SRS16-86

Cat. No.: HY-135430

CAS No.: 1793052-96-6

Molecular Formula: $C_{26}H_{32}N_4O_2$

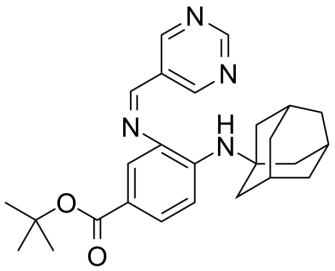
Molecular Weight: 432.56

Target: Ferroptosis

Pathway: Apoptosis

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (77.05 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3118 mL	11.5591 mL	23.1182 mL
	5 mM	0.4624 mL	2.3118 mL	4.6236 mL
	10 mM	0.2312 mL	1.1559 mL	2.3118 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 (5.78 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.78 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.83 mg/mL (1.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

SRS16-86 is a potent inhibitor of ferroptosis[1]. SRS16-86 is more stable than more stable to metabolism and plasma than Ferrostatin-1 in vivo. SRS16-86 can be used for renal ischemia-reperfusion injury (IRI) and spinal cord injury (SCI) research[2][3].

In Vitro

SRS16-86 (1 μM; 24 hours; in the presence or absence of erastin) inhibits ferroptosis in HT-1080 cells and NIH 3T3 cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

SRS16-86 (intraperitoneal injection; 2 mg/kg; 15 min before the onset of surgery) protects mice from functional acute renal failure and structural organ damage after ischemia–reperfusion injury (IRI)[2].

SRS16-86 (intraperitoneal injection; 2 mg/kg; 4 weeks) combination therapy with [Nec-1+SfA] is superior in the prevention of renal IRI compared with the double-combination therapy with [Nec-1+SfA]. Addition of 16-86 reduces plasma levels of serum urea and serum creatinine in IRI mice model[2].

SRS16-86 (intraperitoneal injection; 15 mg/kg; once a day; 7 days) enhances functional recovery after SCI, it decreases the levels of proinflammatory cytokines and the inflammatory adhesion factor in injured spinal cord in rats[3].

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

Animal Model:	Renal IRI model
Dosage:	2 mg/kg; 15 min before the onset of surgery
Administration:	Intraperitoneal injection; 2 mg/kg; 15 min before the onset of surgery
Result:	Was protective from renal IRI.

Animal Model:	Renal IRI model
Dosage:	2 mg/kg
Administration:	Combination with Necrostatin-1/Sanglifehrin A; 2 mg/kg; 4 weeks
Result:	Further increased the protective effect of [Necrostatin-1/Sanglifehrin A] combination therapy in renal IRI model.

REFERENCES

[1]. Sam Hofmans, et al. Novel Ferroptosis Inhibitors with Improved Potency and ADME Properties. J Med Chem. 2016 Mar 10;59(5):2041-53.

[2]. Andreas Linkermann, et al. Synchronized renal tubular cell death involves ferroptosis.Proc Natl Acad Sci U S A. 2014 Nov 25;111(47):16836-41.

[3]. Yan Zhang, et al. Ferroptosis inhibitor SRS 16-86 attenuates ferroptosis and promotes functional recovery in contusion spinal cord injury. Brain Res. 2019 Mar 1;1706:48-57.

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