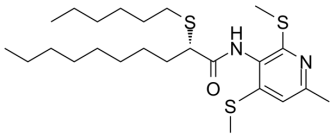
CP-113818

Cat. No.:	HY-105445
CAS No.:	135025-12-6
Molecular Formula:	$C_{24}H_{42}N_2OS_3$
Molecular Weight:	470.8
Target:	Acyltransferase
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	CP-113818 is a potent cholesterol acyltransferase (ACAT) inhibitor. CP-113818 can be used for the research of Alzheimer's disease[1].
In Vitro	CP-113818 inhibits Aβ production in cell-based experiments[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	CP-113818 (0-7.1 mg/kg/day) markedly reduces amyloid pathology in a mouse model of Alzheimer's disease[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6, hAPP (human amyloid precursor protein) transgenic mice[1]
Dosage:	0, 0.2, 1.6, 3.2, 4.8, and 7.1 mg/kg/day
Administration:	Via implantable slow-release biopolymer pellets, 21 days for nontransgenic mice or 60 days for hAPP mice
Result:	Reduced total cholesterol levels by 29% in the serum, hepatic free cholesterol and cholesteryl-esters were also decreased in a dose-dependent manner by up to 37% and 93%, respectively in the nontransgenic mice. Effectively reduced cholesteryl-ester levels of hAPP mice in the absence of adrenal toxicity, reduced plaque numbers, and decreased amyloid load in a gender-independent manner in hAPP mice. Reduced levels of “insoluble” and soluble $A\beta_{1-40}$ and $A\beta_{1-42}$ in the brains of hAPP transgenic mice. Restored normal spatial learning and memory in female hAPP mice in a morris water maze test. Reduced processing of endogenous APP but not notch or N-cadherin, without directly inhibiting β- and γ-secretase activities or Aβ aggregation in nontransgenic littermates.

REFERENCES

[1]. Hutter-Paier B, et al. The ACAT inhibitor CP-113,818 markedly reduces amyloid pathology in a mouse model of Alzheimer's disease. Neuron. 2004 Oct 14;44(2):227-38.

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