Reserpine

Cat. No.: HY-N0480

CAS No.: 50-55-5

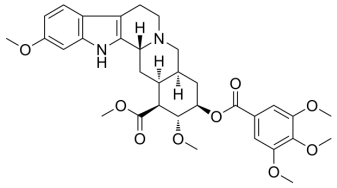
Molecular Formula: $C_{33}H_{40}N_2O_9$

Molecular Weight: 608.68

Target: Monoamine Transporter; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (41.07 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6429 mL	8.2145 mL	16.4290 mL
	5 mM	0.3286 mL	1.6429 mL	3.2858 mL
	10 mM	0.1643 mL	0.8214 mL	1.6429 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 (4.11 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

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

IC_{50} & Target

VMAT2[1]

In Vitro

Reserpine is an inhibitor of the vesicular monoamine transporter 2 (VMAT2). Reserpine displays a significant effect 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 in JB6 P+ and HepG2-C8 cells, respectively. Reserpine 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 (2.5 to 10 μM) also increases the protein expression of Nrf2, HO-1, and NQO1. Reserpine 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 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

Induction of Gastric Ulcer[4][5][6]

Background

Peripheral cholinergic and adrenergic mechanisms are involved in the ulceration induced by reserpine. The ulcerogenic activity of reserpine was significantly reduced by α-adrenoceptor antagonists (phenoxybenzamine (HY-B0431) and phentolamine (HY-12717)) but not by the β-adrenoceptor blocker, propranolol (HY-B0573B)[6].

Specific Mmodeling Methods

Rats: Wistar Rats • male • 200-290 g[4]

Administration: 5 mg/kg • ip • 18 h before sacrifice

Mice: ICR mice • male • 7 weeks old[5]

Administration: 10 mg/kg • ip • once daily for 3 days

Note

(1)Rats were fasted with water ad libitum for 48 h prior to experimentation. Rats were housed and experiments were conducted in a temperature-controlled room (23 ± 1°C).(2)The level of cancer induction was identified by specific biochemical markers such as serum gastrin level, TBARS, and glutathione followed by histopathological analysis at two-time periods for 8 and 16 week.

Modeling Record

Individual phenotypic changes: induced marked gastric glandular ulceration and elicited the release of free /~-glucuronidase from lysosomes in the gastric mucosa.

Molecular changes: In the reserpine-induced gastric ulcer control mice, the gastric secretion volume was increased, the pH value (1.04) was decreased, the serum cytokine levels of IL-6, IL-12, TNF-α and IFN-γ was increased.

Correlated Product(s):

Induction of Depression[7][8]

Background

Reserpine is an irreversible inhibitor of vesicular monoamine transporter 2, which regulates the accumulation of monoamines into the synaptic vesicles and their reuptake from the synapses. Therefore, Reserpine inhibits monoamine pre-synaptic reuptake and storage, leading to monoamine depletion and depressive disorders[7].

Specific Mmodeling Methods

Rats: Wistar Rats • male • 120-150 g[7]

Administration: 0.5 mg/kg • ip • once per day for 14 days

Mice: C57BL/6 mice • male • 7 weeks old[8]

Administration: 0.5 mg/kg • ip • once per day for 10 days

Note

(1)Reserpine was diluted in glacial acetic acid to a final concentration of 0.5% acetic acid in distilled water.

Modeling Record

Individual phenotypic changes: showed a significant decrease in spontaneous locomotor activity in the activity cage, decrease in latency to immobility, and increase in the immobility duration in forced swimming test (FST), indicating motor impairment and worsened depressive phenotype.

Molecular changes: Reserpine administration significantly increased cortical contents of MDA (malondialdehyde), reduced GSH (glutathione), increased TNF-α and reduced BDNF (brain derived neurotropic factor). Showed a significant decrease in cortical nor-epinephrine (NE), serotonin (5-HT), and dopamine (DA)

Correlated Product(s):

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. 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].

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. 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].

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 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 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].

CUSTOMER VALIDATION

- Biomed Pharmacother. 2024 Jul:176:116856.
- Crit Rev Anal Chem. 2021 Mar 10;1-15.
- Nigerian Journal of Scientific Research. 18 (3): 2019.

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[1]. Pfeiffer CJ, et al. Reserpine-induced gastric ulcers: protection by lysosomal stabilization due to zinc. Eur J Pharmacol. 1980 Feb;61(4):347-53.

[2]. Li GJ, et al. Preventive Effect of Polysaccharide of Larimichthys crocea Swim Bladder on Reserpine Induced Gastric Ulcer in ICR Mice. Korean J Physiol Pharmacol. 2014 Apr;18(2):183-90.

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

[4]. Park BK, et al. Antidepressant-Like Effects of Gyejibokryeong-hwan in a Mouse Model of Reserpine-Induced Depression. Biomed Res Int. 2018 Jun 26;2018:5845491.

[5]. El-Marasy SA, et al. Anti-depressant effect of cerebrolysin in reserpine-induced depression in rats: Behavioral, biochemical, molecular and immunohistochemical evidence. Chem Biol Interact. 2021 Jan 25;334:109329.

[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.

[7]. Hong B, et al. Reserpine Inhibit the JB6 P+ Cell Transformation Through Epigenetic Reactivation of Nrf2-Mediated Anti-oxidative Stress Pathway. AAPS J. 2016 May;18(3):659-69.

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