Troglitazone

Cat. No.: HY-50935

CAS No.: 97322-87-7

Molecular Formula: $C_{24}H_{27}NO_5S$

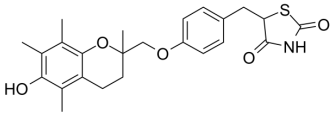
Molecular Weight: 441.54

Target: PPAR; Autophagy; Apoptosis; Ferroptosis

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (283.10 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2648 mL	11.3240 mL	22.6480 mL
	5 mM	0.4530 mL	2.2648 mL	4.5296 mL
	10 mM	0.2265 mL	1.1324 mL	2.2648 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na >> 0.5% Tween-80
   Solubility: 10 mg/mL (22.65 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.66 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (4.71 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Troglitazone is an orally active PPARγ agonist, with EC_{50}s of 550 nM and 780 nM for human and murine PPARγ receptor, respectively. Troglitazone has anticancer activity, prevents and inhibits the development of type 2 diabetes.

IC_{50} & Target

PPARγ
550 nM (EC50, Human PPARγ)

In Vitro	Troglitazone (2-200 μM, 24 h) is cytotoxic to the pancreatic cancer cell lines (MIA Paca2 and PANC-1 cells), with IC_{50}s of 49.9±1.2 and 51.3±5.3 μM, respectively[2]. Troglitazone (50 μM, 24 h) increases chromatin condensation in MIA Paca2 and PANC-1 cells, enhances the activity of caspase-3 and decreases Bcl-2 expression[2]. Troglitazone (0-4 μM, 12 h) sensitizes TRAIL-mediated apoptosis in human lung adenocarcinoma cells (A549, HCC-15 and Calu-3)[3]. Troglitazone (0-4 μM, 12 h) induces autophagy and sensitized apoptosis mediated by TRAIL in A549 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Troglitazone (200 mg/kg, p.o., every day for 5 weeks) shows inhibitory effects on the growth of tumor in the MIA Paca2 xenograft model[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Briefly, cells are seeded into 96-well plates at a density of 1×10^5 cells/well and incubated for 24 h. The cells are treated with Troglitazone in the presence or absence of other chemicals for a further 24 h using FBS-free medium. The assay utilizes the conversion of alamar blue reagent to fluorescent resorufin by metabolically active cells. The resorufin signal is measured at an excitation wavelength of 530 nm and an emission wavelength of 580 nm. The 50% growth inhibitory concentrations (IC_{50}) are calculated according to the sigmoid inhibitory effect model $E = IC_{50}\ \gamma/(IC_{50}\ \gamma + C\gamma)$, where E represents the surviving fraction (% of control), C represents the drug concentration in the medium, and γ represents the Hill coefficient. For co-exposure studies, the Troglitazone dosage is set to approximately the IC_{50} value for each cell line[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Balb/c male mice (4 weeks old) are subcutaneously inoculated in the back with MIA Paca2 cells (5×10^6 cells/100 μL in PBS) 14 days prior to starting Troglitazone administration. Mice are then orally administered 200 mg/kg Troglitazone in 0.5% methylcellulose solution or vehicle daily for 5 weeks. Tumor size is measured bi-dimensionally and the volume is calculated using the formula $(\text{length} \times \text{width}^2) \times 0.5$. Body weights of mice are also monitored throughout the experiment[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Metab. 2023 Jan 3;35(1):200-211.e9.
- Proc Natl Acad Sci U S A. 2019 Feb 19;116(8):2996-3005.
- Cell Rep. 2023 May 5;42(5):112481.
- Front Immunol. 2022 Aug 25;13:958677.
- Antioxidants (Basel). 2022 Mar 31;11(4):682.

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REFERENCES

[1]. Knowler WC, et al. Prevention of type 2 diabetes with troglitazone in the Diabetes Prevention Program. Diabetes. 2005 Apr;54(4):1150-6.

[2]. Willson TM, et al. The PPARs: from orphan receptors to drug discovery. J Med Chem. 2000 Feb 24;43(4):527-50.

[3]. Fujita M, et al. In vitro and in vivo cytotoxicity of troglitazone in pancreatic cancer. J Exp Clin Cancer Res. 2017 Jul 3;36(1):91.

[4]. Nazim UM, et al. PPARγ activation by troglitazone enhances human lung cancer cells to TRAIL-induced apoptosis via autophagy flux. Oncotarget. 2017 Apr 18;8(16):26819-26831.

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