Triciribine

Cat. No.: HY-15457
CAS No.: 35943-35-2
Molecular Formula: C₁₃H₁₆N₆O₄
Molecular Weight: 320.3
Target: DNA/RNA Synthesis; Akt; HIV
Pathway: Cell Cycle/DNA Damage; PI3K/Akt/mTOR; Anti-infection
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: 100 mg/mL (312.21 mM; Need ultrasonic)

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Solvent Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td></td>
<td>3.1221 mL</td>
<td>15.6104 mL</td>
<td>31.2207 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td></td>
<td>0.6244 mL</td>
<td>3.1221 mL</td>
<td>6.2441 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td></td>
<td>0.3122 mL</td>
<td>1.5610 mL</td>
<td>3.1221 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 >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.81 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Triciribine is a DNA synthesis inhibitor, also inhibits Akt and HIV-1/2 with IC₅₀ of 130 nM, and 0.02-0.46 μM, respectively.

<table>
<thead>
<tr>
<th>IC₅₀ &amp; Target</th>
<th>DNA synthesis</th>
<th>Akt</th>
<th>HIV-1</th>
<th>HIV-2</th>
</tr>
</thead>
<tbody>
<tr>
<td>IC₅₀</td>
<td>130 nM (IC₅₀, Cell Assay)</td>
<td>0.02-0.46 μM (IC₅₀)</td>
<td>0.02-0.46 μM (IC₅₀)</td>
<td></td>
</tr>
</tbody>
</table>
### In Vitro

The nucleoside analog Triciribine (TCN) is a purine analog which is initially shown to inhibit DNA synthesis. Triciribine selectively inhibits the phosphorylation and activation of all three Akt isoforms. At a concentration of 10 μM, Triciribine Akt phosphorylation is inhibited at both Thr308 and Ser473. Triciribine effectively inhibits the phosphorylation and consequently the catalytic activity of Akt in PC-3 cells. The Akt inhibitor Triciribine (TCN) does not effectively inhibit the human cell line U87MG but inhibits other astrocytoma cell lines in a grade-dependent manner. The WHO II K1861-10 line is incompletely inhibited (69% maximum inhibition) with a GI₅₀ value of 1.7 μM for Triciribine. Triciribine exhibits maximum growth inhibition around 1-10 μM and inhibits phosphorylation of Akt, as well as downstream p70S6K, to basal levels at 100 μM (IC₅₀ = 130 nM) in KR158 cells. Triciribine (TCN) is a novel tricyclic compound with known antitumor activity. Using a syncytial plaque assay, Triciribine is active against HIV-1 at 0.01-0.02 μM. Using a microtiter XTT assay, Triciribine is active against a panel of HIV-1 and HIV-2 strains at IC₅₀ values ranging from 0.02 to 0.46 μM.

### In Vivo

Triciribine (TCBN) treatment, administered for 7 days after 14 days of hypoxia until 21 days of hypoxia is reached, reversed the vascular thickening as shown by immunohistochemistry and Western analyses. On the other hand, Rapamycin treatment did not prevent hypoxia-induced pulmonary alveolar hemorrhage and congestion. Triciribine partially inhibits progressive pruning of the vasculature.

### PROTOCOL

#### Cell Assay [2]

The human SF295 and U87MG GBM lines and the mouse K1861-10, KR158, and KR130G astrocytoma lines are plated at a density of 2500 cells/100 μL complete media in 96-well plates. Mouse primary astrocytes are plated at 5000 cells/100 μL. Cells are treated in triplicate with serial dilutions of inhibitors (The inhibitors tested are PI-103, Triciribine and Rapamycin) ranging from μM to pM. Cell proliferation is measured after 3 days using the Alamar Blue assay on a Novostar plate reader. Values for 50% inhibitory concentration (IC₅₀) and 50% growth inhibitory concentration (GI₅₀) are calculated using standard procedures in GraphPad Prism v4 and Microsoft Excel. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### Animal Administration [4]

Mice

Akt1⁺/⁺ and Akt1⁻/⁻ mice are subjected to normoxia or hypoxia (10% O₂) for 7 and 14 days (n=2-6 mice per group). Noteworthy, high mortality is observed in Akt1⁻/⁻ mice exposed to hypoxia longer than 14-16 days. For pharmacological inhibition studies, Akt1⁺/⁺ mice, subjected to normoxia or chronic hypoxia for 14 days, received daily i.p. injection of saline, Triciribine (0.5 mg/kg per day) or Rapamycin (1.5 mg/kg per day) for 7 days, and the total continuous exposure to hypoxia or normoxia is 21 days (n=6-8 mice per group). Pharmacological inhibitors are administered daily while the mice are maintained in the hypoxia chamber to minimize exposure to air and spontaneous reversal of pulmonary remodelling.

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

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