Imipramine hydrochloride

Cat. No.: HY-B1490

CAS No.: 113-52-0

Molecular Formula: $C_{19}H_{25}ClN_2$

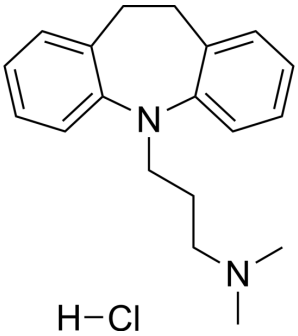
Molecular Weight: 316.87

Target: Serotonin Transporter; Apoptosis; Autophagy

Pathway: Neuronal Signaling; Apoptosis; Autophagy

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

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (315.59 mM)

H_2O : 62.5 mg/mL (197.24 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1559 mL	15.7793 mL	31.5587 mL
5 mM	0.6312 mL	3.1559 mL	6.3117 mL
10 mM	0.3156 mL	1.5779 mL	3.1559 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 60 mg/mL (189.35 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 (7.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.89 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.89 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Imipramine hydrochloride is an orally active tertiary amine tricyclic antidepressant. Imipramine hydrochloride is a Fascin1 inhibitor with antitumor activities. Imipramine hydrochloride also inhibits serotonin transporter with an IC_{50} value of 32 nM. Imipramine hydrochloride stimulates U-87MG glioma cells autophagy and induces HL-60 cell apoptosis. Imipramine hydrochloride shows neuroprotective and immunomodulatory effects[1][2][3][4][5].

IC_{50} & Target	Fascin1, Serotonin Autophagy, Apoptosis[1][2][3][5] IC_{50}: 32 nM (human placental serotonin transporter)[5]

In Vitro

Imipramine (0.5-300 μM, 3 days) inhibits HCT-116 cell viability[1].
?Imipramine (20 μM) inhibits cell migration (7 h) and invasion (48 h)[1].
?Imipramine (50 μM, 0-240 min) inhibites the PI3K/Akt/mTOR signaling pathway in U-87MG glioma cells[2].
?Imipramine (60 μM, 24 h) stimulates U-87MG glioma cells autophagy[2].
?Imipramine (80 μM, 24 h) induces HL-60 cell apoptosis[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	DLD-1, HCT-116, and SW-480
Concentration:	0.5-300 μM
Incubation Time:	3 days
Result:	Inhibited cell viability and HCT-116 was more sensitive than DLD-1 and SW-480.

Cell Migration Assay [1]

Cell Line:	SW-480, DLD-1, and HCT-116
Concentration:	20 μM
Incubation Time:	7 h
Result:	Produced a remarkable inhibition of migration in all assayed cell lines.

Cell Invasion Assay[1]

Cell Line:	HCT-116
Concentration:	20 μM
Incubation Time:	48 h
Result:	Inhibited cell invasion through Matrigel.

Western Blot Analysis[2]

Cell Line:	U-87MG
Concentration:	50 μM
Incubation Time:	0, 15, 30, 60, 120 and 240 min
Result:	Markedly inhibited the phosphorylation of both Akt (Ser473) and mTOR (Ser2481) in a time-dependent manner. Also dephosphorylated p70 S6K, a downstream target of mTOR.

Cell Autophagy Assay[2]

Cell Line:	U-87MG
Concentration:	60 μM
Incubation Time:	24 h
Result:	Stimulated the induction of autophagy through the redistribution of LC3 in U-87MG glioma

	cells.

Cell Autophagy Assay[3]

Cell Line:	HL-60
Concentration:	80 μM
Incubation Time:	24 h
Result:	Induced cell apoptosis.

In Vivo

Imipramine (20 mg/kg, i.p. or 15 mg/kg, p.o.; daily for 24 days) attenuates neuroinflammatory signaling and reverses stress-induced social avoidance in mice[4].

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

Animal Model:	Male C57BL/6 mice (6–8 weeks old) subjected to RSD (repeated social defeat) and HCC (home cage control)[4]
Dosage:	20 mg/kg or 15 mg/kg
Administration:	Intraperitoneal injection or oral administration, daily for 24 days
Result:	Reversed RSD-induced social avoidance behavior, significantly increasing the interaction time, significantly decreased stress-induced mRNA levels for IL-6 in brain microglia.

CUSTOMER VALIDATION

- Nat Chem Biol. 2024 Feb 14.
- Cell Commun Signal. 2023 May 25;21(1):123.
- Inflammation. 2021 Jan 29.
- Pathogens. 2022 May 22;11(5):602.

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REFERENCES

[1]. Alburquerque-González B, et al. New role of the antidepressant imipramine as a Fascin1 inhibitor in colorectal cancer cells. Exp Mol Med. 2020 Feb;52(2):281-292.

[2]. Jeon SH, et al. The tricyclic antidepressant imipramine induces autophagic cell death in U-87MG glioma cells. Biochem Biophys Res Commun. 2011 Sep 23;413(2):311-7.

[3]. Xia Z, et al. The antidepressants imipramine, clomipramine, and citalopram induce apoptosis in human acute myeloid leukemia HL-60 cells via caspase-3 activation. J Biochem Mol Toxicol. 1999;13(6):338-47.

[4]. Ramirez K, et al. Imipramine attenuates neuroinflammatory signaling and reverses stress-induced social avoidance. Brain Behav Immun. 2015 May;46:212-20.

[5]. Balkovetz DF, et al. Evidence for an imipramine-sensitive serotonin transporter in human placental brush-border membranes. J Biol Chem. 1989 Feb 5;264(4):2195-8.

Caution: Product has not been fully validated for medical applications. For research use only.

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