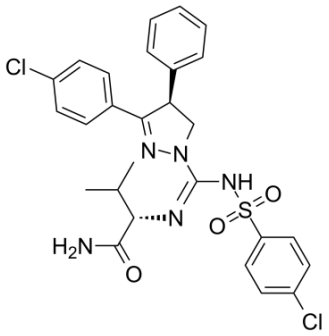
JD-5037

Cat. No.:	HY-18697
CAS No.:	1392116-14-1
Molecular Formula:	$C_{27}H_{27}Cl_2N_5O_3S$
Molecular Weight:	572.51
Target:	Cannabinoid Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
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 : ≥ 100 mg/mL (174.67 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.7467 mL	8.7335 mL	17.4669 mL
5 mM	0.3493 mL	1.7467 mL	3.4934 mL
10 mM	0.1747 mL	0.8733 mL	1.7467 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: ≥ 2.75 mg/mL (4.80 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (4.80 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

JD-5037 is a potent CB_1R antagonist with an IC_{50} of 1.5 nM.

IC_{50} & Target

CB1

1.5 nM (IC_{50})

In Vivo

JD5037 (3 mg/kg/d, i.p.) induces equal reductions in body weight, attenuates the HFD-induced hyperglycemia, and reduces the HFD-induced hepatic injury and steatosis in obese Magel2-null mice[2]. JD5037 (3 mg/kg/day, p.o.) significantly reduces the size of tumors and abrogates the tumor in DEN-treated mice. JD5037 attenuates the AEA levels in HCC samples from mice[3].

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

PROTOCOL

Animal Administration [2]

Mice: JD-5037 is formulated in vehicle (V; 1% Tween80, 4% DMSO, 95% Saline). Obese mice are treated chronically (28 d) with vehicle (V; 1% Tween80, 4% DMSO, 95% Saline), JD5037, or SLV319 at a dose of 3 mg/kg, i.p. Body weight and food intake are monitored daily. Mice are euthanized by cervical dislocation under anesthesia; the brain, hypothalamus, liver, and combined fat pads are removed, weighed, and snap-frozen, and trunk blood is collected for determining the endocrine and biochemical parameters[2].

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

CUSTOMER VALIDATION

- J Am Soc Nephrol. 2017 Dec;28(12):3518-3532.
- Br J Pharmacol. 2020 Jan;177(1):110-127.
- Diabetes. 2020 Oct;69(10):2120-2132.
- Elife. 2020 Nov 19;9:e60771.

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REFERENCES

[1]. Chorvat RJ. Peripherally restricted CB1 receptor blockers. Bioorg Med Chem Lett. 2013 Sep 1;23(17):4751-60.

[2]. Knani I, et al. Targeting the endocannabinoid/CB1 receptor system for treating obesity in Prader-Willi syndrome. Mol Metab. 2016 Oct 22;5(12):1187-1199.

[3]. Mukhopadhyay B, et al. Cannabinoid receptor 1 promotes hepatocellular carcinoma initiation and progression through multiple mechanisms. Hepatology. 2015 May;61(5):1615-26.

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