Costunolide

Cat. No.: HY-N0036

CAS No.: 553-21-9

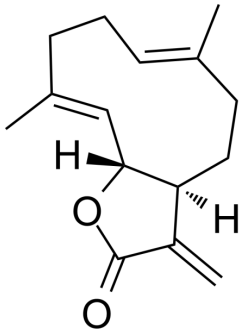
Molecular Formula: $C_{15}H_{20}O_2$

Molecular Weight: 232.32

Target: Apoptosis; Endogenous Metabolite

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage: -20°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

DMSO : ≥ 49 mg/mL (210.92 mM)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3044 mL	21.5220 mL	43.0441 mL
5 mM	0.8609 mL	4.3044 mL	8.6088 mL
10 mM	0.4304 mL	2.1522 mL	4.3044 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: ≥ 2.08 mg/mL (8.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.95 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (8.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Costunolide ((+)-Costunolide) is a naturally occurring sesquiterpene lactone, with antioxidative, anti-inflammatory, antiallergic, bone remodeling, neuroprotective, hair growth promoting, anticancer, and antidiabetic properties. Costunolide can induce cell cycle arrest and apoptosis on breast cancer cells[1][2][3].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Costunolide inhibits the colony formation, migrative and invasive abilities of the H1299 cells in a dose or time dependent

manner[2].
Costunolide (6.7-215.2 μM; 24 hours) inhibits the viability of H1299 cells in a dose-dependent manner, with an IC_{50} of 23.93 μM[2].
Costunolide (12.0-48.0 μM; 48 hours) induces apoptosis in H1299 cells[2].
Costunolide (12-48.0 μM; 6-12 hours) regulates metastasis- and proliferation-associated mRNA expression[2].
Costunolide regulates epithelial-to-mesenchymal transition (EMT)-associated protein expression[2].
Costunolide regulates c-Myc mediated apoptosis signaling and 14-3-3-mediated signaling pathways in breast cancer cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	H1299 cells
Concentration:	6.7 μM, 13.5 μM, 26.9 μM, 107.6 μM, 215.2 μM
Incubation Time:	24 hours
Result:	Inhibited the viability of H1299 cells (MTT assay).

Apoptosis Analysis[2]

Cell Line:	H1299 cells
Concentration:	0 μM, 12.0 μM, 24.0 μM, 48.0 μM
Incubation Time:	48 hours
Result:	Significantly promoted apoptosis at 24.0 μM and 48.0 μM.

RT-PCR[2]

Cell Line:	H1299 cells
Concentration:	0 μM, 12.0 μM, 24.0 μM, 48.0 μM
Incubation Time:	6 hours, 12 hours
Result:	Regulated the metastasis- and proliferation-associated mRNA levels in a dose-dependent manner.

Western Blot Analysis[2]

Cell Line:	H1299 cells
Concentration:	0 μM, 12.0 μM, 24.0 μM, 48.0 μM
Incubation Time:	48 hours
Result:	Significantly inhibited the EMT of H1299 cells.

In Vivo

Costunolide (20 mg/kg; i.p; daily; for 30 days) inhibits breast cancer through c-Myc/p53 and AKT/14-3-3 pathway[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	4 weeks old female BALB/c nude mice, MDA-MB-231 cells xenograft mouse models[3]
Dosage:	20 mg/kg
Administration:	Intraperitoneal injection, daily, for 30 days

Result:	Reduced the expression levels of c-Myc and p-AKT and elevated the expression levels of p53 and p-14-3-3.

CUSTOMER VALIDATION

- Cell Mol Biol Lett. 2019 Aug 14;24:52.
- Molecules. 2020 Jun 19;25(12):2840.
- Gene. 2018 Dec 15;678:261-269.

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REFERENCES

[1]. Dae Yong Kim, et al. Costunolide-A Bioactive Sesquiterpene Lactone with Diverse Therapeutic Potential. Int J Mol Sci. 2019 Jun; 20(12): 2926.

[2]. Minyan Wei, et al. Costunolide induces apoptosis and inhibits migration and invasion in H1299 lung cancer cells. Oncol Rep. 2020 Jun;43(6):1986-1994.

[3]. Zhangxiao Peng, et al. Costunolide and dehydrocostuslactone combination treatment inhibit breast cancer by inducing cell cycle arrest and apoptosis through c-Myc/p53 and AKT/14-3-3 pathway.Sci Rep. 2017; 7: 41254.

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