Senazodan hydrochloride

Cat. No.: HY-101693A

CAS No.: 98326-33-1

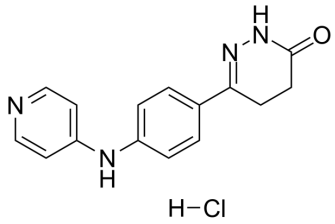
Molecular Formula: $C_{15}H_{15}ClN_4O$

Molecular Weight: 302.76

Target: Phosphodiesterase (PDE)

Pathway: Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 67.5 mg/mL (222.95 mM; Need ultrasonic)
H_2O : 25 mg/mL (82.57 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.3029 mL	16.5147 mL	33.0295 mL
5 mM	0.6606 mL	3.3029 mL	6.6059 mL
10 mM	0.3303 mL	1.6515 mL	3.3029 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 (8.26 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.26 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

PDE III[1]

In Vitro

Senazodan (hydrochloride) seems to affect directly the actin-myosin crossbridge kinetics, and increases myosin ATPase activity[1]. Senazodan (hydrochloride) produces a concentration-dependent increase in tension development. Senazodan (hydrochloride) enhances Ca^{2+} binding to myofilaments and to purified cardiac troponin C. Senazodan (hydrochloride) also enhances contractility in guinea-pig papillary muscles by inhibiting PDE III[2].Senazodan (0.1 nM~0.1 mM) (hydrochloride) 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) (hydrochloride) 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) (hydrochloride) decreases the pressor effect of norepinephrine (NE)[3].

Senazodan (0.1 mg/kg; i.v.) (hydrochloride) 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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