Ciprofibrate

Cat. No.: HY-B0664

CAS No.: 52214-84-3

Molecular Formula: $C_{13}H_{14}Cl_2O_3$

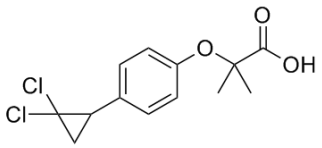
Molecular Weight: 289.15

Target: PPAR

Pathway: Cell Cycle/DNA Damage

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (345.84 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.4584 mL	17.2921 mL	34.5841 mL
5 mM	0.6917 mL	3.4584 mL	6.9168 mL
10 mM	0.3458 mL	1.7292 mL	3.4584 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 (8.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.65 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.65 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ciprofibrate (Win35833) is a potent peroxisome proliferator and increases the phosphorylation level of the PPARalpha[1]. Ciprofibrate acts as an orally active hypolipidaemic agent and can be used for the research of primary hyperlipidaemias[2].

IC_{50} & Target

PPARα

In Vitro

Ciprofibrate (500 μM; 4 hours) increases the PPARa phosphorylation level in rat Fao cells[1].

	In a LucLite assay, Ciprofibrate (10-100μM; 24 hours) induces PPARR activation by existing increased LUC activities in the rat liver H4IIEC3 cells transfected with PPRE-AB LUC reporter gene plasmid[2]. Ciprofibrate (10-100 μM; 24 hours) is not cytotoxic for HepG2 cells, and the cell viability is 99.7%[3]. Ciprofibrate (100 μM; 24 hours) also abolishes FFAs mixture-induced lipid deposition and decreases FFAs mixture-increased TG contents in HepG2 cells[3]. Ciprofibrate (100 μM; 24 hours) almost entirely eliminates the FFAs mixture-induced inflammatory cytokines overproduction, including MCP-1, TNF-α, and IL-6 in HepG2 cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Ciprofibrate (oral administration; 10 mg/kg/day; 3 days) does not result in any significant effects on body weight or absolute liver weight for MCD diet-fed mice. Ciprofibrate improves hepatic steatosis and reduced hepatic necro-inflammation in MCD diet-fed mice. It also reduced hepatic cytokine protein and mRNA levels (MCP-1, TNFα and IL-6) as compared to those of choline-deficient (MCD) diet-fed mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice (six-week-old males)[3]
Dosage:	10 mg/kg
Administration:	Oral administration; 10 mg/kg/day; 3 days
Result:	Decreased MCD diet-resulted hepatic steatosis and hepatic necro-inflammation in mice.

REFERENCES

[1]. Passilly, P., et al., Phosphorylation of peroxisome proliferator-activated receptor alpha in rat Fao cells and stimulation by ciprofibrate. Biochem Pharmacol, 1999. 58(6): p. 1001-8.

[2]. Agnes M Rimando, et al. Pterostilbene, a new agonist for the peroxisome proliferator-activated receptor alpha-isoform, lowers plasma lipoproteins and cholesterol in hypercholesterolemic hamsters. J Agric Food Chem. 2005 May 4;53(9):3403-7.

[3]. Thing-Fong Tzeng, et al. 6-gingerol protects against nutritional steatohepatitis by regulating key genes related to inflammation and lipid metabolism. Nutrients. 2015 Feb 4;7(2):999-1020.

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