Safinamide mesylate

Cat. No.: HY-70057A

CAS No.: 202825-46-5

Molecular Formula: $C_{18}H_{23}FN_2O_5S$

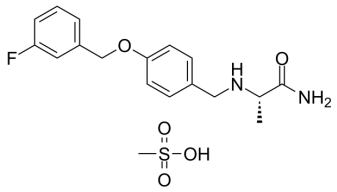
Molecular Weight: 398.45

Target: Monoamine Oxidase

Pathway: Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (627.43 mM; Need ultrasonic)

H_2O : ≥ 20 mg/mL (50.19 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5097 mL	12.5486 mL	25.0972 mL
5 mM	0.5019 mL	2.5097 mL	5.0195 mL
10 mM	0.2510 mL	1.2549 mL	2.5097 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: ≥ 2.08 mg/mL (5.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (5.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Safinamide (FCE 26743; EMD 1195686) mesylate is a potent, selective, and reversible monoamine oxidase B (MAO-B) inhibitor (IC_{50}=0.098 μM) over MAO-A (IC_{50}=580 nM)[1]. Safinamide mesylate 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 mesylate has neuroprotective and neurorescuing effects and can be used for the study of parkinson disease, ischemia stroke et.al[2][3].

IC_{50} & Target	MAO-B	MAO-A

	98 nM (IC_{50})	580 nM (IC_{50})
In Vitro	Safinamide mesylate (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 mesylate 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 mesylate (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 mesylate (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 mesylate 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 mesylate 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.	

CUSTOMER VALIDATION

- Ecotoxicol Environ Saf. 2023 Aug 7;262:115284.

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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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