Reserpine hydrochloride

Cat. No.: HY-N0480A

CAS No.: 16994-56-2

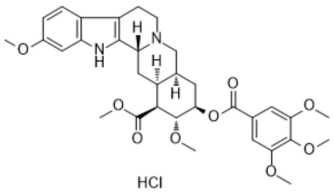
Molecular Formula: $C_{33}H_{41}ClN_2O_9$

Molecular Weight: 645.14

Target: Monoamine Transporter

Pathway: Membrane Transporter/Ion Channel

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (77.50 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.5501 mL	7.7503 mL	15.5005 mL
5 mM	0.3100 mL	1.5501 mL	3.1001 mL
10 mM	0.1550 mL	0.7750 mL	1.5501 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 (3.88 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.88 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (3.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Reserpine hydrochloride is an inhibitor of the vesicular monoamine transporter 2 (VMAT2).

IC_{50} & Target

VMAT2[1]

In Vitro

Reserpine hydrochloride is an inhibitor of the vesicular monoamine transporter 2 (VMAT2). Reserpine hydrochloride displays a significant on the density of dopamine D1 receptors ($F_{2,12}$=8.81, p<0.01) in the rat striatum. The affinity (Kd) for the dopamine D1 and D2 receptors during withdrawal from acute and chronic administration of reserpine is not change[1]. IC_{50}

	values of 43.9 and 54.9 μM are obtained after 1 day of treatment with Reserpine hydrochloride in JB6 P+ and HepG2-C8 cells, respectively. Reserpine hydrochloride induces luciferase activity in a dose-dependent manner at concentrations ranging from 5 to 50 μM, and no significant induction is observed at concentrations lower than 5 μM. Results demonstrate that Reserpine hydrochloride (2.5 to 10 μM) also increases the protein expression of Nrf2, HO-1, and NQO1. Reserpine hydrochloride at concentrations of 2.5 to 10 μM decreases the mRNA expression of DNMT1, DNMT3a, and DNMT3b in a concentration-dependent manner in JB6 P+ cells after 7 days of treatment. Reserpine hydrochloride at 10 μM generates a significant difference for DNMT3a expression ($p<0.05$)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Withdrawal (48 h) from chronic (14-day) but not acute Reserpine hydrochloride administration in a dose of 0.2 mg/kg i.p. produces a significant reduction of the immobility time ($F_{2,18}=3.68$, $p<0.05$), but increases the climbing time ($F_{2,18}=4.48$, $p<0.02$), and does not change the swimming time ($F_{2,18}=1.78$; NS) in the forced swim test (FST) in rats[1]. Reserpine hydrochloride at a dose of 5 mg/kg body weight produces significant increase in the urinary excretion profile of vanillylmandelic acid (VMA) compare to control animals. The amount of 5-hydroxyindoleacetic acid (5-HIAA) excreted in animals treated with Reserpine is found to be more than in the control. Dose dependent hypotension is observed with Reserpine hydrochloride. Reserpine hydrochloride at doses of 0.5, 1, 5, 10 and 15 μg/kg produce significant ($p<0.01$) reduction in blood pressure compare to control[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	After incubation for 24 h, JB6 P+ cells (1×10^5 cells/10-cm dish) are treated with various concentrations of Reserpine hydrochloride. Whole cell lysates are prepared from the treated cells using radioimmunoprecipitation assay buffer supplemented with a protease inhibitor cocktail, and a BCA kit is used to determine protein concentrations[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Cell Assay [2]	JB6 P+ cells are seeded in 96-well plates containing Minimum essential media (MEM) at a density of 1×10^4 cells/mL (100 μL/well) for 1, 3, and 5 days, and HepG2-C8 cells are seeded in plates containing DMEM. After incubation for 24 h, the cells are treated with either DMSO or various concentrations of Reserpine hydrochloride. For JB6 P+ cells, the medium is changed every 2 days for the 3-day and 5-day treatments. Cell viability is assessed using a MTS assay kit according to the manufacturer's instructions. The absorbance of the formazan product is read at 490 nm, and the cell viability is calculated and compared with the DMSO control group[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Albino rats of either sex weighing between 100 to 150 g are used in the study. They are acclimatized to the laboratory conditions for at least 10 days prior to the experiment and are provided with standard diet and water ad libitum with 12 h light and dark cycle. Animals are divided into different groups of six each and are housed individually in metabolic cages. Group 1: Control animals treated with DMSO intraperitoneally at a dose of 0.1 mL/100 g body weight. Group 2: Animals administered intraperitoneally with Reserpine hydrochloride at a dose of 5 mg/kg body weight. The 24 h urine samples from the point of drug administration are collected for each animal[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Crit Rev Anal Chem. 2021 Mar 10;1-15.
- Nigerian Journal of Scientific Research. 18 (3): 2019.

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REFERENCES

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[3]. Gupta MB, et al. Mechanism of ulcerogenic activity of reserpine in albino rats. Eur J Pharmacol. 1974 Jul;27(2):269-71.

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[6]. Antkiewicz-Michaluk L, et al. Withdrawal from repeated administration of a low dose of reserpine induced opposing adaptive changes in the noradrenaline and serotonin system function: a behavioral and neurochemical ex vivo and in vivo studies in the rat. Prog Neuropsychopharmacol Biol Psychiatry. 2015 Mar 3;57:146-54.

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