Coenzyme Q10

Cat. No.: HY-N0111

CAS No.: 303-98-0

Molecular Formula: $C_{59}H_{90}O_4$

Molecular Weight: 863.34

Target: Endogenous Metabolite; Reactive Oxygen Species; Ferroptosis

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Apoptosis

Storage: -20°C, sealed storage, away from moisture and light

* The compound is unstable in solutions, freshly prepared is recommended.

SOLVENT & SOLUBILITY

In Vitro

DMF : 20 mg/mL (23.17 mM; ultrasonic and warming and heat to 60°C)

Ethanol : 2.5 mg/mL (2.90 mM; ultrasonic and warming and heat to 60°C)

H_2O : < 0.1 mg/mL (insoluble)

DMSO : < 1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble or slightly soluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.1583 mL	5.7915 mL	11.5829 mL
5 mM	0.2317 mL	1.1583 mL	2.3166 mL
10 mM	0.1158 mL	0.5791 mL	1.1583 mL

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

In Vivo

1. Add each solvent one by one: 20% HP-β-CD in saline
   Solubility: 13.33 mg/mL (15.44 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMF >> 90% (20% SBE-β-CD in saline)
   Solubility: 3 mg/mL (3.47 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Coenzyme Q10 is an essential cofactor of the electron transport chain and a potent antioxidant agent.

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

Coenzyme Q10 is an obligatory member of the respiratory chain in the mitochondria of all cells. Therefore, it is an essential ingredient in the formation of adenosine triphosphate (ATP), the source of energy in most cellular processes. Coenzyme Q10 is located in the mitochondria, lysosomes, and Golgi and plasma membranes, and provides an antioxidant action either by direct reaction with free radicals or by regeneration of tocopherol and ascorbate from their oxidised state[1]. Coenzyme Q10

	is a popular dietary supplement because of its recognition by the public as an important nutrient in supporting human health. The rationale for the use of Coenzyme Q10 as a therapeutic agent in several cardiovascular and degenerative neurologic and neuromuscular diseases is based upon its fundamental role in mitochondrial function and cellular bioenergetics[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Because of its hydrophobicity and large molecular weight, absorption of dietary Coenzyme Q10 is slow and limited. In the case of dietary supplements, solubilized Coenzyme Q10 formulations show enhanced bioavailability. The Tmax is around 6 h, with an elimination half-life of about 33 h. The reference intervals for plasma Coenzyme Q10 range from 0.40 to 1.91 mM in healthy subjects. With Coenzyme Q10 supplements there is reasonable correlation between increase in plasma Coenzyme Q10 and ingested dose up to a certain point. Animal data show that Coenzyme Q10 in large doses is taken up by all tissues including heart and brain mitochondria[2]. In 12-month-old rats administration of coenzyme Q10 results in significant increases in cerebral cortex mitochondrial concentrations of coenzyme Q10. Oral administration of coenzyme Q10 markedly attenuates striatal lesions produced by systemic administration of 3-nitropropionic acid and significantly increases life span in a transgenic mouse model of familial amyotrophic lateral sclerosis[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [3]	Rats[3] The effects of oral administration of coenzyme Q10 on 3-NP-induced striatal lesions are examined in 300- to 350-g rats (n=5-10). Animals receive either rat chow supplemented with coenzyme Q10 at 200 mg/kg or unsupplemented rat chow. After 1 week they are treated with 3-NP at a dose of 10 mgykg i.p. twice a day until either a control or treated animal became symptomatic with hindlimb dystonia, and then sacrificed in pairs[3]. Mice[3] To determine whether coenzyme Q10 exerts neuroprotective effects in a transgenic mouse model of a human neurodegenerative disorder, coenzyme Q10 (200 mg/kg) is administered orally to 16 transgenic mice overexpressing a human CuyZn superoxide dismutase (SOD1) mutation, as compared with 13 animals that receive unsupplemented rat chow. The mice used are the G1 line, which expresses high levels of human SOD with the G93A mutation. Treatment is started at 50 days after birth. Treatment is continued until mice reach end-stage disease[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Commun Biol. 2023 Nov 25;6(1):1201.

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REFERENCES

[1]. Yang YK, et al. Coenzyme Q10 treatment of cardiovascular disorders of ageing including heart failure, hypertension and endothelial dysfunction. Clin Chim Acta. 2015 Oct 23;450:83-9.

[2]. Bhagavan HN, et al. Coenzyme Q10: absorption, tissue uptake, metabolism and pharmacokinetics. Free Radic Res. 2006 May;40(5):445-53.

[3]. Matthews RT, et al. Coenzyme Q10 administration increases brain mitochondrial concentrations and exerts neuroprotective effects. Proc Natl Acad Sci U S A. 1998 Jul

21;95(15):8892-7.

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