



CFM-2

Catalog No: tcsc3625



Available Sizes

Size: 10mg

Size: 50mg



Specifications

CAS No:

178616-26-7

Formula:

 $C_{17}^{H}_{17}^{N}_{3}^{O}_{3}$

Pathway:

Membrane Transporter/Ion Channel; Neuronal Signaling

Target:

iGluR;iGluR

Purity / Grade:

>98%

Solubility:

10 mM in DMSO

Observed Molecular Weight:

311.34

Product Description

CFM-2 is a selective non-competitive AMPAR antagonist.

IC50 value:

Target: AMPAR antagonist





in vitro: AMPA antagonists GYKI 52466 and CFM-2 inhibit 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.

in vivo: Pretreatment with CFM-2 delayed 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 micromol kg(-1) i.p.) + pentylentetrazole and 1.0 in rats treated repeatedly with CFM-2 (50 micromol kg(-1) i.p.) + pentylentetrazole. CFM-2 was also able to antagonize the long-term increase in sensitivity of the convulsant effects of GABA function inhibitors in pentylentetrazole-kindled animals [1]. CFM-2 has been proven to possess anticonvulsant activity in various models of seizures [2]. Intrathecal application of two selective non-competitive AMPAR antagonists, CFM-2 (25 and 50 microg) and GYKI 52466 (50 microg), 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 affected the contralateral basal responses to thermal and mechanical stimuli [4].

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