Atractylenolide I

Cat. No.: HY-N0201

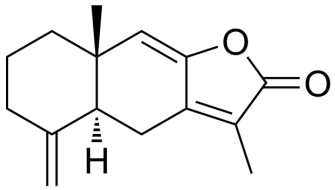
CAS No.: 73069-13-3

Molecular Formula: $C_{15}H_{18}O_2$

Molecular Weight: 230.3

Target: Toll-like Receptor (TLR); JAK; STAT; TNF Receptor

Pathway: Immunology/Inflammation; Epigenetics; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Stem Cell/Wnt; Apoptosis



Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (434.22 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	4.3422 mL	21.7108 mL	43.4216 mL
	5 mM	0.8684 mL	4.3422 mL	8.6843 mL
	10 mM	0.4342 mL	2.1711 mL	4.3422 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 (10.86 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (10.86 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (10.86 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Atractylenolide I is a sesquiterpene derived from the rhizome of Atractylodes macrocephala, possesses diverse bioactivities, such as neuroprotective, anti-allergic, anti-inflammatory and anticancer properties. Atractylenolide I reduces protein levels of phosphorylated JAK2 and STAT3 in A375 cells, and acts as a TLR4-antagonizing agent.

IC_{50} & Target		
	TLR7	TLR9

In Vitro	Atractylenolide I (40, 60, 80, 100, 120, 150 μM) dose- and time-dependently reduces the cell viability in human A375 melanoma cells after treatment for 24, 48 and 72 hours. Atractylenolide I (50 and 100 μM) induces apoptosis of A375 cells in a dose-dependent manner at 48 h of treatment. Atractylenolide I (100 μM) significantly reduces protein levels of phosphorylated JAK2 and STAT3 in A375 cells, without effect on total JAK2 and STAT3. Furthermore, Atractylenolide I inhibits the mRNA expression of STAT3-targeted genes, including Bcl-xL, MMP-2 and MMP-9[1]. Atractylenolide I (up to 100 μM) shows no toxicity in normal cells. Atractylenolide I (25, 50 μM) decreases the Ox-LDL induced TNF-α, IL-6 and NO production in VSMCs. Atractylenolide I (12.5, 25 or 50 μM) significantly reduces the level of MCP-1 and inhibits Ox-LDL-induced VSMCs proliferation and migration. Atractylenolide I (25, 50 μM) inhibits positive staining of foam cells, and also significantly decreases lipid accumulation. Atractylenolide I (50 μM) suppresses p38MAPK and NF-κB p65 expression in VSMCs stimulated by Ox-LDL[3]. Atractylenolide I (1, 10, 100 μM) downregulates paclitaxel-induced expression of VEGF and survivin via MyD88-dependent TLR4 signaling in EOC cells[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Atractylenolide I (5, 10 or 20 mg/kg, p.o.) restores the decreased body weight in mice subjected to chronic unpredictable mild stress (CUMS). Atractylenolide I alleviates CUMS-induced depressive-like behavior, attenuates CUMS-induced imbalances in hippocampal neurotransmitter levels and reduces CUMS-induced increases in hippocampal pro-inflammatory cytokine levels and in the NLRP3 inflammasome in the hippocampi of mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	Briefly, serum starved VSMCs are pre-treated with indicated concentration of Atractylenolide I for 1 h followed by stimulation with Ox-LDL for 24 h. The purple formazan crystals formed after addition of MTT are solubilized in DMSO and absorbance is measured at 540 nm. The viability or proliferation rate is calculated as percentage of control (untreated VSMCs)[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] After adaption for one week, 48 male ICR mice are randomly divided into six groups (eight mice per group): Control group (unstressed + saline vehicle), model group (CUMS + saline vehicle), three Atractylenolide I treatment groups (CUMS + Atractylenolide I) and a fluoxetine group (CUMS + FLU). From the 4th week, Atractylenolide I (5, 10 or 20 mg/kg) or fluoxetine (20 mg/kg) is daily administered by oral gavage for 3 weeks. After the last administration of Atractylenolide I or fluoxetine, behavioral tests are performed[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2019 Mar 4;10(1):1015.
- Phytother Res. 2023 Oct 19.

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REFERENCES

[1]. Atractylenolide I, et al. The JAK2/STAT3 pathway is involved in the anti-melanoma effects of atractylenolide I. Exp Dermatol. 2018 Feb;27(2):201-204.

[2]. Gao H, et al. Anti-depressant-like effect of atractylenolide I in a mouse model of depression induced by chronic unpredictable mild stress. Exp Ther Med. 2018 Feb;15(2):1574-1579.

[3]. Li W, et al. Atractylenolide I restores HO-1 expression and inhibits Ox-LDL-induced VSMCs proliferation, migration and inflammatory responses in vitro. Exp Cell Res. 2017 Apr 1;353(1):26-34.

[4]. Huang JM, et al. Atractylenolide-I sensitizes human ovarian cancer cells to paclitaxel by blocking activation of TLR4/MyD88-dependent pathway. Sci Rep. 2014 Jan 23;4:3840.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA