Fenofibric acid

Cat. No.: HY-B0760

CAS No.: 42017-89-0

Molecular Formula: $C_{17}H_{15}ClO_4$

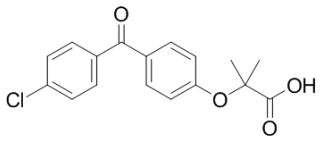
Molecular Weight: 318.75

Target: PPAR; COX

Pathway: Cell Cycle/DNA Damage; Immunology/Inflammation

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 (313.73 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	3.1373 mL	15.6863 mL	31.3725 mL
5 mM	0.6275 mL	3.1373 mL	6.2745 mL
10 mM	0.3137 mL	1.5686 mL	3.1373 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 (7.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fenofibric acid, an active metabolite of fenofibrate, is a PPAR activitor, with EC_{50}s of 22.4 μM, 1.47 μM, and 1.06 μM for PPARα, PPARγ and PPARδ, respectively; Fenofibric acid also inhibits COX-2 enzyme activity, with an IC_{50} of 48 nM.

IC_{50} & Target

PPARδ	PPARγ	PPARα	COX-2
1.06 μM (EC50)	1.47 μM (EC50)	22.4 μM (EC50)	48 μM (IC_{50})

In Vitro

Fenofibric acid is a PPAR activitor, with EC_{50}s of 22.4 μM, 1.47 μM, and 1.06 μM for PPARα, PPARγ and PPARδ, respectively[1]. Fenofibric acid (10, 25, 50, 75, and 100 nM) dose-dependently inhibits COX-2 enzyme, with IC_{50} of 48 nM[2]. Fenofibric acid

	(500 nM) reduces abundance of AOX1 protein in HepG2 cells[3]. Fenofibric acid (100 μM) decreases JNK1/2, c-Jun, and p38 MAPK phosphorylation, and prevents the accumulation of reactive oxygen species, endoplasmic reticulum (ER) stress and disruption of blood retinal barrier (BRB) in response to the combination of high-glucose (HG) and hypoxia in ARPE-19 cells. Fenofibric acid (100 μM) activates IGF-IR/Akt/ERK1/2-mediated survival signaling pathways in ARPE-19 cells under HG conditions and hypoxia[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Fenofibric acid (1, 5, 10 mg/kg, p.o.) shows anti-inflammatory activity in Wistar rats with acute inflammation induced by carrageenan[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [4]	ARPE-19 cells are cultured under normoglycemic (5.5 mM D-glucose) or hyperglycemic (25 mM D-glucose) conditions for 18 days at 37°C under 5% (v/v) CO_2 in medium DMEM/F12 supplemented with 10% (v/v) fetal serum (FS) and penicillin/streptomycin. ARPE-19 cells are used and the media is changed every 3-4 days. The conditions tested are: (1) Control cells which are maintained in 5.5 mM D-glucose (normal glucose) for 18 days. (2) Cells cultured in 5.5 mM D-glucose treated with 100 μM Fenofibric acid for 72 h (days 16, 17, and 18; 1 application/day). (3) Cells cultured as in (1) or (2) and submitted to hypoxia (1% oxygen) for the last 6 or 24 h. (4) Cells maintained in 25 mM D-glucose (HG) for 18 days. (5) Cells cultured in 25 mM D-glucose treated with 100 μM Fenofibric acid for 72 h (days 16, 17, and 18; 1 application/day). (6)Cells cultured as in (4) or (5) and submitted to hypoxia (1% oxygen) for the last 6 or 24 h[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	The anti-inflammatory activity of fenofibrate and its active metabolite fenofibric acid is assessed by injecting 0.1 mL of 1% carrageenan solution prepared in saline (sub-plantar) to the right hind paw of the rats. Rats are divided into 6 groups of six animals each. The first group serves as negative control and receives 1% tween-80 in distilled water, 10 mL/kg body mass. Group 2 and 3 receive a single dose of fenofibrate and standard drug diclofenac at 10 mg/kg body mass, whereas groups 4, 5, and 6 receive 3 doses of Fenofibric acid at 1, 5, and 10 mg/kg body mass, respectively. All the drugs are given orally using gavages 60 min before the injection of 0.1 mL of 1% carrageenan through sub-plantar route. The volume of oedema of test and control groups is measured using plethysmometer at 0, 1, 2, and 3 h after induction of inflammation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Dietz M, et al. Comparative molecular profiling of the PPARα/γ activator aleglitazar: PPAR selectivity, activity and interaction with cofactors. ChemMedChem. 2012 Jun;7(6):1101-11.

[2]. Prasad GS, et al. Anti-inflammatory activity of anti-hyperlipidemic drug, fenofibrate, and its phase-I metabolite fenofibric acid: in silico, in vitro, and in vivo studies. Inflammopharmacology. 2017 Dec 13.

[3]. Neumeier M, et al. Aldehyde oxidase 1 is highly abundant in hepatic steatosis and is downregulated by adiponectin and fenofibric acid in hepatocytes in vitro. Biochem Biophys Res Commun. 2006 Nov 24;350(3):731-5. Epub 2006 Sep 27.

[4]. Miranda S, et al. Beneficial effects of fenofibrate in retinal pigment epithelium by the modulation of stress and survival signaling under diabetic conditions. J Cell Physiol. 2012 Jun;227(6):2352-62.

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

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