RonanRx
PERSONALIZED MEDICINE

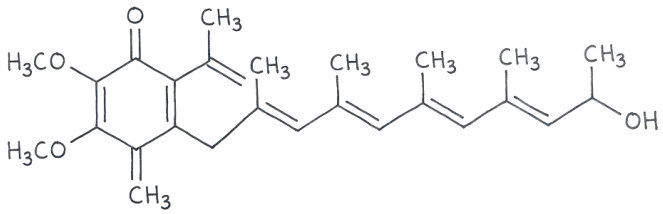
CLINICAL MONOGRAPH · ANTIOXIDANT & MITOCHONDRIAL

Coenzyme Q10 (CoQ10)

Mitochondrial electron carrier and antioxidant

Coenzyme Q10 (CoQ10), also called ubiquinone in its oxidized form and ubiquinol in its reduced form, is a fat-soluble molecule that every mitochondrion in the body uses to make energy [bmjopen2021; nih_ods_coq10]. It shuttles electrons inside the mitochondrial respiratory chain and works as an antioxidant in cell membranes. Levels decline with age, with statin use, and in certain rare genetic diseases.

There is no prescription CoQ10 drug in the United States, it is sold as a dietary supplement. The strongest randomized evidence is in chronic heart failure (Q-SYMBIO trial), in muscle pain from statin drugs, and in preventing migraines [mortensen2014; banach2015]. Evidence in Parkinson disease was disappointing once a large NIH trial was completed [qe3_2014; hargreaves2014]. RonanRx compounds CoQ10 only when an over-the-counter supplement cannot meet a specific clinical need, for example, pharmaceutical-grade purity for a child with a confirmed genetic CoQ10 deficiency, a custom strength above 1,000 mg/day, an allergen-free preparation, or a lipid-based troche when oral capsules don't absorb well.



EVIDENCE POSTURE WELL STUDIED EMERGING

REVIEWED **2026-05-11**

State-licensed 503A | Pharmacist reviewed | Doctor led | Cold-chain ready | Patient choice preserved

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FOR CLINICIANS

Coenzyme Q10 (2,3-dimethoxy-5-methyl-6-decaprenyl-1,4-benzoquinone) is the obligate electron shuttle between Complexes I/II and Complex III of the mitochondrial respiratory chain and a lipid-phase antioxidant in cellular membranes [bentinger2007]. It exists endogenously in equilibrium between ubiquinone (oxidized) and ubiquinol (reduced) and is synthesized via the mevalonate pathway. There is no FDA-approved CoQ10 drug product in the United States; CoQ10 is marketed as a dietary supplement under DSHEA [dshea1994, nih_ods_coq10]. Randomized trial evidence is strongest in chronic heart failure, statin-associated muscle symptoms, migraine prophylaxis, and primary (genetic) CoQ10 deficiency, with emerging signals in adjunctive fertility care and metabolic disease [belardinelli2006].

The Q-SYMBIO trial [mortensen2014] randomized 420 adults with NYHA class III/IV heart failure to CoQ10 300 mg/day or placebo and reported a reduction in the composite of major adverse cardiac events at 2 years (HR 0.50; 95% CI 0.32, 0.80), with reductions in all-cause and cardiovascular mortality [belardinelli2006]. Earlier randomized work [morisco1993] had shown reductions in HF hospitalizations and improvement in endothelial function. The KiSel-10 trial [alehagen2013] in elderly Swedish citizens (CoQ10 200 mg + selenium 200 µg vs placebo for 4 years) reported reduced cardiovascular mortality persisting at 10-, 12-, and post-12-year follow-up [alehagen2016, alehagen2018, alehagen2024]. The Banach et al. meta-analysis of statin-associated muscle symptoms [banach2015] reported modest but statistically significant improvements in muscle pain and weakness with CoQ10 across six small RCTs; a companion meta-analysis [banach2015pharma] confirmed that statin therapy reduces circulating CoQ10 levels. CoQ10 100 mg three times daily for migraine prophylaxis was effective vs placebo in the Sándor RCT [sandor2005]; the pediatric/adolescent open-label work of Hershey [hershey2007] and a 2021 systematic review/meta-analysis [bmjopen2021] support a reduction in headache frequency. The Shults 2002 phase II signal in early Parkinson disease [shults2002] was not confirmed in the NIH-funded QE3 phase III trial [qe3_2014], which was stopped for futility. Evidence in male and female infertility is emerging [lafuente2013, bentov2014, gvozdjakova2013].

Pharmacokinetics are dominated by poor aqueous solubility: bioavailability of crystalline ubiquinone is low and highly formulation-dependent. Bhagavan and Chopra (2007) reviewed plasma CoQ10 responses across commercial formulations [bhagavan2007]; Hosoe et al. (2007) demonstrated that solubilized ubiquinol (Kaneka QH) achieves higher plasma concentrations than equivalent doses of ubiquinone in healthy volunteers [hosoe2007]. CoQ10 is generally well tolerated; the most common adverse events are mild gastrointestinal upset. Drug interactions of clinical concern involve warfarin (potential antagonism via structural similarity to vitamin K) and theoretical additive antihypertensive effect [nih_ods_coq10]. The compounded 503A role for CoQ10 is narrow: pharmaceutical-grade purity for confirmed mitochondrial disease, custom strengths beyond the commercial range, allergen-free formulations, and lipid-based or sublingual/troche delivery for absorption optimization [hargreaves2014]. OTC supplement overlap with these clinical needs is significant and should be discussed honestly with patients [dahri2019].

Why Personalized Coenzyme Q10 (CoQ10)

The CoQ10 doses studied in the major trials were chosen for the average enrolled patient, not for you. Q-SYMBIO landed on 300 mg per day in heart failure. Sándor used 100 mg three times daily in adult migraine. Shults pushed to 1,200 mg per day in early Parkinson disease, and the primary CoQ10 deficiency literature has gone as high as 30 mg per kilogram per day in children with biosynthetic-gene mutations. None of those numbers account for whether you are on a statin, how poorly crystalline ubiquinone absorbs from your gut, whether you carry a COQ2 or COQ6 variant, what excipients you react to, or whether you can swallow a softgel at all.

That gap is what a compounding pharmacy can address when an over-the-counter supplement cannot. RonanRx prepares CoQ10 in custom strengths above the commercial ceiling for confirmed mitochondrial disease, in allergen-free bases for patients who react to the soybean oil or gelatin in retail softgels, as a sublingual troche or lipid-based vehicle when crystalline oral formulations do not produce measurable plasma response, and as ubiquinol rather than ubiquinone when absorption studies in that individual support it. The molecule is the same one Crane isolated in 1957. The strength, the vehicle, and the route are matched to the patient and to a documented clinical reason the supplement aisle cannot meet.

This is what pharmacy looked like before mass manufacturing arrived. A prescriber identified a specific need, wrote an order for a named patient, and a pharmacist prepared it on the bench. Modern 503A oversight, licensed pharmacist accountability, state inspection, and a real recall path, keeps that older arrangement honest.

Quick Facts About Coenzyme Q10 (CoQ10)

Category: Mitochondrial electron carrier (Complex I/II → III shuttle) and lipid-phase antioxidant

Active forms: Ubiquinone (oxidized) and ubiquinol (reduced), endogenously interconverted; both forms are commercially available as oral supplements

FDA-approval status: No FDA-approved coenzyme Q10 drug product. Marketed in the United States as a dietary supplement under the Dietary Supplement Health and Education Act (DSHEA) of 1994.

Best-studied indications: Adjunctive use in chronic heart failure (Q-SYMBIO trial), statin-associated muscle symptoms, migraine prophylaxis, and primary (genetic) coenzyme Q10 deficiency

Typical adult oral dose range: 100, 300 mg/day for most studied indications; up to 1,200 mg/day in Parkinson disease research and 2,400 mg/day in primary CoQ10 deficiency under clinician supervision

OTC overlap: Significant. Pharmaceutical-grade coenzyme Q10 is widely available without prescription. The 503A compounding role is limited to specific clinical situations, pharmaceutical-grade purity for known mitochondrial disease, custom strengths above commercial doses, allergen-free formulations, troche/sublingual delivery, and lipid-based vehicles for absorption.

Compounded under: 503A, patient-specific prescription only when the OTC supplement format cannot meet a documented clinical need

> **SPECIALS: PATIENT-SPECIFIC PRESCRIPTION ONLY**
>
> Coenzyme Q10 (CoQ10) described in this monograph is a 503A compounded preparation. Every dose is made on a prescription, for a named patient, by a licensed pharmacist. It is not a stocked, mass-manufactured product.
>
> - **Made to order, not off a shelf.** No batch sits in a warehouse waiting for buyers. Your prescription triggers the prep.
> - **Named-patient label.** The bottle carries one patient's name. The batch records carry one prescription.
> - **Dose, strength, and route chosen for the patient.** A prescriber decides what gets compounded, not a manufacturer who set the strength for a trial population.
> - **Licensed pharmacist on the hook.** A real person, with a license that can be pulled, signs off on every prep. State inspectors check the facility.
> - **Compounded drugs are not FDA-approved.** They should not be evaluated using branded-drug trial data alone. Availability varies by state and prescribed medication.

How This Differs from a Research-Use-Only Website

A research-use-only website ships a vial from a warehouse. There is no prescription, no pharmacist, no facility inspection, and no way to recall the product if something is wrong with it. If the vial is mislabeled, contaminated, or under-potent, there is nobody whose license is at stake.

A 503A compounding pharmacy is the other thing. The doctor writes the prescription. A licensed pharmacist, whose name is on the label, prepares the medicine in a facility the state inspects. If something goes wrong, there is a person and a license on the hook, and a documented chain of custody on every lot. That accountability is what makes it safe.

What is Coenzyme Q10 (CoQ10)?

Coenzyme Q10 is a lipid-soluble 1,4-benzoquinone with a side chain of ten isoprenoid units (hence the '10'). It was first isolated from beef heart mitochondria by Frederick Crane and colleagues in 1957, and its role in the mitochondrial respiratory chain was established over the following decade [bentinger2007]. The molecule is endogenously synthesized in every nucleated cell via the mevalonate pathway, which is the same pathway inhibited by HMG-CoA reductase (statin) drugs upstream of cholesterol and CoQ10 branchpoints.

CoQ10 exists in vivo in dynamic equilibrium between two redox states: ubiquinone (CoQ10, fully oxidized) and ubiquinol (CoQ10H2, fully reduced) [bentinger2007]. Both forms are commercially available; ubiquinol formulations claim superior absorption based on PK studies in healthy adults [hosoe2007, bhagavan2007]. The endogenous interconversion between forms is rapid, so the practical distinction is one of absorption from the gastrointestinal tract rather than systemic mechanism.

In the United States, coenzyme Q10 is sold as a dietary supplement under the Dietary Supplement Health and Education Act of 1994 [dshea1994]. There is no FDA-approved coenzyme Q10 prescription drug product [bentinger2007]. The Office of Dietary Supplements (NIH) maintains a clinical factsheet summarizing supplement-grade product information [nih_ods_coq10].

How Coenzyme Q10 (CoQ10) Works

Inside the inner mitochondrial membrane, coenzyme Q10 functions as a mobile electron carrier that accepts electrons from Complex I (NADH dehydrogenase) and Complex II (succinate dehydrogenase) and delivers them to Complex III (cytochrome bc1). This shuttle is obligate, without functioning CoQ10 the respiratory chain cannot pump protons, ATP synthesis falls, and reactive oxygen species generation rises. CoQ10 cycles between the oxidized (ubiquinone), one-electron-reduced (semiquinone), and two-electron-reduced (ubiquinol) states many thousands of times per minute in metabolically active cells.

Outside the respiratory chain, CoQ10 acts as a lipid-phase antioxidant in cellular and lipoprotein membranes, where the ubiquinol form donates a hydrogen atom to lipid peroxyl radicals and is regenerated by enzymatic and non-enzymatic recycling systems [bentinger2007]. CoQ10 also regulates the mitochondrial permeability transition pore and participates in pyrimidine biosynthesis via electron acceptance from dihydroorotate dehydrogenase.

Tissue CoQ10 concentrations are highest in mitochondria-rich organs (heart, kidney, liver, skeletal muscle). Plasma concentrations decline with age and are reduced by HMG-CoA reductase inhibitor (statin) therapy because the mevalonate pathway provides the isoprenoid tail; meta-analytic evidence confirms a statin-associated reduction in circulating CoQ10 [banach2015pharma].

Biological Role of Coenzyme Q10 (CoQ10)

Coenzyme Q10 is one of three obligate electron carriers (with NAD/NADH and the cytochromes) that connect substrate oxidation to ATP synthesis. The respiratory chain captures the energy released from the stepwise transfer of electrons from NADH and FADH2 to molecular oxygen and converts it into a transmembrane proton gradient that drives ATP synthase. Without CoQ10, the gradient cannot form and metabolism collapses. Tissue CoQ10 content correlates with mitochondrial density and is highest in cardiomyocytes, hepatocytes, renal tubular cells, and skeletal myocytes.

The CoQ10 antioxidant role is biologically distinct from its electron-carrier role and was characterized later, primarily through work in the 1990s on lipid peroxidation in low-density lipoprotein. Ubiquinol in LDL is regenerated from ubiquinone by intracellular reductases, so a single ubiquinol molecule can scavenge many radical equivalents, a feature shared with tocopherol but unique among membrane antioxidants in its bioenergetic dual function [bentinger2007].

Detailed Mechanism of Coenzyme Q10 (CoQ10)

Biosynthesis of CoQ10 proceeds via the mevalonate pathway through farnesyl pyrophosphate to a decaprenyl tail, which is conjugated to a benzoquinone ring derived from tyrosine. At least ten enzymes (COQ2 through COQ10 plus polyprenyl pyrophosphate synthase) participate in the mitochondrial assembly steps. Loss-of-function mutations in any of these genes produce primary coenzyme Q10 deficiency, a heterogeneous group of mitochondrial disorders that includes nephrotic syndrome (COQ2, COQ6, COQ8B), encephalomyopathy, cerebellar ataxia, and Leigh-like syndromes. Hargreaves's review of CoQ10 as therapy for mitochondrial disease [hargreaves2014] documents the most consistent supplementation responses occur in primary deficiency states caused by biosynthetic-gene mutations, where doses up to 30 mg/kg/day have produced clinical and biochemical improvement.

In the respiratory chain, CoQ10 functions as a 2-electron, 2-proton redox cycler: ubiquinone accepts an electron to form the semiquinone radical (CoQ10·–), and a second electron plus two protons to form ubiquinol. The semiquinone intermediate, while ordinarily short-lived, is a source of mitochondrial superoxide when the respiratory chain is overloaded or partially inhibited, a feature relevant to CoQ10's dual role as electron carrier and antioxidant. The reduced ubiquinol pool in plasma lipoproteins is the only known endogenous lipid-phase antioxidant that is itself regenerated; it accounts for most of the redox-protection of circulating LDL [bentinger2007].

CoQ10 also regulates the mitochondrial permeability transition pore (mPTP), sustained pore opening drives apoptotic and necrotic cell death in ischemia-reperfusion injury and heart failure. Modulation of mPTP sensitivity by membrane CoQ10 content has been advanced as a mechanism for the heart-failure clinical effect observed in Q-SYMBIO [mortensen2014] and the prior Morisco and Belardinelli trials [morisco1993, belardinelli2006]. Endothelial-function improvement with CoQ10 in heart failure has been documented [belardinelli2006] and is consistent with both mitochondrial-bioenergetic and antioxidant mechanisms.

Coenzyme Q10 (CoQ10) Research History

Coenzyme Q10 was isolated by Frederick Crane and colleagues at the University of Wisconsin in 1957 from beef heart mitochondria, and its structure was elucidated by Karl Folkers's group at Merck shortly thereafter [alehagen2016]. Through the 1960s and 1970s the molecule's role as an obligate electron carrier

in the mitochondrial respiratory chain was established by oxidative phosphorylation researchers including Peter Mitchell, whose chemiosmotic hypothesis required a mobile electron shuttle of CoQ10's properties.

Clinical use of supplemental CoQ10 originated in Japan in the 1970s for cardiac applications and spread to the United States and Europe over the following decades. Morisco and colleagues (1993) reported a randomized trial of CoQ10 in congestive heart failure [morisco1993] showing reduced hospitalizations for worsening heart failure. Singh et al. (1999) reported reductions in blood pressure with CoQ10 in adults with hypertension and coronary artery disease [singh1999], and Rosenfeldt et al. (2003) summarized the systematic-review evidence for CoQ10 in hypertension, heart failure, and exercise performance [rosenfeldt2003]. The Belardinelli 2006 trial [belardinelli2006] demonstrated endothelial-function improvement with CoQ10 plus exercise in chronic heart failure. The Q-SYMBIO trial [mortensen2014] provided the most rigorous evidence to date: 420 adults with NYHA III/IV heart failure randomized to CoQ10 300 mg/day vs placebo, with a 2-year reduction in the composite major adverse cardiac event endpoint and in all-cause and cardiovascular mortality [mortensen2014] [alehagen2016].

Independently, the Swedish KiSel-10 trial randomized 443 elderly community-dwelling adults to CoQ10 200 mg + selenium 200 µg vs placebo for 4 years. The primary report [alehagen2013] showed reductions in cardiovascular mortality and N-terminal proBNP that persisted at 10-year [alehagen2016, low-Se subgroup analysis], 12-year [alehagen2018], and post-12-year follow-up [alehagen2024], establishing CoQ10 plus selenium as one of the few combination supplementation regimens with multi-decade randomized mortality data [alehagen2016].

The statin-associated muscle symptom literature developed in parallel. The Banach et al. meta-analysis (2015) of six RCTs reported modest but statistically significant improvements in muscle pain and weakness scores with CoQ10 supplementation [banach2015]; a companion meta-analysis confirmed that statin therapy reduces circulating CoQ10 [banach2015pharma] [alehagen2016]. Migraine prophylaxis evidence began with the Sándor 2005 randomized controlled trial of CoQ10 100 mg three times daily [sandor2005], extended into pediatric and adolescent populations by Hershey et al. (2007) [hershey2007], and was synthesized in subsequent meta-analyses [bmjopen2021, dahri2019].

Parkinson disease has been the most prominent disappointment in the CoQ10 literature [alehagen2016]. Shults et al. (2002) reported a phase II signal of slower functional decline with high-dose CoQ10 (1,200 mg/day) in early Parkinson disease [shults2002], leading to the NIH-funded Parkinson Study Group QE3 phase III trial (2014). QE3 randomized 600 adults with early Parkinson disease to CoQ10 1,200 or 2,400 mg/day vs placebo and was stopped for futility at the planned interim analysis [qe3_2014], producing the definitive null. Evidence in male infertility [lafuente2013, gvozdjakova2013] and oocyte/female fertility [bentov2014] remains emerging, primarily on the basis of small RCTs of sperm parameters and observational IVF outcomes.

Coenzyme Q10 (CoQ10) Timeline

1957 • Crane, Hatefi, Lester, and Widmer isolate coenzyme Q (ubiquinone) from beef heart mitochondria at the University of Wisconsin [bentinger2007]

1958 • Folkers's group at Merck elucidates the structure of coenzyme Q10

1961 • Peter Mitchell's chemiosmotic hypothesis posits the role of a mobile electron carrier matching CoQ10's biophysical properties

1993 • Morisco et al [morisco1993]. publish a randomized trial of CoQ10 in congestive heart failure showing reduced hospitalizations

1994 • U.S [dshea1994]. Congress passes the Dietary Supplement Health and Education Act (DSHEA), under which CoQ10 is marketed as a dietary supplement

1999 • Singh et al [singh1999]. report blood-pressure reduction with hydrosoluble CoQ10 in adults with hypertension and coronary artery disease

2002 • Shults et al [shults2002]. publish a phase II signal of slower functional decline with high-dose CoQ10 (1,200 mg/day) in early Parkinson disease (Arch Neurol)

2003 • Rosenfeldt et al [rosenfeldt2003]. publish a systematic review of CoQ10 in physical exercise, hypertension, and heart failure

2005 • Sándor et al [sandor2005]. publish a randomized controlled trial of CoQ10 100 mg three times daily for migraine prophylaxis (Neurology)

2006 • Belardinelli et al [belardinelli2006]. show CoQ10 plus exercise training improves endothelial function in chronic heart failure (Eur Heart J)

2007 • Bhagavan and Chopra publish their review of plasma CoQ10 response to oral formulations (Mitochondrion); Hosoe et al [bhagavan2007; hosoe2007]. characterize ubiquinol bioavailability in healthy volunteers

2007 • Hershey et al [hershey2007]. (Headache) report CoQ10 deficiency and supplementation response in pediatric and adolescent migraine

2007 • Bentinger, Brismar, and Dallner review the antioxidant role of coenzyme Q (Mitochondrion) [bentinger2007]

2013 • Alehagen et al [alehagen2013]. (KiSel-10) publish the 5-year prospective randomized trial of CoQ10 200 mg + selenium 200 µg vs placebo in elderly Swedish citizens, reporting reduced cardiovascular mortality (Int J Cardiol)

2013 • Lafuente et al. publish a meta-analysis of CoQ10 in male infertility (J Assist Reprod Genet); Gvozdjakova et al [lafuente2013; gvozdjakova2013]. report on CoQ10 in asthenozoospermia

2014 • Mortensen et al [mortensen2014]. publish Q-SYMBIO in JACC Heart Failure, 420 adults with NYHA III/IV heart failure randomized to CoQ10 300 mg/day vs placebo, with reduced major adverse cardiac events and mortality at 2 years

2014 • The NIH-funded Parkinson Study Group QE3 trial of high-dose CoQ10 (1,200 or 2,400 mg/day) in early Parkinson disease is stopped for futility (JAMA Neurol), definitive null result [qe3_2014]

2014 • Bentov et al [bentov2014]. report on CoQ10 supplementation and oocyte aneuploidy in women undergoing IVF-ICSI

2014 • Hargreaves reviews CoQ10 as therapy for mitochondrial disease (Int J Biochem Cell Biol), summarizing dose-response in primary CoQ10 deficiency [hargreaves2014]

2015 • Banach et al [banach2015; banach2015pharma]. publish a meta-analysis of six RCTs of CoQ10 for statin-induced myopathy (Mayo Clin Proc), modest but significant benefit; companion meta-analysis (Pharmacol Res) confirms statin therapy reduces circulating CoQ10

2016 • Alehagen et al [alehagen2016]. publish a low-selenium subgroup secondary analysis of KiSel-10, confirming cardiovascular mortality benefit in adults with the lowest baseline selenium status (PLoS One)

2018 • Alehagen et al [alehagen2018]. publish 12-year follow-up of KiSel-10, validating prior 10-year results that cardiovascular mortality remains reduced years after the 4-year intervention

2019 • Dahri et al [dahri2019]. publish an RCT of CoQ10 supplementation in patients with migraine showing reduced headache severity and inflammatory markers (Nutr Neurosci)

2021 • Sazali et al [bmjopen2021]. publish a systematic review and meta-analysis of CoQ10 for migraine prophylaxis in BMJ Open, supporting a reduction in headache frequency

2024 • Alehagen et al [alehagen2024]. publish post-12-year follow-up of KiSel-10 in BMC Medicine, reporting positive effects on thyroid hormones, cardiovascular mortality, and quality of life

Clinical Contexts for Coenzyme Q10 (CoQ10)

Adjunctive therapy in chronic heart failure (NYHA II, IV) WELL STUDIED

Well-studied adjunct; no FDA-approved CoQ10 drug. Best randomized evidence is from Q-SYMBIO.

Q-SYMBIO [mortensen2014] randomized 420 adults with NYHA class III/IV heart failure to CoQ10 100 mg three times daily (300 mg/day) or placebo on top of guideline-directed medical therapy and reported a 50% reduction in the composite major adverse cardiac event endpoint at 2 years (HR 0.50; 95% CI 0.32, 0.80), with reductions in all-cause and cardiovascular mortality. The earlier Morisco trial [morisco1993] reported reduced HF hospitalizations with CoQ10 in an Italian multicenter randomized study, and the Belardinelli 2006 trial [belardinelli2006] demonstrated endothelial-function improvement with CoQ10 plus exercise. The Swedish KiSel-10 trial [alehagen2013] in elderly community-dwelling adults reported reduced cardiovascular mortality with CoQ10 200 mg + selenium 200 µg over 4 years that persisted at 10-, 12-, and post-12-year follow-up [alehagen2016, alehagen2018, alehagen2024]. CoQ10 is not a substitute for FDA-approved heart-failure therapy (beta-blockers, ACE inhibitors/ARBs, MRAs, SGLT2 inhibitors, sacubitril-valsartan); it is used as an adjunct.

Statin-associated muscle symptoms WELL STUDIED

Well-studied adjunct with mixed individual-trial results; meta-analysis supports modest benefit.

The Banach et al. meta-analysis [banach2015] pooled six small RCTs of CoQ10 in statin-associated muscle symptoms and reported significant improvements in muscle pain (weighted mean difference −1.6 on visual analog or composite scales) and weakness. A companion meta-analysis [banach2015pharma] confirmed that statin therapy reduces circulating CoQ10 concentrations, providing a biological rationale. Effect sizes are modest and not all individual RCTs are positive; CoQ10 is best framed as an evidence-supported empirical option for the substantial minority of statin-intolerant patients in whom the alternative is statin discontinuation.

Migraine prophylaxis WELL STUDIED

Well-studied adjunct; American Headache Society and AAN guidelines classify CoQ10 as 'probably effective.'

The Sándor randomized controlled trial [sandor2005] of CoQ10 100 mg three times daily for migraine prophylaxis in adults reported a reduction in attack frequency. The Hershey pediatric/adolescent study [hershey2007] documented CoQ10 deficiency in a substantial proportion of children with migraine and a reduction in headache frequency with supplementation. The Dahri 2019 RCT [dahri2019] reported reductions in headache severity, frequency, and inflammatory markers. The 2021 BMJ Open systematic review and meta-analysis [bmjopen2021] pooled adult RCT evidence and reported reduced headache frequency with CoQ10 vs placebo. Typical adult prophylactic dosing is 100 mg three times daily (300 mg/day) with onset of effect over 4, 12 weeks.

Primary (genetic) coenzyme Q10 deficiency WELL STUDIED

Confirmed therapeutic role per mitochondrial-disease consensus reviews; rare-disease use.

Primary coenzyme Q10 deficiency comprises a heterogeneous group of mitochondrial disorders caused by loss-of-function mutations in CoQ10 biosynthetic genes (COQ2, COQ4, COQ6, COQ7, COQ8A, COQ8B, COQ9, PDSS1, PDSS2). Phenotypes include steroid-resistant nephrotic syndrome (COQ2, COQ6, COQ8B), encephalomyopathy, cerebellar ataxia, and Leigh-like syndromes. Oral CoQ10 supplementation at doses up to 30 mg/kg/day (commonly 600, 2,400 mg/day in adults) has produced clinical and biochemical improvement in case series and small cohorts, particularly when started early; the Hargreaves review [hargreaves2014] summarizes the dose-response and clinical-improvement literature. Primary deficiency is the use case in which pharmaceutical-grade compounded preparations have the clearest role over OTC supplements.

Hypertension EMERGING

Emerging; modest BP-lowering effect in small randomized trials and pooled reviews.

The Singh 1999 randomized trial [singh1999] reported reductions in systolic and diastolic blood pressure with hydrosoluble CoQ10 in adults with hypertension and coronary artery disease. The Rosenfeldt 2003 systematic review [rosenfeldt2003] aggregated CoQ10 evidence in hypertension, heart failure, and exercise performance with modest pooled BP-lowering effect. Subsequent randomized evidence has been inconsistent; CoQ10 is not a substitute for guideline-directed antihypertensive therapy.

Male infertility (asthenozoospermia, oligospermia) EMERGING

Emerging; small RCTs and meta-analyses report improvement in sperm motility and concentration without confirmed effect on live-birth rates.

The Lafuente meta-analysis [lafuente2013] pooled randomized evidence of CoQ10 in male infertility and reported improvements in sperm concentration and motility. The Gvozdjakova et al. report on asthenozoospermia [gvozdjakova2013] documented reductions in oxidative-stress markers and improvement in motility with combined CoQ10 and vitamin E supplementation. None of the trials are powered to detect a live-birth-rate effect; reproductive-endocrinology use is empirical and not guideline-endorsed.

Female fertility / IVF support (DHEA + CoQ10 protocols) EMERGING

Emerging; small studies in poor-responders or women of advanced reproductive age.

Bentov and Casper's pilot work [bentov2014] reported a numerical reduction in oocyte aneuploidy with pre-cycle CoQ10 supplementation in women undergoing IVF-ICSI. CoQ10 is frequently combined with DHEA in reproductive-endocrinology 'pre-cycle prep' protocols for diminished ovarian reserve and advanced reproductive age; randomized evidence remains limited and effect on live-birth rate is not established.

Parkinson disease WELL STUDIED

Studied and negative, phase II signal not confirmed in the NIH-funded QE3 phase III trial.

Shults et al. (2002) [shults2002] reported a phase II signal that high-dose CoQ10 (300, 600, or 1,200 mg/day) slowed functional decline in early Parkinson disease. The NIH-funded Parkinson Study Group QE3 phase III trial [qe3_2014] randomized 600 adults with early Parkinson disease to CoQ10 1,200 mg/day, 2,400 mg/day, or placebo for 16 months and was stopped at the planned interim analysis for futility, no evidence of slowing of clinical decline. CoQ10 is not recommended as disease-modifying therapy in Parkinson disease.

Off-Label Uses of Coenzyme Q10 (CoQ10)

Adjunctive antioxidant supplementation in cardiovascular and metabolic disease prevention (combined with selenium) EMERGING

Emerging; supported by the KiSel-10 trial and its long-term follow-up in elderly Swedish populations with low baseline selenium status.

KiSel-10 randomized 443 elderly Swedish adults to CoQ10 200 mg + selenium 200 µg vs placebo for 4 years and reported persistent reductions in cardiovascular mortality through more than 12 years of follow-up [alehagen2013; alehagen2018; alehagen2024]. Effects are most pronounced in those with the lowest baseline selenium status [alehagen2016] and may not generalize to populations with adequate selenium intake. Use as a general antioxidant/cardiometabolic prevention strategy outside this specific population is not guideline-endorsed.

FDA-Approved Uses of Coenzyme Q10 (CoQ10)

There is no FDA-approved coenzyme Q10 drug product in the United States [fda503a]. CoQ10 is marketed as a dietary supplement under the Dietary Supplement Health and Education Act (DSHEA) of 1994 [dshea1994]. The NIH Office of Dietary Supplements maintains a clinician/consumer factsheet that summarizes available supplement-grade product information and the evidence base [nih_ods_coq10].

Compounded CoQ10 preparations dispensed under section 503A are not FDA-approved drugs and are not generic versions of any approved product [fda503a]. They are patient-specific compounded preparations dispensed against a prescription written by a licensed prescriber for a documented clinical need that the OTC supplement format cannot meet.

Compounded Coenzyme Q10 (CoQ10) (503A)

OTC overlap is significant [fda503a]. Pharmaceutical-grade coenzyme Q10 is widely available without prescription, in both ubiquinone and ubiquinol forms, at strengths from 30 mg to 600 mg per capsule and at varying claimed bioavailability profiles [sandor2005; bmjopen2021; alehagen2013]. RonanRx does not recommend compounded CoQ10 as a substitute for the supplement-grade product in routine adjunctive use (e.g., for general antioxidant support or empirical statin-myalgia trial), the supplement market is well-served by reputable manufacturers and the clinical evidence base does not distinguish between properly-formulated supplement-grade and pharmaceutical-grade preparations [nih_ods_coq10, bhagavan2007].

Compounded 503A CoQ10 has a narrower clinical role in four documented patient-specific situations [fda503a]. First, pharmaceutical-grade purity with documented active-ingredient identity and assay is

warranted in patients with primary (genetic) coenzyme Q10 deficiency who require high-dose chronic therapy (commonly 600, 2,400 mg/day in adults, up to 30 mg/kg/day in pediatric patients) and whose treatment depends on consistent batch-to-batch bioavailability [hargreaves2014] [mortensen2014; banach2015]. Second, custom strengths beyond the commercially available range (typically >600 mg per unit) can reduce pill burden in patients requiring >1,200 mg/day. Third, allergen-free formulations are useful for patients with documented sensitivity to common supplement excipients (soy, soybean oil, gelatin, common preservatives). Fourth, lipid-based vehicles, sublingual troches, or buccal formulations can improve absorption in patients with documented poor response to standard oral capsules, though evidence that any specific compounded delivery form is superior to the best commercial ubiquinol formulation is limited [hosoe2007, bhagavan2007].

Compounded preparations are not bioequivalent to any commercial supplement-grade reference product. The published clinical evidence for CoQ10 is generated with various supplement-grade and pharmaceutical-grade products, none of which are FDA-approved drugs. Plasma CoQ10 monitoring is available through specialty laboratories and is the only objective basis for documenting compounded-preparation absorption in individual patients [fda503a].

Coenzyme Q10 (CoQ10) Formulations and Routes

Form	Concentration	Description
Oral capsule (compounded)	Custom, typically 100, 200, 300, 400, or 600 mg per capsule of ubiquinone or ubiquinol	Pharmaceutical-grade ubiquinone or ubiquinol in lipid-based or oil-suspension capsule. Used when the clinical situation requires assured potency, allergen-free vehicle, or strength above the commercial range.
Sublingual or buccal troche (compounded)	Custom, typically 50, 200 mg per troche	Lipid-base troche for patients with poor absorption from oral capsules or with difficulty swallowing. Direct mucosal evidence of superior bioavailability vs the best commercial ubiquinol capsule is limited.
Commercial OTC supplement (reference, non-compounded)	30, 600 mg per capsule, ubiquinone or ubiquinol	Available without prescription under DSHEA. Reputable supplement-grade products are the appropriate first-line format for most clinical use; the published RCT evidence base for CoQ10 is generated with such products.

Routes used in published literature: oral, sublingual, buccal, troche.

Coenzyme Q10 (CoQ10) Dosing

Route	Population	Range	Duration	Study type
Oral	Adults with chronic heart failure (NYHA II, IV) as adjunct to guideline-directed therapy	100 mg three times daily (300 mg/day total) as studied in Q-SYMBIO	Indefinite while clinically beneficial; Q-SYMBIO followed participants for 2 years	Phase III RCT (Q-SYMBIO)
Oral	Adults with statin-associated muscle symptoms	100, 300 mg/day; effect onset over 4, 12 weeks; empirical trial	8, 12 week trial, continue if symptomatic improvement	Meta-analysis of 6 RCTs (Banach 2015)
Oral	Adults with episodic migraine, prophylaxis	100 mg three times daily (300 mg/day) as studied in Sándor 2005	Trial for 3 months; continue if effective	RCT (Sándor 2005); meta-analysis (Sazali 2021)
Oral	Pediatric/adolescent migraine prophylaxis	1, 3 mg/kg/day, typically 100 mg once or twice daily by adolescence	Trial for 3 months; continue if effective	Open-label pediatric series (Hershey 2007)
Oral	Primary (genetic) coenzyme Q10 deficiency	Up to 30 mg/kg/day in divided doses; commonly 600, 2,400 mg/day in adults	Indefinite (lifelong)	Case series and consensus reviews (Hargreaves 2014)
Oral	Elderly with low baseline selenium status (combination prevention regimen)	CoQ10 200 mg/day combined with selenium 200 µg/day (KiSel-10 regimen)	4 years intervention with persistent mortality benefit at >12-year follow-up	RCT (KiSel-10, Alehagen 2013)
Oral	Early Parkinson disease, historical research dose, not recommended	1,200 mg/day was studied in Shults 2002 phase II; 1,200 and 2,400 mg/day in QE3 phase III (both futile)	16 months in QE3	Phase II (Shults 2002), signal; Phase III (QE3 2014), futile

Doctor-prescribed and titrated. CoQ10 dosing in clinical trials has used 100, 300 mg/day for most adult indications, 100 mg three times daily being the most studied schedule for both Q-SYMBIO (heart failure) and Sándor (migraine prophylaxis). Higher doses (600, 2,400 mg/day) are used in primary CoQ10 deficiency under specialist supervision and were studied, without benefit, in Parkinson disease in the QE3 trial [qe3_2014].

Onset of clinical effect is slow. In migraine prophylaxis, the Sándor and pediatric studies report onset over 4, 12 weeks; in heart failure, Q-SYMBIO observed divergence of event curves over the first 6 months [mortensen2014; sandor2005; hershey2007]. Patients should be counseled that CoQ10 is not an acute or as-needed therapy. Plasma CoQ10 concentrations correlate poorly with intracellular target-tissue levels but can be useful in patients on chronic high-dose therapy to document absorption [hargreaves2014].

Coenzyme Q10 (CoQ10) Safety

Coenzyme Q10 is well tolerated across the studied dose range. Across the Q-SYMBIO heart failure trial (300 mg/day for 2 years) [1], the Sándor migraine prophylaxis trial (300 mg/day) [10], the Banach meta-analysis of statin-myopathy RCTs [8], and the KiSel-10 trial of CoQ10 plus selenium for 4 years [4], adverse-event rates were not significantly different from placebo. The most common adverse events are mild gastrointestinal symptoms, nausea, dyspepsia, loose stools, and occasional abdominal discomfort, typically dose-related and reversible on dose reduction. Rash and insomnia have been reported occasionally.

Even at very high doses, tolerability is preserved. The QE3 trial [15] administered CoQ10 1,200 or 2,400 mg/day for up to 16 months in adults with early Parkinson disease without a meaningful tolerability signal; the trial was stopped for futility, not for safety. Primary CoQ10 deficiency case series have documented tolerability of doses up to 30 mg/kg/day [19].

Clinically relevant drug interactions are limited but specific. Coenzyme Q10 is structurally similar to vitamin K (both are quinones), and isolated case reports describe reductions in INR with concomitant warfarin therapy [25]. Patients on warfarin who initiate or discontinue CoQ10 should have INR monitored. An additive antihypertensive effect with established antihypertensive agents is theoretical and may be relevant at the high end of the CoQ10 dose range; blood-pressure monitoring is reasonable after initiation in patients with controlled hypertension. CoQ10 does not have well-documented interactions with the CYP450 system at supplement doses.

Contraindications

There are no absolute contraindications to coenzyme Q10 supplementation in adults at the typical clinical dose range. Hypersensitivity to a specific commercial or compounded formulation (typically an excipient, soy, soybean oil, gelatin, rather than CoQ10 itself) is a relative contraindication for that formulation and can usually be managed by switching to an allergen-free preparation [25].

Caution is warranted in patients on warfarin given case-report-level evidence of INR reduction [25]. Use during pregnancy and lactation should be discussed with the prescriber given limited human data, although case reports and observational use in pre-eclampsia and IVF protocols have not raised safety signals.

Drug interactions

Warfarin: case reports describe reduction in INR with concomitant CoQ10, attributed to structural similarity between ubiquinone and vitamin K (both are quinones) and possible competition at the vitamin K cycle. Patients on warfarin who initiate, change dose of, or discontinue CoQ10 should have INR monitored more closely for 2, 4 weeks [25].

Antihypertensive agents: theoretical additive BP-lowering effect at the higher end of the CoQ10 dose range [23][24]. Blood pressure should be checked after initiation in patients with already-controlled hypertension.

Statins: HMG-CoA reductase inhibition reduces endogenous CoQ10 synthesis (the mevalonate pathway is shared), and statin therapy reduces circulating CoQ10 concentrations in meta-analysis [9]. This is the mechanistic rationale for CoQ10 supplementation in statin-associated muscle symptoms [8]; the interaction is favorable rather than harmful.

Chemotherapy: CoQ10 has been used adjunctively with anthracycline chemotherapy to mitigate cardiotoxicity in case series, but theoretical concerns about antioxidant interference with chemotherapy efficacy mean concomitant use should be coordinated with the treating oncologist [25].

Adverse events

Across the major randomized trials, Q-SYMBIO 300 mg/day for 2 years [1], KiSel-10 200 mg/day plus selenium for 4 years [4], Sándor 300 mg/day for migraine [10], the Banach meta-analysis of 6 statin-myopathy RCTs [8], and QE3 1,200, 2,400 mg/day for up to 16 months [15], adverse-event rates were not significantly different from placebo. The most common reported events are mild gastrointestinal: nausea, dyspepsia, loose stools, occasional abdominal discomfort. Rash, insomnia, fatigue, and dizziness have been reported occasionally.

Serious adverse events directly attributable to CoQ10 are rare. The NIH ODS clinical factsheet [25] summarizes the post-marketing supplement-surveillance record. The most clinically significant safety concern is the warfarin, CoQ10 interaction discussed under Drug Interactions.

Monitoring Coenzyme Q10 (CoQ10) Therapy

Routine plasma CoQ10 monitoring is not required for most adult indications. In patients on chronic high-dose therapy (>600 mg/day) for primary CoQ10 deficiency or refractory neurologic indications, periodic plasma CoQ10 measurement through specialty laboratories can document absorption and adherence. Plasma concentrations correlate poorly with intracellular tissue concentrations, so the value is in detecting non-response rather than confirming target-tissue effect.

On therapy: assess clinical response at the indication-appropriate interval (typically 3 months for migraine prophylaxis, 3, 6 months for heart failure adjunct, 6, 12 weeks for statin myalgia) [mortensen2014; sandor2005]. Monitor INR in patients on warfarin who initiate or change CoQ10 dose. Monitor blood

pressure after initiation in patients with controlled hypertension [banach2015]. Continue therapy if benefit is documented; discontinue if no clinical improvement after an adequate trial [hargreaves2014; nih_ods_coq10].

Coenzyme Q10 (CoQ10) in Special Populations

Coenzyme Q10 (CoQ10) Evidence Quality

Evidence for coenzyme Q10 is heterogeneous by indication [nih_ods_coq10] [alehagen2024]. For chronic heart failure (NYHA III/IV), the Q-SYMBIO trial [mortensen2014] is a moderate-sized randomized phase III, like trial with a positive primary endpoint and supportive mortality data; the Morisco [morisco1993] and Belardinelli [belardinelli2006] randomized trials add mechanistic and hospitalization-endpoint support. The KiSel-10 long-term combination trial provides 4-year randomized intervention plus >12-year follow-up cardiovascular mortality data in an elderly low-selenium population, with the caveat that the effect attributable to CoQ10 alone cannot be separated from selenium [alehagen2013; alehagen2016; alehagen2018]. For statin-associated muscle symptoms, the Banach meta-analysis of 6 small RCTs [banach2015] reports modest but significant benefit, supported by a companion meta-analysis confirming statin-induced CoQ10 depletion [banach2015pharma]; individual trials are mixed. For migraine prophylaxis, the Sándor RCT [sandor2005] and the 2021 BMJ Open systematic review/meta-analysis [bmjopen2021] support a reduction in headache frequency, classed as 'probably effective' in current headache-medicine guidelines.

For primary (genetic) CoQ10 deficiency, evidence is from case series and small cohorts [hargreaves2014] but the mechanistic basis is direct (loss-of-function mutations in CoQ10-biosynthetic genes) and clinical-improvement responses are reproducible enough that high-dose CoQ10 is the standard of care [dahri2019]. For Parkinson disease, the NIH-funded QE3 phase III trial [qe3_2014] was definitively negative and supersedes the earlier Shults 2002 phase II signal [shults2002]. For male and female infertility, evidence is from small RCTs and pilot studies [lafuente2013, gvozdjakova2013, bentov2014] without live-birth-rate outcome data. For hypertension and general antioxidant 'wellness' use, evidence is modest and inconsistent.

Across indications, the compounded 503A role for CoQ10 is narrow because over-the-counter pharmaceutical-grade and supplement-grade products meet the clinical need for the vast majority of patients [dahri2019]. Compounded preparations are appropriate when an OTC product cannot deliver the strength, purity assurance, allergen profile, or delivery vehicle that an individual patient documentably requires.

Major Coenzyme Q10 (CoQ10) Clinical Studies

Study	Design	Participants	Duration	Finding
Q-SYMBIO (Mortensen 2014, JACC Heart Failure)	Randomized, double-blind, placebo-controlled, multinational phase III, like trial of CoQ10 100 mg three times daily vs placebo as adjunct to standard heart failure therapy in adults with NYHA class III or IV chronic heart failure	420	2 years	CoQ10 reduced the primary composite of major adverse cardiac events (HR 0.50; 95% CI 0.32, 0.80; p=0.003), with reductions in all-cause and cardiovascular mortality and improvement in NYHA functional class [mortensen2014]
Morisco et al. (1993, Clin Investig)	Multicenter randomized double-blind placebo-controlled trial of CoQ10 2 mg/kg/day in adults with congestive heart failure on standard therapy	641	1 year	CoQ10 reduced hospitalizations for worsening heart failure and episodes of pulmonary edema/cardiac asthma vs placebo; earliest large randomized signal in HF [morisco1993]
Belardinelli et al. (2006, Eur Heart J)	Randomized double-blind crossover trial of CoQ10 100 mg three times daily ± supervised exercise training in adults with chronic heart failure	21	4-week treatment phases with crossover	CoQ10 improved endothelial function (flow-mediated dilation), peak VO2, and left ventricular contractility vs placebo, with additive effect of exercise training [belardinelli2006]
KiSel-10 primary report (Alehagen 2013, Int J Cardiol)	Prospective randomized double-blind placebo-controlled trial of CoQ10 200 mg/day + selenium 200 µg/day vs placebo in community-dwelling elderly Swedish citizens	443	5 years (4-year intervention + follow-up)	Reduced cardiovascular mortality and NT-proBNP vs placebo; positive primary endpoint [alehagen2013]
KiSel-10 low-selenium	Secondary analysis of KiSel-10 stratified by	—	5 years	Cardiovascular mortality reduction concentrated in

Study	Design	Participants	Duration	Finding
subgroup (Alehagen 2016, PLoS One)	baseline serum selenium status			adults with the lowest baseline selenium status, where the active intervention closed the selenium gap [alehagen2016]
KiSel-10 12-year follow-up (Alehagen 2018, PLoS One)	Long-term follow-up of the KiSel-10 RCT	—	12 years from randomization	Cardiovascular mortality reduction persisted at 12 years, well beyond the 4-year intervention period, validating prior 10-year follow-up results [alehagen2018]
KiSel-10 post-12-year follow-up (Alehagen 2024, BMC Medicine)	Extended follow-up of KiSel-10 with additional analyses of thyroid hormones and quality of life	—	>12 years from randomization	Positive effects of CoQ10 plus selenium on thyroid hormones, cardiovascular mortality, and quality of life sustained on long-term follow-up [alehagen2024]
Banach et al. (2015, Mayo Clin Proc), statin myopathy meta-analysis	Systematic review and meta-analysis of 6 randomized placebo-controlled trials of CoQ10 in statin-associated muscle symptoms	302	Trials 4, 12 weeks	Significant reduction in muscle pain scores and improvement in weakness with CoQ10 vs placebo; effect size modest but consistent across positive trials [banach2015]
Banach et al. (2015, Pharmacol Res), statin-induced CoQ10 depletion	Systematic review and meta-analysis of placebo-controlled trials measuring plasma CoQ10 in patients on statin therapy	—	—	Statin therapy reduced circulating CoQ10 concentrations significantly vs placebo, providing biological rationale for supplementation in statin-associated muscle symptoms [banach2015pharma]
Sándor et al. (2005, Neurology),	Randomized double-blind placebo-controlled trial of CoQ10 100 mg three times daily for migraine	42	3 months	CoQ10 reduced attack frequency, headache days, and days with nausea vs placebo; 47.6% of CoQ10-

Study	Design	Participants	Duration	Finding
migraine prophylaxis RCT	prophylaxis in adults with episodic migraine			treated participants had ≥50% reduction in attack frequency vs 14.4% on placebo [sandor2005]
Hershey et al. (2007, Headache), pediatric/ adolescent migraine	Open-label retrospective and prospective evaluation of CoQ10 status and supplementation response in pediatric and adolescent migraine clinic patients	1550	Variable	A substantial proportion of pediatric/adolescent migraine patients had below-reference CoQ10 levels; supplementation reduced headache frequency and disability scores [hershey2007]
Dahri et al. (2019, Nutr Neurosci), migraine RCT	Randomized double-blind placebo-controlled trial of oral CoQ10 in adults with episodic migraine	—	Up to 3 months	Reduced headache severity, duration, and inflammatory markers (TNF-α, CGRP) with CoQ10 vs placebo [dahri2019]
Sazali et al. (2021, BMJ Open), migraine prophylaxis meta-analysis	Systematic review and meta-analysis of randomized trials of CoQ10 for migraine prophylaxis in adults	—	—	CoQ10 reduced headache frequency vs placebo in pooled analysis; AAN/AHS guidelines classify CoQ10 as 'probably effective' for migraine prevention [bmjopen2021]
Shults et al. (2002, Arch Neurol), Parkinson phase II	Randomized double-blind placebo-controlled phase II dose-ranging trial of CoQ10 300, 600, or 1,200 mg/day in early Parkinson disease	80	16 months	Less worsening of total UPDRS score with CoQ10 vs placebo, with greatest effect at 1,200 mg/day, motivated subsequent phase III NIH trial [shults2002]
QE3 (Parkinson Study Group, Beal et al. 2014, JAMA Neurol)	Randomized double-blind placebo-controlled NIH-funded phase III trial of CoQ10 1,200 mg/day, 2,400 mg/day, or placebo in early Parkinson disease	600	16 months (stopped at planned interim for futility)	No evidence of benefit at either dose vs placebo on the primary clinical endpoint; trial stopped for futility, definitive null result that supersedes the Shults 2002 phase II signal [qe3_2014]

Study	Design	Participants	Duration	Finding
Hosoe et al. (2007, Regul Toxicol Pharmacol), ubiquinol PK	Single- and multiple-dose pharmacokinetic study of solubilized ubiquinol (Kaneka QH) in healthy volunteers	—	Single dose plus 4-week multiple-dose phase	Solubilized ubiquinol produced higher plasma CoQ10 concentrations than equivalent doses of ubiquinone formulations; safety profile unremarkable [hosoe2007]
Bhagavan and Chopra (2007, Mitochondrion), bioavailability review	Review of plasma CoQ10 response across commercial oral CoQ10 formulations	—	—	Plasma CoQ10 response is highly formulation-dependent; lipid-based, solubilized, and ubiquinol formulations generally achieve higher plasma levels than crystalline ubiquinone in oil-suspension or dry-powder formats [bhagavan2007]
Hargreaves (2014, Int J Biochem Cell Biol), primary CoQ10 deficiency review	Narrative review of CoQ10 as therapy for primary and secondary mitochondrial disease	—	—	Clinical and biochemical responses to high-dose oral CoQ10 (up to 30 mg/kg/day) are most consistent in primary CoQ10 deficiency due to biosynthetic-gene mutations; secondary deficiency responses are more variable [hargreaves2014]
Lafuente et al. (2013, J Assist Reprod Genet), male infertility meta-analysis	Systematic review and meta-analysis of randomized trials of CoQ10 in male infertility (asthenozoospermia, oligospermia)	—	—	CoQ10 supplementation improved sperm concentration and motility vs placebo; effect on live-birth rate not demonstrated and not powered [lafuente2013]
Bentov et al. (2014, Clin Med Insights Reprod Health), IVF oocyte aneuploidy	Pilot randomized trial of pre-cycle CoQ10 supplementation in women undergoing IVF-ICSI	—	—	Numerical reduction in oocyte aneuploidy with CoQ10 vs placebo; not powered for live-birth-rate outcome [bentov2014]

Study	Design	Participants	Duration	Finding
Singh et al. (1999, J Hum Hypertens), hypertension RCT	Randomized double-blind trial of hydrosoluble CoQ10 60 mg twice daily vs placebo in adults with hypertension and coronary artery disease	59	8 weeks	Reductions in systolic and diastolic blood pressure and improvement in insulin sensitivity vs placebo [singh1999]
Rosenfeldt et al. (2003, Biofactors), exercise/HTN/HF systematic review	Systematic review of CoQ10 in physical exercise, hypertension, and heart failure	—	—	Modest pooled blood-pressure-lowering effect across small trials; supportive but inconsistent evidence in heart failure preceding Q-SYMBIO [rosenfeldt2003]

Coenzyme Q10 (CoQ10) Pharmacokinetics & Pharmacodynamics

Pharmacokinetics

Coenzyme Q10 is a lipid-soluble molecule with poor aqueous solubility, which dominates its absorption pharmacokinetics. Oral bioavailability of crystalline ubiquinone is low and highly formulation-dependent, lipid-based, solubilized, and ubiquinol formulations achieve substantially higher plasma concentrations than dry-powder or oil-suspension formats of crystalline ubiquinone [bhagavan2007]. Hosoe et al. characterized solubilized ubiquinol (Kaneka QH) pharmacokinetics in healthy volunteers and demonstrated higher plasma CoQ10 concentrations than equivalent doses of ubiquinone [hosoe2007]. Time to peak plasma concentration is typically 5, 10 hours after oral administration; biological half-life is approximately 33 hours.

Absorbed CoQ10 is incorporated into chylomicrons, distributed via plasma lipoproteins (predominantly LDL), and taken up into peripheral tissues. Plasma concentrations are higher in patients with hyperlipidemia (lipoprotein-bound fraction) and lower in patients on chronic statin therapy. Plasma CoQ10 correlates poorly with intracellular target-tissue concentrations, so plasma levels are useful for documenting absorption but not for inferring target-tissue effect. CoQ10 is not significantly renally cleared and does not have well-documented CYP450-mediated metabolism at supplement doses.

Compounded sublingual troches and lipid-based capsules are designed to optimize the absorption step. Direct mucosal-route evidence of superior plasma response vs the best commercial ubiquinol formulation is limited; the rationale for compounded delivery is patient-specific (e.g., documented poor response to standard oral capsules, swallowing difficulty, or specific allergen avoidance) rather than population-level superiority.

Pharmacodynamics

Pharmacodynamic endpoints depend on indication. In primary CoQ10 deficiency, target endpoints include leukocyte and muscle CoQ10 concentrations, complex I+III and II+III activity, and clinical phenotype response (renal function in nephrotic-syndrome variants, neurologic function in encephalomyopathic variants) [hargreaves2014]. In heart failure, endpoints are NT-proBNP, NYHA functional class, peak VO2, and cardiovascular event rates [mortensen2014, alehagen2013]. In migraine prophylaxis, endpoints are monthly headache frequency, headache days, and days with nausea [sandor2005, bmjopen2021]. In statin-myopathy, endpoints are muscle pain and weakness scores and adherence to statin therapy [banach2015].

Onset of pharmacodynamic effect is slow across indications, typically 4, 12 weeks. Plasma CoQ10 rises within days of starting supplementation, but intracellular target-tissue accumulation and downstream physiologic effects unfold over weeks to months.

↓↑ Comparing Coenzyme Q10 (CoQ10) Formulations

There is no FDA-approved reference product for coenzyme Q10. The clinical comparators are the wide range of supplement-grade OTC products that span dry-powder crystalline ubiquinone, oil-suspension ubiquinone, solubilized ubiquinone, and solubilized ubiquinol. Plasma response varies several-fold across formulations at the same labeled dose [bhagavan2007]; solubilized ubiquinol generally produces the highest plasma concentrations in healthy adults [hosoe2007].

Compounded preparations should be benchmarked against the best commercial ubiquinol formulation, not against crystalline ubiquinone, when discussing relative absorption with prescribers. The compounded preparation's role is patient-specific (purity assurance, custom strength, allergen avoidance, alternative delivery vehicle) rather than population-level superiority.

🌡 Coenzyme Q10 (CoQ10) Storage and Handling

Coenzyme Q10 is sensitive to light, heat, and oxidative degradation. Capsules and troches should be stored in tight, light-resistant containers at controlled room temperature (15, 30°C / 59, 86°F). Compounded preparations are dispensed with beyond-use dating per USP <795> for nonsterile preparations [usp_795]; refrigeration is not required for capsules and troches but may extend beyond-use dating for lipid-rich preparations.

Coenzyme Q10 (CoQ10) Compounding & Operations

503A compounding

Compounded coenzyme Q10 is prepared under section 503A on patient-specific prescriptions in state-licensed compounding pharmacies [fda503a]. RonanRx prepares nonsterile oral capsules and sublingual/buccal troches per USP General Chapter <795> [usp_795] with documented active-ingredient sourcing (USP-grade or pharmaceutical-grade ubiquinone or ubiquinol with certificate of analysis), gravimetric verification, beyond-use dating per chapter requirements, and full lot traceability. CoQ10 is not a sterile preparation in oral or troche form; USP <797> does not apply unless a sterile injectable is compounded (rare for CoQ10).

RonanRx limits compounded CoQ10 to four documented patient-specific situations: (1) pharmaceutical-grade purity for patients with confirmed primary (genetic) CoQ10 deficiency requiring chronic high-dose therapy; (2) custom strengths beyond the commercially available range (typically >600 mg per unit, to reduce pill burden at >1,200 mg/day total daily dose); (3) allergen-free formulations for patients with documented sensitivity to common supplement excipients; and (4) lipid-based, sublingual, or buccal delivery vehicles for patients with documented poor absorption from standard oral capsules [hargreaves2014]. We do not fill prescriptions for compounded CoQ10 that read as routine substitution for a commercial supplement-grade product.

Pharmacist review

Each prescription for compounded coenzyme Q10 undergoes pharmacist review prior to dispensing [nih_ods_coq10] [mortensen2014; banach2015; alehagen2013]. The review confirms: (1) a documented patient-specific clinical reason that a commercial supplement-grade product is not appropriate (one of the four situations enumerated under Compounding 503A); (2) prescriber rationale for the requested strength and delivery form; (3) absence of relevant drug interactions, particularly warfarin co-prescription; (4) appropriate counseling regarding slow onset of clinical effect and the indication-appropriate trial duration; and (5) honest patient communication that the published RCT evidence for CoQ10 is generated with various supplement-grade and pharmaceutical-grade products and does not establish compounded-preparation superiority over the best commercial ubiquinol formulation [fda503a] [sandor2005; bmjopen2021; hargreaves2014].

Quality and traceability

Active pharmaceutical ingredient (ubiquinone or ubiquinol) is sourced from FDA-registered facilities with documented certificates of analysis confirming identity, potency, residual solvents, heavy metals, and microbial limits per USP monograph requirements. Each batch is recorded with lot numbers traceable to

API source, compounding date, beyond-use date, and dispensing pharmacist of record. Finished product lot records are retained per state board of pharmacy retention requirements.

Cold chain

Compounded oral capsules and troches of coenzyme Q10 are not cold-chain products under typical formulation. Standard storage is controlled room temperature in light-resistant packaging. Lipid-rich preparations and some sublingual troche bases may have extended beyond-use dating with refrigerated storage; the pharmacy assigns storage conditions per USP <795> stability evaluation [usp_795].

Frequently Asked Questions About Coenzyme Q10 (CoQ10)

Is coenzyme Q10 a prescription drug?

No. There is no FDA-approved coenzyme Q10 drug product in the United States. CoQ10 is sold as a dietary supplement under the Dietary Supplement Health and Education Act (DSHEA) of 1994 [dshea1994; nih_ods_coq10]. Compounded CoQ10 is dispensed under section 503A on patient-specific prescriptions when an over-the-counter supplement format cannot meet a documented clinical need [fda503a].

When does compounded CoQ10 make sense over an over-the-counter supplement?

In four documented situations: (1) pharmaceutical-grade purity for confirmed primary (genetic) coenzyme Q10 deficiency requiring chronic high-dose therapy; (2) custom strengths beyond the commercial range (e.g., >600 mg per unit to reduce pill burden at >1,200 mg/day); (3) allergen-free preparations for patients sensitive to common supplement excipients; and (4) lipid-based, sublingual, or buccal delivery vehicles in patients with documented poor absorption from standard oral capsules [hargreaves2014; bhagavan2007; hosoe2007]. For routine adjunctive use, the OTC supplement market is well-served and compounding is rarely indicated.

What's the difference between ubiquinone and ubiquinol?

Ubiquinone is the oxidized form of CoQ10 and ubiquinol is the reduced form. Both are endogenously present and rapidly interconverted in vivo. The practical difference is absorption: solubilized ubiquinol formulations produce higher plasma CoQ10 concentrations than equivalent doses of crystalline ubiquinone in healthy volunteers (Hosoe 2007) [hosoe2007; bhagavan2007]. Once absorbed, the systemic effect is the same.

Does CoQ10 work for statin muscle pain?

Probably modestly. The Banach 2015 meta-analysis of six small randomized trials reported significant improvements in muscle pain and weakness with CoQ10 vs placebo [banach2015]. Effect sizes are modest and not all individual trials are positive. A companion meta-analysis confirmed that statin therapy reduces

circulating CoQ10 concentrations, supporting the biological rationale [banach2015pharma]. CoQ10 is a reasonable empirical option for patients with statin-associated muscle symptoms in whom the alternative is statin discontinuation.

Does CoQ10 help heart failure?

The Q-SYMBIO trial (Mortensen 2014) randomized 420 adults with NYHA III/IV heart failure to CoQ10 300 mg/day or placebo on top of guideline-directed therapy and reported a 50% reduction in major adverse cardiac events at 2 years, with reductions in all-cause and cardiovascular mortality [mortensen2014]. The KiSel-10 trial of CoQ10 plus selenium also reduced cardiovascular mortality in an elderly Swedish population with persistent benefit at >12-year follow-up [alehagen2013; alehagen2018]. CoQ10 is an adjunct, not a substitute for guideline-directed heart failure therapy.

Does CoQ10 prevent migraines?

Yes, modestly. The Sándor 2005 randomized trial of CoQ10 100 mg three times daily showed reductions in migraine frequency and the 2021 BMJ Open systematic review and meta-analysis confirmed reduced headache frequency with CoQ10 vs placebo [sandor2005]. American Headache Society and AAN guidelines classify CoQ10 as 'probably effective' for migraine prophylaxis [bmjopen2021; dahri2019; hershey2007]. Onset of benefit is over 4, 12 weeks.

What about CoQ10 for Parkinson disease?

It does not work. An early phase II trial (Shults 2002) suggested CoQ10 1,200 mg/day might slow functional decline in early Parkinson disease, but the NIH-funded QE3 phase III trial (2014) randomized 600 adults to CoQ10 1,200 mg/day, 2,400 mg/day, or placebo and was stopped at the planned interim analysis for futility, no benefit at either dose [shults2002; qe3_2014]. CoQ10 is not recommended as disease-modifying therapy in Parkinson disease.

Are there drug interactions to worry about?

The most clinically relevant interaction is with warfarin: case reports describe reduction in INR with concomitant CoQ10, likely via structural similarity to vitamin K. INR should be monitored when CoQ10 is initiated, changed, or discontinued in warfarin-treated patients. CoQ10 may have an additive blood-pressure-lowering effect with antihypertensives at the higher dose range. CoQ10 does not have well-documented CYP450 interactions at supplement doses [nih_ods_coq10; singh1999].

Does RonanRx sell compounded CoQ10 directly to patients?

No. Compounded CoQ10 requires a patient-specific prescription written by a licensed prescriber for an identified patient with a documented clinical need that the OTC supplement format cannot meet, plus pharmacist review before dispensing. RonanRx is not a direct-to-consumer storefront and does not substitute compounded CoQ10 for a commercial supplement-grade product [fda503a].

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How to Access Coenzyme Q10 (CoQ10)

Compounded Coenzyme Q10 (CoQ10) is dispensed under 503A on a patient-specific prescription. Depending on your role, the next step looks different.

FOR PRESCRIBING CLINICIANS

Offer this medication

A pharmacist will follow up within two business days. We'll cover state availability, supported formulations, and what integration looks like for your clinic.

ronanrx.com/request-partnership-call

PATIENT WITH A DOCTOR

Receive your prescription

If your doctor has prescribed Coenzyme Q10 (CoQ10), sign up so we can prepare and ship your medication. The signup wizard collects intake and connects you to the prescribing workflow.

ronanrx.com/patients

PATIENT WITHOUT A DOCTOR

Find a partner clinic

RonanRx prescribes through partner clinics — we don't initiate prescriptions on this site. Read how the referral process works and how to find a partner clinic in your state.

ronanrx.com/find-clinic

Other compounds RonanRx makes

This monograph is one of many in the RonanRx formulary. Every compound below is prepared under 503A on a patient-specific prescription. Browse the full catalog at ronanrx.com/medications and ronanrx.com/peptides, or scan the codes at right for each index.

Medications

Peptides

MEDICATIONS (40)

Alpha-Lipoic Acid (ALA) — Antioxidant & mitochondrial
Coenzyme Q10 (CoQ10) — Antioxidant & mitochondrial
Glutathione — Antioxidant & mitochondrial
NAD+ / NMN — Antioxidant & mitochondrial
Compounded Topical Anesthetics (BLT, LET) — Dermatology
Topical Minoxidil — Dermatology
Topical Tretinoin — Dermatology
Compounded Magnesium — Energy & nutritional
Cyanocobalamin — Energy & nutritional
High-Dose Vitamin D — Energy & nutritional
Hydroxocobalamin — Energy & nutritional
Iron (Compounded) — Energy & nutritional
L-Carnitine — Energy & nutritional
Methylcobalamin (B12) — Energy & nutritional
Methylfolate — Energy & nutritional
Anastrozole — Hormone optimization
Clomiphene & Enclomiphene — Hormone optimization
DHEA — Hormone optimization
Estradiol — Hormone optimization
Estriol — Hormone optimization
Human Chorionic Gonadotropin (HCG) — Hormone optimization
Pregnenolone — Hormone optimization
Progesterone — Hormone optimization
Testosterone — Hormone optimization
Compounded Metformin — Metabolic & weight
Compounded Semaglutide — Metabolic & weight
Compounded Tirzepatide — Metabolic & weight
Lipotropic Injection (MIC, MICC) — Metabolic & weight
Low-Dose Naltrexone (LDN) — Metabolic & weight
Naltrexone-Bupropion Combination — Metabolic & weight
Topiramate — Metabolic & weight
Bremelanotide / PT-141 — Sexual health
Compounded Sildenafil — Sexual health
Compounded Tadalafil — Sexual health
Trimix Injection — Sexual health
Compounded Gabapentin — Sleep & recovery
Compounded Melatonin — Sleep & recovery
Compounded T3 (Liothyronine) — Thyroid
Compounded T3/T4 Combinations — Thyroid
Compounded T4 (Levothyroxine) — Thyroid

PEPTIDES (21)

Sermorelin — Available now

Tesamorelin — Available now

AOD-9604 — Growth-hormone axis (under FDA review)

CJC-1295 — Growth-hormone axis (under FDA review)

GHRP-2 / GHRP-6 — Growth-hormone axis (under FDA review)

Hexarelin — Growth-hormone axis (under FDA review)

Ipamorelin — Growth-hormone axis (under FDA review)

MK-677 / Ibutamoren — Growth-hormone axis (under FDA review)

5-Amino 1MQ — Metabolic & longevity (under FDA review)

Epitalon / Epithalon — Metabolic & longevity (under FDA review)

MOTS-C — Metabolic & longevity (under FDA review)

Thymosin Alpha-1 / Thymalin — Metabolic & longevity (under FDA review)

DSIP, Delta Sleep-Inducing Peptide — Neuro & cognitive (under FDA review)

Selank — Neuro & cognitive (under FDA review)

Semax — Neuro & cognitive (under FDA review)

Vasoactive Intestinal Peptide (VIP) — Neuro & cognitive (under FDA review)

BPC-157 — Tissue repair (under FDA review)

KPV — Tissue repair (under FDA review)

LL-37 — Tissue repair (under FDA review)

Pentadeca Arginate (PDA) — Tissue repair (under FDA review)

TB-500 / Thymosin Beta-4 — Tissue repair (under FDA review)