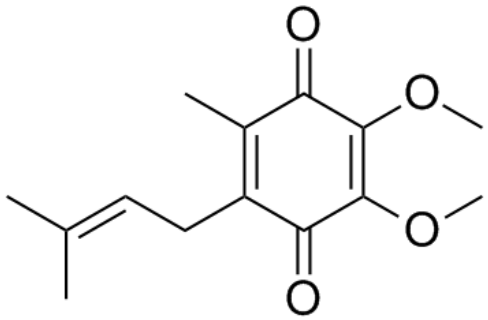
Product Data Sheet

Product Name: Ubiquinone-1
Cat. No.: GC30723

Chemical Properties

Cas. No.	727-81-1		
SMILES	O=C1C(OC)=C(OC)C(C(C)=C1C/C=C(C)\C)=O		
Formula	$C_{14}H_{18}O_4$	M.Wt	250.29
Solubility	DMF: 10 mg/ml,Ethanol: 10 mg/ml	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines rat pulmonary microvascular endothelial cells (PMVEC)

Product Data Sheet

Preparation Method	Rat pulmonary microvascular endothelial cells (PMVEC) were grown in DMEM containing 10% FCS, 100U/ml penicillin, 100μg/ml streptomycin, and 30mg/ml L-glutamine on either 6-well gelatin-coated (2% w/w) plastic cell culture dishes, on gelatin-coated (2% vol/wt) Biosilon (polystyrene) microcarrier beads (230mm mean diameter; 160cm^2/ml beads) or on commercially available collagen-coated transwell inserts. Confluent monolayers of PMVEC in individual wells of 6-well culture dishes (for ATP measurements), or on microcarrier beads (for Δψm or O_2 consumption measurements) were washed free of culture medium three times in Hanks balanced salt solution containing 10mM 4-(2-hydroxyethyl)-1-piperazineethanesulfonic acid, pH 7.4, warmed to 37°C (HBSS/Hepes). The HBSS/Hepes contained 0.2mM glucose plus 5.3mM mannitol (low glucose). Experimental treatments were administered in the HBSS/Hepes at 37°C over a 6h time course. The concentrations of each substance, used either alone or in combination, were rotenone (0.5μM), Ubiquinone-1 (10μM). PMVEC ATP levels were measured using the ATPlite kit. Mitochondrial membrane potential (Δψm) measurements were made using TMRE as the Δψm sensitive dye.
Reaction Conditions	10μM; 6h
Applications	Treatment of rat pulmonary microvascular endothelial cells (PMVEC) cultured in low glucose medium with Ubiquinone-1 prevented ATP depletion and mitochondrial membrane depolarization caused by the complex I inhibitor rotenone.

References:

[1] Bongard RD, Townsley MI, Merker MP. The effects of mitochondrial complex I blockade on ATP and permeability in rat pulmonary microvascular endothelial cells in culture (PMVEC) are overcome by coenzyme Q1 (CoQ1). *Free Radic Biol Med*. 2015;79:69-77.

Background

Ubiquinone-1 is an intermediate in the biosynthetic pathway of coenzyme Q [1]. Ubiquinone-1 is

mainly reduced to ubiquinol by the action of complex I and II in the mitochondrial respiratory chain, acting as an electron carrier in the inner mitochondrial membrane to mediate electron transfer within the aerobic respiratory chain, promote oxidative phosphorylation and generate ATP and energy for cells[2]. Ubiquinone-1 also plays a role in antioxidant defense by scavenging reactive oxygen species (ROS) and reducing oxidative stress damage[3]. Ubiquinone-1 is usually used in research on energy metabolism and neurological diseases, immune system diseases[4][5].

In vitro, treatment of rat pulmonary microvascular endothelial cells (PMVEC) cultured in low glucose medium with Ubiquinone-1 (10μM; 6h) prevented ATP depletion, mitochondrial membrane depolarization, and increased monolayer permeability caused by the complex I inhibitor rotenone[6]. Treatment of primary rat astrocyte cultures with Ubiquinone-1 (5μM; 30min) significantly inhibited the recycling of ascorbate from dehydroascorbic acid (DHAA) in a dose-dependent manner under glucose-free conditions[7].

References:
[1] Fato R, Estornell E, Di Bernardo S, Pallotti F, Parenti Castelli G, Lenaz G. Steady-state kinetics of the reduction of coenzyme Q analogs by complex I (NADH:ubiquinone oxidoreductase) in bovine heart mitochondria and submitochondrial particles. *Biochemistry*. 1996;35(8):2705-2716.
[2] Mantle D, Dewsbury M, Hargreaves IP. The Ubiquinone-Ubiquinol Redox Cycle and Its Clinical Consequences: An Overview. *Int J Mol Sci*. 2024;25(12):6765.
[3] Olaru G, Buga AM, Sandu RE, Padureanu V, Popa DG, Calina D. Harnessing Mitochondrial Function for Post-Stroke Rehabilitation: Unlocking Antioxidant Power. *Antioxidants (Basel)*. 2025;14(9):1080.
[4] Roginsky VA, Mohr D, Stocker R. Reduction of ubiquinone-1 by ascorbic acid is a catalytic and reversible process controlled by the concentration of molecular oxygen. *Redox Rep*. 1996;2(1):55-62.
[5] Rauchova H. Coenzyme Q10 effects in neurological diseases. *Physiol Res*. 2021;70(Suppl4):S683-S714.
[6] Bongard RD, Townsley MI, Merker MP. The effects of mitochondrial complex I blockade on ATP and permeability in rat pulmonary microvascular endothelial cells in culture (PMVEC) are overcome by coenzyme Q1 (CoQ1). *Free Radic Biol Med*. 2015;79:69-77.
[7] Dragan M, Dixon SJ, Jaworski E, Chan TS, O'brien PJ, Wilson JX. Coenzyme Q(1) depletes NAD(P)H and impairs recycling of ascorbate in astrocytes. *Brain Res*. 2006;1078(1):9-18.