Iguratimod

**Cat. No.:** HY-17009  
**CAS No.:** 123663-49-0  
**Molecular Formula:** C₁₇H₁₄N₂O₆S  
**Molecular Weight:** 374.37  
**Target:** COX  
**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**  
DMSO: 33.33 mg/mL (89.03 mM; Need ultrasonic)  
H₂O: < 0.1 mg/mL (insoluble)

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>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>2.6712 mL</td>
<td>13.3558 mL</td>
<td>26.7115 mL</td>
</tr>
<tr>
<td></td>
<td>5 mM</td>
<td>0.5342 mL</td>
<td>2.6712 mL</td>
<td>5.3423 mL</td>
</tr>
<tr>
<td></td>
<td>10 mM</td>
<td>0.2671 mL</td>
<td>1.3356 mL</td>
<td>2.6712 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% (20% SBE-β-CD in saline)  
Solubility: 2.5 mg/mL (6.68 mM); Suspended solution; Need ultrasonic

### BIOLOGICAL ACTIVITY

**Description**  
Iguratimod is an antirheumatic agent, acts as an inhibitor of COX-2, with an IC₅₀ of 20 μM (7.7 μg/mL), but shows no effect on COX-1. Iguratimod also inhibits macrophage migration inhibitory factor (MIF) with an IC₅₀ of 6.81 μM.

**IC₅₀ & Target**  
- COX-2: 20 μM (IC₅₀)  
- MIF: 6.81 μM (IC₅₀)

**In Vitro**  
Iguratimod (T-614) is an antirheumatic agent, acts as an inhibitor of COX-2, with an IC₅₀ of 20 μM (7.7 μg/mL), but shows no effect on COX-1. Iguratimod (0.1, 1, 10 μg/mL) inhibits bradykinin-stimulated PGE2 release from fibroblasts. Iguratimod suppresses the COX activity from bradykinin stimulated fibroblasts in a concentration-dependent manner, with an IC₅₀ of 48 μg/mL. Iguratimod (10 and 30 μg/mL) also dose-dependently inhibits COX-2 mRNA levels[1].
addition, Iguratimod potently inhibits macrophage migration inhibitory factor (MIF) with an IC₅₀ of 6.81 μM. Iguratimod is synergetic with glucocorticoids in vitro[3].

In Vivo

Iguratimod (5 or 20 mg/kg) shows analgesic effect, significantly improves the pain withdrawal threshold of the left hind paw in dose-dependent manner in rats. Iguratimod (5 or 20 mg/kg) reduces the elevation of pERK1/2 and c-Fos in the spinal cord induced by cancer cell inoculation. Iguratimod also dose-dependently decreases the IL-6 levels in rats. In Iguratimod-treated rats, the activity of osteoclasts is weaker than the control group[2]. Iguratimod (20 mg/kg i.p.) shows significantly increased survival in BALB/c mice that are vulnerable to endotoxemia, and attenuates TNFα release measured in serum isolated 90 min post-LPS administration in wild-type C57BL/6 mice[3].

PROTOCOL

Cell Assay [3]

Briefly, human Raji B cells are plated at a density of 0.5 × 10⁴ cells/well in a 96-well plate and synchronized by incubation for 24 h in RPMI 1640 medium supplemented with 0.1-0.5% FBS. Synchronized cells are pretreated with Iguratimod or vehicle for 30 min prior to stimulation with macrophage migration inhibitory factor (MIF) for 24 h. At 20 h BrdU is added to cells and quantified using a BrdU Cell proliferation assay kit[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Administration [3]

Mice[3].

Endotoxemia is induced by intraperitoneal injection of LPS from E. coli O111:B4. In BALB/c animals, 5 mg/kg LPS is used as a lethal dose for survival experiments; animals are treated with Iguratimod (20 mg/kg i.p.) 0.5 h prior to LPS, 6 h after LPS, and then once daily for 3 days and monitored for survival over 2 weeks. In C57BL/6 animals, 20 mg/kg LPS is used as non-lethal dose for plasma cytokine experiments; animals are pretreated with Iguratimod (20 mg/kg i.p.) twice, one dose each at 2 and 0.5 h prior to LPS administration, and euthanized at 90 min post-LPS by CO₂ asphyxiation with cervical dislocation. Blood is collected by cardiac puncture and allowed to clot 20 min at room temperature and 20 min at 4°C; sera are isolated by centrifugation at 300 × g for 10 min and stored at −20°C for further analysis by TNFα ELISA (1:3 dilution)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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REFERENCES


