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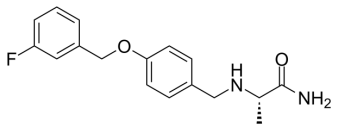
Safinamide

Cat. No.:	HY-70057
CAS No.:	133865-89-1
Molecular Formula:	$C_{17}H_{19}FN_2O_2$
Molecular Weight:	302.34
Target:	Monoamine Oxidase
Pathway:	Neuronal Signaling
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 (330.75 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Concentration / Solvent / Mass	1 mg	5 mg	10 mg
	1 mM	3.3075 mL	16.5377 mL	33.0753 mL
	5 mM	0.6615 mL	3.3075 mL	6.6151 mL
	10 mM	0.3308 mL	1.6538 mL	3.3075 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 (8.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.27 mM); Suspended solution

BIOLOGICAL ACTIVITY

Description

Safinamide is a potent, selective, and reversible monoamine oxidase B (MAO-B) inhibitor (IC_{50}=0.098 μM) over MAO-A (IC_{50} =580 μM)[1]. Safinamide also blocks sodium channels and modulates glutamate (Glu) release, showing a greater affinity at depolarized (IC_{50}=8 μM) than at resting (IC_{50}=262 μM) potentials. Safinamide has neuroprotective and neurorescuing effects and can be used for the study of parkinson disease, ischemia stroke etc.al[2][3].

IC_{50} & Target	MAO-B	MAO-A
	98 nM (IC_{50})	580 μM (IC_{50})

In Vitro

Safinamide (1–300 μM) reduces the amplitude of the peak sodium currents in a concentration-dependent manner. When

currents are stimulated to a V_{test} of +10 mV from a V_h of -110 mV, the IC_{50} value was 262 μM. When the holding potential is depolarized to -53 mV, the inhibitory effect of safinamide with a lower IC_{50} value (8 μM) in rat cortical neurons[1].

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

In Vivo

Safinamide (intraperitoneal injection; 90 mg/kg; once daily; 14 days) treatment prior to MCAO significantly ameliorates MCAO-caused cerebral infarction volume, neurological deficit, disruption of the brain-blood barrier (BBB), and impairs expression of tight junction protein occludin and ZO-1 in mice[3].

Safinamide (intraperitoneal injection; 5 mg/kg, 15 mg/kg and 30 mg/kg) dose dependently inhibits the veratridine-induced GABA release and Glu release in vivo. At the dose 30 mg/kg, Safinamide prevents the effect of veratridine both on Glu (treatment F1,8=1.31; time×treatment interaction F8,64=2.4) and GABA (treatment F1,8=4.04; time F8,64=3.76, time×treatment interaction F8,64 = 2.83) release.

Safinamide causes a slight, albeit not significant, reduction of veratridine-stimulated Glu release at 0.5 mg/kg and full inhibition at 5 and 15 mg/kg in rat[3].

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

Animal Model:	Focal cerebral ischemia C57/BL6 male mouse Model[3]
Dosage:	90 mg/kg
Administration:	Intraperitoneal injection; once daily; 14 days
Result:	Significantly decreased infarction volume in brain areas.

CUSTOMER VALIDATION

- Ecotoxicol Environ Saf. 2023 Aug 7;262:115284.
- Behav Brain Res. 2023 Nov 30:114787.

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REFERENCES

[1]. Leonetti F, et al. Solid-phase synthesis and insights into structure-activity relationships of safinamide analogues as potent and selective inhibitors of type B monoamine oxidase. J Med Chem, 2007, 50(20), 4909-4916.

[2]. C Caccia, et al.Safinamide: from molecular targets to a new anti-Parkinson drug. Neurology. 2006 Oct 10;67(7 Suppl 2):S18-23.

[3]. Michele Morari, et al. Safinamide Differentially Modulates In Vivo Glutamate and GABA Release in the Rat Hippocampus and Basal Ganglia.J Pharmacol Exp Ther. 2018 Feb;364(2):198-206.

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