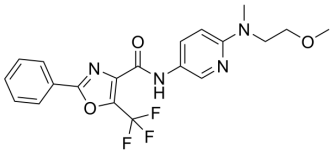
DGAT1-IN-3

Cat. No.:	HY-16434
CAS No.:	939375-07-2
Molecular Formula:	$C_{20}H_{19}F_3N_4O_3$
Molecular Weight:	420.39
Target:	Acyltransferase
Pathway:	Metabolic Enzyme/Protease
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (237.87 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3787 mL	11.8937 mL	23.7874 mL
	5 mM	0.4757 mL	2.3787 mL	4.7575 mL
	10 mM	0.2379 mL	1.1894 mL	2.3787 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 (5.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

DGAT1-IN-3 is a potent, selective and orally bioavailable inhibitor of DGAT-1, with IC_{50}s of 38 nM for human DGAT-1 and 120 nM for rat DGAT-1. DGAT1-IN-3 could be used to research of obesity, dyslipidemia, and metabolic syndrome[1][2].

IC_{50} & Target

IC50: 38 nM (human DGAT-1); 120 nM (rat DGAT-1)[2]

In Vitro

DGAT1-IN-3 blocks the human ether-a-go-go-related gene (hERG) encoded potassium channel with an IC_{20} of 0.2 μM[1].
DGAT1-IN-3 inhibits human DGAT-1 in CHOK1 cells with an EC_{50} of 0.66 μM[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

DGAT1-IN-3 (5-50 mg/kg; p.o once daily for three weeks) reduces weight gain and plasma triglycerides, and improves lipid profile[2].
DGAT1-IN-3 (50 mg/kg; p.o) exhibits good oral bioavailability (77%) and the maximum exposure level in plasma (C_{max}) is 24 μM[2].
DGAT1-IN-3 (5 mg/kg; i.v) exhibits terminal elimination half-lives (1.95 h) and low clearance (13.5 mL/min/kg)[2].

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

Animal Model:	Three-month-old male Sprague Dawley DIO rats (fed with a high-fat diet)[2]
Dosage:	0, 5, 25, 50 mg/kg; once daily for three weeks
Administration:	P.o. administration
Result:	Reduced cumulative body weight gain in a dose-dependent manner and was well tolerated in rats.

Animal Model:	Male Wistar rats[2]
Dosage:	50 mg/kg for p.o. and 5 mg/kg for i.v. (Pharmacokinetic Analysis)
Administration:	P.o. and i.v. administration
Result:	C_{max} (24 μM); $T_{1/2}$ (1.95 h).

REFERENCES

[1]. Yimin Q, et, al. Discovery of orally active carboxylic acid derivatives of 2-phenyl-5-trifluoromethyloxazole-4-carboxamide as potent diacylglycerol acyltransferase-1 inhibitors for the potential treatment of obesity and diabetes. J Med Chem. 2011 Apr 14; 54(7): 2433-46.

[2]. Weiya Y, et, al. Discovery and optimization of 2-phenyloxazole derivatives as diacylglycerol acyltransferase-1 inhibitors. Bioorg Med Chem Lett. 2011 Dec 1; 21(23): 7205-9.

[3]. Gómez-Outes A, et al. New parenteral anticoagulants in development. Ther Adv Cardiovasc Dis. 2011 Feb;5(1):33-59.

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