Senazodan

Cat. No.: HY-101693

CAS No.: 98326-32-0

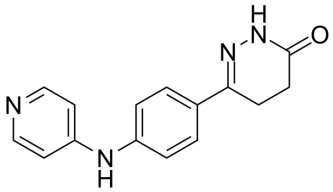
Molecular Formula: $C_{15}H_{14}N_4O$

Molecular Weight: 266.3

Target: Phosphodiesterase (PDE)

Pathway: Metabolic Enzyme/Protease

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (93.88 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7552 mL	18.7758 mL	37.5516 mL
	5 mM	0.7510 mL	3.7552 mL	7.5103 mL
	10 mM	0.3755 mL	1.8776 mL	3.7552 mL

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

BIOLOGICAL ACTIVITY

Description

Senazodan (MCI 154) is a Ca^{2+} sensitiser, and also shows inhibition effect on PDE III[1][2].

IC_{50} & Target

PDE III[1]

In Vitro

Senazodan seems to affect directly the actin-myosin crossbridge kinetics, and increase myosin ATPase activity[1]. Senazodan produces a concentration-dependent increase in tension development. Senazodan enhances Ca^{2+} binding to myofilaments and to purified cardiac troponin C. Senazodan also enhances contractility in guinea-pig papillary muscles by inhibiting PDE III[2]. Senazodan (0.1 nM~0.1 mM) shows that the contractile response of superior mesenteric arterie (SMA) to norepinephrine (NE) after hemorrhagic shock is significantly decreased as compared with the normal control group. Senazodan (0.01 mM) pretreatment prevents the effects of Ang II, and the concentration-response curve of Ca^{2+} is shifted to the right as compared with Ang II-alone group[3].

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

In Vivo

Senazodan (0.1~2.0 mg/kg; left femoral vein catheterization infusion) decreases the pressor effect of norepinephrine (NE)[3]. Senazodan (0.1 mg/kg; i.v.) makes LVSP, IP, MC, and Lo all increased significantly, while heart rate is not obviously changed and left ventricular end-diastolic pressure (LVEDP) is reduced remarkably[4].

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

Animal Model:	Wistar rats (200~250 g)[3]
Dosage:	0.1~2.0 mg/kg
Administration:	Left femoral vein catheterization infusion
Result:	Decreased the pressor effect of norepinephrine (NE).

Animal Model:	Rabbits[4]
Dosage:	0.1 mg/kg
Administration:	I.v.
Result:	LVSP, IP, MC, and Lo all were increased significantly while heart rate was not obviously changed and left ventricular end-diastolic pressure (LVEDP) was reduced remarkably.

REFERENCES

[1]. Lehtonen LA, et al. Pharmacokinetics and pharmacodynamics of intravenous inotropic agents. Clin Pharmacokinet. 2004;43(3):187-203.

[2]. Erhardt L. An emerging role for calcium sensitisation in the treatment of heart failure. Expert Opin Investig Drugs. 2005 Jun;14(6):659-70.

[3]. Yang G, et al. Effects of MCI-154 on vascular reactivity and its mechanisms after hemorrhagic shock in rats. J Cardiovasc Pharmacol. 2006;47(6):751-757.

[4]. Ming MJ, et al. Effects of MCI-154, a calcium sensitizer, on cardiac dysfunction in endotoxic shock in rabbits. Shock. 2000;13(6):459-463.

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