Emodin

Cat. No.: HY-14393

CAS No.: 518-82-1

Molecular Formula: $C_{15}H_{10}O_5$

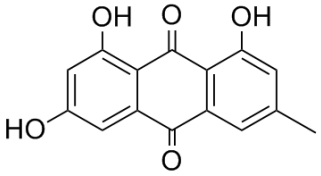
Molecular Weight: 270.24

Target: Casein Kinase; SARS-CoV; Autophagy

Pathway: Cell Cycle/DNA Damage; Stem Cell/Wnt; Anti-infection; Autophagy

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 12.5 mg/mL (46.26 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.7004 mL	18.5021 mL	37.0041 mL
5 mM	0.7401 mL	3.7004 mL	7.4008 mL
10 mM	0.3700 mL	1.8502 mL	3.7004 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 1.25 mg/mL (4.63 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (4.63 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Emodin (Frangula emodin) is a broad-spectrum anticancer agent. Emodin inhibits casein kinase II (CKII) activity with IC_{50} of 2 μM[1]. Emodin blocks the SARS coronavirus (SARS-CoV)[2].

IC_{50} & Target

CKII

2 μM (IC_{50})

In Vitro

Emodin, an anthraquinone derivative, selectively inhibits casein kinase II(CKII), a Ser/Thr kinase, as a competitive inhibitor. Emodin inhibits CKII activity with IC_{50} of 2 μM, which is two to three orders of magnitude lower than those against the other kinases. Enzyme kinetic assays show that Emodin inhibits CKII activity as acompetitive inhibitor against ATP with K_i of 7.2 μ

	M[1]. Emodin is a broad-spectrum inhibitory agent of cancer cells, including leukemia, lung cancer, human tongue squamous cancer, colon cancer, gallbladder cancer, pancreatic cancer, breast cancer, human cervical cancer and hepatic carcinoma cells. Emodin inhibits A549, HepG2, OVCAR-3, HeLa and Madin-Darby Canine Kidney (MDCK) cells with IC_{50} of 19.54, 12.79, 25.82, 12.14 and 5.81 μg/mL. The anticancer mechanisms of Emodin are involved in many biological pathways, such as casein kinase II and ERK1/2[3]. Emodin is applied as a Reactive oxygen species (ROS) generator in combination with cisplatin in T24 and J82 human bladder cancer cells. Emodin kills T24 and J82 cells in the dose-dependent and time-dependent manner, and it is less toxic to HCV-29 cells. The concentration of 20 and 15 μM is selected as appropriate doses for investigating chemotherapeutic sensitivity of T24 and J82 cells at 24 h, respectively[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mice treated with Emodin (50 mg/kg) and Cisplatin (1 mg/kg) have significantly smaller tumors than those from the other groups. In addition, no notable differences on the body weight loss are observed among groups and no obvious necrosis and abnormity are observed in the sections of liver, kidney and heart. These results demonstrate that Emodin/cisplatin co-treatment can significantly suppress tumor growth in vivo with no distinct side effects. Consistent with in vitro experiment, TUNEL assay shows that Emodin/cisplatin combination significantly increased cell apoptosis in xenograft tumors. Emodin/Cisplatin co-treatment group also has lower MRP1 expression than the other groups[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [4]	The T24 human bladder cancer cells, the HCV-29 normal bladder epithelial cells and J82 human bladder cancer cells are are cultured in RPMI 1640 medium supplemented with 10 % fetal bovine serum at 37°C in a humidified atmosphere containing 5 % CO_2. Cells are seeded in 96-well plates with 2×10^4 cells per well. The cells are incubated with Emodin for 24 h at different concentrations (0, 5, 10, 20, 30, 40, 50, 60, 70 μM) and chose the critical concentration (20 μM) treated with cells for 0, 6, 12, 24, 48, 72, 96 h. The cells are incubated with cisplatin for 24 h at different concentrations (0, 0.5, 1.0, 1.5, 2.0, 2.5, 3.0 μg/mL). MTT assay is used to analyze the cell viability. Cells are treated with drugs for 24 h and apoptotic rates are assessed with flow cytometry using AnnexinV-fluorescein isothiocyanate (AnnexinV-FITC)/propidium iodide (PI) kit. Samples are prepared according to the manufacturer's instruction and analyzed by a flow cytometry (FCM) Calibur[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [4]	Mice[4] 3×10^6 T24 cells are harvested, washed, and resuspended in serum-free optimum medium and then injected subcutaneously into 6-week old BALB/c-nu/nu mice (n=8 mice per group). Three days after inoculation, the mice are intraperitoneally administered with PBS, Emodin (50 mg/kg), Cisplatin (1 mg/kg), or Emodin/cisplatin every two days. On day 18, every mouse is sacrificed. After body weight measurement, tumors are isolated, weighted and fixed in 4 % paraformaldehyde (PFA). Hearts, livers and kidneys are stained with Hematoxylin & Eosin to determine the systemic toxicity. Terminal deoxynucleotidyl transferase(TdT)-mediated dUTP nick end label (TUNEL) assay is performed on paraformaldehyde-fixed and paraffin-embedded tumor sections. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2020 Nov 9;gkaa969.
- Acta Pharmacol Sin. 2020 May 12.
- Int Immunopharmacol. 2020, 107020.
- Exp Cell Res. 2020 May 3:112054.
- Am J Transl Res. 2020 May 15;12(5):1851-1861.

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REFERENCES

[1]. Yim H, et al. Emodin, an anthraquinone derivative isolated from the rhizomes of Rheum palmatum, selectively inhibits the activity of casein kinase II as a competitive inhibitor. Planta Med. 1999 Feb;65(1):9-13.

[2]. Xing JY, et al. Antitumor Effects and Mechanism of Novel Emodin Rhamnoside Derivatives against Human Cancer CellsIn Vitro. PLoS One. 2015 Dec 18;10(12):e0144781.

[3]. Li X, et al. Emodin enhances cisplatin-induced cytotoxicity in human bladder cancer cells through ROS elevation and MRP1 downregulation. BMC Cancer. 2016 Aug 2;16:578.

[4]. Tin-Yun Ho, et al. Emodin Blocks the SARS Coronavirus Spike Protein and Angiotensin-Converting Enzyme 2 Interaction. Antiviral Res. 2007 May;74(2):92-101.

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