Flunarizine

Cat. No.: HY-B0358

CAS No.: 52468-60-7

Molecular Formula: $C_{26}H_{26}F_2N_2$

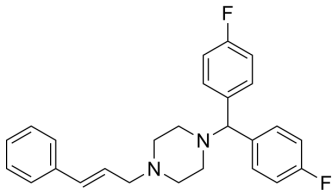
Molecular Weight: 404.49

Target: Calcium Channel; Sodium Channel; Dopamine Receptor

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; GPCR/G Protein

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 : 100 mg/mL (247.22 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4722 mL	12.3612 mL	24.7225 mL
5 mM	0.4944 mL	2.4722 mL	4.9445 mL
10 mM	0.2472 mL	1.2361 mL	2.4722 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 (6.18 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Flunarizine is a potent dual Na^+/Ca^{2+} channel (T-type) blocker. Flunarizine is a D_2 dopamine receptor antagonist. Flunarizine shows anticonvulsive and antimigraine activity, and peripheral vasodilator effects[1][2][3][4][5].

IC_{50} & Target

D_2 Receptor

In Vitro

Flunarizine blocks sodium currents (I_{Na}) and calcium currents (I_{Ca}) with IC_{50} values of 0.94 μM and 1.77 μM in cultured rat cortical neurons, respectively[2].

Flunarizine (10 and 30 μM; 24 h) shows cytotoxic effects to chromaffin cells[4].

Flunarizine (1-30 μM) causes clear cytoprotection of chromaffin cell at concentrations of 3-10 μM[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cytotoxicity Assay[4]

Cell Line:	Chromaffin cells[4]
Concentration:	10 and 30 μM
Incubation Time:	24 hours
Result:	Showed a tendency to increase cell death at the concentration of 10 μM, and showed near 100% cell loss at the concentration of 30 μM.

In Vivo

Flunarizine (intraperitoneal injection; 30 mg/kg; once) protects mice from lipopolysaccharide- (LPS-) induced acute lung injury (ALI)[5].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male BALB/c mice (6-8 weeks old) with acute lung injury induced by lipopolysaccharide[5]
Dosage:	30 mg/kg
Administration:	Intraperitoneal injection; 30 mg/kg; once
Result:	Suppressed the LPS-induced cell influx, protein leakage, and inflammatory cytokines release. Inhibited the pulmonary inflammation.

CUSTOMER VALIDATION

- J Leukoc Biol. 2021 Sep 17.
- J Ethnopharmacol. 2020 May 23;254:112727.
- Sci Rep. 2018 Nov 16;8(1):16932.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Hong-Seob So, et al. Protective effect of T-type calcium channel blocker flunarizine on cisplatin-induced death of auditory cells. Hear Res. 2005 Jun;204(1-2):127-39.

[2]. Qing Ye, et al. Flunarizine blocks voltage-gated Na(+) and Ca(2+) currents in cultured rat cortical neurons: A possible locus of action in the prevention of migraine. Neurosci Lett. 2011 Jan 10;487(3):394-9.

[3]. Celia M Santi, et al. Differential inhibition of T-type calcium channels by neuroleptics. J Neurosci. 2002 Jan 15;22(2):396-403.

[4]. Novalbos J, et al. Effects of dotarizine and flunarizine on chromaffin cell viability and cytosolic Ca2+. Eur J Pharmacol. 1999 Feb 5;366(2-3):309-17.

[5]. Wan L, et al. Mibefradil and Flunarizine, Two T-Type Calcium Channel Inhibitors, Protect Mice against Lipopolysaccharide-Induced Acute Lung Injury. Mediators Inflamm. 2020 Nov 10;2020:3691701.

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

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