

Amoxapine

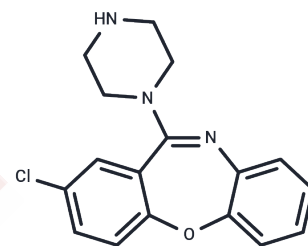
Chemical Properties

CAS No. : 14028-44-5

Formula: C₁₇H₁₆ClN₃O

Molecular Weight: 313.78

Storage: Store at low temperature, Keep away from direct sunlight, Keep away from moisture
 Powder: -20°C for 3 years | In solvent: -80°C for 1 year
 Actual storage temperature shall be subject to the COA.



Biological Description

Description	Amoxapine (CL-67772) exerts its antidepressant effect by inhibiting the re-uptake of norepinephrine and, to a lesser degree, of serotonin, at adrenergic nerve endings and blocks the response of dopamine receptors to dopamine. Amoxapine is a tricyclic antidepressant of the dibenzoxazepine class. This drug is used to treat symptoms of depression and may cause tardive dyskinesia. Amoxapine also binds to alpha-adrenergic, histaminergic, and cholinergic receptors which accounts for many of the side effects seen with this agent.
Targets(IC50)	Others, Antibacterial, Autophagy, GlyT
In vitro	Amoxapine, administered intraperitoneally (i.p.) at doses of 1, 5, and 10 mg/kg, notably at lower doses, was found to decrease paradoxical sleep and increase slow-wave sleep. Throughout the treatment period, a consistent reduction in paradoxical sleep was observed with Amoxapine (10 mg/kg, i.p.), although tolerance to the inhibition effect of cericlamine was noted in this sleep phase. Additionally, Amoxapine (10 mg/kg/day) did not affect the levels of Substance P, dynorphin, and cholecystokinin, but significantly increased leucine-enkephalin levels in the rat cortex, spinal cord, and hypothalamus. Despite not altering opioid receptor binding in the rat cortex, Amoxapine (10 mg/kg/day, i.p.) increased the density of δ - and μ -opioid receptor binding sites in the spinal cord and decreased it in the hypothalamus. Moreover, Amoxapine attenuated spontaneous activity, induced catalepsy and ptosis, and exhibited inhibitory effects on the dysknetic movements induced by Apomorphine and stereotypy behaviors induced by amphetamine, through altering avoidance behaviors discerned by monkeys.
In vivo	In both oocytes and HEK 293 cells, Amoxapine induces acute hERG channel blockade with IC ₅₀ values of 21.6 and 5.1 μ M, respectively. In human embryonic kidney 293 cells, it selectively inhibits GLYT2a over its isotype GLYT1b by a factor of 10. Amoxapine leads to a blockade of reverse frequency dependence and causes a leftward shift with accelerated inactivation. Treatment with Amoxapine results in a gradual reduction of hERG transport to the cell membrane surface in HEK 293 cells, with an IC ₅₀ of 15.3 μ M.

Solubility Information

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Solubility	DMSO: 8.06 mg/mL (25.69 mM), Sonication is recommended. H2O: < 1 mg/mL (insoluble or slightly soluble), Ethanol: < 1 mg/mL (insoluble or slightly soluble), (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 1 mg/mL (3.19 mM), Sonication is recommended. <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.1869 mL	15.9347 mL	31.8695 mL
5 mM	0.6374 mL	3.1869 mL	6.3739 mL
10 mM	0.3187 mL	1.5935 mL	3.1869 mL
50 mM	0.0637 mL	0.3187 mL	0.6374 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

- Núñez E, et al. Br J Pharmacol. 2000 Jan;129(1):200-6.
Obers S, et al. Naunyn Schmiedebergs Arch Pharmacol, 2010, 381(5), 385-400.
Hamon M, et al. Neuropharmacology, 1987, 26(6), 531-539.
Maudhuit C, et al. Neuropharmacology, 1994, 33(8), 12017-1025.
Greenblatt EN, et al. Arch Int Pharmacodyn Ther, 1978, 233(1), 107-135.

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