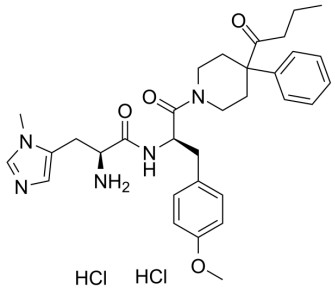
BMS-470539 dihydrochloride

Cat. No.:	HY-115644
CAS No.:	2341796-82-3
Molecular Formula:	$C_{32}H_{43}Cl_2N_5O_4$
Molecular Weight:	632.62
Target:	Melanocortin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	-20°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

DMSO : ≥ 125 mg/mL (197.59 mM)

H_2O : ≥ 100 mg/mL (158.07 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.5807 mL	7.9036 mL	15.8073 mL
5 mM	0.3161 mL	1.5807 mL	3.1615 mL
10 mM	0.1581 mL	0.7904 mL	1.5807 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

BMS-470539 dihydrochloride is a highly potent and selective melanocortin-1 receptor (MC-1R) agonist with an IC_{50} of 120 nM, an EC_{50} of 28 nM. BMS-470539 dihydrochloride does not activate MC-3R and is a very weak partial agonist at MC-4R and MC-5R. BMS-470539 dihydrochloride has potently anti-inflammatory properties[1][2][3].

IC_{50} & Target

IC50: 120 nM (Melanocortin-1 receptor); EC50: 28 nM (Melanocortin-1 receptor)[1]

In Vitro

An HBL melanoma cell line is established that stably expresses a NF-κB luciferase reporter. In these cells, 0.5 ng/mL TNF-α induces a dose-dependent increase in NF-κB luciferase activity. Treatment of HBL-NF-κB cells with BMS-470539 elicits a dose-dependent, statistically significant reduction in TNF-α-stimulated NF-κB luciferase activity. BMS-470539 has no effect on luciferase reporter activity in the absence of TNF-α stimulation. In nontransfected HBL cells, treatment with BMS-470539 results in a dose-dependent inhibition of NF-B nuclear translocation[2].

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

In Vivo

BMS-470539 (2.05-18.47 mg/kg; intravenous injection; for 125 minutes; WT and MC1 receptor recessive e/e mice) treatment inhibits cell adhesion and emigration with no effect on cell rolling. And also inhibits the tissue expression of both CXCL1 and

CCL2[3].

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

Animal Model:	Wild-type (WT) and MC1 receptor recessive e/e mice induced with ischaemia-reperfusion[3]
Dosage:	2.05 mg/kg, 6.16 mg/kg and 18.47 mg/kg
Administration:	Intravenous injection; for 125 minutes
Result:	Inhibited cell adhesion and emigration with no effect on cell rolling. Inhibited tissue expression of both CXCL1 and CCL2.

REFERENCES

[1]. Herpin TF, et al. Discovery of tyrosine-based potent and selective melanocortin-1 receptor small-molecule agonists with anti-inflammatory properties. J Med Chem. 2003 Mar 27;46(7):1123-6.

[2]. Kang L, et al. A selective small molecule agonist of the melanocortin-1 receptor inhibits lipopolysaccharide-induced cytokine accumulation and leukocyte infiltration in mice. J Leukoc Biol. 2006 Oct;80(4):897-904. Epub 2006 Aug 3.

[3]. Leoni G, et al. The melanocortin MC(1) receptor agonist BMS-470539 inhibits leucocyte trafficking in the inflamed vasculature. Br J Pharmacol. 2010 May;160(1):171-80.

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