Rosiglitazone maleate

Cat. No.: HY-14600

CAS No.: 155141-29-0

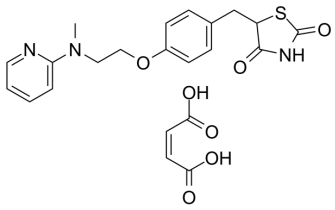
Molecular Formula: $C_{22}H_{23}N_3O_7S$

Molecular Weight: 474

Target: PPAR; TRP Channel; Autophagy; Autophagy; Ferroptosis

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; Membrane Transporter/Ion Channel; Neuronal Signaling; Autophagy; Apoptosis

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (210.97 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (ultrasonic) (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1097 mL	10.5485 mL	21.0970 mL
	5 mM	0.4219 mL	2.1097 mL	4.2194 mL
	10 mM	0.2110 mL	1.0549 mL	2.1097 mL

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 (5.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.27 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.27 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rosiglitazone maleate (BRL 49653C) is a potent and selective activator of PPARγ, with EC_{50}s of 30 nM, 100 nM and 60 nM for PPARγ1, PPARγ2, and PPARγ, respectively, and a K_d of appr 40 nM for PPARγ; Rosiglitazone maleate is also an modulator of TRP channels, inhibits TRP melastatin 2 (TRPM2), TRPM3 and activates TRP canonical 5 (TRPC5).

IC_{50} & Target	PPARγ1	PPARγ2
	30 nM (EC50)	100 nM (EC50)

In Vitro	Rosiglitazone maleate is a potent and selective activator of PPARγ, with EC_{50}s of 30 nM and 100 nM for PPARγ1 and PPARγ2, respectively, and a K_d 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 EC_{50} of 60 nM[2]. Rosiglitazone (1 μM) activates PPARγ, which binds to NF-α1 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-α1-dependent manner[3]. Rosiglitazone completely inhibits TRPM3 with IC_{50} 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 IC_{50} of appr 22.5 μM. Rosiglitazone is a strong stimulator of TRPC5 channels, with an EC_{50} of -30 μM[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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 [^{3}H]-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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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 celleluse 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]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Circulation. 2022 Nov 30.
- Cell Metab. 2023 Dec 5;35(12):2165-2182.e7.
- Cell Metab. 2023 Sep 7;S1550-4131(23)00304-2.
- Cell Metab. 2021 Mar 2;33(3):581-597.e9.
- Nat Commun. 2023 Jun 2;14(1):3208.

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REFERENCES

[1]. Lehmann JM, et al. An antidiabetic thiazolidinedione is a high affinity ligand for peroxisome proliferator-activated receptor gamma (PPAR gamma). J Biol Chem. 1995 Jun 2;270(22):12953-6.

[2]. Willson TM, et al. The structure-activity relationship between peroxisome proliferator-activated receptor gamma agonism and the antihyperglycemic activity of thiazolidinediones. J Med Chem. 1996 Feb 2;39(3):665-8.

[3]. Thouennon E, et al. Rosiglitazone-activated PPARγ induces neurotrophic factor-α1 transcription contributing to neuroprotection. J Neurochem. 2015 Aug;134(3):463-70.

[4]. Majeed Y, et al. Rapid and contrasting effects of rosiglitazone on transient receptor potential TRPM3 and TRPC5 channels. Mol Pharmacol. 2011 Jun;79(6):1023-30.

[5]. Ateyya H, et al. Beneficial effects of rosiglitazone and losartan combination in diabetic rats. Can J Physiol Pharmacol. 2018 Mar;96(3):215-220.

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