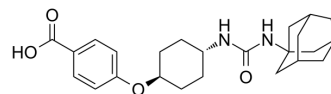


trans-AUCB

Cat. No.:	HY-113974		
CAS No.:	885012-33-9		
Molecular Formula:	C ₂₄ H ₃₂ N ₂ O 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)			
		Solvent Concentration	Mass	
			1 mg	5 mg
			10 mg	
Preparing Stock Solutions	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	<ol style="list-style-type: none"> 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 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 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	IC ₅₀ : 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 μ M; 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 μ M; 48 hours) suppresses U251 and U87 cell growth by activating NF- κ B-p65^[1].

trans-AUCB (10 μ M; 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 μ M
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 μ M
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 μ M
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 \cdot 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- κ B-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.

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

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