GSK-872 hydrochloride

Cat. No.: HY-101872A

CAS No.: 2703752-81-0

Molecular Formula: $C_{19}H_{18}ClN_3O_2S_2$

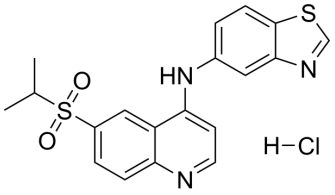
Molecular Weight: 419.95

Target: RIP kinase

Pathway: Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10 mg/mL (23.81 mM; ultrasonic and warming and heat to 60°C)

H_2O : 2.5 mg/mL (5.95 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	2.3812 mL	11.9062 mL	23.8124 mL
5 mM	0.4762 mL	2.3812 mL	4.7625 mL
10 mM	0.2381 mL	1.1906 mL	2.3812 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: ≥ 0.21 mg/mL (0.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GSK-872 hydrochloride is a RIPK3 inhibitor, which binds RIP3 kinase domain with an IC_{50} of 1.8 nM, and inhibits kinase activity with an IC_{50} of 1.3 nM. GSK-872 hydrochloride decreases the RIPK3-mediated necroptosis and subsequent cytoplasmic translocation and expression of HMGB1, as well as ameliorates brain edema and neurological deficits in early brain injury[1][2][3].

IC_{50} & Target

RIPK3

In Vitro

GSK-872 hydrochloride (0.01-3 μM; 24 hours) blocks TNF-induced necroptosis in human HT-29 cells in a concentration-dependent manner[1].

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

Cell Viability Assay[1]

Cell Line:	HT-29 cells
Concentration:	0.01, 0.03 , 0.1, 0.3, 1, and 3 μM
Incubation Time:	24 hours
Result:	Blocked TNF-induced necroptosis in a concentration-dependent manner.

In Vivo

GSK-872 hydrochloride (25 mM; intracerebroventricular injection) can attenuate brain edema and improve neurological function following subarachnoid hemorrhage (SAH) and reduce the number of necrotic cells. GSK-872 hydrochloride can also decrease the protein levels of RIPK3 and MLKL, and cytoplasmic translocation and expression of HMGB1, an important pro-inflammatory protein[3].

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

Animal Model:	Eight weeks old Sprague-Dawley male rats with 300-320 g body weight (rat SAH model)[3]
Dosage:	25 mM/6 μL
Administration:	Syringe pump (intracerebroventricular) at 30 min after SAH
Result:	Attenuated brain edema, improved neurological function and decreased the number of necrotic cells in the ipsilateral cortex. Decreased the expression of RIPK3, MLKL and cytoplasmic HMGB1 at 72 h after SAH in the ipsilateral cortex.

CUSTOMER VALIDATION

- Nature. 2020 Apr;580(7803):386-390.
- Cell Res. 2023 Aug 14.
- Cell Res. 2023 Mar;33(3):201-214.
- Signal Transduct Target Ther. 2020 Oct 9;5(1):235.
- Nat Cell Biol. 2022 Apr;24(4):471-482.

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REFERENCES

[1]. Mandal P, et al. RIP3 induces apoptosis independent of pronecrotic kinase activity. Mol Cell. 2014 Nov 20;56(4):481-95.

[2]. Arora D, et al. Deltamethrin induced RIPK3-mediated caspase-independent non-apoptotic cell death in rat primary hepatocytes. Biochem Biophys Res Commun. 2016 Oct 14;479(2):217-223.

[3]. Chen T, et al. Inhibiting of RIPK3 attenuates early brain injury following subarachnoid hemorrhage: Possibly through alleviating necroptosis. Biomed Pharmacother. 2018;107:563-570.

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