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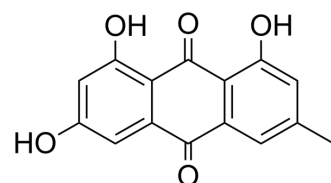
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Emodin

Cat. No.:	HY-14393
CAS No.:	518-82-1
Molecular Formula:	C ₁₅ H ₁₀ O ₅
Molecular Weight:	270.24
Target:	Casein Kinase; Autophagy; SARS-CoV; 11β-HSD
Pathway:	Cell Cycle/DNA Damage; Stem Cell/Wnt; Autophagy; Anti-infection; Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 1 year -20°C 6 months



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	<div>Solvent Concentration</div> <div>Mass</div>	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); Suspended 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)	CK2α Wild-type 5.9 μM (IC ₅₀ , at ATP)	mouse 11β-HSD1 86 nM (IC ₅₀)

		concentration is 10 μ M)	concentration is 50 μ M)	
	human 11 β -HSD1 186 nM (IC ₅₀)			
In Vitro	Emodin (10-400 μ M) blocks the binding of S protein to ACE2 in a dose-dependent manner with the IC ₅₀ value of 200 μ M ^[1] . Emodin (5-50 μ M) 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 μ M for CK2 α Wild-type, Ile174Ala mutant, and His160Ala mutant at ATP concentration is 50 μ M, respectively. The IC ₅₀ s are 1.40 and 38.00 μ M for CK2 α Wild-type, and Val66Ala mutant at ATP concentration is 10 μ M ^[2] . Emodin exhibits low inhibitory activity against mouse and human 11 β -hydroxysteroid dehydrogenase type 2 (11 β -HSD2), with an IC ₅₀ higher than 1 mM, indicating that Emodin is more than 5000-fold selective for the human and mouse 11 β -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 μ M		
	Incubation Time:	24 hours		
	Result:	Vero cells treated with 50 μ M remained 82.4 \pm 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 11 β -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 11 β -HSD1 enzymatic activity by 17.6 and 31.3% and mesenteric fat 11 β -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.
- Acta Pharmacol Sin. 2021 Jan;42(1):108-114.
- Phytother Res. 2024 Jan 10.
- Fertil Steril. 2020 May;113(5):1067-1079.e5.
- Int Immunopharmacol. 2020 Dec 23;91:107277.

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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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