(S)-Selisistat

Cat. No.: HY-15452A

CAS No.: 848193-68-0

Molecular Formula: $C_{13}H_{13}ClN_2O$

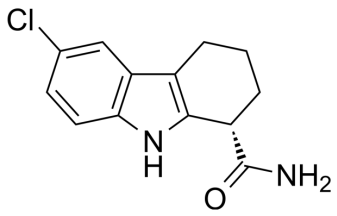
Molecular Weight: 248.71

Target: Sirtuin

Pathway: Cell Cycle/DNA Damage; Epigenetics

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 (402.07 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.0207 mL	20.1037 mL	40.2075 mL
5 mM	0.8041 mL	4.0207 mL	8.0415 mL
10 mM	0.4021 mL	2.0104 mL	4.0207 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 (10.05 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.05 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.05 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

(S)-Selisistat ((S)-EX-527) is a potent and selective SIRT1 inhibitor, with an IC_{50} of 98 nM.

IC_{50} & Target

SIRT1

98 nM (IC_{50})

In Vitro

(S)-Selisistat is an inhibitor of SIRT1 enzymatic activity (IC_{50}, 98 nM), identified in a high-throughput screen using bacterially

	expressed human SIRT1. (S)-Selisistat inhibits SIRT1 in a concentration-dependent manner with an IC_{50} of 38 nM, in agreement with the activity on bacterially expressed SIRT1. (S)-Selisistat has much lower potency against SIRT2 (IC_{50}, 19.6 μM) or SIRT3 (IC_{50}, 48.7 μM) but does not inhibit class I/II HDAC activity at concentrations up to 100 μM[1]. (S)-Selisistat exerts an inhibitory effect on SIRT1 activity without affecting SIRT1 expression on both mRNA and protein levels[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	(S)-Selisistat abolishes Resveratrol (RSV)-induced attenuation of microvascular inflammation in ob/ob septic mice. Finally, ob/ob mice in Sepsis+RSV group has significantly increased 7-day survival vs. Sepsis+Vehicle group[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Napper AD, et al. Discovery of indoles as potent and selective inhibitors of the deacetylase SIRT1. J Med Chem. 2005 Dec 15;48(25):8045-54.

[2]. Solomon JM, et al. Inhibition of SIRT1 catalytic activity increases p53 acetylation but does not alter cell survival following DNA damage. Mol Cell Biol. 2006 Jan;26(1):28-38.

[3]. Jia Y, et al. SIRT1 is a regulator in high glucose-induced inflammatory response in RAW264.7 cells. PLoS One. 2015 Mar 20;10(3):e0120849.

[4]. Wang X, et al. Resveratrol attenuates microvascular inflammation in sepsis via SIRT-1-Induced modulation of adhesion molecules in ob/ob mice. Obesity (Silver Spring). 2015 Jun;23(6):1209-17.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA