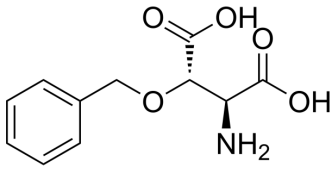
DL-TBOA

Cat. No.:	HY-107522
CAS No.:	205309-81-5
Molecular Formula:	$C_{11}H_{13}NO_5$
Molecular Weight:	239.23
Target:	EAAT
Pathway:	Membrane Transporter/Ion Channel
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 : 200 mg/mL (836.02 mM; Need ultrasonic)

H_2O : 5 mg/mL (20.90 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1801 mL	20.9004 mL	41.8008 mL
5 mM	0.8360 mL	4.1801 mL	8.3602 mL
10 mM	0.4180 mL	2.0900 mL	4.1801 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: ≥ 5 mg/mL (20.90 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (20.90 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 5 mg/mL (20.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DL-TBOA is a potent non-transportable inhibitor of excitatory amino acid transporters with IC_{50}s of 70 μM, 6 μM and 6 μM for excitatory amino acid transporter-1 (EAAT1), EAAT2 and EAAT3, respectively. DL-TBOA inhibits the uptake of [^{14}C]glutamate in COS-1 cells expressing the human EAAT1 and EAAT2 with K_i valuesof 42 μM and 5.7 μM, respectively. DL-TBOA blocks EAAT4 and EAAT5 in a competitive manner with K_i values of 4.4 μM and 3.2 μM, respectively[1][2][3].

IC_{50} & Target	EAAT1	EAAT2	EAAT3

In Vitro

DL-TBOA (70-350 μM; 48 hours; HCT116 and LoVo cell lines) treatment concentration-dependently enhances SN38-induced loss of viability. DL-TBOA reversed Oxaliplatin-induced loss of viability[4].
DL-TBOA (350 μM; 24 hours; HCT116 and LoVo cell lines) treatment decreases p53 induction by SN38 and oxaliplatin[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[4]

Cell Line:	HCT116 and LoVo cell lines
Concentration:	70 μM, 350 μM
Incubation Time:	48 hours
Result:	Enhanced SN38-induced, and counteracted Oxaliplatin-induced, cell death.

Cell Viability Assay[4]

Cell Line:	HCT116 and LoVo cell lines
Concentration:	350 μM
Incubation Time:	24 hours
Result:	p53 induction by SN38 and oxaliplatin was decreased.

In Vivo

DL-TBOA (10 nmol; i.c.v., rats) significantly facilitates the expression of naloxone-precipitated somatic signs and conditioned place aversion[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (180-250 g)[5]
Dosage:	1 nmol, 3 nmol, 10 nmol
Administration:	Intracerebroventricularly injection (i.c.v.)
Result:	Dose dependently facilitated various somatic signs induced by Naloxone (0.1 mg/kg).

REFERENCES

[1]. Yumiko Sekiya, et al. Facilitation of morphine withdrawal symptoms and morphine-induced conditioned place preference by a glutamate transporter inhibitor DL-threo-beta-benzyloxyaspartate in rats. Eur J Pharmacol. 2004 Feb 6;485(1-3):201-10.

[2]. Shimamoto K, et al. DL-threo-beta-benzyloxyaspartate, a potent blocker of excitatory amino acid transporters. Mol Pharmacol. 1998 Feb;53(2):195-201.

[3]. Jabaudon D, et al. Inhibition of uptake unmasks rapid extracellular turnover of glutamate of nonvesicular origin. Proc Natl Acad Sci U S A. 1999 Jul 20;96(15):8733-8.

[4]. Shigeri Y, et al. Effects of threo-beta-hydroxyaspartate derivatives on excitatory amino acid transporters (EAAT4 and EAAT5). J Neurochem. 2001 Oct;79(2):297-302.

[5]. Pedraz-Cuesta E, et al. The glutamate transport inhibitor DL-Threo-β-Benzyloxyaspartic acid (DL-TBOA) differentially affects SN38- and oxaliplatin-induced death of drug-resistant colorectal cancer cells. BMC Cancer. 2015 May 16;15:411.

Caution: Product has not been fully validated for medical applications. For research use only.

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