Rosiglitazone hydrochloride

Cat. No.: HY-17386A

CAS No.: 302543-62-0

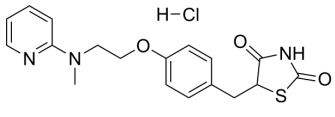
Molecular Formula: $C_{18}H_{20}ClN_3O_3S$

Molecular Weight: 393.89

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

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

Storage: -20°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 : 62.5 mg/mL (158.67 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5388 mL	12.6939 mL	25.3878 mL
	5 mM	0.5078 mL	2.5388 mL	5.0776 mL
	10 mM	0.2539 mL	1.2694 mL	2.5388 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: ≥ 10 mg/mL (25.39 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 10 mg/mL (25.39 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% saline
   Solubility: ≥ 10 mg/mL (25.39 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rosiglitazone (BRL 49653) hydrochloride is an orally active selective PPARγ agonist (EC_{50}: 60 nM, K_d: 40 nM). Rosiglitazone hydrochloride is a TRPC5 activator (EC_{50}: 30 μM) and TRPM3 inhibitor. Rosiglitazone hydrochloride can be used in the research of obesity and diabetes, senescence, ovarian cancer[1][2][4][7].

IC_{50} & Target				
	PPARγ 40 nM (Kd)	PPARγ 60 nM (EC50)	TRPC5 30 μM (EC50)	TRPM3

In Vitro

Rosiglitazone hydrochloride (0.1-10 μM, 72 h) results in pluripotent C3H10T1/2 stem cell differentiation to adipocytes[1].
Rosiglitazone hydrochloride (1 μM, 24 h) activates PPARγ, which binds to NF-α1 promoter to activate gene transcription in neurons[3].
Rosiglitazone hydrochloride (1 μM, 24 h) protects Neuro2A cells and hippocampal neurons against oxidative stress, and up-regulates BCL-2 expression in an NF-α1-dependent manner[3].
Rosiglitazone hydrochloride (0.01-100 μM, 15 min) inhibits TRPM3 with IC_{50} values of 9.5 and 4.6 μM against nifedipine- and PregS-evoked activity respectively[4].
Rosiglitazone hydrochloride (0.5-50 μM, 7 days) inhibits ovarian cancer cell proliferation[7].
Rosiglitazone hydrochloride (5 μM, 7 days) suppresses Olaparib (HY-10162)‐induced alterations of cellular senescence and promotes apoptosis in A2780 and SKOV3 cells[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[7]

Cell Line:	A2780 and SKOV3 cells
Concentration:	0.5-50 μM
Incubation Time:	1-7 days
Result:	Inhibited cell proliferation in a time‐dependent and concentration‐dependent manner.

Western Blot Analysis[3]

Cell Line:	Hippocampal neurons
Concentration:	1 μM
Incubation Time:	1 μM
Result:	Increased NF-α1 and BCL-2 protein level.

In Vivo

Rosiglitazone hydrochloride (oral administration, 5 mg/kg, daily for 8 weeks) decreases the serum glucose in diabetic rats[5].
Rosiglitazone hydrochloride (intraperitoneal injection, 3 mg/kg/day) ameliorates airway inflammation induced by cigarette smoke via inhibiting the M1 macrophage polarization by activating PPARγ and RXRα in male Wistar rats[6].
Rosiglitazone hydrochloride (intraperitoneal injection, 10 mg/kg, once every 2 days) inhibits subcutaneous ovarian cancer growth in A2780 and SKOV3 mouse subcutaneous xenograft models[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Streptozotocin (STZ)-induced diabetic rats[5]
Dosage:	5 mg/kg
Administration:	Oral administration, daily for 8 weeks.
Result:	Decreased IL-6, TNF-α, and VCAM-1 levels in diabetic group. Displayed lower levels of lipid peroxidation and NOx with an increase in aortic GSH and SOD levels compared to diabetic groups.

Animal Model:	Male Wistar rats[6]
Dosage:	3 mg/kg/day
Administration:	Intraperitoneal injection, twice a day, 6 days per week for 12 consecutive weeks
Result:	Ameliorated emphysema, elevated PEF, and higher level of total cells, neutrophils and

	cytokines (TNF-α and IL-1β) induced by cigarette smoke (CS). Inhibited CS-induced M1 macrophage polarization and decreased the ratio of M1/M2.

CUSTOMER VALIDATION

- Cancer Cell. 2024 Apr 4:S1535-6108(24)00090-4.
- 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.

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REFERENCES

[1]. Haoshen Feng, et al. Rosiglitazone ameliorated airway inflammation induced by cigarette smoke via inhibiting the M1 macrophage polarization by activating PPARγ and RXRα. Int Immunopharmacol. 2021 Aug;97:107809.

[2]. Zehua Wang, et al. Rosiglitazone ameliorates senescence and promotes apoptosis in ovarian cancer induced by olaparib. Cancer Chemother Pharmacol. 2020 Feb;85(2):273-284.

[3]. 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.

[4]. 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.

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

[6]. 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.

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

Caution: Product has not been fully validated for medical applications. For research use only.

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