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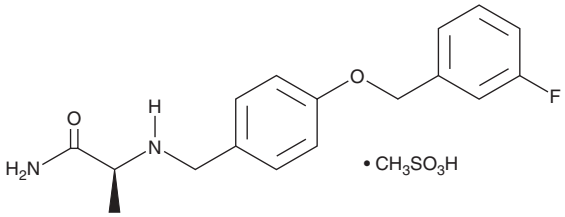
PRODUCT INFORMATION

Safinamide (mesylate)

Item No. 21546

CAS Registry No.: 202825-46-5
Formal Name: 2S-[[[4-[(3-fluorophenyl)methoxy] phenyl]methyl]amino]-propanamide, monomethanesulfonate
Synonyms: EMD 1195686, FCE 26743, NW-1015, PNU-151774E
MF: $C_{17}H_{19}FN_2O_2 \bullet CH_3SO_3H$
FW: 398.4
Purity: ≥98%
UV/Vis.: λ_{max}: 228, 263, 269 nm
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Safinamide (mesylate) is supplied as a crystalline solid. A stock solution may be made by dissolving the safinamide (mesylate) in the solvent of choice. Safinamide (mesylate) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of safinamide (mesylate) in ethanol is approximately 5 mg/ml and approximately 30 mg/ml in DMSO and DMF.

Safinamide (mesylate) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, safinamide (mesylate) should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Safinamide (mesylate) has a solubility of approximately 0.1 mg/ml in a 1:9 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Safinamide is an inhibitor of monoamine oxidase B (MAO-B; IC_{50} = ~0.1 µM).[1] It is selective for MAO-B over MAO-A (IC_{50} = >10 µM). It also inhibits radioligand binding to sodium channel binding site 2, sigma-1, and sigma-2 receptors in rat brain membranes (IC_{50}s = 8.2, 0.019, and 1.59 µM, respectively).[2] Safinamide inhibits high voltage-activated calcium currents and depolarization-induced tetrodotoxin-sensitive fast sodium currents in rat hippocampal neurons in a concentration-dependent manner. It inhibits veratrine-induced glutamate release in rat hippocampal slices (IC_{50} = 56 µM). Safinamide inhibits maximal electroshock-induced tonic extension seizures in mice and rats (ED_{50}s = 8 and 11.8 mg/kg, p.o.) as well as maximal seizures induced by bicuculline (Item No. 11727), picrotoxin (Item No. 20771), 3-mercaptopropionic acid, and strychnine in mice (ED_{50}s = 26.9, 60.6, 21.5, and 104.1 mg/kg, p.o., respectively).[3] Formulations containing safinamide have been used as adjunctive treatments to levodopa and carbidopa in the treatment of "off" episodes associated with Parkinson's disease.

References

1. Strolin Benedetti, M.S., Marrari, P., Colombo, M., *et al. J. Pharm. Pharmacol.* **46(10)**, 814-819 (1994).
2. Salvati, P., Maj, R., Caccia, C., *et al. J. Pharmacol. Exp. Ther.* **288(3)**, 1151-1159 (1999).
3. Fariello, R.G., McArthur, R.A., Bonsignori, A., *et al. J. Pharmacol. Exp. Ther.* **285(2)**, 397-403 (1998).

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