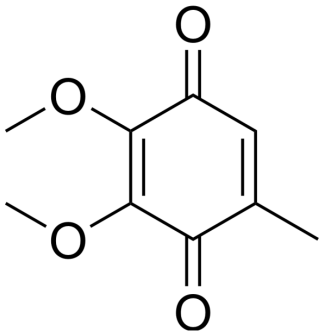
Coenzyme Q0



Cat. No.: HY-W016412

CAS No.: 605-94-7

Molecular Formula: $C_9H_{10}O_4$

Molecular Weight: 182.18

Target: Apoptosis; Autophagy; EGFR; Akt; mTOR; Caspase; Bcl-2 Family; Reactive Oxygen Species; PARP; COX; NO Synthase; TNF Receptor; Interleukin Related; MMP; NF-κB

Pathway: Apoptosis; Autophagy; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; PI3K/Akt/mTOR; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Cell Cycle/DNA Damage; Epigenetics

Storage:

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

SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (274.45 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	5.4891 mL	27.4454 mL	54.8908 mL
	5 mM	1.0978 mL	5.4891 mL	10.9782 mL
	10 mM	0.5489 mL	2.7445 mL	5.4891 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1.67 mg/mL (9.17 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.67 mg/mL (9.17 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Coenzyme Q0 (CoQ0) is a potent, oral active ubiquinone compound can be derived from Antrodia cinnamomea. Coenzyme Q0 induces apoptosis and autophagy, suppresses of HER-2/AKT/mTOR signaling to potentiate the apoptosis and autophagy mechanisms. Coenzyme Q0 regulates NFκB/AP-1 activation and enhances Nrf2 stabilization in attenuation of inflammation and redox imbalance. Coenzyme Q0 has anti-angiogenic activity through downregulation of MMP-9/NF-κB and upregulation of HO-1 signaling[1][2][3].

In Vitro

Coenzyme Q0 (0-40 μM; 24 h) and inhibits viability and growth of human ovarian carcinoma cells[1].

Coenzyme Q0 (CoQ0) (0-30 μM; 24 h; SKOV-3 cells) has anti-proliferative activity through induction of G2/M cell-cycle arrest and reduction of cell-cycle regulatory proteins[1].
Coenzyme Q0 (CoQ0) (0-30 μM; 0-30 min; SKOV-3 cells) increases intracellular ROS levels to promote SKOV-3 cell death[1].
Coenzyme Q0 (CoQ0) (0-30 μM; 24 h; SKOV-3 cells) induces autophagy by increase accumulation of LC3-II, GFP-LC3 puncta, AVOs formation and Beclin-1/Bcl-2 dysregulation[1].
Coenzyme Q0 (CoQ0) (0-30 μM; 24 h; SKOV-3 cells) induces apoptosis by mitochondrial (caspase-3, PARP and Bax/Bcl-2 dysregulation) and ER stress (caspase-12 and Hsp70) signals[1].
Coenzyme Q0 (CoQ0) (30 μM; 24 h; SKOV-3 cells) suppresses of HER-2/AKT/mTOR signaling to potentiate the apoptosis and autophagy mechanisms[1].
Coenzyme Q0 (CoQ0) (0-10 μM; 0.5-18 h; RAW264.7 cells) regulates NFκB/AP-1 activation and enhances Nrf2 stabilization[2].
Coenzyme Q0 (CoQ0) (5 μM; 0-12 h; EA.hy 926 cells) has anti-angiogenic activity in EA.hy 926 cells[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	SKOV-3, A2780 and A2870/CP70 cells
Concentration:	0, 10, 20, 30 and 40 μM
Incubation Time:	24 hours
Result:	Decreased viability with the IC_{50} values of 26.6 μM, 27.3 μM and 28.4 μM for SKOV-3, A2780 and A2870/CP70 cells, respectively.

Cell Cycle Analysis[1]

Cell Line:	SKOV-3, A2780 and A2870/CP70 cells
Concentration:	0, 10, 20 and 30 μM
Incubation Time:	24 hours
Result:	Arrested cell cycle at G2/M phase and reduced cell-cycle proteins in SKOV-3 cells.

Apoptosis Analysis[1]

Cell Line:	SKOV-3, A2780 and A2870/CP70 cells
Concentration:	0, 5, 15 and 30 μM
Incubation Time:	24 hours
Result:	Promoted the conversion of LC3–1 to LC3-II and increased the LC3-II accumulation. Increased Bax/Bcl-2 ratio in a dose-dependent manner.

Apoptosis Analysis[1]

Cell Line:	SKOV-3 cells
Concentration:	0, 10, 20 and 30 μM
Incubation Time:	24 hours
Result:	Had the percentage of early apoptotic cells are 25.1%, 34% and 36% for 10, 20 and 30 μM, respectively.

Western Blot Analysis[1]

Cell Line:	SKOV-3 cells

Concentration:	0, 5, 15 and 30 μM
Incubation Time:	24 hours
Result:	Activated of caspase-3 and cleavaged of PARP. Increased the expressions of caspase-12, HSP-70 and Bax in a dose-dependent manner, decreased the expressions of Bcl-2.

Western Blot Analysis[1]

Cell Line:	SKOV-3 cells
Concentration:	30 μM
Incubation Time:	24 hours
Result:	Decreased the phosphorylated HER-2 (Y1221) levels, p-AKT (Ser473) and p-mTOR (S2448) levels.

Western Blot Analysis[2]

Cell Line:	RAW264.7 cells
Concentration:	0, 2.5, 5 and 10 μM
Incubation Time:	0.5-18 hours
Result:	Inhibited iNOS/COX-2 protein expressions with reductions of NO, PGE2, TNF-α and IL-1β secretions.

Western Blot Analysis[3]

Cell Line:	EA.hy 926 cells
Concentration:	5 μM
Incubation Time:	0, 1, 3, 6 and 12 hours
Result:	Increased expressions of heme oxygenase-1 (HO-1) and γ-glutamylcysteine synthetase (γ-GCLC), inhibits protein expressions of matrix metalloproteinase-9 (MMP-9), reduces TNF-α-induced nuclear translocation and transcriptional activation of nuclear factor-κB (NF-κB).

In Vivo

Coenzyme Q0 (CoQ0) (1.5 and 2.5 mg/kg; i.p.; once every four days, for 52 d) suppresses tumor growth in SKOV-3 xenografted nude mice[1].

Coenzyme Q0(CoQ0) (5 mg/kg; p.o.; for 4 h) has anti-inflammatory activities through Nrf2 activation and NFκB inhibition in liver and spleen of LPS-treated mice[2].

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Animal Model:	SKOV-3 xenografted nude mice[1]
Dosage:	1.5 and 2.5 mg/kg
Administration:	Intraperitoneal injection; Once every four days, for 52 days
Result:	Inhibited the tumor growth at 1.5 and 2.5 mg/kg.

Animal Model:	LPS-treated female FVB mice[2]
Dosage:	5 mg/kg
Administration:	Oral administration; for 4 hours
Result:	Down-regulates inflammatory genes in liver and spleen tissues of LPS injected mice.

CUSTOMER VALIDATION

- Emerg Microbes Infect. 2024 Jan 2:2300525.

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REFERENCES

[1]. Yang HL, et, al. Coenzyme Q0 regulates NFκB/AP-1 activation and enhances Nrf2 stabilization in attenuation of LPS-induced inflammation and redox imbalance: Evidence from in vitro and in vivo studies. Biochim Biophys Acta. 2016 Feb;1859(2):246-61.

[2]. Yang HL, et, al. Coenzyme Q0 regulates NFκB/AP-1 activation and enhances Nrf2 stabilization in attenuation of LPS-induced inflammation and redox imbalance: Evidence from in vitro and in vivo studies. Biochim Biophys Acta. 2016 Feb;1859(2):246-61.

[3]. Yang HL, et, al. Anti-angiogenic properties of coenzyme Q0 through downregulation of MMP-9/NF-κB and upregulation of HO-1 signaling in TNF-α-activated human endothelial cells. Biochem Pharmacol. 2015 Nov 1;98(1):144-56.

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

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