Cucurbitacin I

Cat. No.: HY-N1405

CAS No.: 2222-07-3

Molecular Formula: $C_{30}H_{42}O_7$

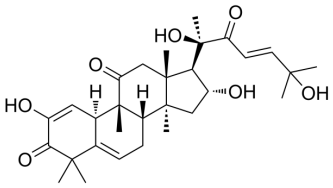
Molecular Weight: 514.65

Target: STAT; JAK

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

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 (194.31 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9431 mL	9.7153 mL	19.4307 mL
5 mM	0.3886 mL	1.9431 mL	3.8861 mL
10 mM	0.1943 mL	0.9715 mL	1.9431 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: ≥ 3 mg/mL (5.83 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (5.83 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3 mg/mL (5.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cucurbitacin I is a natural selective inhibitor of JAK2/STAT3, with potent anti-cancer activity.

IC_{50} & Target

JAK2 | STAT3

In Vitro

Exposure of the COLO205 cells to Cucurbitacin I significantly decreases cell viability. The anticancer activity of Cucurbitacin I is accomplished by downregulating p-STAT3 and MMP-9 expression[1]. PE-induced cell enlargement and upregulation of

	ANF and β-MHC are significantly suppressed by pretreatment of the cardiomyocytes with Cucurbitacin I. Notably, Cucurbitacin I also impaires connective tissue growth factor (CTGF) and MAPK signaling, pro-hypertrophic factors, as well as TGF-β/Smad signaling, the important contributing factors to fibrosis[2]. Incubation of the Seax cell line with the Jak/Stat3 inhibitor Cucurbitacin I result in a time- and concentration-dependent decrease of P-Stat3 and Stat3. In freshly isolated Sz cells (n=3), Cucurbitacin I induces a concentration-dependent decrease in Stat3 expression whereas P-Stat3 is undetectable. Finally, incubation of freshly isolated Sz cells (n=4) with 30 μM Cucurbitacin I for 6 hours induces apoptosis in the large majority (73-91%) of tumor cells[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	No major side effects are noted throughout the study. It is shown that average tumor volumes at the end of the study are as follows: control, 616 mm^3 (±130); CQ, 580 mm^3 (±107); Cucurbitacin I, 346mm^3 (±79); and combination, 220mm^3 (±62). The differences in tumor volume between the Cucurbitacin I and control, combination and control, and combination and Cucurbitacin I arms are significant. Furthermore, combination-treated tumors exhibit a significantly lower average tumor weight at study termination than the control. Moreover, there was no effect on the body weights of mice[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [4]	Mice[4] BALB/c nude (nu/nu) female mice are used. U251 cells (5×10^6 cells in 50 μL of serum-free DMEM) are inoculated subcutaneously into the right flank of 5-week-old female mice after acclimatization for a week. Tumor growth is measured daily with calipers. When the tumors reach a mean volume of 90-120 mm^3, animals are randomized into groups. In the first experiment, 16 mice are randomly assigned to Cucurbitacin I (1 mg/kg/day in 20% DMSO in PBS) or drug vehicle control (20% DMSO in PBS) and dosed intraperitoneally with 100 μL of vehicle or drug once daily for 18 days, whereas, in the second, 20 mice are assigned to four groups. Control animals receive 20% DMSO in PBS vehicle, whereas treated animals are injected with Cucurbitacin I (1 mg/kg/day) in 20% DMSO in PBS, CQ (25 mg/kg/day) in 20% DMSO in PBS, and Cucurbitacin I (1 mg/kg/day) plus CQ (25 mg/kg/day) in 20% DMSO in PBS and dosed intraperitoneally with 100 μL of vehicle or drug once daily for 15 days[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nature. 2023 Sep;621(7980):830-839.
- J Neuroinflammation. 2021 Nov 5;18(1):256.
- Cancer Cell Int. 2023 Sep 2;23(1):191.
- Chem Biol Interact. 21 October 2022, 110226.
- Cell Cycle. 2019 Nov;18(21):3010-3029.

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REFERENCES

[1]. Song J, et al. Cucurbitacin I inhibits cell migration and invasion and enhances chemosensitivity in colon cancer. Oncol Rep. 2015 Apr;33(4):1867-71.

[2]. Moon Hee Jeong, et al. Cucurbitacin I Attenuates Cardiomyocyte Hypertrophy via Inhibition of Connective Tissue Growth Factor (CCN2) and TGF- β/Smads Signalings. PLoS One. 2015 Aug 21;10(8):e0136236.

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

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