FICZ

Cat. No.: HY-12451
CAS No.: 172922-91-7
Molecular Formula: C₁₉H₁₂N₂O
Molecular Weight: 284.31
Target: Aryl Hydrocarbon Receptor
Pathway: Immunology/Inflammation
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>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></td>
<td>1 mM</td>
<td>3.5173 mL</td>
<td>17.5864 mL</td>
<td>35.1729 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.7035 mL</td>
<td>3.5173 mL</td>
<td>7.0346 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.3517 mL</td>
<td>1.7586 mL</td>
<td>3.5173 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: ≥ 0.83 mg/mL (2.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
FICZ is a potent aryl hydrocarbon receptor (AhR) agonist with a $K_d$ of 70 pM.

In Vitro
FICZ (0.01 nM-1 μM) alone or in combination with 50 nM MNF induces sustained CYP1A1 activity and leads to oxidative stress and activation of apoptosis via a mitochondrial-dependent pathway. In HepG2 cells, FICZ stimulates cell growth at low concentrations but inhibits cell growth at high concentrations[1]. FICZ (10,000-30,000 nM) significantly decreases CEH viability with an estimated LC₅₀ (95% confidence intervals) of 14,000 nM. FICZ shows concentration-dependent effects on EROD activity in CEH cultures, with the mean EC₅₀ values at 3, 8, and 24 h of 0.016 nM, 0.80 nM, and 11 nM, respectively[2]. FICZ treatment increases transcript expression of CYP1A1 in a dose-dependent manner in both the parental iPSC line and the CYP1A1 targeted clone[3]. CYP1 inhibition in the presence of FICZ results in enhanced AHR activation, suggesting that FICZ accumulates in the cell when its metabolism is blocked. CYP1 enzymes plays a role in regulating biological effects of FICZ[4].
Nuclear export and degradation of the AHR protein are two additional steps in the AHR-mediated signal transduction pathway\([5]\). Exposure to AhR agonists causes AhR-expressing cells to downregulate the receptor through the ubiquitin/proteasome degradation pathway\([6]\).

**PROTOCOL**

**Cell Assay** \(^\[2\]\)

The cell viability of CEH treated with FICZ or TCDD is studied with the untreated cells (used as a live cell control) and sodium hypochlorite (5\%)-treated cells (used as a dead cell control). This assay is based upon the bioluminescent measurement of adenosine triphosphate (ATP) that is present in all metabolically active cells. Luciferase is utilized in this method to catalyze the formation of light from ATP and luciferin. CEH are lysed 24 h after dosing and the luminescence emitted from the ATP-dependent oxidation of luciferin is measured with a LuminoSkan Ascent luminometer.

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

**REFERENCES**


