AK-1

Cat. No.: HY-101465
CAS No.: 330461-64-8
Molecular Formula: C₁₉H₂₁N₃O₅S
Molecular Weight: 403.45
Target: Sirtuin
Pathway: Cell Cycle/DNA Damage; Epigenetics
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: ≥ 50 mg/mL (123.93 mM) *

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Solvent Mass Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>2.4786 mL</td>
<td>12.3931 mL</td>
<td>24.7862 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4957 mL</td>
<td>2.4786 mL</td>
<td>4.9572 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2479 mL</td>
<td>1.2393 mL</td>
<td>2.4786 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: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (6.20 mM); Clear solution

**BIOLOGICAL ACTIVITY**

Description
AK-1 is a potent, specific and cell-permeable SIRT2 inhibitor, with an IC₅₀ of 12.5 μM.

IC₅₀ & Target
SIRT2
12.5 μM (IC₅₀)

In Vitro
AK-1 achieves significant neuroprotection in Huntington’s disease flies at 10 μM, improving the number of rhabdomeres from 5.2 to 5.6.[1] AK-1 is a potent, specific and cell-permeable SIRT2 inhibitor, with an IC₅₀ of 12.5 μM.[2] AK-1 treatment induces proteasomal degradation of the Snail transcription factor through inactivation of the NF-κB/CSN2 pathway. Reduction in the level of Snail results in upregulation of p21, leading to G1 arrest, slow proliferation, and slow wound-healing activity. The regulation of Snail-p21 axis by AK-1 also occurs in HT-29 colon cancer cells.[3]
Under hypoxic conditions, AK-1 increases the ubiquitination of HIF-1α in a VHL-dependent manner, leading to the degradation of HIF-1α via a proteasomal pathway. Downregulation of HIF-1α expression reduces its transcriptional activity and, eventually, reduces the expression of BNIP3, one of HIF-1 target genes, in AK-1-treated cells[4].

**PROTOCOL**

**Cell Assay**[3]

HEK293 cells are co-transfected with 3 μg of pGL2-PGK1-HRE-Luc and 1 μg of pCMV-β-galactosidase plasmids. Twenty-four hours later, the cells are incubated under hypoxic conditions for 24 hr in the presence of 10 μM AK-1 and then lysed with luciferase cell lysis buffer. Luciferase and β-galactosidase activities are measured using luciferin and o-nitrophenyl-β-d-galactopyranoside, respectively, as substrates. Transfection efficiency is normalized according to β-galactosidase activity[3].

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

**REFERENCES**


Caution: Product has not been fully validated for medical applications. For research use only.

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