Rosiglitazone

Cat. No.: HY-17386
CAS No.: 122320-73-4
Molecular Formula: C₁₈H₁₉N₃O₃S
Molecular Weight: 357.43
Target: Ferroptosis; PPAR; TRP Channel; Autophagy
Pathway: Apoptosis; Cell Cycle/DNA Damage; Membrane Transporter/Ion Channel; Neuronal Signaling; Autophagy
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 : ≥ 110 mg/mL (307.75 mM)
* "≥" means soluble, but saturation unknown.

<table>
<thead>
<tr>
<th>Preparing Stock Solutions</th>
<th>Mass</th>
<th>1 mg</th>
<th>5 mg</th>
<th>10 mg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 mM</td>
<td></td>
<td>2.7978 mL</td>
<td>13.9888 mL</td>
<td>27.9775 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td></td>
<td>0.5596 mL</td>
<td>2.7978 mL</td>
<td>5.5955 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td></td>
<td>0.2798 mL</td>
<td>1.3989 mL</td>
<td>2.7978 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: ≥ 2.75 mg/mL (7.69 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.75 mg/mL (7.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (7.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description
Rosiglitazone (BRL 49653) is a selective PPARγ agonist with EC₅₀s of 30 nM, 100 nM and 60 nM for PPARγ1, PPARγ2, and PPARγ, respectively.

IC₅₀ & Target

<table>
<thead>
<tr>
<th>PPARγ1</th>
<th>PPARγ2</th>
<th>TRPC5</th>
<th>TRPM2</th>
</tr>
</thead>
</table>
Rosiglitazone is a potent and selective activator of PPARγ, with EC50s of 30 nM and 100 nM for PPARγ1 and PPARγ2, respectively, and a Kd of appr 40 nM for PPARγ. Rosiglitazone (BRL49653, 0.1, 1.10 μM) promotes differentiation of C3H10T1/2 stem cells to adipocytes[1]. Rosiglitazone (Compound 6) activates PPARγ, with an EC50 of 60 nM[2]. Rosiglitazone (1 μM) activates PPARγ, which binds to NF-κα promoter to activate gene transcription in neurons. Rosiglitazone (1 μM) also protects Neuro2A cells and hippocampal neurons against oxidative stress, and up-regulates BCL-2 expression in an NF-κα-dependent manner[3]. Rosiglitazone completely inhibits TRPM3 with IC50 values of 9.5 and 4.6 μM against nifedipine- and PregS-evoked activity, but such effects are not via PPARγ. Rosiglitazone inhibits TRPM2 at higher concentration, with an IC50 of appr 22.5 μM. Rosiglitazone is a strong stimulator of TRPC5 channels, with an EC50 of -30 μM[4].

Rosiglitazone (5 mg/kg, p.o.) decreases the serum glucose in diabetic rats. Rosiglitazone also decreases IL-6, TNF-α, and VCAM-1 levels in diabetic group. Rosiglitazone in combination with losartan increases glucose compared to diabetic and Los-treated groups. Rosiglitazone significantly ameliorates endothelial dysfunction indicated by a significantly lower contractile response to PE and Ang II and enhancement of ACh-provoked relaxation in aortas isolated from diabetic rats[5].

**PROTOCOL**

**Kinase Assay**[1]

cDNA encoding amino acids 174-475 of PPARγ1 is amplified via polymerase chain reaction and inserted into bacterial expression vector pGEX-2T. GST-PPARγ LBD is expressed in BL21(DE3)plysS cells and extracts. For saturation binding analysis, bacterial extracts (100 μg of protein) are incubated at 4°C for 3 h in buffer containing 10 mM Tris (pH 8.0), 50 mM KCl, 10 mM dithiothreitol with [3H]-BRL49653 (specific activity, 40 Ci/mmol) in the presence or absence of unlabeled Rosiglitazone. Bound is separated from free radioactivity by elution through 1-mL Sephadex G-25 desalting columns. Bound radioactivity eluted in the column void volume and is quantitated by liquid scintillation counting[1].

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

**Cell Assay**[1]

C3H10T1/2 cells are grown in a 24-well plate in DME medium supplemented with 10% fetal calf serum. Medium and compound (Rosiglitazone) are exchanged every 3 days. Cells are stained at day 7 with Oil Red O and photographed[1].

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**Animal Administration**[2]

Rats are intravenously injected with 38 mg/kg streptozotocin and after 48 h, diabetes is identified by urinary glucosuria and then random blood sugar is measured and this day is regarded as day 0. Animals with a serum glucose level of 220-300 mg/dL are selected to be used in this study. Rats are randomly separated into five groups for daily drug administration for 8 weeks: group 1: control nondiabetic rats given a vehicle only (0.5 mL/kg of 0.5% carboxy methyl cellcellose orally), group 2: control diabetic rats given a vehicle, group 3: diabetic rats receiving Rosiglitazone (5 mg/kg orally), group 4: diabetic rats receiving losartan (2 mg/kg, orally), and group 5: diabetic rats receiving both Rosiglitazone and losartan[2].

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REFERENCES


