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Lieferung & Zahlungsart

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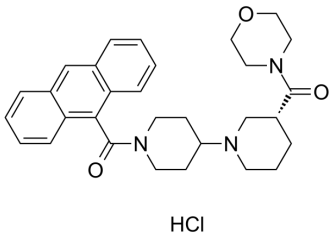
CP-640186 hydrochloride

Cat. No.:	HY-15259A
CAS No.:	591778-70-0
Molecular Formula:	$C_{30}H_{36}ClN_3O_3$
Molecular Weight:	522.08
Target:	Acetyl-CoA Carboxylase
Pathway:	Metabolic Enzyme/Protease
Storage:	4°C, sealed storage, away from moisture * In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (95.77 mM; Need ultrasonic)
DMSO : ≥ 48 mg/mL (91.94 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9154 mL	9.5771 mL	19.1542 mL
5 mM	0.3831 mL	1.9154 mL	3.8308 mL
10 mM	0.1915 mL	0.9577 mL	1.9154 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (191.54 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.79 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.79 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.79 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CP-640186 hydrochloride is an orally active and cell-permeable Acetyl-CoA carboxylase (ACC) inhibitor with IC_{50}s of 53 nM and 61 nM for rat liver ACC1 and rat skeletal muscle ACC2 respectively. Acetyl-CoA carboxylase (ACC) is a key enzyme of fatty acid metabolism that enables the synthesis of malonyl-CoA. CP-640186 hydrochloride can also stimulate muscle fatty acid oxidation[1][2].

IC_{50} & Target	IC50: 53 nM (rat liver ACC1) and 61 nM (rat skeletal muscle ACC2)[1]

In Vitro

CP-640186 (20 μM; 48 h) treatment can inhibit H460 cell growth[3].
CP-640186 (0.1 nM-100 μM; 2 h) treatment increases fatty acid metabolism in a concentration-dependent manner in C2C12 cells and muscle strips[1].
CP-640186 (0.62-1.8 μM; 2 h) treatment inhibits fatty acid synthesis and TG synthesis in HepG2 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[3]

Cell Line:	Human fibroblasts and H460 cells
Concentration:	20 μM
Incubation Time:	48 hours
Result:	Led to a ⊠30% decrease in cell number compared to vehicle-treated controls.

Cell Viability Assay[1]

Cell Line:	C2C12 cells and muscle strips
Concentration:	0.1 nM-100 μM
Incubation Time:	2 hours
Result:	Stimulated palmitate acid oxidation with an EC_{50} of 57 nM and a maximal stimulation of 280% in C2C12 cells. Stimulated palmitate acid oxidation with an EC_{50} of 1.3 μM and a maximal stimulation of 240% in isolated rat epitrochlearis muscle.

Cell Viability Assay[1]

Cell Line:	HepG2 cells
Concentration:	0.62-1.8 μM
Incubation Time:	6 hours
Result:	Inhibited fatty acid synthesis and TG synthesis in HepG2 cells with EC_{50}s of 0.62 μM and 1.8 μM, respecticely.

In Vivo

CP-640186 (oral gavage; 4.6-21 mg/kg; once) demonstrates acute efficacy[1].
CP-640186 (intravenous injection and oral gavage; Intravenous dose, 5 mg/kg; oral dose, 10 mg/kg; once) shows lowe drug exposure in the rat than the ob/ob mouse at equal doses[1].
CP-640186 (oral gavage; 100 mg/kg; once) treatment shows a complete shift from carbohydrate utilization to fatty acid utilization as a source of energy at high exposure level[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male ob/ob mice[1]
Dosage:	4.6-21 mg/kg
Administration:	Oral gavage; 4.6-21 mg/kg; once
Result:	Demonstrated acute efficacy for up to 8 h after oral administration, exhibiting ED_{50} values of 4.6, 9.7, and 21 mg/kg, at 1, 4, and 8 h, respectively, after treatment.

Animal Model:	Male Sprague-Dawley rats[1]
Dosage:	Intravenous dose, 5 mg/kg; oral dose, 10 mg/kg
Administration:	Intravenous injection and oral gavage; intravenous dose, 5 mg/kg; oral dose, 10 mg/kg; once
Result:	Showed a plasma half-life of 1.5 h, a bioavailability of 39%, a Cl_p of 65 ml/min/kg, a V_{dss} of 5 liters/kg, an oral T_{max} of 1.0 h, an oral C_{max} of 345 ng/mL, and an oral $AUC_{0-\infty}$ of 960 ng•h/mL.

Animal Model:	Male ob/ob mice[1]
Dosage:	Intravenous dose, 5 mg/kg; oral dose, 10 mg/kg
Administration:	Intravenous injection and oral gavage; Intravenous dose, 5 mg/kg; oral dose, 10 mg/kg; once
Result:	Showed a plasma half-life of 1.1 h, a bioavailability of 50%, a Cl_p of 54 ml/min/kg, an oral T_{max} of 0.25 h, an oral C_{max} of 2177 ng/mL, and an oral $AUC_{0-\infty}$ of 3068 ng•h/mL.

Animal Model:	Twenty male Sprague-Dawley rats (350-400 g) fasted and then refed a high sucrose diet for 2 days; additional eight rats fasted for 24 h[1]
Dosage:	100 mg/kg
Administration:	Oral gavage; 100 mg/kg; once
Result:	Resulted in time-dependent reductions in RQ (a ratio of CO_2 production to O_2 consumption) of up to 64%.

CUSTOMER VALIDATION

- J Exp Med. 2021 Dec 6;218(12):e20210639.
- Nutrients. 2021 May 21;13(6):1740.
- Front Oncol. 2021 Apr 22;11:665763.
- Front Oncol. 2021 Apr 6.
- Viruses. 2019 Dec 10;11(12):1145.

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REFERENCES

[1]. Daniel Hess, et al. Inhibition of stearoylCoA desaturase activity blocks cell cycle progression and induces programmed cell death in lung cancer cells. PLoS One. 2010 Jun 30;5(6):e11394.

[2]. Harwood HJ Jr, et al. Isozyme-nonselective N-substituted bipiperidylcarboxamide acetyl-CoA carboxylase inhibitors reduce tissue malonyl-CoA concentrations, inhibit fatty acid synthesis, and increase fatty acid oxidation in cultured cells and in experiment

[3]. Yamashita T, et al. Design, synthesis, and structure-activity relationships of spirolactones bearing 2-ureidobenzothiophene as acetyl-CoA carboxylases inhibitors. Bioorg Med Chem Lett. 2011 Nov 1;21(21):6314-8.

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