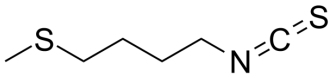
Erucin

Cat. No.:	HY-121323
CAS No.:	4430-36-8
Molecular Formula:	$C_6H_{11}NS_2$
Molecular Weight:	161.29
Target:	Apoptosis
Pathway:	Apoptosis
Storage:	Pure form -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (620.00 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	6.2000 mL	31.0001 mL	62.0001 mL
	5 mM	1.2400 mL	6.2000 mL	12.4000 mL
	10 mM	0.6200 mL	3.1000 mL	6.2000 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 (15.50 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.50 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Erucin (ERU) is an isothiocyanate particularly abundant in arugula. Erucin shows anticancer, neuroprotective, and anti-inflammatory activities[1][2][3][4].

In Vitro

Erucin (ERU) (0-100 μM) releases H_2S and inhibits cell viability in AsPC□1 cells in a concentration-dependent manner[1].
Erucin inhibits cell migration and altered the AsPC□1 cell cycle, reducing G0/G1 phase and increasing G2/M and S phases[1].
Erucin (30 μM, 72 h) induces AsPC□1 cell apoptosis and inhibits cell migration[1].
Erucin reduces levels of phosphorylated ERK1/2 in AsPC□1 cells[1].

Erucin (0-200 μM, 24 h) shows antiproliferative activity with an IC_{50} of 97.7 μM in A549 cells[2].
Erucin (0-50 μM, 24 h) induces the cleavage of PARP-1 at 50 μM, and increases p53 and p21 protein expression in A549 cells[2].
Erucin decreases LPS-induced production of NO, prostaglandin E_2 (PGE_2), TNF-α, IL-6 and IL-1β in RAW 264.7 cells[3].
Erucin decreases LPS-induced expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase (COX)-2 in RAW 264.7 cells[3].
Erucin inhibits LPS-induced activation of NFκB Signaling in RAW 264.7 cells[3].

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

Cell Viability Assay[1]

Cell Line:	AsPC□1
Concentration:	10, 30, and 100 μM
Incubation Time:	72 h
Result:	Showed a significant and concentration□dependent reduction of cell viability.

Cell Cycle Analysis[1]

Cell Line:	AsPC□1
Concentration:	30 μM
Incubation Time:	72 h
Result:	Showed a particular increase of cells number in the G2/M phase (36.6% ± 3.5 vs. vehicle□ treated cells in the G2/M phase: 24.0% ± 1.3) and in the S□phase (18.1% ± 1.5 vs. vehicle□ treated cells in the S phase: 11.0% ± 0.7) and a consequent significant reduction of cells in the G0/G1 phase (35.1% ± 5.0 vs. vehicle□treated cells in the G0/G1 phase: 59.5% ± 1.8.

Apoptosis Analysis[1]

Cell Line:	AsPC□1
Concentration:	30 μM
Incubation Time:	72 h
Result:	Significantly increased the number of total apoptotic cells (apoptotic dead cells and apoptotic live cells; vehicle: 17.7% ± 2.5 vs. Erucin: 28.7% ± 4.2).

Cell Proliferation Assay[2]

Cell Line:	A549
Concentration:	0-200 μM
Incubation Time:	24 h
Result:	Showed antiproliferative effect with an IC_{50} of 97.7 μM.

Western Blot Analysis[2]

Cell Line:	A549
Concentration:	0-50 μM
Incubation Time:	24 h

Result:	Induced the cleavage of PARP-1 at 50 μM. Increased p53 and p21 protein expression.

Western Blot Analysis[3]

Cell Line:	RAW 264.7
Concentration:	0, 2.5, and 5 μM
Incubation Time:	30 min
Result:	Decreased the expression of iNOS and COX-2 induced by LPS. Suppressed the LPS-induced reduction in IκB-α. Suppressed NFκB DNA binding and transcriptional activity.

RT-PCR[3]

Cell Line:	RAW 264.7
Concentration:	0, 2.5, and 5 μM
Incubation Time:	24 h
Result:	Decreased LPS-induced TNF-α, IL-6 and IL-1β production.

In Vivo

Erucin (ERU) (0-300 nM) significantly inhibits TPA-induced edema formation[3].
Erucin (30 μmol/kg; i.p.; twice a week for 4 week) shows neuroprotective effects[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female ICR mice (4 weeks of age), TPA (12-O-tetradecanoylphorbol-13-acetate)-induced mouse ear edema model[3]
Dosage:	0, 100, and 300 nM
Administration:	Topically applied to the mouse ear 30 min prior to the topical application of TPA
Result:	Significantly inhibited TPA-induced edema formation.

Animal Model:	Male C57Bl/6 mice (9 weeks old, 25–30 g body weight)[4]
Dosage:	30 μmol/kg
Administration:	Intraperitoneal administration, twice a week, 4 weeks (Induce brain lesion by intrastriatal injection of 6-OHDA)
Result:	Induced a partial recovery in the rotational behavior test. Upregulated the expression of TH. Counteract neuronal death and DNA fragmentation in 6-OHDA lesioned mice. increase total GSH and Nrf2 levels in 6-OHDA lesioned mice.

REFERENCES

[1]. Valentina Citi, et al. Anticancer properties of erucin, an H2 S-releasing isothiocyanate, on human pancreatic adenocarcinoma cells (AsPC-1). Phytother Res. 2019 Mar;33(3):845-855.

[2]. A. Melchini, et al. Erucin, a new promising cancer chemopreventive agent from rocket salads, shows anti-proliferative activity on human lung carcinoma A549 cells. Food Chem Toxicol. 2009 Jul;47(7):1430-6.

[3]. Han Jin Cho, et al. Erucin exerts anti-inflammatory properties in murine macrophages and mouse skin: possible mediation through the inhibition of NFκB signaling. Int J Mol Sci. 2013 Oct 15;14(10):20564-77.

[4]. Fabiana Morroni, et al. Comparison of Adaptive Neuroprotective Mechanisms of Sulforaphane and its Interconversion Product Erucin in in Vitro and in Vivo Models of Parkinson's Disease. J Agric Food Chem. 2018 Jan 31;66(4):856-865.

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