Clofibrate

Cat. No.: HY-B0287

CAS No.: 637-07-0

Molecular Formula: $C_{12}H_{15}ClO_3$

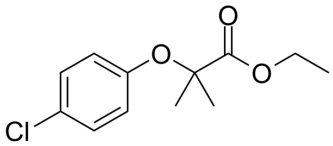
Molecular Weight: 242.7

Target: PPAR

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

Storage:

Pure form	-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 (412.03 mM)

H_2O : < 0.1 mg/mL (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1203 mL	20.6016 mL	41.2031 mL
5 mM	0.8241 mL	4.1203 mL	8.2406 mL
10 mM	0.4120 mL	2.0602 mL	4.1203 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 (10.30 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.30 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.30 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Clofibrate is an agonist of PPAR, with EC_{50}s of 50 μM, -500 μM for murine PPARα and PPARγ, and 55 μM, -500 μM for human PPARα and PPARγ, respectively.

IC_{50} & Target

PPARα	PPARγ
50 μM (EC50)	500 μM (EC50)

In Vitro	Clofibrate is a PPAR agonist, with E_{50}s of 50 μM, -500 μM for murine PPARα and PPARγ, and 55 μM, -500 μM for human PPARα and PPARγ, respectively[1]. Clofibrate (0.5, 1, 2 mM) increases FABP1 expression in two fatty acid (FA)-treated rat hepatoma cells. Clofibrate lowers ROS levels after early treatment, much more than late treatment in FA-treated cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Clofibrate (0.5%) up-regulates serum concentrations and hepatic expression of FGF21 in fetuses, with a return to basal levels after Clofibrate administration withdrawal. Clofibrate administration-offspring have significantly higher expression of thermogenic genes (Ucp1, Cidea, Ppara Ppargc1a, Cpt1b) and UCP1 protein levels in response to HFD in inguinal fat, but not in retroperitoneal (combined with perirenal) or epididymal fat[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cells are seeded at a density of 2.5×10^4 cells/well (for WST-1, intracellular lipid droplet quantification and dichlorofluorescein (DCF) assay, 96-well plates) and 1×10^5 cells/well (for Nile Red Staining, 12-well plates) in MEM/EBSS medium and incubated overnight for adherence. The next day cell culture medium is replaced with freshly prepared medium containing the fatty acid mixture oleate:palmitate (2:1) in presence of 3% fatty-acid-free bovine serum albumin. Cells are treated with 0, 0.5, 1, 2, and 3 mM fatty acid (FA) mixture for 24 and 48 hr at 37°C in a humidified incubator in an atmosphere of 95% air and 5% CO_2. Clofibrate is used to increase levels of FABP1 in treated cell cultures. Clofibrate (500 μM) is dissolved in DMSOand later added to the medium (DMSO < 0.1% v/v in final volume). Control cells are incubated with DMSO alone. Four different cell treatments includ 1-day FA treatment, 2-day FA treatment, early clofibrate intervention and late clofibrate intervention[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Female and male C57BL/6JNarl mice are used for breeding. Females with parity from 1 to 5 are used. Pregnant females are fed either a control (C) or experimental (CF) diet from breeding to parturition. The C diet is based on an AIN-93M diet with a slight modification to contain 21 kcal% fat from soybean oil, whereas the CF diet is the C diet with addition of 0.5% clofibrate. Pregnancy is dated by the presence of a vaginal plug (defined as pregnancy day 1). After spontaneous parturition (pregnancy day 19.5 ± 0.5), all littermates are uniformly nursed by dams fed the C diet for 3 wk, with litter sizes adjusted to 8-10, weaned onto a nonpurified standard diet for 4 wk, and then switched to a HFD (51 kcal% fat, butter-based) for 5 wk. In this study, only male offspring are used and 2 groups of offspring are designated, according to their mother's diet (C or CF). All mice are kept in a room maintained at 23 ± 2°C, with a controlled 12-h-light:-dark cycle with ad libitum to feed and drinking water. Body weight and feed intake are recorded weekly[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

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]. Chen Y, et al. Clofibrate Attenuates ROS Production by Lipid Overload in Cultured Rat Hepatoma Cells. J Pharm Pharm Sci. 2017;20(0):239-251.

[3]. Chen SH, et al. Prenatal PPARα activation by clofibrate increases subcutaneous fat browning in male C57BL/6J mice fed a high-fat diet during adulthood. PLoS One. 2017 Nov 2;12(11):e0187507.

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