Necrostatin-1

**Product Data Sheet**

**Cat. No.:** HY-15760  
**CAS No.:** 4311-88-0  
**Molecular Formula:** C₁₃H₁₃N₃OS  
**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 6 months  
-20°C 1 month

**SOLVENT & SOLUBILITY**

**In Vitro**  
DMSO: ≥ 46 mg/mL (177.38 mM)  
*"≥" means soluble, but saturation unknown.*

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>Mass 1 mg</th>
<th>Mass 5 mg</th>
<th>Mass 10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 mM</td>
<td>3.8561 mL</td>
<td>19.2805 mL</td>
<td>38.5609 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.7712 mL</td>
<td>3.8561 mL</td>
<td>7.7122 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3856 mL</td>
<td>1.9280 mL</td>
<td>3.8561 mL</td>
</tr>
</tbody>
</table>

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: ≥ 2.5 mg/mL (9.64 mM); Clear solution  
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)  
Solubility: ≥ 2.5 mg/mL (9.64 mM); Clear solution

**BIOLOGICAL ACTIVITY**

**Description**  
Necrostatin-1 (Nec-1) is a potent necroptosis inhibitor with an EC₅₀ of 490 nM in Jurkat cells. Necrostatin-1 inhibits RIP1 kinase (EC₅₀=182 nM). Necrostatin-1 is also an IDO inhibitor[1].

**IC₅₀ & 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].

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### 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].

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#### 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.

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### REFERENCES


