AdipoRon

Cat. No.: HY-15848
CAS No.: 924416-43-3
Molecular Formula: C₂₇H₂₈N₂O₃
Molecular Weight: 428.52
Target: Adiponectin Receptor
Pathway: GPCR/G Protein
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 : ≥ 44 mg/mL (102.68 mM)
H₂O : < 0.1 mg/mL (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

<table>
<thead>
<tr>
<th>Concentration</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td>2.3336 mL</td>
<td>11.6681 mL</td>
<td>23.3361 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.4667 mL</td>
<td>2.3336 mL</td>
<td>4.6672 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.2334 mL</td>
<td>1.1668 mL</td>
<td>2.3336 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 (5.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
AdipoRon is an orally active adiponectin receptor (AdipoR) agonist, binding to AdipoR1 and AdipoR2 with Kᵦₛ of 1.8 and 3.1 μM, respectively.

IC₅₀ & Target
Kᵦ: 1.8 μM (AdipoR1), 3.1 μM (AdipoR2) [1]
### In Vitro

AdipoRon is an orally active and specific AdipoR agonist, binds to AdipoR1 and AdipoR2, with $K_d$s of 1.8 and 3.1 μM. AdipoRon (50 nM-50 μM) increases AMPK phosphorylation via AdipoR1. AdipoRon (50 μM) dose-dependently attenuates the expression of TNF-α and TGF-β1 in the L02 cells. AdipoRon exhibits significant and dosage-dependent growth suppression on macrophages. AdipoRon treatment significantly improves cardiac functional recovery after reperfusion, and inhibits post-MI apoptosis. AdipoRon exerts vasodilation by mechanisms distinct to adiponectin and induces vasorelaxation without a marked decrease in VSMC [$Ca^{2+}$].

### In Vivo

AdipoRon (50 mg/kg, i.v.) causes significant phosphorylation of AMPK in skeletal muscle and liver of wild-type mice but not Adipop1−/− Adipop2−/− double-knockout mice. AdipoRon (0.02, 0.1, and 0.5 mg/kg, i.g.) alleviates D-GalN induced hepatotoxicity in mice, and prevents hepatic architecture distortion against D-GalN challenge. The hepatoprotective potential of AdipoRon is particularly evident in higher dosages (0.1 and 0.5 mg/kg). Enhanced cardiomyocyte apoptosis in APN-deficient mice is rescued by AdipoRon (50 mg/kg, p.o.) administration. Antiapoptotic effect of AdipoRon is attenuated but not lost in AMPK-DN mice.

### PROTOCOL

#### Cell Assay [2]

The effects of AdipoRon on the proliferation of parenchymal and non-parenchymal hepatocytes are evaluated in vitro via L02 and RAW264.7, by MTT assay as described with slight modification: 100 μL cells suspension ($6 \times 10^4$/mL) are seeded in a 96-well plate and incubated for 18 h. Fresh media with AdipoRon are added at specified concentrations, and the incubations continue for a further 24 h. Then cells are incubated for 4 h with 0.5 mg/mL of MTT, and analyzed in a microplate reader at 490 nm. Each group is performed in six replications. The mean absorbance values corrected for a blank (medium only) are calculated as percentages of survival. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

#### Animal Administration [2]

After 3 days of acclimation, mice are randomly divided into six groups (9 mice in each): control, model, bicyclol (20 mg/kg), AdipoRon (0.02 mg/kg, 0.1 mg/kg, 0.5 mg/kg). The synthetic AdipoRon and bicyclol are dissolved in DMSO and diluted by saline containing 0.5% sodium carboxymethyl cellulose (CMC-Na) [final vehicle: 5% DMSO (v/v) saline solution]. All test groups are administered with vehicle (control and model groups) or therapeutic agents (bicyclol or AdipoRon groups) at a dosing volume of 10 mL/kg, by intragastric (i.g.) gavage twice per day for three consecutive days prior to D-GalN administration. 2 h after last treatment, mice are challenged with a single intraperitoneal (i.p.) administration of D-GalN saline solution at a dose of 600 mg/kg to induce acute liver injury, while the control group mice receive saline instead. Then mice are fasted for 20 h before orbital blood collection. Finally, all animals are sacrificed by cervical dislocation, and livers are harvested for biochemical or histopathology analysis. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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