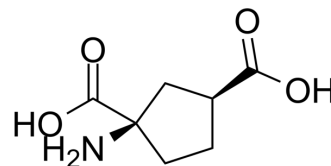


## trans-ACPD

Cat. No.:	HY-19434		
CAS No.:	67684-64-4		
Molecular Formula:	C <sub>7</sub> H <sub>11</sub> NO <sub>4</sub>		
Molecular Weight:	173.17		
Target:	mGluR		
Pathway:	GPCR/G Protein; 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 (288.73 mM; Need ultrasonic)  
 H<sub>2</sub>O : 3.57 mg/mL (20.62 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent		1 mg	5 mg	10 mg
	Concentration	Mass			
	1 mM		5.7747 mL	28.8734 mL	57.7467 mL
	5 mM		1.1549 mL	5.7747 mL	11.5493 mL
	10 mM		0.5775 mL	2.8873 mL	5.7747 mL

Please refer to the solubility information to select the appropriate solvent.

#### In Vivo

- Add each solvent one by one: PBS  
Solubility: 5 mg/mL (28.87 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline  
Solubility: ≥ 2.5 mg/mL (14.44 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.5 mg/mL (14.44 mM); Clear solution

### BIOLOGICAL ACTIVITY

#### Description

trans-ACPD, a metabotropic receptor agonist, produces calcium mobilization and an inward current in cultured cerebellar Purkinje neurons.

#### IC<sub>50</sub> & Target

mGluR

#### In Vitro

Excitatory amino acid (EAA) analogues activate receptors that are coupled to the increased hydrolysis of phosphoinositides

(PIs). In these studies, hippocampal slices are prepared from neonatal rats (6-11 days old) to characterize the effects of EAA analogues on these receptors. The concentrations of trans-ACPD required to evoke half-maximal stimulation ( $EC_{50}$  value) is 51  $\mu$ M. DL-2-Amino-3-phosphonopropionate (DL-AP3) is also equipotent as an inhibitor of PI hydrolysis stimulated by ibotenate, quisqualate, and trans-ACPD ( $IC_{50}$  values are 480-850  $\mu$ M)<sup>[2]</sup>.

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

#### In Vivo

Intrathecal injection of NMDA, kainate, and trans-ACPD, TNF- $\alpha$ , or IL-1 $\beta$  causes significant ( $p < 0.001$ ) biting behaviour in mice compared to animals injected intrathecally with saline. In all groups, systemic pre-treatment with GM (100 mg/kg, i.p.) significantly ( $p < 0.001$ ) reduces the biting behaviour compared to mice treated with saline (10 mL/kg, i.p.). The greatest effect of GM is observed on the pro-inflammatory cytokines and NMDA, with the following inhibition percentages: TNF- $\alpha$  (92 $\pm$ 7%), IL-1 $\beta$  (91 $\pm$ 5%), NMDA (69 $\pm$ 1%), and trans-ACPD (71 $\pm$ 12%). By contrast, at the same dose, GM has no significant effect on the kainate-mediated biting response<sup>[3]</sup>.

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

## PROTOCOL

### Animal

#### Administration <sup>[3]</sup>

Mice<sup>[3]</sup>

Male Swiss mice (25-35 g) are used. Intrathecal injections are given to fully conscious mice. Briefly, the animals are manually restrained, and a 30-gauge needle connected by a polyethylene tube to a 25  $\mu$ L Hamilton gas-tight syringe is inserted through the skin and between the vertebrae into the subdural space of the L5-L6 spinal segments. Intrathecal injections (5  $\mu$ L/site) are administered over a period of 5 s. Biting behaviour is defined as a single head movement directed at the flanks or hind limbs, resulting in contact of the animal's snout with the target organ. The nociceptive response is elicited by NMDA (450 pmol/site, a selective agonist of the NMDA glutamatergic ionotropic receptor), kainate (110 pmol/site, a selective agonist of the kainate subtype of glutamatergic ionotropic receptors), and trans-ACPD (50 nmol/site, a non-selective agonist of metabotropic glutamate receptors, which is active at group I and group II), TNF- $\alpha$  (0.1 pmol/site) and IL-1 $\beta$  (1 pmol/site) or saline (5  $\mu$ L/site, i.t.). The amount of time the animal spent biting or licking the caudal region is taken as evidence of nociception and is evaluated following local post injections of the following agonists: NMDA (5 min), kainate (4 min), and trans-ACPD TNF- $\alpha$ , and IL-1 $\beta$  (15 min). Animals received GM (100 mg/kg, i.p.) 0.5 h before intrathecal injection of 5  $\mu$ L of the drugs, while control animals received a similar volume of saline (10 mL/kg, i.p.).

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

## REFERENCES

- [1]. Linden DJ, et al. Trans-ACPD, a metabotropic receptor agonist, produces calcium mobilization and an inward current in cultured cerebellar Purkinje neurons. *J Neurophysiol.* 1994 May;71(5):1992-8.
- [2]. Littman L, et al. Multiple mechanisms for inhibition of excitatory amino acid receptors coupled to phosphoinositide hydrolysis. *J Neurochem.* 1992 Nov;59(5):1893-904.
- [3]. Córdova MM, et al. Polysaccharide glucomannan isolated from *Heterodermia obscurata* attenuates acute and chronic pain in mice. *Carbohydr Polym.* 2013 Feb 15;92(2):2058-64.

**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