Mirtazapine

Cat. No.: HY-B0352

CAS No.: 85650-52-8

Molecular Formula: $C_{17}H_{19}N_3$

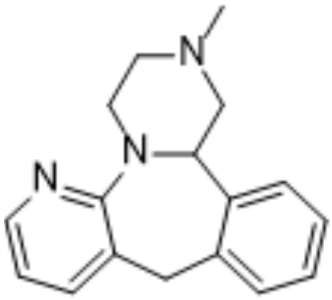
Molecular Weight: 265.35

Target: 5-HT Receptor; Histamine Receptor; Adrenergic Receptor

Pathway: GPCR/G Protein; Neuronal Signaling; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (188.43 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.7686 mL	18.8430 mL	37.6861 mL
	5 mM	0.7537 mL	3.7686 mL	7.5372 mL
	10 mM	0.3769 mL	1.8843 mL	3.7686 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 (9.42 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.42 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.42 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mirtazapine (Org3770) is a potent and orally active noradrenergic and specific serotonergic antidepressant (NaSSA) agent. Mirtazapine is also a 5-HT_2, 5-HT_3, histamine H1 receptor and α2-adrenoceptor antagonist with pK_i values of 8.05, 8.1, 9.3 and 6.95, respectively[1][2].

IC_{50} & Target				
	5-HT_3 Receptor	5-HT_2 Receptor	H_1 Receptor	α2-adrenergic receptor
	8.1 (pKi)	8.05 (pKi)	9.3 (pKi)	6.95 (pKi)

In Vitro	Mirtazapine can antagonize the adrenergic α2-autoreceptors and α2-heteroreceptors as well as block 5-HT_2 and 5-HT_3 receptors. Mirtazapine enhances the release of norepinephrine and 5-HT1A-mediated serotonergic transmission[1]. The cytochrome (CYP) P450 isoenzymes CYP1A2, CYP2D6, and CYP3A4 are mainly responsible for Mirtazapine's metabolism [1]. Mirtazapine (10 μM) significantly reduces activation-induced release of cytokine/chemokine mediators from human $CD14^+$ monocytes in vitro[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mirtazapine (1-20 mg/kg; intraperitoneal injection; once; C57BL/6 mice) treatment strikingly and dose-dependently inhibits Con A-induced liver injury[3]. Mirtazapine treatment inhibits hepatic macrophage/monocyte activation, decreases hepatic macrophage/monocyte-derived pro-inflammatory cytokine (e.g., TNFα) and chemokine (e.g., CXCL1 and CXCL2) production, suppression of Con A-induced increases in the hepatic expression of the neutrophil relevant endothelial cell adhesion molecule ICAM-1, with the resultant significant reduction in neutrophil recruitment into the liver[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C57BL/6 mice (8-10 week old) treated with concanavalin A (Con A)[3]
Dosage:	1 mg/kg, 10 mg/kg, and 20 mg/kg
Administration:	Intraperitoneal injection; once
Result:	Strikingly and dose-dependently inhibited Con A-induced liver injury.

CUSTOMER VALIDATION

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

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REFERENCES

[1]. S A Anttila, et al. A review of the pharmacological and clinical profile of mirtazapine. CNS Drug Rev. Fall 2001;7(3):249-64.

[2]. T H de Boer, et al. Neurochemical and autonomic pharmacological profiles of the 6-aza-analogue of mianserin, Org 3770 and its enantiomers. Neuropharmacology. 1988 Apr;27(4):399-408.

[3]. Wagdi Almishri, et al. The Antidepressant Mirtazapine Inhibits Hepatic Innate Immune Networks to Attenuate Immune-Mediated Liver Injury in Mice. Front Immunol. 2019 Apr 12;10:803.

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