BMS-309403 sodium

Cat. No.: HY-101903A

CAS No.: 2802523-05-1

Molecular Formula: $C_{31}H_{25}N_2NaO_3$

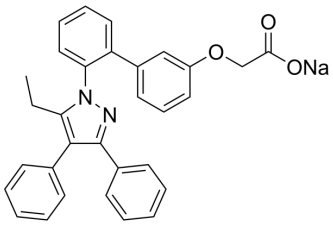
Molecular Weight: 496.53

Target: FABP

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (201.40 mM; Need ultrasonic)

DMSO : 50 mg/mL (100.70 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0140 mL	10.0699 mL	20.1398 mL
5 mM	0.4028 mL	2.0140 mL	4.0280 mL
10 mM	0.2014 mL	1.0070 mL	2.0140 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 (5.03 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.03 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

BMS-309403 sodium 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 sodium interacts with the fatty-acid-binding pocket within the interior of the protein and competitively inhibits the binding of endogenous fatty acids. BMS-309403 sodium improves endothelial function in apolipoprotein E-deficient mice and in cultured human endothelial cells[1][2][3].

In Vitro

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

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.
- 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.
- Antioxidants (Basel). 2023, 12(1), 74.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Sulsky R, et al. Potent and selective biphenyl azole inhibitors of adipocyte fatty acid binding protein (aFABP). Bioorg Med Chem Lett. 2007;17(12):3511-3515.

[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;162(7):1564-1576.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA