Chicoric acid

Cat. No.:	HY-N0457		
CAS No.:	6537-80-0		
Molecular Formula:	$C_{22}H_{18}O_{12}$		
Molecular Weight:	474.37		
Target:	Reactive O>	kygen Spe	ecies; Apoptosis
Pathway:	Immunolog	gy/Inflam	mation; Metabolic Enzyme/Protease; NF-кB; Apoptosis
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month

SOLVENT & SOLUBILITY

Preparing Stock Solutions	Solvent Mass	1 mg	5 mg	10 mg	
	Concentration	2 1001	10 5402 ml	21.0000 ml	
		1 mM	2.1081 mL	10.5403 mL	21.0806 mL
	5 mM	0.4216 mL	2.1081 mL	4.2161 mL	
		10 mM	0.2108 mL	1.0540 mL	2.1081 mL
	Please refer to the so	lubility information to select the app	propriate solvent.		

BIOLOGICAL ACTIVITY Description Chicoric acid (Cichoric acid), an orally active dicaffeyltartaric acid, induces reactive oxygen species (ROS) generation. Chicoric acid inhibits cell viability and induces mitochondria-dependent apoptosis in 3T3-L1 preadipocytes through ROS-mediated PI3K/Akt and MAPK signaling pathways. Chicoric acid increases glucose uptake, improves insulin resistance, and attenuates glucosamine-induced inflammation. Chicoric acid has antidiabetic properties and antioxidant, anti-inflammatory effects^{[1][2][3]}. In Vitro Chicoric acid (Cichoric acid; 10-200 µM; for 24, 48, and 72 h) causes a dose- and time-dependent decrease in cell viability^[1]. Chicoric acid (100 µM; 48 h) induces apoptosis through caspase-3-dependent pathway^[1]. Chicoric acid (100 µM; 48 h) decreases the protein level of p-Akt^[1]. Chicoric acid (25, 50, 100 µM; for 24 hours) dramatically improves glucose uptake in a dose-dependent manner, and Chicoric acid further enhances insulin-induced (100 nM; 30 min) glucose uptake by 57.7% in HepG2 cells^[2].



Chicoric acid (100 μ M; for 24 hours) restores glucosamine-induced impairment of GLUT2 translocation through activating PI3K/Akt pathway in HepG2 cells^[2].

Chicoric acid (100 μ M) has no effects on HepG2 cell viability^[2].

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

Cell Viability Assay^[1]

Cell Line:	3T3-L1 preadipocytes
Concentration:	10-200 μΜ
Incubation Time:	24, 48, and 72 hours
Result:	Had no effect on the viability of 3T3-L1 preadipocytes with 10-50 μM for 24 h, but significantly decreased cell viability with 100 μM and 200 $\mu M.$

Apoptosis Analysis^[1]

Cell Line:	3T3-L1 preadipocytes
Concentration:	100 μΜ
Incubation Time:	48 hours
Result:	Demonstrated typical characteristics of apoptosis such as cell shrinkage, chromatin condensation, and the increased permeability of cell membranes after DAPI and AO/EB staining.

Western Blot Analysis^[1]

Cell Line:	3T3-L1 preadipocytes
Concentration:	100 μΜ
Incubation Time:	48 hours
Result:	Decreased the protein level of p-Akt in a dose- and time-dependent manner. The protein level of total Akt was not affected

In Vivo

Chicoric acid (Cichoric acid; 60 mg/kg/day; drinking water for 4 weeks) inhibits pancreas apoptosis and adjusts islet function in diabetic mice, leading to an increase in insulin generation and secretion in C57BL/6J mice with Streptozotocin (STZ; 50 mg/kg; ip; for consecutive 5 days)^[3].

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Animal Model:	C57BL/6J mice with STZ (50 mg/kg; ip; for consecutive 5 days) ^[3]
Dosage:	60 mg/kg
Administration:	Drinking water; daily; for 4 weeks
Result:	Inhibited pancreas apoptosis and adjusted islet function in diabetic mice, leading to an increase in insulin generation and secretion. Regulated mitochondrial biogenesis, glycogen synthesis, and inhibited inflammation via activating antioxidant responses.
	Showed a remarkable increase in body weight starting at week 7.

REFERENCES

[1]. Haifang Xiao, et al. Chicoric acid induces apoptosis in 3T3-L1 preadipocytes through ROS-mediated PI3K/Akt and MAPK signaling pathways. J Agric Food Chem. 2013 Feb 20;61(7):1509-20.

[2]. Di Zhu, et al. Cichoric Acid Reverses Insulin Resistance and Suppresses Inflammatory Responses in the Glucosamine-Induced HepG2 Cells. J Agric Food Chem. 2015 Dec 30;63(51):10903-13.

[3]. Di Zhu, et al. Cichoric acid improved hyperglycaemia and restored muscle injury via activating antioxidant response in MLD-STZ-induced diabetic mice. Food Chem Toxicol. 2017 Sep;107(Pt A):138-149.

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

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