanger. Biochem Pharmacol. 1988 Nov 15;37(22):4399-403.

[2]. Ruiz A, et al. CGP37157, an inhibitor of the mitochondrial Na+/Ca2+ exchanger, protects neurons from excitotoxicity by blocking voltage-gated Ca2+ channels. Cell Death Dis. 2014 Apr 10;5:e1156.

[3]. Scherzed A, et al. Effects of salinomycin and CGP37157 on head and neck squamous cell carcinoma cell lines in vitro. Mol Med Rep. 2015 Sep;12(3):4455-61.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA