ITE

Cat. No.: HY-19317

CAS No.: 448906-42-1

Molecular Formula: $C_{14}H_{10}N_2O_3S$

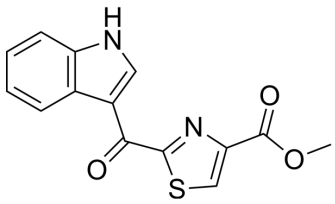
Molecular Weight: 286.31

Target: Aryl Hydrocarbon Receptor

Pathway: Immunology/Inflammation

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 : ≥ 41 mg/mL (143.20 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.4927 mL	17.4636 mL	34.9272 mL
5 mM	0.6985 mL	3.4927 mL	6.9854 mL
10 mM	0.3493 mL	1.7464 mL	3.4927 mL

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

In Vivo

1. Add each solvent one by one: 17% Polyethylene glycol 12-hydroxystearate in saline
   Solubility: 10 mg/mL (34.93 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.67 mg/mL (9.33 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 2.08 mg/mL (7.26 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

ITE is a potent endogenous agonist of aryl hydrocarbon receptor (AhR), binding directly to AHR, with a K_i of 3 nM. ITE also has immunosuppressive activity.

IC_{50} & Target

Ki: 3 nM (AhR)[1]

In Vitro

ITE is an endogenous agonist of AhR, binding directly to AHR, with a K_i of 3 nM[1]. ITE (0.03-30 mg/mL) decreases the antigen-

	specific T-cell proliferative responses[2]. ITE potently inhibits human pulmonary artery endothelial (HPAECs) growth at 10 and 20 μM, but shows no effect at 0.01-5 μM. ITE does not affect cell cycle progress of HPAECs at 10 and 20 μM, or induce expression of cleaved caspase-3 protein in HPAECs at 20 μM. In addition, ITE (20 μM) elevates CYP1A1 and CYP1B1 mRNA levels and decreases the levels of AhR protein in HPAECs[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	ITE (200 μg, i.p.) significantly suppresses the development of experimental autoimmune uveitis (EAU) in mice. ITE reduces the proportions of cells expressing IFN-γ, IL-17, or IL-10 in mice. ITE also suppresses the secretion of inflammatory cytokines by LN cells in mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	Subconfluent cells (25, 000 cells/well) are seeded in 96-well plates. Cells are treated with ITE at 5, 10 and 20 μM or DMSO (0.1% v/v) in ECM for 2, 4 or 6 days with a change of ECM containing DMSO or ITE every other day (5 wells/treatment). At the end of treatment, cells are incubated with MTT reagent for 4 hr, and solubilized in crystal dissolving solution (100 μL/well) for 20 min. The absorbance is determined at 570 nm using the microplate reader[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Eight- to 12-week-old female B10.A mice is used in the assay. Daily treatment starts on day 0 and consists of 200 μg of ITE suspended in 0.2 mL PBS, given intraperitoneally. Control mice are similarly treated with 0.2 mL of the vehicle, PBS containing 3.6% DMSO[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Microbiome. 2023 Nov 7;11(1):245.
- EMBO Mol Med. 2022 Oct 28;e15677.
- EMBO Mol Med. 2021 Mar 16;e13466.
- Phytomedicine. 14 September 2021, 153751.
- Clin Epigenetics. 2022 Sep 2;14(1):109.

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REFERENCES

[1]. Song J, et al. A ligand for the aryl hydrocarbon receptor isolated from lung. Proc Natl Acad Sci U S A. 2002 Nov 12;99(23):14694-9.

[2]. Nugent LF, et al. ITE, a novel endogenous nontoxic aryl hydrocarbon receptor ligand, efficiently suppresses EAU and T-cell-mediated immunity. Invest Ophthalmol Vis Sci. 2013 Nov 13;54(12):7463-9.

[3]. Pang LP, et al. ITE inhibits growth of human pulmonary artery endothelial cells. Exp Lung Res. 2017 Oct;43(8):283-292.

Caution: Product has not been fully validated for medical applications. For research use only.

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