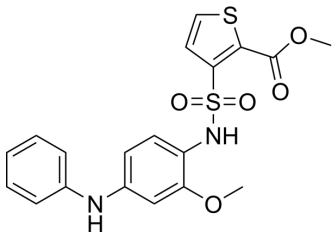
GSK0660

Cat. No.:	HY-12377
CAS No.:	1014691-61-2
Molecular Formula:	$C_{19}H_{18}N_2O_5S_2$
Molecular Weight:	418.49
Target:	PPAR
Pathway:	Cell Cycle/DNA Damage; 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 : ≥ 49 mg/mL (117.09 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.3895 mL	11.9477 mL	23.8954 mL
5 mM	0.4779 mL	2.3895 mL	4.7791 mL
10 mM	0.2390 mL	1.1948 mL	2.3895 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 (5.97 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

GSK0660 is a potent antagonist of PPARβ and PPARδ, with IC_{50}s of 155 nM for both isoforms.

IC_{50} & Target

PPARβ/δ
155 nM (IC_{50})

In Vitro

GSK0660 is a potent antagonist of PPARβ and PPARδ, with IC_{50}s of both 155 nM, and is nearly inactive on PPARα and PPARγ with IC_{50}s of both >10 μM. GSK0660 antagonizes 100% of the activity of PPARβ/δ with a pIC_{50} of 6.8. GSK0660 (100 nM) reduces CPT1a (a PPARβ/δ target gene) expression below the basal vehicle-treated level by approximately 50%, but shows no effect on PDK4 expression, which is also a PPARβ/δ target gene in skeletal muscle cells[1].
GSK0660 (0.5 μM) reduces the levels of AMPK and eNOS phosphorylation, and BMP-2, Runx-2 mRNA expression in MC3T3-E1 cells. GSK0660 (0.1 and 0.5 μM) reverses the bezafibrate-induced enchancement of ALP activity on d 7 in MC3T3-E1 cells[2].

GSK0660 (1 μM) markedly blocks GW501516-mediated attenuation of glutamate release, and the effect of GW501516 on ROS generation in BV-2 cells stimulated with LPS. Furthermore, GSK0660 significantly reduces inhibitory effect of GW501516 on the LPS-induced expression of gp91phox mRNA in BV-2 cells[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]

Cell viability is determined by the MTT dye. MC3T3-E1 cells are incubated with bezafibrate (1−1000 μM) for 24, 48, or 72 h, and are pretreated with the AMPK inhibitor compound C (5 μM), PPARβ inhibitor GSK0660 (0.5 μM), PPARα inhibitor MK886 (10 μM), or NOS inhibitor L-NAME (1000 μM) followed by bezafibrate (100 μM) incubation for 48 h. After the incubations, 10 μL of MTT is added to each well of a 96-well microplate, and the microplates are placed in an incubator at 37°C for 4 h. One hundred fifty microliters of DMSO is added to all wells and mixed thoroughly to lyse the cells and dissolve the dark blue crystals. After 10 min, the absorbance is measured at 570 nm using a microplate reader[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Adv Res. 2020 Jun 20;27:115-125.
- Brain Res Bull. 2018 Jun;140:378-391.
- Microvasc Res. 2023 Mar 22;148:104531.
- PPAR Res. 2020 Dec 28.

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REFERENCES

[1]. Shearer BG, et al. Identification and characterization of a selective peroxisome proliferator-activated receptor beta/delta (NR1C2) antagonist. Mol Endocrinol. 2008 Feb;22(2):523-9. Epub 2007 Nov 1.

[2]. Zhong X, et al. Bezafibrate enhances proliferation and differentiation of osteoblastic MC3T3-E1 cells via AMPK and eNOS activation. Acta Pharmacol Sin. 2011 May;32(5):591-600.

[3]. Lee WJ, et al. Activation of PPARδ attenuates neurotoxicity by inhibiting lipopolysaccharide-triggered glutamate release in BV-2 microglial cells. J Cell Biochem. 2018 Feb 1.

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