Tomatidine hydrochloride

Cat. No.: HY-N2149A

CAS No.: 6192-62-7

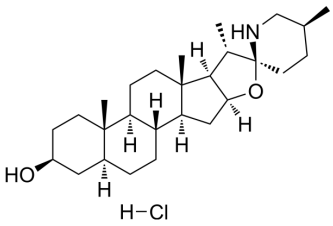
Molecular Formula: $C_{27}H_{46}ClNO_2$

Molecular Weight: 452.11

Target: NF-κB; JNK; Autophagy; Endogenous Metabolite

Pathway: NF-κB; MAPK/ERK Pathway; Autophagy; Metabolic Enzyme/Protease

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

Methanol : 12.5 mg/mL (27.65 mM; Need ultrasonic)
Ethanol : 3.45 mg/mL (7.63 mM; Need ultrasonic)
DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)
H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2119 mL	11.0593 mL	22.1185 mL
	5 mM	0.4424 mL	2.2119 mL	4.4237 mL
	10 mM	0.2212 mL	1.1059 mL	2.2119 mL

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

BIOLOGICAL ACTIVITY

Description

Tomatidine hydrochloride acts as an anti-inflammatory agent by blocking NF-κB and JNK signaling[1]. Tomatidine hydrochloride activates autophagy either in mammal cells or C elegans[2].

IC_{50} & Target	p65	JNK

In Vitro

Tomatidine decreases inducible NO synthase and COX-2 expression through suppression of I-κBα phosphorylation, NF-κB nuclear translocation and JNK activation, which in turn inhibits c-jun phosphorylation and Oct-2 expression. Tomatidine, solasodine and diosgenin (40 μM) show 66%, 22% and 41% inhibition of nitrite production, respectively. The iNOS protein is barely detectable in unstimulated cells but markedly increases after LPS treatment, and Tomatidine causes dose-dependent inhibition of LPS-induced iNOS expression. p65 is the major component of NF-κB in LPS-stimulated macrophages, the effect of Tomatidine on p65 DNA-binding activity is determined. In the presence of Tomatidine at 10-40 μM, the binding activity of NF-κB is suppressed in a dose-dependent manner. Tomatidine inhibits the phosphorylation of I-κB, blocks the I-κB production, and furthermore suppresses p65 NF-κB translocation to the nucleus and modulated binding activity[1].

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

PROTOCOL

Cell Assay [1]

RAW 264.7 cells, derived from murine macrophages, are cultured in DMEM supplemented with 10% endotoxin-free, heat-inactivated fetal calf serum, Penicillin (100 units/mL), and Streptomycin (100 μg/mL) in a 5% CO_2 atmosphere at 37 °C in a humidified incubator. For all assay, cell is plated at 2×10^5 cells/cm^2 in culture dishes or plates. Treatment with vehicle (0.1% DMSO or 0.1% ethanol), test compounds and/or LPS is carried out under serum-free conditions[1].

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

CUSTOMER VALIDATION

- iScience. 2023 Jul 13.
- Aging. 2020 Jul 5;12(13):12799-12811.
- Eur J Pharmacol. 2020 Sep 5;882:173280.
- FASEB J. 2019 Feb;33(2):2574-2586.
- Research Square Preprint. 2023 Apr 27.

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REFERENCES

[1]. Chiu FL, et al. Tomatidine inhibits iNOS and COX-2 through suppression of NF-kappaB and JNK pathways in LPS-stimulated mouse macrophages. FEBS Lett. 2008 Jul 9;582(16):2407-12.

[2]. Anil Ahsan, et al. Tomatidine Protects Against Ischemic Neuronal Injury by Improving Lysosomal Function. Eur J Pharmacol. 2020 Jun 21;173280.

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