Anatabine dicitrate

Cat. No.: HY-19918A

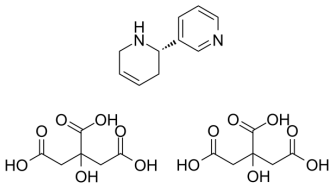
Molecular Formula: $C_{22}H_{28}N_2O_{14}$

Molecular Weight: 544.46

Target: nAChR; Amyloid-β; NF-κB

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; NF-κB

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

H_2O : 100 mg/mL (183.67 mM; Need ultrasonic)
DMSO : 30 mg/mL (55.10 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8367 mL	9.1834 mL	18.3668 mL
5 mM	0.3673 mL	1.8367 mL	3.6734 mL
10 mM	0.1837 mL	0.9183 mL	1.8367 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 50 mg/mL (91.83 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Anatabine dicitrate is a tobacco alkaloid that can cross the blood-brain barrier. Anatabine dicitrate is a potent α4β2 nAChR agonist. Anatabine dicitrate inhibits NF-κB activation lower amyloid-β (Aβ) production by preventing the β-cleavage of amyloid precursor protein (APP). Anatabine dicitrate has anti-inflammatory effects and has the potential for neurodegenerative disorders treatment[1][2][3].

IC_{50} & Target

NF-κB[1]
Amyloid-β (Aβ)[1]
α4β2 nAChR[2]

In Vitro

Anatabine (600 μg/mL; 24 hours; SHSY-5Y cells) treatment shows an inhibition of p65 NF-κB phosphorylation[1].
Anatabine (500-1000μg/mL; 30 minutes; SHSY-5Y cells) treatment fully prevents the increase in BACE-1 mRNA levels induced by TNF-α. After 24 hours, Anatabine treatment shows a dose-dependent inhibition of BACE-1 protein levels[1].
Anatabine dose dependently inhibits $Aβ_{1-40}$ and $Aβ_{1-42}$ with an approximate half maximal inhibitory concentration of 640 μ

g/mL for both $A\beta_{1-40}$ and $A\beta_{1-42}$ in 7W CHO cells. Anatabine inhibits sAPPβ secretion without impacting sAPPα suggesting that Anatabine is preventing the β-cleavage of amyloid precursor protein (APP)[1].

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

Western Blot Analysis[1]

Cell Line:	SHSY-5Y cells
Concentration:	600 μg/mL
Incubation Time:	24 hours
Result:	An inhibition of p65 NF-κB phosphorylation was observed.

RT-PCR[1]

Cell Line:	SHSY-5Y cells
Concentration:	500 μg/mL, 1000 μg/mL
Incubation Time:	30 minutes
Result:	Fully prevented the increase in BACE-1 mRNA levels induced by TNF-α.

In Vivo

Anatabine (0.5-2 mg/kg; intraperitoneal injection; daily; for 4 days; transgenic mouse) treatment significantly lowers brain soluble $A\beta_{1-40}$ and $A\beta_{1-42}$ levels in a transgenic mouse model of Alzheimer's disease[1].

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

Animal Model:	Transgenic mice overexpressing the human APP695sw mutation and the presenilin-1 mutation M146L (Tg PS1/APPsw) (50 week-old)[1]
Dosage:	0.5 mg/kg, 2 mg/kg
Administration:	Intraperitoneal injection; daily; for 4 days
Result:	Significantly lowered brain soluble $A\beta_{1-40}$ and $A\beta_{1-42}$ levels in a transgenic mouse model of Alzheimer's disease.

REFERENCES

[1]. Paris D, et al. Anatabine lowers Alzheimer's Aβ production in vitro and in vivo. Eur J Pharmacol. 2011 Nov 30;670(2-3):384-91.

[2]. Xing H, et al. A Pharmacological Comparison of Two Isomeric Nicotinic Receptor Agonists: The Marine Toxin Isoanatabine and the Tobacco Alkaloid Anatabine. Mar Drugs. 2020 Feb 11;18(2). pii: E106.

[3]. eo EJ, et al. Phytochemicals as inhibitors of NF-κB for treatment of Alzheimer's disease. Pharmacol Res. 2018 Mar;129:262-273.

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