Coenzyme Q9

Cat. No.: HY-101415

CAS No.: 303-97-9

Molecular Formula: $C_{54}H_{82}O_4$

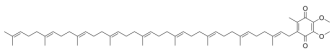
Molecular Weight: 795.23

Target: Endogenous Metabolite; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

Ethanol : 12.5 mg/mL (15.72 mM; Need ultrasonic and warming)
DMSO : 2 mg/mL (2.51 mM; ultrasonic and warming and heat to 80°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2575 mL	6.2875 mL	12.5750 mL
5 mM	0.2515 mL	1.2575 mL	2.5150 mL
10 mM	0.1257 mL	0.6287 mL	1.2575 mL

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

BIOLOGICAL ACTIVITY

Description

Coenzyme Q9 (Ubiquinone Q9), the major form of ubiquinone in rodents, is an amphipathic molecular component of the electron transport chain that functions as an endogenous antioxidant. Coenzyme Q9 attenuates the diabetes-induced decreases in antioxidant defense mechanisms. Coenzyme Q9 improves left ventricular performance and reduces myocardial infarct size and cardiomyocyte apoptosis[1][2].

IC_{50} & Target

Human Endogenous Metabolite

In Vivo

Coenzyme Q9 (5 mg/kg; p.o.; once a day for 4 weeks) reduces myocardial ischemia/reperfusion injury[2].
CoQ_{10} and CoQ_9 are components of themitochondrial respiratory chain in mammals and can regulate some mitochondrial proteins/functions. Soybean, corn, and rapeseed oils are very rich sources of CoQ_{10}, whereas CoQ_9 has been found in high concentrations in corn oil[3].
The lack of a functional CoQ_9 protein in homozygous CoQ_9 mutant (CoQ_9(X/X)) mice causes a severe reduction in the CoQ_7 protein and a widespread CoQ deficiency and accumulation of demethoxyubiquinone. The deficit in CoQ induces a brain-specific impairment of mitochondrial bioenergetics performance, a reduction in respiratory control ratio, ATP levels and

ATP/ADP ratio and specific loss of respiratory complex I. These effects lead to neuronal death and demyelinization with severe vacuolization and astrogliosis in the brain of CoQ_9 (X/X) mice that consequently die between 3 and 6 months of age[4].

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

Animal Model:	Male Hartley guinea pigs of about 350-400 g body weight[2]
Dosage:	5 mg/kg of body weight
Administration:	P.o.; once a day for 4 weeks
Result:	Nutritional supplementation of CoQ9 can reduce myocardial ischemia/reperfusion injury to the same extent as CoQ10.

REFERENCES

[1]. Lekli I, et al. Coenzyme Q9 provides cardioprotection after converting into coenzyme Q10. J Agric Food Chem. 2008 Jul 9;56(13):5331-7.

[2]. Venegas C, et al. Determination of coenzyme Q10, coenzyme Q9, and melatonin contents in virgin argan oils: comparison with other edible vegetable oils. J Agric Food Chem. 2011 Nov 23;59(22):12102-8.

[3]. García-Corzo L, et al. Dysfunctional Coq9 protein causes predominant encephalomyopathy associated with CoQ deficiency. Hum Mol Genet. 2013 Mar 15;22(6):1233-48.

[4]. Wold LE, et al. Insulin-like growth factor I (IGF-1) supplementation prevents diabetes-induced alterations in coenzymes Q9 and Q10. Acta Diabetol. 2003;40(2):85-90.

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