Necrostatin-1

Cat. No.: HY-15760

CAS No.: 4311-88-0

Molecular Formula: $C_{13}H_{13}N_3OS$

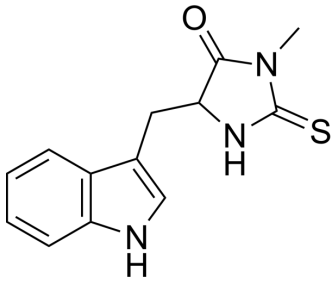
Molecular Weight: 259.33

Target: RIP kinase; Autophagy; Indoleamine 2,3-Dioxygenase (IDO); Ferroptosis

Pathway: Apoptosis; Autophagy; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (482.01 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8561 mL	19.2805 mL	38.5609 mL
	5 mM	0.7712 mL	3.8561 mL	7.7122 mL
	10 mM	0.3856 mL	1.9280 mL	3.8561 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 12.5 mg/mL (48.20 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (8.02 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.02 mM); Clear solution
4. Add each solvent one by one: 10% (50% EtOH >> 50% Cremophor EL) >> 90% saline
   Solubility: 1.67 mg/mL (6.44 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Necrostatin-1 (Nec-1) is a potent and cross the blood-brain barrier necroptosis inhibitor with an EC_{50} of 490 nM in Jurkat cells. Necrostatin-1 inhibits RIP1 kinase (EC_{50}=182 nM). Necrostatin-1 is also an IDO inhibitor[1].

IC_{50} & Target

EC50: 182 nM (RIP1 kinase)[1]

In Vitro	Necrostatin-1 (Nec-1) efficiently inhibits the TNFα-induced necrotic death of L929 cells, which does not require exogenous caspase inhibitors[1]. ?Necrostatin-1 (Nec-1) prevents radiocontrast media (RCM)-induced dilation of peritubular capillaries, suggesting a novel role unrelated to cell death for the RIP1 kinase domain in the regulation of microvascular hemodynamics and pathophysiology of contrast-induced AKI (CIAKI)[2]. ?Necrostatin-1 (Nec-1) (30 μM) increases the survival of cardiomyocyte progenitor cell (CMPCs) by inhibiting necrotic cell death[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Necrostatin-1 (Nec-1) induces tubular bilation and affects the kinetics of the dilation of peritubular capillaries after RCM application. Upon a single intraperitoneal application of a single dose of Necrostatin-1 (1.65 mg/kg body weight, i.p.) 15 minutes before RCM, the return to baseline levels is prevented within the observation period[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	C6 (3×10^5 cells/well) and U87 (1.5×10^5 cells/well) glioma cells are seeded onto 96-well microplate and cultured 24 h. PBS is added into the control group and Shikonin is added into experimental group to reach the final concentration. Cellular viability is assessed using an MTT assay after Shikonin treatment at indicated time point. The absorbance value (A) at 570 nm is read using an automatic multi-well spectrophotometer. Two groups of glioma cells from the same cell line are treated with Shikonin at lower or higher concentration, respectively; other two groups of glioma cells are treated 1 h with 100 μM Necrostatin-1 or 40 μM z-VAD-fmk prior to co-incubation with Shikonin at indicated concentration. Additionally, another two groups of glioma cells are treated only with 100 μM Necrostatin-1 or 40 μM Z-VAD-fmk at corresponding time point[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration	Mice[2] 8-10 week old male C57BL/6 mice (average weight approx.23 g) are used. Mice receive intravenous application of 200 μL PBS or radiocontrast media (RCM) via the tail vein. A single dose of Z-VAD-fmk (10 mg/kg body weight) or Necrostatin-1 (1.65 mg/kg body weight) is applied intraperitoneally 15 min. before RCM-injection. Mice are harvested another 24 hours after RCM-application (48 hours after reperfusion). Blood samples are obtained from retroorbital bleeding and serum levels of urea and creatinine are determined. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nature. 2023 Mar;615(7950):158-167.
- Nature. 2020 Apr;580(7803):386-390.
- Cell. 2024 Feb 1;187(3):624-641.e23.
- Signal Transduct Target Ther. 2020 May 8;5(1):51.
- Circulation. 2022 Nov 30.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Degterev A, et al. Chemical inhibitor of nonapoptotic cell death with therapeutic potential for ischemic brain injury. Nat Chem Biol. 2005 Jul;1(2):112-9.

[2]. Linkermann A, et al. The RIP1-kinase inhibitor necrostatin-1 prevents osmotic nephrosis and contrast-induced AKI in mice. J Am Soc Nephrol. 2013 Oct;24(10):1545-57.

[3]. Huang C, et al. Shikonin kills glioma cells through necroptosis mediated by RIP-1. PLoS One. 2013 Jun 28;8(6):e66326.

[4]. Feyen D, et al. Increasing short-term cardiomyocyte progenitor cell (CMPC) survival by necrostatin-1 did not further preserve cardiac function. Cardiovasc Res. 2013 Jul 1;99(1):83-91.

[5]. Zhou K, et al. RIP1-RIP3-DRP1 pathway regulates NLRP3 inflammasome activation following subarachnoid hemorrhage. Exp Neurol. 2017 Sep;295:116-124.

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