Balaglitazone

Cat. No.: HY-16086

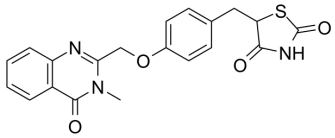
CAS No.: 199113-98-9

Molecular Formula: $C_{20}H_{17}N_3O_4S$

Molecular Weight: 395.43

Target: PPAR

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



Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (252.89 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5289 mL	12.6445 mL	25.2889 mL
	5 mM	0.5058 mL	2.5289 mL	5.0578 mL
	10 mM	0.2529 mL	1.2644 mL	2.5289 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 (6.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.32 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Balaglitazone is a selective partial PPARγ agonist with an EC_{50} of 1.351 μM for human PPARγ.

IC_{50} & Target

PPARγ
351 nM (EC50, Human PPARγ)

In Vitro

Balaglitazone is a selective partial PPARγ agonist with an EC_{50} of 1.351 μM[1]. Balaglitazone (5-100 μM) has equal cytotoxicity

	towards K562 and K562/DOX cells. Balaglitazone decreases doxorubicin cytotoxicity in K562 and K562/DOX cells, with IC_{50}s of 0.117 μM and 0.53 μM, respectively. Balaglitazone reverses multidrug resistance (MDR) in K562/DOX cells. Balaglitazone (25 μM) increases Rh123 accumulation in K562/DOX cells, but does not increases MFI in K562 cells. Balaglitazone downregulates P-gp expression in K562/DOX cells, and such effects are via upregulation of PTEN in K562/DOX cells, and be abolished by PTEN inhibition[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Balaglitazone (3 mg/kg, p.o.) shows antihyperglycaemic activity in fully diabetic and insulin resistant db/db mice, and is more potent than the full PPARγ agonist rosiglitazone[1]. Balaglitazone (10 mg/kg, p.o.) suppresses overall glucose, decreases insulin levels, and increases bodyweight in male diet-induced obese rats, and such effects are equal to that of 30 mg/kg pioglitazone[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	MTT assay is used for cell viability analyses. Briefly, K562 and K562/DOX cells are seeded in a 96-well plate in RPMI-1640 medium supplemented with 10% FBS at the density of 2×10^4 cells/well. After 24 h incubation, various concentrations of doxorubicin (DOX) with or without balaglitazone are diluted in RPMI-1640 medium (without FBS) and added into each well. Experiments for each group are performed in triplicates and with a blank control. After 48 h of treatment, the medium is removed and 200 μL of RPMI-1640 medium supplemented with 10% FBS and 10% MTT (5 mg/mL) is added. After incubation for another 4 h, the reduced intracellular formazan product is dissolved by replacing 100 μL of RPMI-1640 medium with the same volume of dimethyl sulfoxide (DMSO). Absorbance values are measured at 570 nm with a micro plate reader. The half maximal inhibitory concentration (IC_{50}) of each experiment is calculated. The resistance fold (RF) is calculated by dividing the IC_{50} value of treatment in resistant cells by the IC_{50} value of treatment in corresponding parental cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Antihyperglycaemic effects of balaglitazone and rosiglitazone are assessed in adult male diabetic db/db mice. At 14 weeks of age, animals are randomised according to fasting blood glucose into 11 groups (n = 6). Mice are dosed orally once daily for 9 days with vehicle (0.2% carboxymethyl cellulose (CMC) + 0.4% Tween-80 in saline) or increasing doses of either balaglitazone (0.1; 0.3; 1.0; 3.0; 10.0 mg/kg/day) or rosiglitazone (0.2; 0.6; 2.0; 6.0 mg/kg/day). After 7 days of treatment, plasma samples obtained in the morning (between 8:00 and 10:00 AM) are analysed for glucose and insulin. After 9 days of treatment, animals are exposed to an oral glucose tolerance test (OGTT; 3.0 g/kg). The resulting area under the curve is calculated for each of the doses[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Microbiol. 2019 Jan 8;9:3257.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Larsen PJ, et al. Dissociation of antihyperglycaemic and adverse effects of partial perioxisome proliferator-activated receptor (PPAR-gamma) agonist balaglitazone. Eur J Pharmacol. 2008 Oct 31;596(1-3):173-9.

[2]. Yousefi B, et al. Balaglitazone reverses P-glycoprotein-mediated multidrug resistance via upregulation of PTEN in a PPARγ-dependent manner in leukemia cells. Tumour Biol. 2017 Oct;39(10):1010428317716501.

[3]. Henriksen K, et al. A comparison of glycemic control, water retention, and musculoskeletal effects of balaglitazone and pioglitazone in diet-induced obese rats. Eur J Pharmacol. 2009 Aug 15;616(1-3):340-5.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA