Cucurbitacin E

Cat. No.: HY-N0417

CAS No.: 18444-66-1

Molecular Formula: $C_{32}H_{44}O_8$

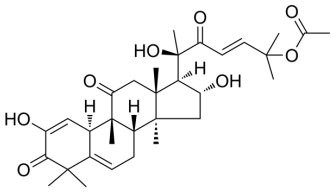
Molecular Weight: 556.69

Target: CDK; Autophagy

Pathway: Cell Cycle/DNA Damage; Autophagy

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 : 50 mg/mL (89.82 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7963 mL	8.9817 mL	17.9633 mL
	5 mM	0.3593 mL	1.7963 mL	3.5927 mL
	10 mM	0.1796 mL	0.8982 mL	1.7963 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 (4.49 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.49 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Cucurbitacin E is a natural compound which from Cucurbitaceae plants. Cucurbitacin E significantly suppresses the activity of the cyclin B1/CDC2 complex.

IC_{50} & Target		
	cyclin B1/CDC2	Autophagy

In Vitro

To explore the antitumor activity of Cucurbitacin E (CuE) against colorectal cancer (CRC) cells, an in vitro study is initiated in which each of the CRC cell lines is exposed to increasing doses of Cucurbitacin E (0, 2.5, 5, and 7.5 μM) over a period of 24 h. The proliferation of the Cucurbitacin E-treated cancer cells is then measured using the MTT method. Cucurbitacin E is shown to induce morphological changes in the primary colon cancer cells. Microscopic observation showed that following exposure to Cucurbitacin E (5 μM) between 6 and 24 h, the primary colon cancer cells underwent a remarkable change in

	morphology. Cucurbitacin E inhibits tumor growth by arresting the cell cycle in the G_2/M phase via GADD45γ gene expression and the blockage of cyclin B1/CDC2 complex in primary CRC cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	A high fat diet mice model of metabolic syndrome (HFD-MetS) is developed to assess the role of Cucurbitacin E (CuE) on body weight and fat tissue biology. Significant decrease in body weights of HFD-MetS mice treated with Cucurbitacin E (0.5mg/kg) are found as compared to HFD-MetS mice treated with vehicle alone. Cucurbitacin E treatment reduces all fat pads weights in HFD-MetS mice. 55% reduction is observed in total fat in mice, after treatment with Cucurbitacin E in comparison to HFD-MetS mice. Abdominal obesity is strongly associated with metabolic syndrome. Central obesity is reduced to 50% after Cucurbitacin E treatment as compared to HFD MetS mice, elucidating the effectiveness of Cucurbitacin E in targeting MetS[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	The colorectal cancer (CRC) cells are seeded into 96-well culture plates at 5000 cells/well. The cells are treated with 0, 2.5, 5, and 7.5 μM Cucurbitacin E for 1-3 days. MTT dye (1 mg/mL) is added to each well for at least 4 h of treatment. The reaction is stopped by the addition of DMSO, and optical density is measured at 540 nm on a multi-well plate reader. Background absorbance of the medium in the absence of cells is subtracted. All samples are assayed in triplicate, and the mean for each experiment is calculated. Results are expressed as a percentage of control, which is considered as 100%. Each assay is carried out in triplicate, and the results are expressed as the mean[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] C57BL/6 male mice are used. The mice are designated as metabolic syndrome mice (HFD-MetS-mice). Briefly, the mice are randomly assigned into two groups according to their diet for 8 weeks (n = 10-12): high fat diet group (HFD) (60% fat, 20% carbohydrate, 20% protein) or the matched low fat, standard diet group (SD) (10% fat, 70% carbohydrate, 20% protein). After eight weeks on high fat diet, the mice with significant obese phenotype and fasting blood glucose levels ≥126 mg/dL are considered MetS mice. The MetS mice are continued on the HFD throughout the study. The MetS mice are then randomly divided into three additional groups, according to the treatment administered by oral gavage for 10 weeks (n=10-12): a low dose 0.25 mg/kg/day of Cucurbitacin E designated as HFD+Cucurbitacin E (L) or high dose 0.5 mg/kg/day of Cucurbitacin E, designated as HFD+Cucurbitacin E (H) or 50 mg/kg/day Orlistat (HFD+Orlistat). Animals on SD are administered 0.5% CMC by oral gavage[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2020 May 20;5(1):56.
- J Transl Med. 2023 Dec 4;21(1):880.
- J Transl Med. 2022 Oct 2;20(1):444.
- Chin Med. 2022 Feb 22;17(1):28.
- Curr Issues Mol Biol. 2023 Oct 7, 45(10), 8138-8151.

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REFERENCES

[1]. Hsu YC, et al. Therapeutic ROS targeting of GADD45γ in the induction of G2/M arrest in primary human colorectal cancer cell lines by cucurbitacin E. Cell Death Dis. 2014 Apr 24;5:e1198.

[2]. Murtaza M, et al. Cucurbitacin E reduces obesity and related metabolic dysfunction in mice by targeting JAK-STAT5 signaling pathway. PLoS One. 2017 Jun 9;12(6):e0178910.

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