Yonkenafil hydrochloride

Cat. No.: HY-125095

CAS No.: 804519-64-0

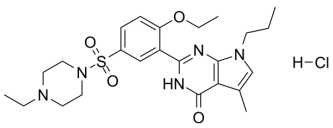
Molecular Formula: $C_{24}H_{34}ClN_5O_4S$

Molecular Weight: 524.08

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 : 100 mg/mL (190.81 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.9081 mL	9.5405 mL	19.0811 mL
	5 mM	0.3816 mL	1.9081 mL	3.8162 mL
	10 mM	0.1908 mL	0.9541 mL	1.9081 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.77 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.77 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Yonkenafil (Tunodafil) hydrochloride, a novel phosphodiesterase 5 (PDE5) inhibitor, is effective in reducing cerebral infarction, neurological deficits, edema, and neuronal damage in the infarcted area. Yonkenafil (Tunodafil) hydrochloride may improve cognitive function by modulating neurogenesis and has a potential therapeutic effect on Alzheimer's disease[1].

IC_{50} & Target

PDE5

In Vivo

Yonkenafil (Tunodafil) hydrochloride (4-32 mg/kg, i.v. daily for 7 days) improves behavioral outcomes after stroke and reduces cerebral infarct volume, inhibits neuronal apoptosis, and significantly enhances synaptic function in ischemic brain by modulating the expression of BDNF/TrkB and NGF/TrkA[1].

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

Animal Model:	Male Sprague-Dawley (SD) Rat[1]
Dosage:	4, 8, 16 and 32 mg/kg
Administration:	i.v. daily for 7 days
Result:	Induced a dose-dependent decrease in infarct volume, with an ED_{50} of 12.27 mg/kg. Increased hsp70 expression, decreased apaf-1 expression, and inhibited caspase-3 and caspase-9 cleavage. Significantly prevented neuronal damage and increases the number of surviving neurons after stroke. Prevented decrease in synaptophysin levels and increase in PSD-95 and nNOS levels.

REFERENCES

[1]. Xuemei Chen, et al. Yonkenafil: a novel phosphodiesterase type 5 inhibitor induces neuronal network potentiation by a cGMP-dependent Nogo-R axis in acute experimental stroke. Exp Neurol. 2014 Nov;261:267-77.

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