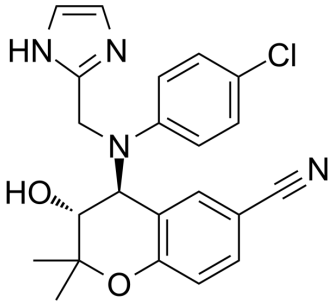
BMS-191095

Cat. No.:	HY-14256
CAS No.:	166095-21-2
Molecular Formula:	$C_{22}H_{21}ClN_4O_2$
Molecular Weight:	408.88
Target:	Potassium Channel
Pathway:	Membrane Transporter/Ion Channel
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (244.57 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4457 mL	12.2285 mL	24.4571 mL
	5 mM	0.4891 mL	2.4457 mL	4.8914 mL
	10 mM	0.2446 mL	1.2229 mL	2.4457 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 (6.11 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.11 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BMS-191095 is a selective activator of mitochondrial ATP-sensitive potassium (mitoKATP) channels. BMS-191095 inhibits human platelet aggregation by opening mitochondrial K(ATP) channels[1][2][3].

In Vitro

BMS-191095 (50 μmol/L) induces mitochondrial depolarization of vascular smooth muscle (VSM) cells from SD rats[1].
BMS-191095 (10-100 μmol/L) dose-dependently induces vasodilation in endothelium denuded cerebral arteries[1].
BMS-191095 (50 μmol/L) increases the frequency of calcium sparks in VSM cells[1].
BMS-191095 (0-1500 μM) inhibits human platelet aggregation induced by collagen and thrombin with IC_{50} values of 63.9 and

	104.8 μM, respectively[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	BMS-191095 (2.5 or 25 μg; intraventricular infusion, 30 min/60 min/24 hours before the induction of ischemia, once) reduces neuronal damage in rats with transient focal cerebral ischemia[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Wistar rats with the induction of ischemia induced by middle cerebral artery occlusion (MCAO)[3]
Dosage:	2.5 or 25 μg
Administration:	Intraventricular infusion; 30 min/60 min/24 hours before the induction of ischemia, once
Result:	Reduced total infarct volume in rats with of pretreat dose of 25 mg and 24 h before MCA. Induced a rapid mitochondrial depolarization.

CUSTOMER VALIDATION

- Pharmaceuticals. 2023, 16(2), 225.

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REFERENCES

[1]. Cho MR, et al. BMS-191095, a cardioselective mitochondrial K(ATP) opener, inhibits human platelet aggregation by opening mitochondrial K(ATP) channels. Arch Pharm Res. 2005 Jan;28(1):61-7.

[2]. Mayanagi K, et al. The mitochondrial K(ATP) channel opener BMS-191095 reduces neuronal damage after transient focal cerebral ischemia in rats. J Cereb Blood Flow Metab. 2007 Feb;27(2):348-55.

[3]. Katakam PV, et al. Diversity of mitochondria-dependent dilator mechanisms in vascular smooth muscle of cerebral arteries from normal and insulin-resistant rats. Am J Physiol Heart Circ Physiol. 2014 Aug 15;307(4):H493-503.

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