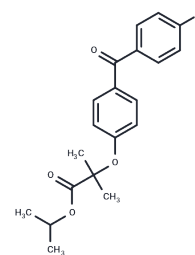


Fenofibrate

Chemical Properties

CAS No. :	49562-28-9
Formula:	C ₂₀ H ₂₁ ClO ₄
Molecular Weight:	360.83
Appearance:	no data available
Storage:	Powder: -20°C for 3 years In solvent: -80°C for 1 year



Biological Description

Description	Fenofibrate (Lipanthyl) is a PPAR α agonist (EC ₅₀ =30 μ M) and is selective. Fenofibrate also inhibits cytochrome P450 isoforms, such as CYP2C19, CYP2B6, CYP2C9, CYP2C8, and CYP3A4. Fenofibrate exhibits antihyperlipidemic activity.
Targets(IC ₅₀)	Autophagy,Cytochromes P450,PPAR
In vitro	<p>METHODS: MOLM13 cells were treated with LCL161 (1000-160000 nM) and PKC412 (2.5-40 nM) for 3 days, and cell viability was measured by trypan blue exclusion.</p> <p>RESULTS: LCL161 moderately inhibited cell growth when administered alone. the combination of LCL161 and PKC412 killed significantly more MOLM13-luc+ cells than either drug alone, and the Calculusyn Combination Index suggests a synergistic effect. [1]</p> <p>METHODS: NSCLC cells A549 and H460 were treated with LCL161 (10 μM) and paclitaxel (10 μM) for 48 h and apoptosis was detected by flow cytometry.</p> <p>RESULTS: Apoptosis was significantly reduced in the LCL161/paclitaxel combination treatment group compared to cells treated with LCL161 or paclitaxel alone. [2]</p>
In vivo	<p>METHODS: To test the antitumor activity in vivo, LCL161 (50 mg/kg twice daily) and PKC412 (40 mg/kg once daily) were administered by gavage to NCr nude mice injected with Ba/F3-FLT3-ITD-luc+ tumor cells for seven days.</p> <p>RESULTS: LCL161 significantly enhanced the ability of PKC412 to inhibit the growth of Ba/F3-FLT3-ITD-luc+ cells in vivo. the difference between the inhibition of leukemia growth by PKC412 or LCL161 alone and the combined inhibition of leukemia growth by PKC412+LCL161 was also significant. The weight of spleens of mice treated with PKC412+LCL161 percentage was less than that of mice treated with vector alone or either drug. [1]</p>
Kinase Assay	The half-maximal inhibitory concentrations (IC ₅₀ s) of Fenofibrate, statins (atorvastatin, lovastatin, pravastatin, simvastatin and simvastatin acid, the active form of simvastatin) and glipizide for recombinant human CYP1A2, CYP2B6, CYP2C8, CYP2C9, CYP2C19, CYP2D6, and CYP3A4 are determined using fluorometric CYP450 inhibition assays. Briefly, the drugs are dissolved in methanol or acetonitrile. In 96 well assay plates, the drugs are diluted to a series of concentrations in a solution containing cofactors including NADP ⁺ (final concentration 1.3 mM), MgCl ₂ (final concentration 3.3 mM), glucose-6-phosphate (G6P, final concentration 3.3 mM) and glucose 6-phosphate dehydrogenase (final concentration 0.4 U/mL). The mixture is pre-incubated at 37°C for 10 min. The enzymes and fluorogenic substrates are diluted to desired concentrations in sodium phosphate reaction buffer (pH 7.4, final concentration 200 mM) and mixed.

Reactions are initiated with addition of the enzyme and substrate mixture to the cofactor and drug mixture. The final reaction volume of all assays is 200 μ L. After incubating at 37°C for a pre-specified period of time (15 to 45 min), the reactions are stopped with addition of 75 μ L quenching solution (0.5 M Tris base or 2N NaOH). Fluorescence is determined using a BioTek Synergy 2 fluorescence reader. Each of the drugs is tested at eight concentrations in duplicate. To estimate IC50s, percent of inhibition is calculated using net fluorescence that is corrected for the background. The values of percent of inhibition are then fitted to a three or four parameter log-logistic model[1].

Solubility Information

Solubility	Ethanol: 36.1 mg/mL (100.05 mM), Sonication is recommended. DMSO: 55 mg/mL (152.43 mM), Sonication is recommended. 10% DMSO+40% PEG300+5% Tween 80+45% Saline: 5.5 mg/mL (15.24 mM), Solution. (< 1 mg/ml refers to the product slightly soluble or insoluble)
------------	--

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.7714 mL	13.8569 mL	27.7139 mL
5 mM	0.5543 mL	2.7714 mL	5.5428 mL
10 mM	0.2771 mL	1.3857 mL	2.7714 mL
50 mM	0.0554 mL	0.2771 mL	0.5543 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Reference

- Wilk A, et al. Molecular mechanisms of fenofibrate-induced metabolic catastrophe and glioblastoma cell death. *Mol Cell Biol.* 2015 Jan;35(1):182-98.
- Zeng X, Zhu S, Lu W, et al. Target identification among known drugs by deep learning from heterogeneous networks. *Chemical Science.* 2020, 11(7): 1775-1797.
- Zhang Y, Li T, Pan M, et al. SIRT1 prevents cigarette smoking-induced lung fibroblasts activation by regulating mitochondrial oxidative stress and lipid metabolism. *Journal of Translational Medicine.* 2022, 20(1): 1-13
- You BJ, et al. Fenofibrate induces human hepatoma Hep3B cells apoptosis and necroptosis through inhibition of thioesterase domain of fatty acid synthase. *Sci Rep.* 2019 Mar 1;9(1):3306.
- Kong R, Wang N, Han W, et al. Fenofibrate Exerts Antitumor Effects in Colon Cancer via Regulation of DNMT1 and CDKN2A. *PPAR research.* 2021, 2021.
- Frias FT, et al. Fenofibrate reverses changes induced by high-fat diet on metabolism in mice muscle and visceral adipocytes. *J Cell Physiol.* 2018 Apr;233(4):3515-3528.
- Zeng X, Zhu S, Lu W, et al. Target identification among known drugs by deep learning from heterogeneous networks[J]. *Chemical Science.* 2020, 11(7): 1775-1797.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

This product is for Research Use Only· Not for Human or Veterinary or Therapeutic Use

Tel:781-999-4286 E_mail:info@targetmol.com Address:36 Washington Street,Wellesley Hills,MA 02481