Atraric acid

Cat. No.: HY-N2908

CAS No.: 4707-47-5

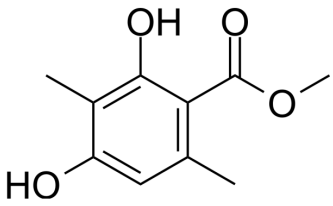
Molecular Formula: $C_{10}H_{12}O_4$

Molecular Weight: 196.2

Target: Androgen Receptor; NO Synthase; p38 MAPK; NF-κB

Pathway: Vitamin D Related/Nuclear Receptor; Immunology/Inflammation; MAPK/ERK Pathway; NF-κB

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (509.68 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	5.0968 mL	25.4842 mL	50.9684 mL
	5 mM	1.0194 mL	5.0968 mL	10.1937 mL
	10 mM	0.5097 mL	2.5484 mL	5.0968 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (12.74 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Atraric acid (Methyl atrarate) is a specific androgen receptor (AR) antagonist with anti-inflammatory and anticancer effects. Atraric acid represses the expression of the endogenous prostate specific antigen gene in both LNCaP and C4-2 cells. Atraric acid can also inhibit the synthesis of NO and cytokine, and suppress the MAPK-NFκB signaling pathway. Atraric acid can be used to research prostate diseases and inflammatory diseases[1][2].

IC_{50} & Target

Androgen receptor, NO synthesis, MAPK-NFκB pathway[1][2]

In Vitro

Atraric acid (10 μM; CV1 cells) represses the transactivation function mediated by Dihydrotestosterone-induced human AR[1].

Atraric acid (10 μM; PCa cells) inhibits the expression of the PSA gene in both androgen-dependent and androgen-independent PCa cells[1].

Atraric acid (1-300 μM; 24 h) dose-dependently inhibits pro-inflammatory cytokine, nitric oxide, prostaglandin E2 in LPS-stimulated RAW264.7 cells, but does not influence the cell viability[2].

Atraric acid (100 and 300 μM; 18 h or 4 h) downregulates the expression of phosphorylated IκB, extracellular signal-regulated kinases (ERK) and nuclear factor kappa B (NFκB) signaling pathway to exhibit anti-inflammatory effects in LPS-stimulated RAW264.7 cells[2].

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

Cell Viability Assay[2]

Cell Line:	RAW264.7 cells
Concentration:	1-300 μM
Incubation Time:	24 h
Result:	Did not influence the cell viability.

Western Blot Analysis[2]

Cell Line:	RAW264.7 cells
Concentration:	100 and 300 μM
Incubation Time:	18 h or 4 h
Result:	Inhibited LPS-Induced expression of iNOS and COX-2 in a dose-dependent manner. Suppressed LPS-stimulated phosphorylation of the Nfκb signaling pathway.

In Vivo

Atraric acid (10, 30 mg/kg; i.p.; single dosage) inhibits the production of pro-inflammatory cytokines and reduces pathological damages in LPS-induced endotoxin shock mice[2].

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

Animal Model:	Female BALB/c mice (7 weeks old, 17-20 g; LPS-induced endotoxin shock)[2]
Dosage:	10, 30 mg/kg
Administration:	i.p.; single dosage
Result:	Inhibited the production of pro-inflammatory cytokines. Reduced pathological damages such as vasodilation and bleeding.

REFERENCES

[1]. Roell D, Baniahmad A. The natural compounds atraric acid and N-butylbenzene-sulfonamide as antagonists of the human androgen receptor and inhibitors of prostate cancer cell growth. Mol Cell Endocrinol. 2011 Jan 30;332(1-2):1-8.

[2]. Papaioannou M, et al. The natural compound atraric acid is an antagonist of the human androgen receptor inhibiting cellular invasiveness and prostate cancer cell growth. J Cell Mol Med. 2009 Aug;13(8B):2210-2223.

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