MDL-29951

Cat. No.: HY-16312

CAS No.: 130798-51-5

Molecular Formula: $C_{12}H_9Cl_2NO_4$

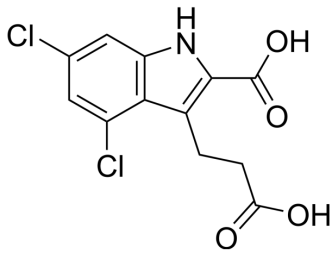
Molecular Weight: 302.11

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 (165.50 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3101 mL	16.5503 mL	33.1005 mL
5 mM	0.6620 mL	3.3101 mL	6.6201 mL
10 mM	0.3310 mL	1.6550 mL	3.3101 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 (8.28 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MDL-29951 is a novel glycine antagonist of NMDA receptor activation, with K_i of 0.14 μM for [^{3}H]glycine binding in vitro and in vivo.

IC_{50} & Target

NMDA Receptor

In Vitro

MDL 100,748 and MDL 29,951 are approximately 2000-fold selective for the glycine binding site relative to the glutamate

recognition sites[1]. MDL-29951 is found to inhibit the human F16Bpase under these conditions (IC_{50}=2.5 μM). MDL-29951 inhibits the human liver (IC_{50}=2.5 μM), porcine kidney (IC_{50}=1.0 μM), and rabbit liver (IC_{50}=0.21 μM) isoforms of the enzyme, but is significantly less potent against the rat liver isoform (IC_{50}=11 μM)[2]. The MDL29951-activated receptor exhibits other activities associated with GPCR-mediated signaling, including G protein-dependent activation of extracellular signal-regulated kinase 1 and 2 (ERK1/2) and recruitment of β-arrestin. As with recombinant cell systems, MDL29951 promotes Ca^{2+} signaling responses and inhibition of cyclic adenosine monophosphate (cAMP) accumulation in rat oligodendrocyte precursor cells during the period of peak GPR17 abundance. Effects of MDL29951 are markedly reduced in cells with low GPR17 abundance and are blocked by pranlukast[3].

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

PROTOCOL

Kinase Assay [1]

[^{3}H]JCPP (30.7 Ci/mmol) binding assays are conducted in minivials, incubated for 15 mm at 25°C in 1 mL of 50 mM Tris-HC1 (pH 7.4) containing 10 nM [^{3}H]JCPP, 200 g of membrane protein and unlabeled ligands as indicated. Nonspecific binding is defined using 1 mM L-glutamate. Bound ligand is separated by centrifugation. Specific binding accounted for approximately 80% of total binding.

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

CUSTOMER VALIDATION

- Cell Death Dis. 2021 Jun 12;12(6):610.
- J Neurosci. 2016 Oct 12;36(41):10560-10573.

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REFERENCES

[1]. Baron BM, et al. Potent indole- and quinoline-containing N-methyl-D-aspartate antagonists acting at the strychnine-insensitive glycine binding site. J Pharmacol Exp Ther. 1992 Sep;262(3):947-56.

[2]. Wright SW, et al. 3-(2-carboxyethyl)-4,6-dichloro-1H-indole-2-carboxylic acid: an allosteric inhibitor of fructose-1,6-bisphosphatase at the AMP site. Bioorg Med Chem Lett. 2003 Jun 16;13(12):2055-8.

[3]. Harden TK. Enigmatic GPCR finds a stimulating drug. Sci Signal. 2013 Oct 22;6(298):pe34.

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