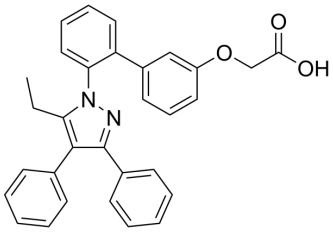
BMS-309403

Cat. No.:	HY-101903
CAS No.:	300657-03-8
Molecular Formula:	$C_{31}H_{26}N_2O_3$
Molecular Weight:	475
Target:	FABP
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 1 year -20°C 6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (210.53 mM; Need ultrasonic and warming)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1053 mL	10.5263 mL	21.0526 mL
	5 mM	0.4211 mL	2.1053 mL	4.2105 mL
	10 mM	0.2105 mL	1.0526 mL	2.1053 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: ≥ 2.08 mg/mL (4.38 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-309403 is a potent, orally active and selective adipocyte fatty acid binding protein (also known as FABP4, aP2) inhibitor with K_is of <2, 250, and 350 nM for FABP4, FABP3, and FABP5, respectively. BMS-309403 interacts with the fatty-acid-binding pocket within the interior of the protein and competitively inhibits the binding of endogenous fatty acids. BMS-309403 improves endothelial function in apolipoprotein E-deficient mice and in cultured human endothelial cells[1][2][3].

IC_{50} & Target

Ki: less than 2 nM (FABP4), 250 nM (FABP3), 350 nM (FABP5)[1]

In Vitro

Treatment with BMS-309403 significantly decreased MCP-1 production from THP-1 macrophages in a dose- and time-dependent manner[2].

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

In Vivo

BMS-309403 sodium (15 mg/kg; chronic treatment; daily for 6 weeks) improves endothelial function, phosphorylated and total eNOS and reduced plasma triglyceride levels but did not affect endothelium-independent relaxations[3].

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

Animal Model:	C57BL/6J mice (ApoE$^{-/-}$ mice)[3]
Dosage:	15 mg/kg
Administration:	Chronic treatment ; daily for 6 weeks
Result:	Significantly increased the phosphorylated eNOS (Ser1177) and total eNOS but not the phosphorylated to total eNOS ratio in aortae of 18 weeks old ApoE$^{-/-}$ mice.

CUSTOMER VALIDATION

- Bone Res. 2022 Jun 22;10(1):45.
- Cancer Lett. 2023 Sep 21;216403.
- Int J Biol Sci. 2021 Oct 11;17(15):4207-4222.
- Cell Death Dis. 2019 May 16;10(6):382.
- Free Radic Biol Med. 2022 Jul 15;S0891-5849(22)00474-9.

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REFERENCES

[1]. Sulsky R, et al. Potent and selective biphenyl azole inhibitors of adipocyte fatty acid binding protein (aFABP). Bioorg Med Chem Lett. 2007 Jun 15;17(12):3511-5.

[2]. Lin W, et al. BMS309403 stimulates glucose uptake in myotubes through activation of AMP-activated protein kinase. PLoS One. 2012;7(8):e44570.

[3]. Lee MY, et al. Chronic administration of BMS309403 improves endothelial function in apolipoprotein E-deficient mice and in cultured human endothelial cells. Br J Pharmacol. 2011 Apr;162(7):1564-76.

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