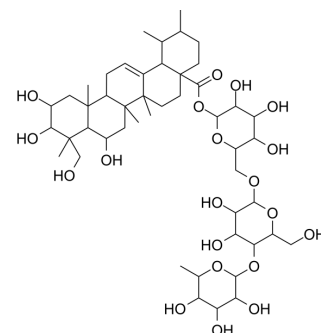


Madecassoside

Cat. No.:	HY-N0568		
CAS No.:	34540-22-2		
Molecular Formula:	C ₄₈ H ₇₈ O ₂₀		
Molecular Weight:	975.12		
Target:	Endogenous Metabolite; Apoptosis; Autophagy; Keap1-Nrf2; p38 MAPK; Caspase		
Pathway:	Metabolic Enzyme/Protease; Apoptosis; Autophagy; NF-κB; MAPK/ERK Pathway		
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 (102.55 mM; Need ultrasonic)
H₂O : 33.33 mg/mL (34.18 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent Concentration	Mass		
		1 mg	5 mg	10 mg
	1 mM	1.0255 mL	5.1276 mL	10.2551 mL
	5 mM	0.2051 mL	1.0255 mL	2.0510 mL
	10 mM	0.1026 mL	0.5128 mL	1.0255 mL

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

In Vivo

- Add each solvent one by one: PBS
Solubility: 25 mg/mL (25.64 mM); Clear solution; Need ultrasonic
- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (2.56 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Madecassoside is a pentacyclic triterpene isolated from *Centella asiatica* and has anti-inflammatory properties. Antioxidant and anti-aging effects. Madecassoside is a pentacyclic triterpene isolated from *Centella asiatica*. Madecassoside is orally active and has inhibitory properties against inflammation, oxidation, apoptosis and autophagy. Madecassosid inhibits activities of p38 MAPK and NF-κB^{[5][6]}, exhibits an anti-apoptotic property, activates Nrf2 expression to reduce the

neurotoxicity^[10]. Madecassoside can be used in endocrine diseases, cardiovascular diseases, skin diseases and other diseases.

In Vitro

Madecassosid (30, 100 µmol/L, 12 h) increases cell viability of oxidative injured human umbilical vein endothelial cells (HUVECs)^[5].

Madecassosid (10-100 µmol/L, 12 h) inhibits phosphorylation of p38 MAPK and activity of Caspase-3, and therefore exhibits an antiapoptotic activity^[5].

Madecassosid (10-100 µg/L) exhibits antioxidative activity against melanocyte dendrites retraction, maintaining mitochondrial membrane potential and Ca²⁺ homeostasis^[7] /sup>.</sup>

Madecassosid (30 µM) improves Insulin secretion by increasing expressions of p-IRS1, Akt and p-Akt under glucotoxic conditions^[8].

Madecassosid (10 µM, 24 h) prevents inflammation and autophagy of neuronal cells induced by Aβ₂₅₋₃₅ through the class III PI3K/Beclin-1/Bcl-2 pathway^[9].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay^[5]

Cell Line:	HUVECs
Concentration:	10, 30, 100 µmol/L
Incubation Time:	12 h
Result:	Increased cell viability to 68.9% and 78.3%, with concentration of 30 and 100 µmol/L, respectively.

Western Blot Analysis^[5]

Cell Line:	HUVECs
Concentration:	10, 30, 100 µmol/L
Incubation Time:	12 h
Result:	Inhibited phosphorylation in p38 MAPK.

In Vivo

Madecassosid (6, 12, 24 mg/kg, i.v.) resolves neurological deficit and ameliorates neuronal apoptosis after focal cerebral ischemia reperfusion^[6].

Madecassosid (6, 12, 24 mg/kg, i.v.) inhibits activity of NF-κB and so prevents the brain injury^[6].

Madecassosid (120 mg/kg, i.g.) reduces LPS-induced neurotoxicity and enhances heme oxygenase proteins via upregulation of Nrf2 in LPS-stimulated neurotoxicity^[10].

Madecassosid (10-40 mg/kg, p.o.) ameliorates oxidative damage and inflammation after bleomycin (BLM) instillation, inhibits TGF-β1 overexpression and collagen synthesis, improves collagen degradation^[11].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Focal cerebral ischemia reperfusion injury in Sprague Dawley rats ^[6]
Dosage:	6, 12, 24 mg/kg
Administration:	Intravenous injection
Result:	Reduced neurological deficit, infarct area, brain damage and neuronal apoptosis.

Animal Model:	Lipopolysaccharide-induced neurotoxicity in Sprague-Dawley rats ^[10]
Dosage:	30-120 mg/kg for 14 days
Administration:	Intraperitoneal injection
Result:	Reduced LPS-induced cognitive impairment and inflammatory cytokine, promoted Nrf2

pathway at concentration of 120 mg/kg.

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

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