CaCCinh-A01

Cat. No.: HY-100611

CAS No.: 407587-33-1

Molecular Formula: $C_{18}H_{21}NO_4S$

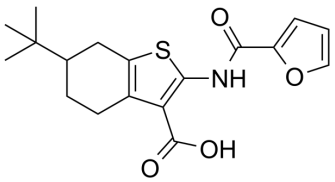
Molecular Weight: 347.43

Target: Chloride Channel

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 : 100 mg/mL (287.83 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8783 mL	14.3914 mL	28.7828 mL
	5 mM	0.5757 mL	2.8783 mL	5.7566 mL
	10 mM	0.2878 mL	1.4391 mL	2.8783 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: ≥ 2.5 mg/mL (7.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.20 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CaCCinh-A01 is an inhibitor of both TMEM16A and calcium-activated chloride channel (CaCC) with IC_{50}s of 2.1 and 10 μM, respectively.

IC_{50} & Target

IC50: 2.1 μM (TMEM16A)[1], 10 μM (CaCC)[2]

In Vitro

30 μM CaCCinh-A01 and 100 μM tannic acid strongly inhibit CaCC current following ATP stimulation[1]. Calcium-dependent chloride current is reduced by 38±14, 66±10, and 91±1% by 0.1, 1, and 10 μM CaCCinh-A01, respectively. ATP-induced short-

	circuit currents are reduced by 38±7 and 78±3% at 10 and 30 μM CaCCinh-A01, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	CaCCinh-A01 (vein injection; 5 mg/kg; caudal vein injection within 15 min after the onset of reperfusion) significantly reduces infarction when compared with MCAO-saline treatment at 24 h or 72 h in middle cerebral artery occlusion model in mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Two-month-old male C57/BL6J mice[3]
Dosage:	5 mg/kg
Administration:	Vein injection; 5 mg/kg; caudal vein injection within 15 min after the onset of reperfusion
Result:	Attenuated brain infarct size, improved neurological outcomes and lowered BBB permeability after ischemic stroke in mice.

CUSTOMER VALIDATION

- Br J Pharmacol. 2021 Dec 27.
- Biochem Pharmacol. 2020 Aug;178:114062.
- SSRN. 2023 Nov 14.
- bioRxiv. 2023 Jul 3.

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REFERENCES

[1]. TMEM16A inhibitors reveal TMEM16A as a minor component of calcium-activated chloridechannel conductance in airway and intestinal epithelial cells. J Biol Chem. 2011 Jan 21;286(3):2365-74.

[2]. De La Fuente R, et al. Small-molecule screen identifies inhibitors of a human intestinal calcium-activated chloridechannel. Mol Pharmacol. 2008 Mar;73(3):758-68.

[3]. Pin-Yi Liu, et al. TMEM16A Inhibition Preserves Blood-Brain Barrier Integrity After Ischemic Stroke.Front Cell Neurosci. 2019 Aug 6;13:360.

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