Anisomycin

Cat. No.: HY-18982
CAS No.: 22862-76-6
Molecular Formula: \( \text{C}_{14}\text{H}_{19}\text{NO}_{4} \)
Molecular Weight: 265.31
Target: DNA/RNA Synthesis; JNK; Bacterial; Apoptosis; Antibiotic
Pathway: Cell Cycle/DNA Damage; MAPK/ERK Pathway; Anti-infection; Apoptosis
Storage: Powder -20°C 3 years
- 4°C 2 years
In solvent -80°C 6 months
- -20°C 1 month

SOLVENT & SOLUBILITY

In Vitro

<table>
<thead>
<tr>
<th>Solvent &amp; Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>3.7692 mL</td>
<td>18.8459 mL</td>
<td>37.6918 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.7538 mL</td>
<td>3.7692 mL</td>
<td>7.5384 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.3769 mL</td>
<td>1.8846 mL</td>
<td>3.7692 mL</td>
</tr>
</tbody>
</table>

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

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 (9.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.42 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Anisomycin is a potent protein synthesis inhibitor which interferes with protein and DNA synthesis by inhibiting peptidyl transferase or the 80S ribosome system\(^1\). Anisomycin is a JNK activator, which increases phospho-JNK\(^2\)[3]. Anisomycin is a bacterial antibiotic isolated from Streptomyces griseolus\(^4\).

<table>
<thead>
<tr>
<th>IC₅₀ &amp; Target</th>
<th>JNK</th>
<th>DNA synthesis</th>
</tr>
</thead>
</table>

Page 1 of 2
### In Vitro

To examine whether JNK has a core role in colistin-induced neurotoxicity in PC-12 cells, an SP600125 (a highly selective inhibitor of JNK) and Anisomycin (a potent activator) are used in this study. In order to select an appropriate concentration, PC-12 cells are treated with a range of SP600125 (0-80 μM) and Anisomycin (0-20 μM) respectively for 24 h. The results show that the cells viability significantly decreases by SP600125 treatment in a concentration-dependent manner, observed at the concentrations greater than 20 μM (p<0.01). Similarly the cells viability is inhibited by Anisomycin treatment (≥8 μM) (p<0.05) \(^1\).

### In Vivo

Disruption of TNFRp55/p75 attenuates Anisomycin-induced ventricular functional improvements. Anisomycin results in an improvement in left ventricular developed pressure (LVDP), which disappears in animals with disruption of TNFR p55/p75. In addition, the Anisomycin-induced improvement in LVEDP in wild-type animals is eliminated by deletion of TNFR p55/p75. Likewise, disruption of TNFR p55/p75 abrogates the recovery of rate pressure product (RPP) elicited by pretreatment of Anisomycin. TNFR p55/p75\(^{-/-}\) mice without Anisomycin treatment do not show differences in cardiac functional recovery compared with the control wild-type mice. There are no significant differences in heart rate between wild-type and TNFR p55/p75-deficient mice. To see whether Nox2 is involved in Anisomycin-induced myocardial protection, Nox2-deficient mice are treated with Anisomycin. The improvement in the LVEDP in Anisomycin-treated mice is eliminated in Nox2\(^{-/-}\) mice compared with wild-type mice. In addition, recovery of RPP in wild-type mice treated with Anisomycin is mitigated in Nox2\(^{-/-}\) mice. Nox2\(^{-/-}\) mice without Anisomycin treatment do not show the differences in cardiac functional recovery compared with wild-type control mice\(^2\).

### PROTOCOL

#### Cell Assay \(^1\)

PC-12 cells are seeded in 96-well plates at a concentration of 1×10\(^4\) cells/well and cultured in an incubator at 37°C with 5% CO\(_2\) for at least 12 h prior to exposure to different concentrations of SP600125 (0-80 μM) or Anisomycin (0-20 μM) for 24 h. Subsequently, the culture medium is added to 20 μL of 5 mg/mL MTT working solution and the plate is incubated for 2 h at 37°C. The culture supernatant is removed and the formazan crystals are dissolved in 150 μL DMSO. Finally, the absorbance of each well is measured at 490 nm by a microplate reader. Cell viability is expressed as the percentage of the control group, which is set to 100%\(^1\).

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

#### Animal Administration \(^2\)

Adult male TNFRp55/p75 mice, adult male wild-type C57/BL and homozygous Nox2\(^{-/-}\) mice are used in this study. Mice are randomized into six experimental groups that undergo the following treatments. Animals are divided into six groups: group 1: control ischemia/reperfusion, wild-type mice are injected with DMSO (0.1 mL); group 2: Anisomycin+wild-type mice, wild-type mice are injected with Anisomycin (0.1 mg/kg ip); group 3: Anisomycin+TNFR p55/p75\(^{-/-}\) mice, TNFR p55/p75\(^{-/-}\) mice are injected with Anisomycin (0.1 mg/kg ip); group 4: TNFR p55/p75\(^{-/-}\) mice, TNFR p55/p75\(^{-/-}\) mice are not injected with Anisomycin; group 5: Anisomycin+Nox2\(^{-/-}\) mice, Nox2\(^{-/-}\) mice are injected with Anisomycin (0.1 mg/kg ip); and group 6: Nox2\(^{-/-}\) mice, Nox2\(^{-/-}\) mice are not injected with Anisomycin. Later (24 h), the hearts are subjected to 30 min of ischemia followed by 30 min of reperfusion\(^2\).

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

### CUSTOMER VALIDATION

- J Hazard Mater. 2020 May.
REFERENCES


