TCN 201

Cat. No.: HY-13457

CAS No.: 852918-02-6

Molecular Formula: $C_{21}H_{17}ClFN_3O_4S$

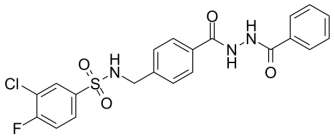
Molecular Weight: 462

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture and light

* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (541.13 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1645 mL	10.8225 mL	21.6450 mL
	5 mM	0.4329 mL	2.1645 mL	4.3290 mL
	10 mM	0.2165 mL	1.0823 mL	2.1645 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: ≥ 2.08 mg/mL (4.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TCN 201 is a potent, selective and non-competitive antagonist of GluN1/GluN2A NMDA receptor, with a pIC_{50} of 6.8. TCN 201 is selective for GluN1/GluN2A NMDA receptor over GluN1/GluN2B NMDA receptor (pIC_{50}<4.3)[1][2].

IC_{50} & Target

pIC50: 6.8 (GluN1/GluN2A NMDA receptor)[1]

In Vitro

TCN 201 (compound 1) is selective for GluN1/GluN2A NMDAR over GluN1/GluN2B NMDAR, with pIC_{50}s of 6.8 and <4.3, respectively[1].

TCN 201 (10 μM) produces only slight inhibition of GluN1/GluN2B NMDAR-mediated currents in oocytes[2].

TCN 201 (10-30 μM) antagonism of NMDAR-mediated responses is both subtype- and glycine-dependent and more potent

	than TCN 213 in oocytes[2]. TCN 201 (0.1-100 μM) does not produce complete block of NMDAR-mediated responses in oocytes[2]. TCN 201 (10 μM) antagonism of NMDAR-mediated currents shows a negative correlation with their ifenprodil sensitivity in rat cortical neurons[2]. TCN 201 (1-9 μM) suppresses cortical spreading depression (CSD) in chick retina[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	TCN-201 (10 mg/kg; i.p.) is ineffective in CSD blood-oxygen level-dependent (BOLD) response in rats[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Edman S, et, al. TCN 201 selectively blocks GluN2A-containing NMDARs in a GluN1 co-agonist dependent but non-competitive manner. Neuropharmacology. 2012 Sep; 63(3): 441-9.

[2]. Bu F, et, al. NR2A contributes to genesis and propagation of cortical spreading depression in rats. Sci Rep. 2016 Mar 22;6:23576.

[3]. Shatillo A, et, al. Involvement of NMDA receptor subtypes in cortical spreading depression in rats assessed by fMRI. Neuropharmacology. 2015 Jun; 93:164-70.

[4]. Bettini E, et, al. Identification and characterization of novel NMDA receptor antagonists selective for NR2A- over NR2B-containing receptors. J Pharmacol Exp Ther. 2010 Dec; 335(3): 636-44.

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