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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

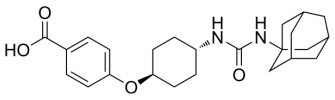
mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

trans-AUCB

Cat. No.:	HY-113974
CAS No.:	885012-33-9
Molecular Formula:	$C_{24}H_{32}N_2O_4$
Molecular Weight:	412.52
Target:	Epoxide Hydrolase
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (242.41 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4241 mL	12.1206 mL	24.2412 mL
	5 mM	0.4848 mL	2.4241 mL	4.8482 mL
	10 mM	0.2424 mL	1.2121 mL	2.4241 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 (5.04 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.04 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.04 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

trans-AUCB (t-AUCB) is a potent, orally active and selective soluble epoxide hydrolase (sEH) inhibitor with IC_{50}s of 1.3 nM, 8 nM, 8 nM for hsEH, mouse sEH and rat sEH, respectively. trans-AUCB has anti-glioma activity[1][2].

IC_{50} & Target

IC50: 1.3 nM (hsEH), 8 nM (mouse sEH) and 8 nM (rat sEH)[2]

In Vitro

trans-AUCB (t-AUCB; 25-300 μM; 48 hours) suppresses U251 and U87 cell growth in a dose-dependent manner[1].
trans-AUCB (200 μM; 48 or 96 hours) induces cell-cycle G0/G1 phase arrest in U251 and U87 cells[1].

trans-AUCB (200 μM; 10 min-4 hours) can increase the phosphorylation levels of p65 after 10 min, reaching to peak after 30 min and lasting for at least 2 hours[1].
trans-AUCB (200 μM; 48 hours) suppresses U251 and U87 cell growth by activating NF-jB-p65[1].
trans-AUCB (10 μM; 30 min) efficiently inhibits sEH activities in human glioblastoma cell lines (U251, U87) and human hepatocellular carcinoma cell line (HepG2 cells)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	U251, U87 cells
Concentration:	25, 50, 100, 200, or 300 μM
Incubation Time:	48 hours
Result:	Suppressed U251 and U87 cell growth in a dose-dependent manner.

Cell Cycle Analysis[1]

Cell Line:	U251, U87 cells
Concentration:	200 μM
Incubation Time:	48 or 96 hours
Result:	Induced cell-cycle G0/G1 phase arrest in U251 and U87 cells.

Western Blot Analysis[1]

Cell Line:	U251, U87 cells
Concentration:	200 μM
Incubation Time:	10 min, 30 min, 1 hour, 2 hours, or 4 hours
Result:	Increased the phosphorylation levels of p65 after 10 min, reached to peak after 30 min and lasted for at least 2 hours.

In Vivo

trans-AUCB (t-AUCB; p.o.; 0.1, 0.5, 1 mg/kg) ameliorates the LPS-induced hypotension in a dose-dependent manner[2].
trans-AUCB (p.o.; 0.1, 0.5, 1 mg/kg) has $t_{1/2}$ values of 20, 30, 15 min and C_{max} values of 30, 100, 150 nmol/L for p.o. of 0.1, 0.5, 1 mg/kg[2].
trans-AUCB (s.c.; 1, 3, 10 mg/kg) has $t_{1/2}$ values of 60, 85, 75 min and C_{max} values of 245, 2700, 3600 nmol/L for s.c. of 1, 3, 10 mg/kg[2].
trans-AUCB (i.v.; 0.1 mg/kg) has $t_{1/2}$ values of 70 min and 10 hours for distribution (α) and elimination (β) phases. trans-AUCB has a CL of 0.7 L/h kg and a V_{dss} was 17 L/kg[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mice (male CFW strain, 7 weeks old, 24-30 g; and male C57BL/6 strain, 8 weeks old, 22-25 g) [2]
Dosage:	0.1, 0.5, 1 mg/kg
Administration:	PO
Result:	Ameliorated the LPS-induced hypotension in a dose-dependent manner.

Animal Model:	Mice (male CFW strain, 7 weeks old, 24-30 g; and male C57BL/6 strain, 8 weeks old, 22-25 g) [2]
Dosage:	0.1, 0.5, 1 mg/kg (Pharmacokinetic Analysis)
Administration:	PO
Result:	Had $t_{1/2}$ values of 20, 30, 15 min and C_{max} values of 30, 100, 150 nmol/L for p.o. of 0.1, 0.5, 1 mg/kg, respectively.

CUSTOMER VALIDATION

- Mol Metab. 2021 Dec 28;101426.

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REFERENCES

[1]. Li J, et al. t-AUCB, an improved sEH inhibitor, suppresses human glioblastoma cell growth by activatingNF-κB-p65. J Neurooncol. 2012 Jul;108(3):385-93.

[2]. Liu JY, et al. Pharmacokinetic optimization of four soluble epoxide hydrolase inhibitors for use in a murinemodel of inflammation. Br J Pharmacol. 2009 Jan;156(2):284-96.

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