Emodin

Cat. No.: HY-14393

CAS No.: 518-82-1

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

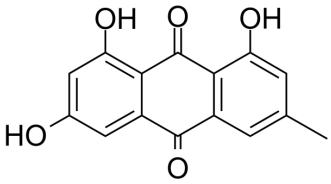
Molecular Weight: 270.24

Target: SARS-CoV; Casein Kinase; Autophagy

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

Acetone : 10.87 mg/mL (40.22 mM; Need ultrasonic)
DMSO : 5.41 mg/mL (20.02 mM; Need ultrasonic)
Ethanol : < 1 mg/mL (ultrasonic) (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: 0.5% MC >> 0.5% Tween-80
   Solubility: 10 mg/mL (37.00 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 3.33 mg/mL (12.32 mM); Suspened solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Emodin (Frangula emodin), an anthraquinone derivative, is an anti-SARS-CoV compound. Emodin blocks the SARS coronavirus spike protein and angiotensin-converting enzyme 2 (ACE2) interaction[1]. Emodin inhibits casein kinase-2 (CK2). Anti-inflammatory and anticancer effects[2]. Emodin is a potent selective 11β-HSD1 inhibitor with the IC_{50} of 186 and 86 nM for human and mouse 11β-HSD1, respectively. Emodin ameliorates metabolic disorder in diet-induced obese mice[3].

IC_{50} & Target				
	SARS-CoV	CK2α Wild-type 1.4 μM (IC_{50}, at ATP concentration is 10 μM)	CK2α Wild-type 5.9 μM (IC_{50}, at ATP concentration is 50 μM)	mouse 11β-HSD1 86 nM (IC_{50})

	human 11β-HSD1 186 nM (IC_{50})

In Vitro

Emodin (10-400 μM) blocks the binding of S protein to ACE2 in a dose-dependent manner with the IC_{50} value of 200 μM[1].
Emodin (5-50 μM) inhibits the S protein-pseudotyped retrovirus infectivity in a dose-dependent manner. Emodin blocks the SARS-CoV S protein binding to Vero E6 cells[1].
Emodin inhibits casein kinase-2 (CK2) with IC_{50}s of 5.9, 30.0, and 7.1 μM for CK2α Wild-type, Ile174Ala mutant, and His160Ala mutant at ATP concentration is 50 μM, respectively. The IC_{50}s are 1.40 and 38.00 μM for CK2α Wild-type, and Val66Ala mutant at ATP concentration is 10 μM[2].
Emodin exhibits low inhibitory activity against mouse and human 11β-hydroxysteroid dehydrogenase type 2 (11β-HSD2), with an IC_{50} higher than 1 mM, indicating that Emodin is more than 5000-fold selective for the human and mouse 11β-HSD1 enzymes over the type 2 isoenzyme[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	Vero E6 cells transfected with the plasmid encoding ACE2
Concentration:	0, 5, 25, 50 μM
Incubation Time:	24 hours
Result:	Vero cells treated with 50 μM remained 82.4±3.8% viability, the anti-SARS-CoV activity was not due to toxicity.

In Vivo

Emodin (single oral administration of 100 or 200 mg/kg) inhibits 11β-HSD1 activity in normal C57BL/6J male mice[3].
Emodin (100 mg/kg; oral administration; b.i.d.) improves insulin sensitivity and lipid metabolism, and lowers blood glucose and hepatic PEPCK, and glucose-6-phosphatase mRNA in diet-induced obese (DIO) mice[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6J male mice[3]
Dosage:	100 or 200 mg/kg
Administration:	Acute administered p.o. ; Two hours later, the mice were killed by cervical dislocation,
Result:	Significantly inhibited liver 11β-HSD1 enzymatic activity by 17.6 and 31.3% and mesenteric fat 11β-HSD1 enzymatic activity by 21.5 and 46.7% at 100 or 200 mg/kg, respectively.

Animal Model:	DIO mice (C57BL/6J male mice were fed a formulated research diet)[3]
Dosage:	100 mg/kg
Administration:	Oral gavage; twice per day; for 35 days
Result:	Reduced fasting glucose concentrations to 77.2% of the vehicle control mice after 7 days of treatment, and these remained significantly lower throughout the treatment period. Exhibited a significant reduction in blood glucose levels at all time-points following oral glucose challenge after 24 days of treatment. Evoked a significantly greater reduction in blood glucose values 40 and 90 min after insulin injection after 28 days of treatment. The serum insulin level was also significantly reduced, to 66.2% of control mice, after 35 days of treatment. Improved the lipid profiles. The serum triglyceride and total cholesterol levels were significantly reduced by 19.3 and 12.5% after 35 days of treatment, respectively.

	Caused a 22.7% reduction of non-esterified free fatty acid (NEFA) level. Lowered body weight and appetite from day 18 of the treatment; their body weights were reduced by 13.9% at the end of treatment.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Jan 8;49(D1):D1113-D1121.
- Fertil Steril. 2020 May;113(5):1067-1079.e5.
- Acta Pharmacol Sin. 2021 Jan;42(1):108-114.
- Int Immunopharmacol. 2020 Dec 23;91:107277.
- Int Immunopharmacol. 2020 Nov;88:107020.

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REFERENCES

[1]. 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.

[2]. Ying Feng, et al. Emodin, a natural product, selectively inhibits 11beta-hydroxysteroid dehydrogenase type 1 and ameliorates metabolic disorder in diet-induced obese mice. Br J Pharmacol. 2010 Sep;161(1):113-26.

[3]. Stefania Sarno, et al. Toward the rational design of protein kinase casein kinase-2 inhibitors. Pharmacol Ther. Feb-Mar 2002;93(2-3):159-68.

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

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