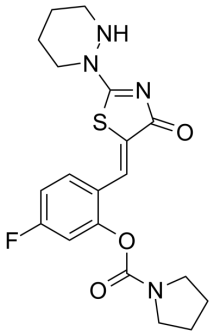
CLP290

Cat. No.:	HY-103023
CAS No.:	1181083-81-7
Molecular Formula:	$C_{19}H_{21}FN_4O_3S$
Molecular Weight:	404.46
Target:	Potassium 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 : 33.33 mg/mL (82.41 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4724 mL	12.3622 mL	24.7243 mL
5 mM	0.4945 mL	2.4724 mL	4.9449 mL
10 mM	0.2472 mL	1.2362 mL	2.4724 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 (5.14 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.14 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CLP290 is an orally available activator of the neuron-specific K^+-Cl^- cotransporter KCC2, displays potential for treatment of a wide range of neurological and psychiatric indications. CLP290 can significantly lower blood arginine-vasopressin (AVP) and glucose levels in STZ rats[1][2].

IC_{50} & Target

KCC2[1]

In Vivo

CLP290 (oral gavage; 100 mg/kg; twice a day; 7 day) enhances KCC2 activity and restores Cl^- transport in superficial dorsal

horn (SDH) neurons of morphine-treated rats, and prevents morphine-induced hyperalgesia (MIH) in rat[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male rats (300 g, >postnatal day 60)[1]
Dosage:	100 mg/kg
Administration:	Oral gavage; 100 mg/kg; twice a day; 7day
Result:	Rescued established MIH and prevented its development by restoring Cl^- transport or preventing its deficiency in the SDH.

CUSTOMER VALIDATION

- Exp Neurol. 2022 Mar 1;114027.

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REFERENCES

[1]. Ferrini F, et al. Enhancing KCC2 function counteracts morphine-induced hyperalgesia. Sci Rep. 2017 Jun 20;7(1):3870.

[2]. Gagnon M, et al. Chloride extrusion enhancers as novel therapeutics for neurological diseases. Nat Med. 2013 Nov;19(11):1524-8.

[3]. Kim YB, et al. Excitatory GABAergic Action and Increased Vasopressin Synthesis in Hypothalamic Magnocellular Neurosecretory Cells Underlie the High Plasma Level of Vasopressin in Diabetic Rats. Diabetes. 2018 Mar;67(3):486-495.

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

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