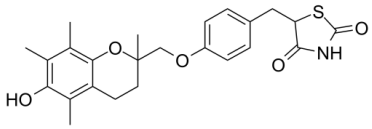
Data Sheet

Product Name:	Troglitazone
Cat. No.:	CS-1634
CAS No.:	97322-87-7
Molecular Formula:	C24H27NO5S
Molecular Weight:	441.54
Target:	Apoptosis; Autophagy; Ferroptosis; PPAR
Pathway:	Apoptosis; Autophagy; Cell Cycle/DNA Damage
Solubility:	DMSO : ≥ 100 mg/mL (226.48 mM)



BIOLOGICAL ACTIVITY:

Troglitazone is a **PPARγ** agonist, with **EC_{50}**s of 550 nM and 780 nM for human and murine PPARγ receptor, respectively. IC50 & Target: EC50: 550 nM (Human PPARγ), 780 nM (Murine PPARγ)[1] **In Vitro:** Troglitazone is a **PPARγ** agonist, with EC_{50}s of 550 nM and 780 nM for human and murine PPARγ receptor, respectively[1]. Troglitazone (2-200 μM) 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. Troglitazone (50 μM) increases chromatin condensation in MIA Paca2 and PANC-1 cells, enhances the activity of caspase-3 and decreases Bcl-2 expression[2]. Troglitazone (0, 1, 2, and 4 μM) sensitizes TRAIL-mediated apoptosis in human lung adenocarcinoma cells. Troglitazone enhancement of TRAIL-induced apoptosis is blocked by inhibition of autophagy, via activation of autophagy flux. In addition, the effects of troglitazone are induced by PPARγ activation in A549 cells[3]. **In Vivo:** Troglitazone (200 mg/kg, p.o.) shows inhibitory effects on the growth of tumor in the MIA Paca2 xenograft model[2].

PROTOCOL (Extracted from published papers and Only for reference)

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]. **Animal Administration:** Troglitazone is formulated in 0.5% methylcellulose solution.[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 (length × $width^2$) × 0.5. Body weights of mice are also monitored throughout the experiment[2].

References:

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

[2]. 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.

[3]. 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.

CAIndexNames:

2,4-Thiazolidinedione, 5-[[4-[(3,4-dihydro-6-hydroxy-2,5,7,8-tetramethyl-2H-1-benzopyran-2-yl)methoxy]phenyl]methyl]-

SMILES:

O=C(SC1CC(C=C2)=CC=C2OCC3(C)OC(C(CC3)=C4C)=C(C)C(C)=C4O)NC1=O

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

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