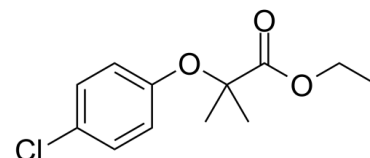


## Data Sheet

Product Name:	Clofibrate
Cat. No.:	CS-2300
CAS No.:	637-07-0
Molecular Formula:	C <sub>12</sub> H <sub>15</sub> ClO <sub>3</sub>
Molecular Weight:	242.70
Target:	PPAR
Pathway:	Cell Cycle/DNA Damage
Solubility:	DMSO : ≥ 100 mg/mL (412.03 mM); H <sub>2</sub> O : < 0.1 mg/mL (insoluble)



### BIOLOGICAL ACTIVITY:

Clofibrate is an agonist of **PPAR**, with **EC<sub>50</sub>s** of 50  $\mu$ M,  $\square$ 500  $\mu$ M for murine PPAR $\alpha$  and PPAR $\gamma$ , and 55  $\mu$ M,  $\square$ 500  $\mu$ M for human PPAR $\alpha$  and PPAR $\gamma$ , respectively. IC<sub>50</sub> & Target: EC<sub>50</sub>: 50  $\mu$ M (Murine PPAR $\alpha$ ),  $\square$ 500  $\mu$ M (Murine PPAR $\gamma$ ), 55  $\mu$ M (Human PPAR $\alpha$ ),  $\square$ 500  $\mu$ M (Human PPAR $\gamma$ )<sup>[1]</sup> **In Vitro**: Clofibrate is a PPAR agonist, with **E<sub>50</sub>s** of 50  $\mu$ M,  $\square$ 500  $\mu$ M for murine PPAR $\alpha$  and PPAR $\gamma$ , and 55  $\mu$ M,  $\square$ 500  $\mu$ M for human PPAR $\alpha$  and PPAR $\gamma$ , respectively<sup>[1]</sup>. 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<sup>[2]</sup>. **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<sup>[3]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay**: Clofibrate is dissolved in DMSO, and added to the medium (DMSO < 0.1% v/v in final volume).<sup>[1]</sup> Cells are seeded at a density of **2.5 × 10<sup>4</sup> cells/well** (for WST-1, intracellular lipid droplet quantification and dichlorofluorescein (DCF) assay, 96-well plates) and **1 × 10<sup>5</sup> 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<sub>2</sub>. Clofibrate is used to increase levels of FABP1 in treated cell cultures. **Clofibrate (500  $\mu$ M) is dissolved in DMSO** and later added to the medium (DMSO < 0.1% v/v in final volume). Control cells are incubated with DMSO alone. Four different cell treatments include 1-day FA treatment, 2-day FA treatment, early clofibrate intervention and late clofibrate intervention<sup>[1]</sup>. **Animal Administration**: Clofibrate is prepared in diet containing 21 kcal% fat from soybean oil.<sup>[3]</sup> **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<sup>[3]</sup>.

## 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 PPAR $\alpha$  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.

## CAIndexNames:

Propanoic acid, 2-(4-chlorophenoxy)-2-methyl-, ethyl ester

## SMILES:

CC(C)(OC1=CC=C(C1)C=C1)C(OCC)=O

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

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