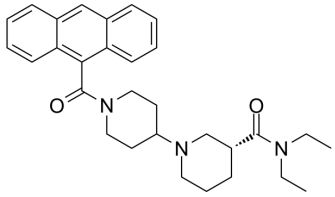
CP-610431

Cat. No.:	HY-16946
CAS No.:	591778-83-5
Molecular Formula:	$C_{30}H_{37}N_3O_2$
Molecular Weight:	471.63
Target:	Acetyl-CoA Carboxylase
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

CP-610431 is a reversible, ATP-uncompetitive, isozyme-nonselective acetyl-CoA carboxylase (ACC) inhibitor. CP-610431 inhibits ACC1 and ACC2 with IC_{50}s of ~50 nM. CP-610431 can be used for the research of metabolic syndrome[1].

In Vitro

CP-610431 is the active R-enantiomer of CP-497485. CP-610431 is more potent than the racemate CP-497485 in inhibiting rat ACC1 (IC_{50}=35.7 nM) and ACC2 (IC_{50}=55 nM), whereas the S-enantiomer, CP-610432, does not substantially inhibit either ACC isoform at concentrations of up to 3 μM. CP-610431 is more potent than CP-497485 in inhibiting HepG2 cell fatty acid and triglyceride (TG) synthesis and in inhibiting TG and apoB secretion[1].

CP-610431 inhibits fatty acid synthesis, triglyceride synthesis, TG secretion, and apolipoprotein B secretion in HepG2 cells (ACC1) with EC_{50}s of 1.6, 1.8, 3.0, and 5.7 μM, without affecting either cholesterol synthesis or apolipoprotein CIII secretion[1].

CP-610431 inhibits both liver and skeletal muscle ACC activity from all three species with essentially equal potency (rat, 36 versus 55 nM; mouse, 50 versus 63 nM; cynomolgus macaque, 70 versus 26 nM) [1].

CP-610431 inhibits mouse primary hepatocyte fatty acid and TG synthesis with IC_{50} values of 0.11 and 1.2 μM and inhibits TG secretion with an IC_{50} of 10 μM[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HepG2 cells
Concentration:	0.1, 1, 10 μM
Incubation Time:	24 hours
Result:	Dose-dependently inhibited HepG2 cell fatty acid synthesis with an IC_{50} of 1.6 μM, TG synthesis with an IC_{50} of 1.8 μM, TG secretion with an IC_{50} of 3.0 μM, and apoB secretion with an IC_{50} of 5.7 μM.

In Vivo

CP-610431 inhibits fatty acid synthesis in CD1 mice and ob/ob mice within 1 h after dose with ED_{50}s of 22 and 4 mg/kg, respectively[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	CD1 mice[1]

Dosage:	30 and 100 mg/kg for fasting CD1 mice; 10, 30, and 100 mg/kg for non-fasting CD1 mice
Administration:	Intraperitoneal administration; 1 hour
Result:	Inhibited hepatic fatty acid synthesis in fasting CD1 mice by 64±12%, and 77±4% at doses of 30 and 100 mg/kg, respectively. Inhibited hepatic fatty acid synthesis in non-fasting CD1 mice by 18%, 51%, and 75% at doses of 10, 30 and 100 mg/kg, respectively.

REFERENCES

[1]. H James Harwood 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 experimental animals. J Biol Chem. 2003 Sep 26;278(39):37099-111.

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