DL-TBOA ammonium

Cat. No.: HY-107522B

CAS No.: 2093503-71-8

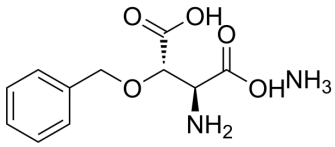
Molecular Formula: $C_{11}H_{16}N_2O_5$

Molecular Weight: 256.26

Target: EAAT

Pathway: Membrane Transporter/Ion Channel

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

DL-TBOA ammonium 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 ammonium 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 ammonium 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 ammonium (70-350 μM; 48 hours) treatment concentration-dependently enhances SN38-induced loss of viability. DL-TBOA reversed Oxaliplatin-induced loss of viability[4].

DL-TBOA ammonium (350 μM; 24 hours) 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 cells, LoVo cells
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 cells, LoVo cells
Concentration:	350 μM
Incubation Time:	24 hours
Result:	Decreased p53 induction by SN38 and oxaliplatin.

In Vivo

DL-TBOA ammonium (10 nmol; i.c.v.) to morphine-dependent 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)-precipitated morphine withdrawal.

REFERENCES

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

[2]. 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.

[3]. 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.

[4]. 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.

[5]. 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.

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