Clomipramine

Cat. No.: HY-B0457A

CAS No.: 303-49-1

Molecular Formula: $C_{19}H_{23}ClN_2$

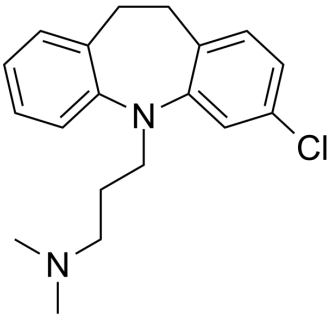
Molecular Weight: 314.85

Target: Serotonin Transporter

Pathway: Neuronal Signaling

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (317.61 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1761 mL	15.8806 mL	31.7612 mL
	5 mM	0.6352 mL	3.1761 mL	6.3522 mL
	10 mM	0.3176 mL	1.5881 mL	3.1761 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 (7.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Clomipramine (Chlorimipramine) is a potent 5-HT reuptake blocker with the IC_{50} value of 1.5 nM. Clomipramine is a tricyclic antidepressant that can be used for the research of depression and obsessive compulsive disorder (OCD)[1].

In Vitro

Clomipramine can inhibit reuptake of both noradrenaline and 5-HT, although Clomipramine inhibits 5-HT reuptake more strongly than it inhibits noradrenaline reuptake[1].

The antidepressant Clomipramine inhibits both venom AChE as well as human serum BChE in a concentration-dependent manner but has no effect on AChE in the rat brain striatum[2]. Clomipramine interferes with the autophagic flux and severely compromises the viability of tumorigenic cells upon cytotoxic stress[3].

Clomipramine reduces autophagy in neuronal primary cultures. Clomipramine (1 and 5 μM) negatively regulates neuronal autophagic pathway in primary cultured cells[3].

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

Western Blot Analysis[3]

Cell Line:	Primary cortical neurons
Concentration:	1 and 5 μM
Incubation Time:	12, 24 and 48 hours
Result:	Enhanced the LC3-I conversion to LC3-II in a concentration-dependent manner at all analyzed time points.

In Vivo

Clomipramine (5-20 mg/kg; i.p) elicits significant hyperglycemia in mice. Clomipramine induces hyperglycemia in mice by blocking the 5-HT2B and/or 5-HT2C receptors, which results in facilitation of adrenaline release. In mice, Clomipramine reduces immobility in the forced swimming test, which is the behavioral model for antidepressants. Clomipramine also inhibits the OCD animal model, marble burying behavior in mice[1]. Clomipramine (20 mg/kg) decreases autophagic flux in murine tissues[3].

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

Animal Model:	C57BL/6 J mice (6 weeks of age and 22 to 25 g)[3]
Dosage:	20 mg/kg
Administration:	Treated intraperitoneally for 21 days
Result:	Both LC3-II and p62 were significantly increased in the liver of Clomipramine treated mice compared to vehicle treated ones.

CUSTOMER VALIDATION

- Cell Commun Signal. 2023 May 25;21(1):123.

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REFERENCES

[1]. Yumi Sugimoto, et al. The tricyclic antidepressant Clomipramine increases plasma glucose levels of mice. J Pharmacol Sci. 2003 Sep;93(1):74-9.

[2]. Mushtaq Ahmed, et al. Comparative study of the inhibitory effect of antidepressants on cholinesterase activity in Bungarus sindanus (krait) venom, human serum and rat striatum. J Enzyme Inhib Med Chem. 2008 Dec;23(6):912-7.

[3]. Federica Cavaliere,The tricyclic antidepressant Clomipramine inhibits neuronal autophagic flux. Sci Rep. 2019 Mar 19;9(1):4881.

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