CFM-2

Cat. No.: HY-12503

CAS No.: 178616-26-7

Molecular Formula: $C_{17}H_{17}N_3O_3$

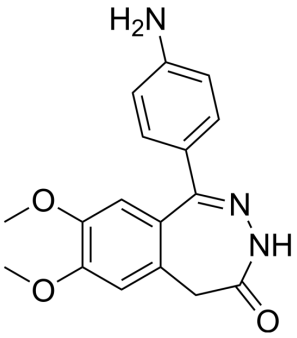
Molecular Weight: 311.34

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

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 : ≥ 50 mg/mL (160.60 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2119 mL	16.0596 mL	32.1192 mL
5 mM	0.6424 mL	3.2119 mL	6.4238 mL
10 mM	0.3212 mL	1.6060 mL	3.2119 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: ≥ 2.75 mg/mL (8.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CFM-2 is a potent and selective non-competitive AMPAR antagonist[1]. CFM-2 possesses anticonvulsant activity in various models of seizures[2].

In Vitro

CFM-2 inhibits the extracellular signal regulated kinase (ERK1/2) pathway, CFM-2 reduced phosphorylation of cAMP-responsive element binding protein (CREB), suppressed expression of cyclin D1, upregulated the cell cycle regulators and tumor suppressor proteins p21 and p53 and decreased number of lung adenocarcinoma cells in G2 and S phases of the cell cycle.

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

In Vivo

Pretreatment with CFM-2 delays the progression of seizure rank during repeated administration of pentylentetrazole. At the end of the period of repeated pentylentetrazole treatment (6 weeks) the mean seizure score was 0 in vehicle treated

controls, 4.3 in animals treated with vehicle + pentylentetrazole, 2.2 in rats treated chronically with CFM-2 (20 μmol/kg; i.p.) + pentylentetrazole and 1.0 in rats treated repeatedly with CFM-2 (50 μmol/kg; i.p.) + pentylenetetrazole. CFM-2 is also able to antagonize the long-term increase in sensitivity of the convulsant effects of GABA function inhibitors in pentylentetrazole-kindled animals[1].

Intrathecal application of two selective non-competitive AMPAR antagonists, CFM-2 (25 and 50 μg) and GYKI 52466 (50μg), significantly attenuated mechanical and thermal hypersensitivities on the ipsilateral hind paw at 2 and 24 h post-CFA injection. Neither CFM-2 nor GYKI 52466 affects the contralateral basal responses to thermal and mechanical stimuli[4].

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

CUSTOMER VALIDATION

- Research Square Print. 2022 Apr.

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REFERENCES

[1]. De Sarro G, et al. Effects of some AMPA receptor antagonists on the development of tolerance in epilepsy-prone rats and in pentylenetetrazole kindled rats. Eur J Pharmacol. 1999 Mar 5;368(2-3):149-59.

[2]. Rizzo M, et al. Determination of new 2,3-benzodiazepines in rat plasma using high-performance liquid chromatography with ultraviolet detection. J Chromatogr B Biomed Sci Appl. 1999 Aug 20;731(2):207-15.

[3]. Stepulak A, et al. AMPA antagonists inhibit the extracellular signal regulated kinase pathway and suppress lung cancer growth. Cancer Biol Ther. 2007 Dec;6(12):1908-15.

[4]. Park JS, et al. Role of spinal cord alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors in complete Freund's adjuvant-induced inflammatory pain. Mol Pain. 2008 Dec 30;4:67.

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