Doxepin Hydrochloride

Cat. No.: HY-B0725

CAS No.: 1229-29-4

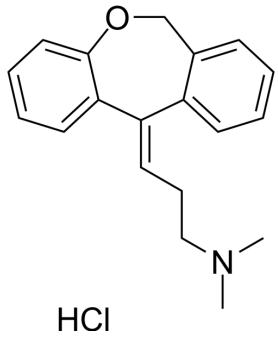
Molecular Formula: $C_{19}H_{22}ClNO$

Molecular Weight: 315.84

Target: Histamine Receptor; Cytochrome P450

Pathway: GPCR/G Protein; Immunology/Inflammation; Neuronal Signaling; Metabolic Enzyme/Protease

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 : ≥ 100 mg/mL (316.62 mM)
H_2O : ≥ 50 mg/mL (158.31 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1662 mL	15.8308 mL	31.6616 mL
5 mM	0.6332 mL	3.1662 mL	6.3323 mL
10 mM	0.3166 mL	1.5831 mL	3.1662 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 140 mg/mL (443.26 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.92 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.92 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.92 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Doxepin hydrochloride is an orally active tricyclic antidepressant agent. Doxepin hydrochloride is a potent and selective histamine receptor H1 antagonist. Doxepin hydrochloride is also a potent CYP450 inhibitor and significantly inhibits CYP450 2C19 and 1A2[1][2]. Doxepin inhibits reuptake of serotonin and norepinephrine as a tricyclic antidepressant[3].
. Doxepin has therapeutic effects in atopic dermatitis，chronic urticaria，can improve cognitive processes， protect central

	nervous system[4]. . Doxepin has also been proposed as a protective factor against oxidative stress[5]. .
IC_{50} & Target	H_1 Receptor
In Vitro	The protective effect of doxepin is associated with the enhancement of PSD-95 and synapsin 1 expression via PI3K/AKT/mTOR signaling pathway[6]. . MCE has not independently confirmed the accuracy of these methods. They are for reference only. Western Blot Analysis

Cell Line:	SH-SY5Y human neuroblastoma cell line
Concentration:	10 ng/ml
Incubation Time:	2 h
Result:	Improved the protein expre-ssion levels of PSD-95, synapsin 1 and p-AKT in SH-SY5Y cells, and decreased the protein expression level of p-mTOR in SH-SY5Y cells.

In Vivo

Doxepin (intraperitoneal injection of 1 mg/kg and 5 mg/kg doxepin once a day for 21 days) can protect against the Aβ1-42-induced memory impairment in rats[6].

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

Animal Model:	SD male rats[6].
Dosage:	1, 5mg/kg
Administration:	Doxepin (intraperitoneal injection of 1 mg/kg and 5 mg/kg doxepin once a day for 21 days)
Result:	Improved the protein expression levels of PSD-95 and synapsin 1 in hippocampus and tem-poral lobe, and decreased the protein expression level of p-AKT in hippocampus and temporal lobe after treatment of 1 mg/kg of doxepin.

CUSTOMER VALIDATION

- Nat Commun. 2022 Nov 10;13(1):6796.
- Cell Commun Signal. 2023 May 25;21(1):123.
- Virus Res. 2022 Aug;317:198816.
- J Appl Toxicol. 2023 Apr 14.

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REFERENCES

[1]. AnnemiekVermeeren, etal. Effects of the use of hypnotics on cognition. Progress in brain research vol. 190 (2011): 89-103.

[2]. G Hajak, etal. Doxepin in the treatment of primary insomnia: a placebo-controlled, double-blind, polysomnographic study. The Journal of clinical psychiatry vol. 62,6

(2001): 453-63.

[3]. Mahsa Gharzi, etal. Effects of different doses of doxepin on passive avoidance learning in rats. Advanced biomedical research vol. 2 66. 30 Jul. 2013.

[4]. Jimei Bu, etal. Mechanism underlying the effects of doxepin on β-amyloid -induced memory impairment in rats. Iran J Basic Med Sci. 2017 Sep;20(9):1044-1049.

[5]. http://pdsp.med.unc.edu/pdsp.php

[6]. Hajak, G., et al., Doxepin in the treatment of primary insomnia: a placebo-controlled, double-blind, polysomnographic study. J Clin Psychiatry, 2001. 62(6): p. 453-63.

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