Methyllycaconitine citrate

Cat. No.: HY-N2332A

CAS No.: 351344-10-0

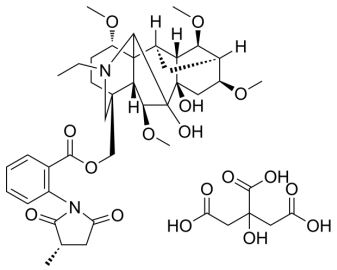
Molecular Formula: $C_{43}H_{58}N_2O_{17}$

Molecular Weight: 874.92

Target: nAChR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (142.87 mM; Need ultrasonic)
H_2O : 2.18 mg/mL (2.49 mM; Need ultrasonic and warming)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.1430 mL	5.7148 mL	11.4296 mL
5 mM	0.2286 mL	1.1430 mL	2.2859 mL
10 mM	0.1143 mL	0.5715 mL	1.1430 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: ≥ 2.08 mg/mL (2.38 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (2.38 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (2.38 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Methyllycaconitine citrate is a specific antagonist of α7 neuronal nicotinic acetylcholine receptor (α7nAChR) with blood-brain barrier permeability.

IC_{50} & Target

α7nAChR[1]

In Vitro

Pretreatment with 5 and 10 μM Methyllycaconitine citrate (MLA) inhibits the decreased cell viability induced by $Aβ_{25-35}$. Cell viability does not decrease after exposure to Methyllycaconitine citrate (2.5, 5, 10, 20 μM). $Aβ_{25-35}$ treatment increases LC3-II levels, which is inhibited by administration of Methyllycaconitine citrate. Methyllycaconitine citrate also inhibits Aβ-induced

	autophagosome accumulation in SH-SY5Y cells. Flow cytometry also demonstrates decreased MDC-labeled vacuoles with Methyllycaconitine citrate treatment[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Methyllycaconitine citrate (MLA) (6 mg/kg) given alone intraperitoneally does not cause climbing behavior when compare with the saline group. Pretreatment with Methyllycaconitine citrate significantly inhibits methamphetamine (METH)-induced climbing behavior, by about 50%. Methyllycaconitine citrate does not modify either basal locomotor activity or METH-induced hyperlocomotion. The METH-induced depletion of dopamine neuron terminals is attenuated in mice pretreated with Methyllycaconitine citrate (250±43 fmol/mg, n=7). A direct effect of Methyllycaconitine citrate on body temperature is ruled out because Methyllycaconitine citrate does not affect basal body temperature (37.0±0.5°C, n=5) or reduce the METH-induced hyperthermia (38.2±0.4°C, n=6, MLA+METH group, n.s. versus METH group)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cells are plated in 96-well plates containing complete medium and cultured for 24 h. Then cells are treated with Methyllycaconitine citrate at the indicated concentrations for specified times. After drug treatment, cell viability is measured by MTT assay. Briefly, 10 μL of the MTT solution (5 mg/mL) is added to each well and incubated for 4 h at 37°C. After removing the supernatant, 100 μL DMSO is added into each well. The absorbance is measured at 570 nm with a microplate reader. All experiments are repeated 3 times[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Adult male Swiss CD-1 mice are used in all experiments. They are housed at 22±1°C under a 12-h light/dark cycle with free access to food and drinking water. Climbing behavior is measured. Briefly, mice of 20 to 26 g are intraperitoneally administered saline (5 mL/kg) or Methyllycaconitine citrate (MLA) (6 mL/kg) at the beginning of the test. Twenty minutes later, the animals receive a single dose of saline or methamphetamine (METH) (1 mL/kg) subcutaneously and are placed individually, for habituation, into the experimental chamber consisting of a cylindrical cage with the wall made of plastic bars and covered with a lid. After a 20-min period of exploratory activity, stereotypy measurement is performed for a period of 30 min[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Neuroinflammation. 2021 Dec 17;18(1):291.
- Cell Rep. 2022 Aug 30;40(9):111308.
- Acta Pharmacol Sin. 2024 Mar 19.
- Cell Death Discov. 2022 Mar 30;8(1):141.
- Cell Death Discov. 2022 Feb 8;8(1):54.

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REFERENCES

[1]. Lockman PR, et al. Chronic nicotine exposure alters blood-brain barrier permeability and diminishes brain uptake of methyllycaconitine. J Neurochem. 2005 Jul;94(1):37-44.

[2]. Zheng X, et al. Methyllycaconitine alleviates amyloid-β peptides-induced cytotoxicity in SH-SY5Y cells. PLoS One. 2014 Oct 31;9(10):e111536.

[3]. Escubedo E, et al. Methyllycaconitine prevents methamphetamine-induced effects in mouse striatum: involvement of alpha7 nicotinic receptors. J Pharmacol Exp Ther. 2005 Nov;315(2):658-67.

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