Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Necrostatin-1

Catalog No: tcsc1666

Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
4311-88-0

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

Pathway:
Apoptosis; Autophagy; Metabolic Enzyme/Protease

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

Form:
Light yellow to yellow (Solid)

Purity / Grade:
98.69%

Solubility:
DMSO : ≥ 46 mg/mL (177.38 mM)

Storage Instruction:
2-8°C

Alternative Names:
4-Imidazolidinone, 5-(1H-indol-3-ylmethyl)-3-methyl-2-thioxo

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Observed Molecular Weight:
259.33

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.

Product Description

Necrostatin-1 is a potent, selective and cell-permeable **necroptosis** inhibitor with an $\mathbf{EC_{50}}$ of 490 nM in Jurkat cells. It acts by inhibiting the death domain kinase RIP (**RIP1**) in the necroptosis pathway.

IC50 & Target: RIP1 kinase[1]

In Vitro: Necrostatin-1 (Nec-1) is a specific and potent small-molecule inhibitor of cell death caused by death-domain receptor (DR) stimulation in the presence of caspase inhibition in multiple cell types. Necrostatin-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]. The decreased viability of C6 glioma cells caused by 3.0 μM and 6.0 μM shikonin is improved by pretreatment with Necrostatin-1 (Nec-1) to 92.3% and 82.9% at 1.5 h and 84.4% and 78.6% at 3.0 h, respectively. Similarly, the viability of U87 glioma cells is elevated by Necrostatin-1 to 91.6% and 81.5% at 1.5 h, and 81.8% and 71.2% at 3.0 h, respectively[3]. Necrostatin-1 (Nec-1) (30 μM) increases the survival of cardiomyocyte progenitor cell (CMPCs) by inhibiting necrotic cell death[4].

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].