Pradigastat

Cat. No.: HY-16278

CAS No.: 956136-95-1

Molecular Formula: $C_{25}H_{24}F_3N_3O_2$

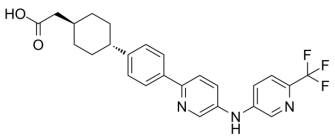
Molecular Weight: 455.47

Target: Acyltransferase

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (137.22 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1955 mL	10.9777 mL	21.9553 mL
5 mM	0.4391 mL	2.1955 mL	4.3911 mL
10 mM	0.2196 mL	1.0978 mL	2.1955 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 (5.49 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.5 mg/mL (5.49 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.49 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Pradigastat (LCQ-908) is a potent, selective and orally active diacylglycerol acyltransferase 1 (DGAT1) inhibitor. Pradigastat has anti-obesity and anti-diabetic effects[1][2].

IC_{50} & Target

Diacylglycerol acyltransferase 1 (DGAT1)[1]

In Vitro

Pradigastat inhibits breast cancer resistance protein (BCRP)-mediated efflux activity in a dose-dependent fashion in a BCRP

	over-expressing human ovarian cancer cell line with an IC_{50} value of 5 μM. Pradigastat inhibits OATP1B1, OATP1B3, and OAT3 activity in a concentration-dependent manner with estimated IC_{50} values of 1.66 μM, 3.34 μM, and 0.973 μM, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pradigastat (LCQ-908) inhibits the postprandial triglyceride levels in rats, dogs and monkeys. In rats whose lipoprotein lipase (LPL) activity has been abolished, Pradigastat reduces the postprandial accumulation of plasma triglyceride. Pradigastat decreases the postprandial rate of chylomicron triglyceride (CM-TG) secretion into the lymphatic duct and reduces the size of chylomicrons[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Neuron. 2020 Jan 22;105(2):276-292.e5.
- Curr Atheroscler Rep. 2022 Feb 2.

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REFERENCES

[1]. Meyers CD, et al. Effect of the DGAT1 inhibitor pradigastat on triglyceride and apoB48 levels in patients with familial chylomicronemia syndrome. Lipids Health Dis. 2015 Feb 18;14:8.

[2]. Kulmatycki K, et al. Evaluation of a potential transporter-mediated drug interaction between ZD 4522 and pradigastat, a novel DGAT-1 inhibitor. Int J Clin Pharmacol Ther. 2015 May;53(5):345-55.

[3]. Charles DanielMeyersMD, et al. The DGAT1 inhibitor pradigastat decreases chylomicron secretion and prevents postprandial triglyceride elevation in humans. Journal of Clinical Lipidology. Volume 7, Issue 3, May-June 2013, Page 285.

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