



REVIEW PAPER

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Coenzyme q10: A multifaceted therapeutic molecule in neurological, cardiovascular, and reproductive health

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ABSTRACT

Coenzyme Q10 (ubiquinone), is a lipid-soluble molecule derived primarily from endogenous synthesis. It plays an essential role in the electron-transport chain of mitochondrial respiratory chain. It also works as an antioxidant and is often used as an anti-aging supplement. It can also help with the symptoms of many health problems that are linked to mitochondrial malfunction. Evidence indicates that CoQ10 supplementation may alleviate neurodegenerative processes, enhance reproductive results in both sexes, and promote cardiovascular function. This review is to deliver a thorough examination of the existing evidence about the therapeutic uses of CoQ10, emphasising its functions in neuroprotection, fertility, and cardiovascular well-being. Its translational potential will be evaluated by looking at mechanistic discoveries, evidence from clinical trials, and where the field is headed in the future. Although promising, additional large-scale, randomised controlled trials are required to formulate definite clinical guidelines.

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INTRODUCTION

Coenzyme Q10 (CoQ10), or ubiquinone, is a naturally occurring benzoquinone molecule that is lipid-soluble and plays a key role in cellular respiration and antioxidant defence (Martelli *et al.*, 2020). The name coenzyme Q10 derives from its chemical structure, a benzoquinone ring with a side chain composed of 10 isoprene units (Fig. 1) (Raizner, 2019). It is mostly found in the inner mitochondrial membrane, where it is a major electron transporter in the mitochondrial respiratory chain. It moves electrons from complexes I and II to complex III, which helps in ATP production (Gutierrez-Mariscal *et al.*, 2020).

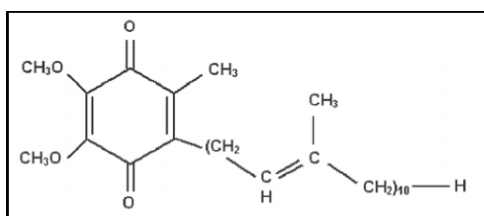


Fig. 1. Chemical structure of coenzyme Q10

CoQ10 has a bioenergetic role, it exists in two forms (Fig. 2): oxidised (ubiquinone) and reduced (ubiquinol) which makes it a strong antioxidant. It neutralises reactive oxygen species (ROS) and restores other antioxidants, including vitamin E and vitamin C, thus preserving cellular redox equilibrium (Bentinger *et al.*, 2010).

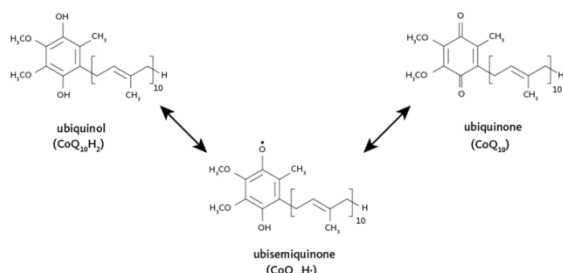


Fig. 2. The different redox forms of coenzyme Q10

Coenzyme Q10 is naturally found in many foods (Fig. 3), with the highest amounts in fatty fish like salmon, tuna, and mackerel, organ meats like liver, heart, and kidney, and to a lesser level in whole grains, nuts, and vegetable oils (Saini, 2011). High concentrations of CoQ10 are found in metabolically active organs like the heart, liver, kidneys, and

brain, emphasizing its physiological importance (Crane, 2001).

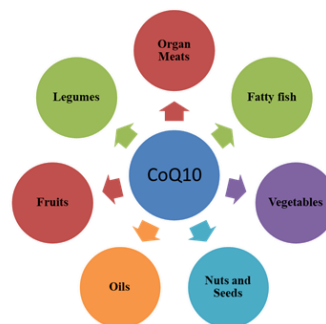


Fig. 3. Sources of coenzyme Q10

Although dietary sources contribute to CoQ10 levels, it is mainly produced endogenously in the inner mitochondrial membrane through the action of nuclear-encoded CoQ proteins (Staiano *et al.*, 2023). However, endogenous synthesis of CoQ10 declines significantly with age and is further exacerbated in individuals with chronic illnesses or those on statin therapy (Bhagavan and Chopra, 2006). Statins inhibit HMG-CoA reductase, a key enzyme in the mevalonate pathway, which is also responsible for CoQ10 biosynthesis, leading to reduced CoQ10 levels and associated mitochondrial dysfunction (Marcoff and Thompson, 2007).

The therapeutic relevance of CoQ10 stems from its role in ameliorating oxidative stress and mitochondrial dysfunction, which are common pathophysiological features across multiple chronic conditions. One area of growing interest is neuroprotection. Neurodegenerative diseases such as Parkinson's and Alzheimer's are associated with impaired mitochondrial function and increased oxidative damage (Beal, 2004). Early studies and clinical trials have shown that CoQ10 supplementation can help preserve mitochondrial integrity, reduce neuronal apoptosis, and slow the progression of disease symptoms (Shults *et al.*, 2002; Matthews *et al.*, 1998).

CoQ10 supplementation has also demonstrated advantageous effects in the domain of reproductive health for both males and females. Oxidative stress

damages sperm function in men, while CoQ10 has been shown to boost mitochondrial activity, which improves sperm motility, morphology, and concentration (Safarinejad, 2009). In females, particularly those undergoing assisted reproductive technology (ART) or exhibiting reduced ovarian reserve, CoQ10 supplementation has been linked to enhanced oocyte quality, fertilisation rates, and embryo development (Xu *et al.*, 2018; Bentov *et al.*, 2014).

The cardiovascular system is perhaps the area of CoQ10 therapy that has been explored the most. Because the heart needs a lot of energy and has a lot of mitochondria, it is especially susceptible to CoQ10 shortage. Supplementation has been demonstrated to enhance left ventricular ejection fraction, diminish oxidative stress, and decrease blood pressure (Kumar *et al.*, 2009; Fotino *et al.*, 2013). The Q-SYMBIO trial, a multicenter randomized controlled study, demonstrated that long-term CoQ10 use significantly reduced hospitalizations and mortality in patients with chronic heart failure (Mortensen *et al.*, 2014).

Although CoQ10 supplementation has a positive profile, obstacles in standardising its use in clinical practice persist due to dose variability, differing bioavailability of formulations (e.g., ubiquinone vs. ubiquinol), and study design issues. The aim of this review is to thoroughly assess the existing literature about the therapeutic uses of CoQ10 in neuroprotection, fertility, and cardiovascular health, emphasising molecular insights, clinical outcomes.

Neuroprotective Effects of CoQ10

Neurological diseases pose a significant threat to human health and quality of life, affecting millions of people worldwide (World Health Organization, 2006). Several causes and mechanisms have been proposed to explain the onset and progression of neurological disorders. These include lifestyle factors, genetic predisposition, infections, dietary habits, environmental exposures, and physical trauma (World Health Organization, 2006). Neurological disorders often present with a range of physical and

cognitive symptoms, such as partial or complete paralysis, seizures, muscle weakness, sensory loss, reading and writing impairments, diminished cognitive abilities, unexplained pain, and decreased alertness. Among the underlying mechanisms, oxidative stress and impaired energy metabolism are recognized as key contributors to the pathogenesis of several neurodegenerative diseases, including Parkinson's disease (PD), Alzheimer's disease (AD), multiple sclerosis (MS), epilepsy, depression and stroke (Table 1).

Furthermore, the prevalence of age-related neurological disorders has been steadily increasing in recent years (Fig. 4). These pathological features are tightly linked to energy metabolism deficits and increased production of reactive oxygen species (ROS), making mitochondrial-targeted therapies a promising approach (Ciurea *et al.*, 2023). Coenzyme Q10 (CoQ10), due to its role in mitochondrial electron transport and antioxidant activity, has emerged as a potential neuroprotective agent (Bagheri *et al.*, 2023b).

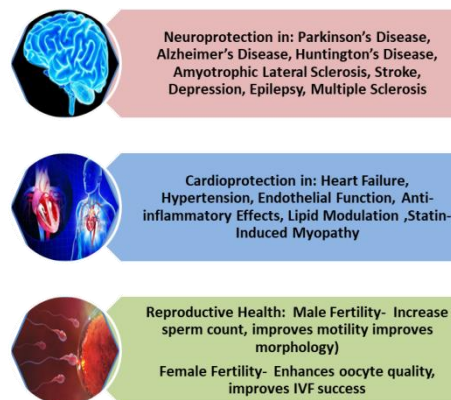


Fig. 4. Major roles of coenzyme Q10 in brain, heart and reproductive health

Cells constantly produce energy, and in doing so, they generate reactive oxygen species (ROS) molecules that can damage cell components like DNA, proteins, or fats. Normally, our bodies balance ROS with antioxidant defenses, repairing or removing damage (Zhao and Zhao, 2013). In Alzheimer's, this balance is disturbed: ROS production is higher, antioxidant defenses are weaker, and damage accumulates. Markers of oxidative damage are found early in

people who go on to develop neurodegenerative diseases, even before major symptoms appear. Damage to mitochondrial DNA (mtDNA), oxidation of proteins, lipid peroxidation (damage to fats in cell membranes) are all elevated. This damage accelerates cell aging and makes neurons more vulnerable to degeneration (Fanlo-Ucar *et al.*, 2024).

Parkinson's disease

Parkinson's disease (PD) is a brain disorder in which certain neurons, especially those that produce dopamine, gradually die. The dying off is not random: it affects neurons in a part of the brain called the substantia nigra, which is important for controlling movement (Kouli *et al.*, 2018). As dopamine levels fall, people begin to have symptoms like tremor, stiffness, slowness of movement, and problems with balance. Alongside there are non-movement issues such as mood changes, disturbed sleep, and sometimes thinking difficulties (Ramesh and Perera, 2023).

Mitochondrial dysfunction is implicated in PD, with coenzyme Q10 playing a significant role as an electron acceptor in the mitochondrial electron transport chain and an antioxidant. Its levels are reduced in PD patients' blood and platelet mitochondria. Preclinical studies indicate that coenzyme Q10 can protect dopaminergic systems in PD. A phase II trial suggests that it may slow progressive disability in early, untreated PD (Shults, 2005). Several studies have shown that Complex I (part of electron transport chain) present inside the mitochondria does not work well in people who have PD. Because neurons need a lot of energy, when Complex I is impaired, the energy supply becomes poor (Kotrasová *et al.*, 2021). Also, damaged or leaky Complex I allows production of harmful molecules called reactive oxygen species (ROS). These can damage cell components (proteins, fats, DNA), making neurons more fragile. Over time, this oxidative damage, together with energy shortage, protein aggregation (like alpha-synuclein clumping), and other cellular stressors, leads to neuron loss (Terman *et al.*, 2010).

There has also been a study that looked at biomarkers (Biomarkers are objective, measurable indicators of normal biological processes, pathogenic processes, or pharmacological responses to therapeutic interventions) and symptom scores at the same time. In one dose-escalation research, persons with early PD were given more and more CoQ10. At mid-range dosages, this seemed to lower several markers of oxidative stress, but these benefits weren't always shown at higher doses (Seet *et al.*, 2014).

Studies indicate that CoQ10 therapy may improve motor symptoms and cognitive impairment in selected PD patients, with a potential link to personalized medicine. A preliminary study highlighted that patients showing notable improvements in Unified Parkinson's Disease Rating Scale (UPDRS) scores had lower baseline plasma ubiquinol levels and F2-isoprostanes, emphasizing the need for individualized treatment based on these biomarkers, particularly at higher CoQ10 dosages (Jiménez-Jiménez *et al.*, 2022).

A double-blind randomized, phase II, placebo-controlled study utilizing an omics-based approach with CoQ10 was conducted. This study focuses on stratifying treatment groups based on "mitochondrial risk burden" among different categories of Parkin/PINK1 mutation carriers and PD patients, classified as homozygous or compound heterozygous (P++), heterozygous (P+), and omics positive (omics+) or negative (omics-). The primary endpoint assessed changes in UPDRS motor subscores, while secondary endpoints included monitoring motor fluctuations, non-motor symptoms via 31P-magnetic resonance spectroscopy (31P-MRS), and structural and functional brain anatomy changes assessed through MRI (Jiménez-Jiménez *et al.*, 2022).

Alzheimer's disease

Alzheimer's disease (AD) is a brain disorder that gradually destroys memory and other thinking skills. Scientists believe several things happen together in the AD brain that worsen over time-oxidative stress, amyloid-beta (A β) build up, and problems with mitochondria (Breijyeh and Karaman, 2020).

Table 1. Table showing neuroprotective effects of CoQ10

Neurological condition	Pathology	Mechanism/role of CoQ10	Key findings	Key points	References
Parkinson's Disease (PD)	Dopaminergic neuron loss	Supports Complex I function; reduces ROS	May slow disease progression	Reduced CoQ10 levels observed in PD patients	(Shults, 2005; Kotrasová <i>et al.</i> , 2021)
	Oxidative stress	Prevents oxidative damage; improves ATP production	Improves motor and cognitive symptoms in some patients	Personalized response based on biomarkers	(Jiménez-Jiménez <i>et al.</i> , 2022)
		Reduces oxidative stress markers	Mid-range doses more effective than very high doses	Dose optimization required	(Seet <i>et al.</i> , 2014)
Alzheimer's Disease (AD)	Amyloid-beta pathology	Reduces ROS; improves mitochondrial function	↓ Aβ plaques; improved cognition in animal models	Works better in early disease stages	(Dumont <i>et al.</i> , 2011; da Rosa <i>et al.</i> , 2022)
	Tau pathology and oxidative stress	Enhances oxidative phosphorylation	May prevent amyloid and tau pathology	Supports mitochondrial bioenergetics	(Fišar and Hroudová, 2024)
	Cellular studies	Reduces apoptosis; activates survival pathways	Improves neuronal survival <i>in vitro</i>	Dose-dependent neuroprotection	(Nankivell <i>et al.</i> , 2025)
Huntington's Disease (HD)	Energy metabolism defect	Increases ATP production; reduces oxidative damage	Improved survival, motor function in mice	High doses show stronger effects	(Smith <i>et al.</i> , 2006)
Amyotrophic Lateral Sclerosis	Motor neuron degeneration	Antioxidant; mitochondrial support	Increased survival in animal models	Human trials inconclusive	(Kaufmann <i>et al.</i> , 2009)
Stroke	Ischemia/reperfusion injury	Reduces oxidative stress; increases BDNF and SOD	Improved cognition; reduced brain edema (animal study)	Potential neuroprotective therapy	(Fatemi <i>et al.</i> , 2022)
Depression	Oxidative and inflammatory imbalance	Antioxidant; anti-inflammatory	Improved depressive symptoms (200 mg/day)	Lower CoQ10 levels in patients	(Maes <i>et al.</i> , 2009; Mehrpooya <i>et al.</i> , 2018)
Epilepsy	Neuronal hyperexcitability	Reduces oxidative stress; protects neurons	Reduced neurodegeneration in models	Protective against seizure-induced damage	(Baluchnejadmoja rad and Roghani, 2012)
Multiple Sclerosis (MS)	Inflammation and demyelination	Reduces oxidative stress and inflammation	Improved fatigue and depressive symptoms	Promising adjunct therapy	(Hong <i>et al.</i> , 2019; Salekzamani <i>et al.</i> , 2024)

Amyloid-beta is a peptide that is produced normally but tends to accumulate in AD in abnormal forms: oligomers and plaques. These Aβ aggregates are toxic in several ways: they can disrupt cell-to-cell signalling, trigger inflammation, and interfere with mitochondria dysfunction. Mitochondrial dysfunction can stimulate increased Aβ synthesis by altering the processing of amyloid precursor protein (APP) and elevating oxidative stress, which facilitates Aβ formation (Ratan *et al.*, 2023). One of these is CoQ10,

which is naturally found in mitochondria. It helps electrons go through the energy-making cycle and also helps get rid of ROS.

Studies have shown that amyloid beta pathology, tau pathology, mitochondrial dysfunction, and oxidative stress are key contributors to the development AD, highlighting the importance of CoQ10, a vital lipid antioxidant essential for mitochondrial function and bioenergetics.

The regulation of oxidative phosphorylation efficiency via CoQ10 may restore impaired mitochondrial function and prevent amyloid and tau pathology in AD. The article aims to evaluate the potential of CoQ10 and its analogues in AD pharmacotherapy (Fišar and Hroudová, 2024).

Animal studies, particularly in mice, elucidate the potential benefits of CoQ10. In a study utilising the Tg19959 mice model (a transgenic AD model designed to simulate amyloid pathology), characterized by high levels of β -amyloid, early plaque deposition, and cognitive deficits. They express human amyloid precursor protein (APP) with familial AD mutations (KM670/671NL and V717F) under the control of the hamster PrP promoter) (Dumont *et al.*, 2009). Researchers administered CoQ10 through the diet at a dosage comparable to a high dose (Dumont *et al.*, 2011). After a few months several beneficial changes were observed: markers of oxidative stress in the brain decreased; the area and quantity of amyloid-beta plaques significantly diminished in regions such as the hippocampus and the overlying cortex; and levels of the A β 42 peptide, a form particularly linked to toxicity, declined (Félix Javier Jiménez-Jiménez *et al.*, 2023).

Another in vitro study on cultured rat neurons exposed to an amyloid fragment (A β 25-35) which is known to damage neurons showed that adding CoQ10 reduced oxidative stress in a dose-dependent way (higher dose, more protection), reduced signs of cell death (measured by markers like cytochrome c release, caspase activity), and helped survival pathways (e.g. PI3K/Akt signalling) (Nankivell *et al.*, 2025).

In general, these research on animals and cells suggest that CoQ10 can lower ROS damage, lower amyloid-beta levels or plaque load, and make behaviour better (for example, thinking or memory tasks) if treatment starts early. It appears to decelerate or mitigate certain damage rather than reverse highly advanced disease. Animal studies indicate that CoQ10 may penetrate the brain, at least

partially, and enhance mitochondrial function (Yang *et al.*, 2009).

These studies are useful because they reveal that CoQ10 can aid by lowering oxidative stress and making mitochondria work better. This, in turn, lowers amyloid-beta pathology and helps memory and learning in these models (da Rosa *et al.*, 2022).

Huntington's disease (HD) and amyotrophic lateral sclerosis

Huntington's disease (HD) and amyotrophic lateral sclerosis (ALS) are both terrible brain diseases that get worse over time. They have very different clinical pictures, but they are both linked to mitochondrial stress and oxidative damage in early research (Farag *et al.*, 2025).

Researchers therefore investigated whether CoQ10, a key component of the mitochondrial electron transport chain that supports cellular energy production and functions as an antioxidant, could slow disease progression or improve functional outcomes (Tada *et al.*, 2012).

Evidence suggests a bioenergetic defect may contribute to HD pathogenesis. CoQ10 has shown neuroprotective effects in the R6/2 mouse model of HD. A dose-ranging study demonstrated that high doses of CoQ10 significantly improved survival rates, motor performance, grip strength, and reduced weight loss, brain atrophy, and Huntington inclusions in R6/2 mice. CoQ10 treatment elevated brain levels of CoQ10 and ATP while decreasing oxidative damage markers. The results indicate a greater therapeutic benefit from high-dose CoQ10, prompting the need for clinical trials in HD patients (Smith *et al.*, 2006).

Co Q10 was assessed for tolerability and blood levels in a trial involving 28 participants (20 with Huntington's disease and 8 healthy) at dosages of 1,200 to 3,600 mg/day over 20 weeks.

While previous studies showed a potential trend in slowing disease progression at 600 mg/day, higher

doses were evaluated. Subjects started at 1,200 mg/day, with monthly evaluations noting that 82% reached the maximum dosage. Six participants withdrew due to gastrointestinal symptoms, worsening HD, or a fall, but serious adverse events were unrelated to CoQ10. Gastrointestinal symptoms were the most frequently reported adverse effects (Hyson *et al.*, 2010).

CoQ10 is a naturally occurring compound that may aid in the treatment of ALS due to its role as a mitochondrial cofactor and its ability to scavenge free radicals, reducing oxidative stress damage. Animal studies indicate increased survival in ALS transgenic mice treated with CoQ10. Human Phase II trials for Parkinson's Disease suggest potential shared mechanisms with ALS, showing CoQ10's safety and tolerability in ALS patients. Despite its popularity among ALS patients, its efficacy remains unproven in clinical trials, and the ideal dosage is yet to be determined (Kaufmann *et al.*, 2009).

Studies indicate that there is insufficient evidence to support a Phase III trial of high dose CoQ10 for treating ALS. In Stage 1, a dose of 2,700 mg/day was preferred over 1,800 mg/day due to a lower mean decline in efficacy and absence of safety issues. Stage 2 revealed conflicting results regarding futility; while the primary null hypothesis that CoQ10 is at least 20% superior to placebo was not rejected, sensitivity tests did reject it, indicating substantial evidence of futility. Secondary analyses also showed no significant differences between CoQ10 and placebo. Although a Phase III trial with the same primary endpoint scoring for deceased patients may not be futile, the sensitivity of results to the scoring method raises concerns. Thus, while CoQ10 at 2,700 mg/day is deemed safe and tolerable, it does not warrant Phase III testing, as the post hoc slope analysis supports the conclusion of futility (Kaufmann *et al.*, 2009).

CoQ10 shows potential in treating neurodegenerative disorders like ALS, due to mitochondrial dysfunction. A 75-year-old scientist with ALS self-administered CoQ10 (500 mg twice daily, later reduced to 200 mg) beginning in 2005, while tracking his symptoms.

Initially presenting with significant weakness and muscle wasting, he experienced improvements in hand grip power, mood, and sensations within a month of treatment. By 2006, his condition was more stable, indicating slowed progression of muscle wasting and grip strength. These findings suggest that solubilized ubiquinol may benefit ALS patients, necessitating further controlled trials to validate these results (Kawasaki *et al.*, 2012).

Newer trials are refining questions about which patient groups might benefit, which formulations deliver better brain penetration, and whether combining mitochondrial support with other targeted therapies could be more effective. Until a clear, replicated clinical benefit is demonstrated, CoQ10 should be regarded as experimental for Huntington's and ALS safe for most people in typical trial doses, but not yet a replacement for proven symptomatic and supportive care.

Researchers are increasingly looking beyond well-known uses of CoQ10 to see whether it might help in other brain conditions including multiple sclerosis, epilepsy, depression and stroke (Fig. 4).

Stroke

Ischemia/reperfusion (I/R) injury plays a critical role in brain injuries from certain strokes. Chronic post-stroke treatment with CoQ10 (200 mg/kg; PO) for 6 weeks in rats decreased brain edema, reduced anxiety and depressive-like behavior, and improved cognitive impairments. CoQ10 also increased superoxide dismutase (SOD) activities and brain-derived neurotrophic factor (BDNF) levels in the brain tissues of stroke animals (Fatemi *et al.*, 2022). Intravenous injection of solubilized CoQ10 has been shown to quickly and lastingly increase its levels in the brain, providing a basis for further study as a neuroprotective agent in ischemic stroke (Kalenikova *et al.*, 2014).

Depression

Depression is majorly associated with inflammation, oxidative and nitrosative stress, and a lowered antioxidant status. Plasma CoQ10 levels were

significantly lower in depressed patients, particularly those with treatment-resistant depression (TRD) and chronic fatigue syndrome (Maes *et al.*, 2009). CoQ10 supplementation has been suggested as beneficial for depressed patients. Adjuvant CoQ10 (200 mg/d) improved depression symptoms in patients with bipolar depression after 8 weeks of treatment, likely due to its antioxidant and anti-inflammatory properties (Mehrpooya *et al.*, 2018).

Epilepsy

Neurological disorders, including epilepsy, are characterized by biochemical, structural, or electrical abnormalities. CoQ10 has neuroprotective effects against neuronal damage. Studies have investigated the neuroprotective effect of CoQ10 in experimental models of epilepsy, where it has been shown to ameliorate neurodegeneration, mossy fiber sprouting, and oxidative stress (Baluchnejadmojarad and Roghani, 2013, Bhardwaj and Kumar, 2016).

Multiple sclerosis (MS)

Inflammation and immune attacks on myelin are the central problems, yet mitochondrial dysfunction and oxidative stress also contribute to nerve damage and fatigue. Small clinical trials and pilot studies have tested CoQ10 in people with MS and found some promising signals. For example, short courses of CoQ10 (often several hundred milligrams per day for a few months) have reduced blood markers of oxidative stress and inflammation, and in some studies patients reported less fatigue and milder depressive symptoms. CoQ10 may effectively mitigate negative events in multiple sclerosis (MS) and shows promise as a supplement for other neurodegenerative disorders. Research highlights its influence on reducing oxidative stress and inflammation, as well as its potential to alleviate clinical symptoms similar to those of MS, prompting further investigation into its relevance for MS patients (Hong *et al.*, 2019, Salekzamani *et al.*, 2024).

CoQ10 and fertility enhancement

Infertility affects approximately 15% of couples worldwide and is frequently associated with oxidative

stress and mitochondrial dysfunction in both males and females (Mannucci *et al.*, 2022). CoQ10, a lipid-soluble antioxidant and crucial component of mitochondrial bioenergetics, plays a significant role in cellular energy production and the neutralization of ROS, both of which are essential for optimal reproductive function like gamete quality, motility, and fertilization competence (Fig. 4). Clinical and translational evidences show consistent improvements in semen parameters (concentration, total and progressive motility, morphology) and reductions in sperm DNA fragmentation after ≥ 3 months of CoQ10 supplementation, with several randomized and controlled trials and systematic reviews reporting higher pregnancy rates in treated men (Alahmar *et al.*, 2020). Studies suggest that CoQ10 supplementation may reduce cumulus cells apoptosis, resulting in an increase of oocyte quantity and quality. It may also contribute to the increase of IVF success rates by inhibiting DNA oxidation and, thus, oocyte apoptosis. Furthermore, CoQ10 could act as a protector of ovarian reserve from the adverse effects of aging (Florou *et al.*, 2020) (Table 2).

Male fertility

Male infertility is frequently associated with elevated levels of ROS in the seminal fluid. These ROS are unstable molecules that attack critical components of sperm. First, they can cause lipid peroxidation, meaning they oxidize the fats in the sperm cell membrane, making it lose flexibility and integrity. Next, they damage the mitochondrial membranes inside the sperm, undermining the mitochondria's ability to produce ATP — the energy that powers sperm motility (Mannucci *et al.*, 2022). Finally, ROS can break DNA strands inside the sperm's nucleus, causing fragmentation and reducing genetic integrity (Tvrdá *et al.*, 2011).

Because CoQ10 is essential in the mitochondrial electron transport chain (where it helps shuttle electrons to make ATP) and also functions as a potent antioxidant, scientists have tested whether oral supplementation of CoQ10 might improve semen quality (Akhigbe *et al.*, 2025). Several well-designed human trials report

positive outcomes, though not every parameter improves equally (Salvio *et al.*, 2021).

One landmark randomized, placebo-controlled trial by Safarinejad in 2009 enrolled 212 infertile men with idiopathic oligoasthenoteratospermia. These men were randomized to receive either 300 mg CoQ10 daily or a placebo over 26 weeks. The results were convincing: the CoQ10 group showed significantly better sperm concentration and motility compared to placebo. Importantly, CoQ10 levels in seminal plasma rose in treated men and those levels correlated with improved motility, suggesting a dose–effect relationship between CoQ10 in semen and sperm function.

A meta-analysis combining several randomized trials showed that, overall, CoQ10 supplementation improves sperm concentration and motility significantly compared to placebo. But one caveat: data on pregnancy or live birth rates are very limited, so we don't yet know whether these improvements translate into higher chances of fathering a child.

CoQ10 has been shown to be safe and to produce noticeable improvements in sperm count, shape, and movement, especially when taken over several weeks to months in adequate amounts (Lafuente *et al.*, 2013).

Another important study explored ubiquinol, which is the reduced, more “active” form of CoQ10, often believed to be absorbed more readily by the body. In that randomized, placebo-controlled trial for 26 weeks. Men receiving ubiquinol showed significant improvements in several semen parameters: sperm count increased, motility improved, and the percentage of morphologically normal sperm rose compared to baseline and placebo. When the treatment was stopped, those gains gradually slipped, but for some time the values remained above the starting point, hinting at a lasting effect, at least transiently (Safarinejad, 2009).

Putting these trials side by side, a few key points emerge. First, the reduced form (ubiquinol) seems promising for men with idiopathic infertility,

producing clear gains in all three main semen parameters (count, motility, morphology). Second, when compared with another antioxidant (selenium), CoQ10 still shows a stronger or at least comparable effect, indicating it doesn't simply compete but may act in a complementary role. Third, upon stopping supplementation, sperm parameters do decline, although often not back to the very baseline immediately—this suggests repeated or extended dosing might be needed to sustain benefits (Safarinejad *et al.*, 2012).

Recently, a trial compared oral CoQ10 (200 mg/day) to selenium (200 µg/day) over a period of three months in men with idiopathic oligoasthenoteratozoospermia. The CoQ10 group achieved notable gains: sperm concentration rose significantly, both total and progressive motility improved, and antioxidant markers in semen showed favorable shifts. The selenium group also had improvements, but in many endpoints CoQ10 delivered stronger effects. This suggests that CoQ10 not only improves the sperm themselves but also enhances the oxidative environment around them, reducing oxidative damage and boosting antioxidant defence (Alahmar *et al.*, 2020).

Though promising, these trials are relatively small and short in duration. What we don't yet know is the ideal dose, how long treatment should continue, whether combining CoQ10 with other antioxidants or fertility interventions gives better results, and critically whether these laboratory improvements translate into greater pregnancy and live birth rates. But as of now, ubiquinol and CoQ10 remain among the leading candidates for safe, adjunctive therapy in idiopathic male infertility (Florou *et al.*, 2020).

Female fertility

CoQ10, which helps mitochondria produce ATP and neutralize damaging free radicals, has been studied for its potential to boost egg quality and fertility outcomes (Florou *et al.*, 2020). It boosts energy within egg cells by reducing oxidative stress and has been shown to increase fertilization rates (Xu *et al.*, 2018).

Table 2. Table showing role of CoQ10 on male and female fertility and overall fertility enhancement

Key aspect	Mechanism/Role of CoQ10	Key findings	Key points	References
Role in Improving Male Fertility	Prevents lipid peroxidation; protects mitochondrial function; reduces DNA damage	Improves sperm concentration, motility, morphology	Addresses root cause: ROS damage	(Mannucci <i>et al.</i> , 2022; Tvrdá <i>et al.</i> , 2011)
	Enhances mitochondrial ATP production and antioxidant defense	Significant improvement in semen parameters (300 mg/day, 26 weeks)	Dose–response relationship observed	(Safarinejad, 2009)
	Enhances antioxidant environment	CoQ10 shows stronger improvements in semen parameters	May act synergistically with other antioxidants	(Alahmar <i>et al.</i> , 2020)
Role in Improving Female Fertility	Improves mitochondrial ATP production; reduces oxidative stress	Increases fertilization rates and embryo quality	Supports egg maturation	(Xu <i>et al.</i> , 2018)
	Enhances ovarian response to stimulation	↑ oocytes retrieved, embryo quality, pregnancy rates	Effective especially in diminished ovarian reserve (DOR)	(Shang <i>et al.</i> , 2024)
	Restores mitochondrial gene expression	Improved ovarian reserve and offspring in aged mice	Strong mechanistic support	(Ben-Meir <i>et al.</i> , 2015)
	Improves follicular fluid antioxidant status	↑ oocyte maturity, especially >35 years	Direct ovarian microenvironment improvement	(Giannubilo <i>et al.</i> , 2018)
Role in overall Fertility Enhancement	Antioxidant action; supports mitochondrial ATP production; reduces ROS	Improves gamete quality, motility, fertilization competence	Targets oxidative stress and mitochondrial dysfunction in both sexes	(Mannucci <i>et al.</i> , 2022; Alahmar <i>et al.</i> , 2020)
	Enhances semen parameters; reduces sperm DNA fragmentation	Improved pregnancy rates in some male studies	Requires ≥3 months supplementation	(Alahmar <i>et al.</i> , 2020)
	Reduces oocyte apoptosis; protects ovarian reserve	Improves IVF success and oocyte quality	Beneficial in aging ovaries	(Florou <i>et al.</i> , 2020)

In a mouse model, older mice showed a drop in mitochondrial gene expression, lower ATP production, and fewer eggs ovulated. When these aged mice were given CoQ10 in their diets, many of those aging effects were reversed: more follicles were preserved, mitochondrial function picked up again, more eggs matured, and more live offspring were produced. In mice that had a genetic disruption in *Pdss2*, a gene essential for CoQ production, the animals closely mirrored many features of aging ovaries: reduced ATP, increased oxidative damage, and loss of ovarian reserve. Supplementation with CoQ10 restored mitochondrial performance and maintained ovarian reserve (Ben-Meir *et al.*, 2015).

A total of 20 randomized clinical trials involving 2617 participants demonstrated that antioxidants significantly improved the number of retrieved oocytes and high-quality embryos while reducing gonadotropin doses, leading to higher clinical pregnancy rates.

Subgroup analyses indicated enhanced effects with lower doses of CoQ10, which outperformed melatonin, myo-inositol, and vitamins. CoQ10 showed notable advantages over placebo treatments, particularly for women with diminished ovarian reserve, especially those under 35 years. The optimal regimen for CoQ10 is 30 mg per day for three months before controlled ovarian stimulation, suggesting that antioxidants are a safe and effective complementary therapy for ovarian aging (Shang *et al.*, 2024).

A previous study in women aged 31–46 years undergoing IVF-ET compared those receiving 200 mg/day CoQ10 supplementation with unsupplemented controls. In the supplemented group, ovarian follicular fluid showed improved antioxidant status and CoQ10 levels; oocyte (egg) maturity was higher, especially in women over 35. This suggests that CoQ10 reaches the ovary and changes the local environment in ways that support egg maturation (Giannubilo *et al.*, 2018).

Meta-analyses combining multiple randomized controlled trials confirm that CoQ10 pretreatment in women with diminished ovarian reserve can increase the number of oocytes retrieved, improve embryo quality, raise clinical pregnancy rates, and reduce cycle cancellation. However, effect on live birth rates was not clearly proven in all studies aside from pooled studies, individual trials support these benefits at the ovarian level. For example, in women aged roughly between 31 and 46 undergoing IVF or embryo transfer, those taking 200 mg/day of CoQ10 saw higher CoQ10 concentrations in follicular fluid. This increased CoQ10 in the follicular fluid was accompanied by better antioxidant status locally, which means less oxidative stress. Crucially, these women also had improved egg maturity (Lin *et al.*, 2024).

A similar analysis showed that CoQ10 pretreatment reduces the odds of cancelled cycles and also may lower miscarriage rates in women with poor ovarian reserve. Another systematic review found that among various antioxidants used in subfertility settings, CoQ10 tends to outperform others like melatonin or vitamins when it comes to increasing retrieved oocytes, improving embryo quality, and boosting clinical pregnancy rates (Lin *et al.*, 2024; Jiang *et al.*, 2025).

To summarize, evidences from animal studies and human trials together point toward CoQ10's benefit in women's reproductive health by strengthening egg mitochondria, cutting down oxidative stress in the ovary, and ultimately improving both the number and quality of eggs. The strongest effects appear when supplementation starts before or early in an assisted reproduction cycle (Nie *et al.*, 2023). A growing body of recent human studies has reinforced the idea that Coenzyme Q10 (CoQ10) supplementation improves outcomes for women undergoing IVF or embryo transfer, especially those with diminished ovarian reserve (DOR).

One large meta-analysis, titled Clinical evidence of coenzyme Q10 pretreatment for women with

diminished ovarian reserve undergoing IVF/ICSI, pooled data from six randomized controlled trials involving around 1,529 women with DOR. The findings were clear: CoQ10 pretreatment was linked with higher clinical pregnancy rates, more optimal embryos, more oocytes retrieved, and a significant reduction in cycle cancellation. Researchers also saw improvements in hormone levels (estradiol on trigger day) and a lower miscarriage rate (Lin *et al.*, 2024).

Cardiovascular benefits of CoQ10

Cardiovascular diseases (CVDs) remain the leading cause of mortality worldwide, accounting for approximately 17.9 million deaths annually (World Health Organization, 2021). A hallmark of CVDs—including heart failure, hypertension, ischemic heart disease, and atherosclerosis is mitochondrial dysfunction and elevated oxidative stress. These disturbances impair myocardial energy production, increase endothelial dysfunction, and accelerate inflammatory processes (Fig. 4). CoQ10, a key component of the mitochondrial electron transport chain and a powerful lipid-soluble antioxidant, has emerged as a potential therapeutic agent in the prevention and treatment of various cardiovascular conditions (Martelli *et al.*, 2020; Gutierrez-Mariscal *et al.*, 2021) (Table 3).

Heart failure is a chronic condition in which the heart cannot pump blood effectively enough to meet the body's needs. This inefficiency arises in large part because the heart muscle, or myocardium, is unable to generate sufficient energy. Normally, the heart depends on a steady supply of ATP, the molecule that fuels muscle contraction and cellular processes (Sharma *et al.*, 2016). In heart failure, ATP production is significantly impaired, leading to weakened contractile function, disturbed cellular balance, and ultimately, progressive deterioration of cardiac performance (Lee *et al.*, 2012).

CoQ10 plays a central role in mitochondrial energy production. It acts as an essential component of the electron transport chain, the cellular system responsible for generating ATP (Xu *et al.*, 2024).

Beyond this energy-related role, CoQ10 also functions as a powerful antioxidant, protecting cardiac tissue from oxidative stress and cellular damage. Because of this dual function—boosting energy metabolism and reducing oxidative injury—CoQ10 has been investigated extensively as a therapeutic supplement for patients with heart failure (Al Saadi *et al.*, 2021).

One of the most important clinical investigations in this area is the Q-SYMBIO trial. This large, multicenter, randomized, double-blind, placebo-controlled study enrolled 420 patients diagnosed with chronic heart failure. The Q-SYMBIO trial is particularly significant because it was one of the first large-scale studies to demonstrate a mortality benefit with CoQ10 supplementation. Prior to this, much of the supporting evidence came from smaller clinical trials or observational studies. While those earlier works hinted at benefits, they lacked the scale and methodological rigor needed to convince the medical community. By employing a randomized and double-blind design across multiple centers, Q-SYMBIO minimized bias and strengthened the reliability of its conclusion (Mortensen *et al.*, 2014).

The mechanisms behind these benefits are tied to the biology of CoQ10. By enhancing mitochondrial ATP production, CoQ10 helps restore the energy supply needed for effective heart muscle contraction. At the same time, its antioxidant properties reduce oxidative burden within cardiac tissue (Madmani *et al.*, 2014). Oxidative stress is a major contributor to progressive heart muscle damage, as it disrupts cell membranes, impairs protein function, and accelerates inflammation (Mortensen *et al.*, 2014).

Another important consideration is that CoQ10 levels are often found to be reduced in patients with chronic illnesses, including those taking statin medications for cholesterol control. Statins inhibit the mevalonate pathway, which is also involved in CoQ10 synthesis, potentially compounding deficiencies in patients with heart failure. This reduction makes supplementation even more relevant for this population (Claxton *et al.*, 2022).

Despite the encouraging results, some limitations remain. Not all clinical trials have shown equally strong benefits, and questions still exist about the optimal dosage, formulation (ubiquinone versus ubiquinol), and duration of treatment. Moreover, CoQ10 supplementation is generally considered safe, but as with any therapy, patient responses can vary (Claxton *et al.*, 2022).

In summary, heart failure is a condition marked by impaired energy production and oxidative stress, both of which directly weaken heart function. CoQ10, by supporting mitochondrial bioenergetics and reducing oxidative damage, offers a biologically sound and clinically promising therapeutic option (Jafari *et al.*, 2018).

Hypertension and endothelial function

High blood pressure (hypertension) is one of the most controllable risk factors for cardiovascular disease and stroke. It damages blood vessels, forces the heart to work harder, and often leads to other serious complications. Researchers believe that coenzyme Q10 (CoQ10), a natural antioxidant with a central role in energy production, can help lower blood pressure and improve the function of the endothelium—the inner lining of blood vessels—through several mechanisms (Gallo *et al.*, 2022).

Recent studies show that CoQ10 supplementation can effectively reduce systolic blood pressure, particularly at dosages below 200 mg/day and during extended treatment periods.

Its effects on diastolic blood pressure and heart rate are minimal. Given its safety profile, CoQ10 may be a viable option for managing hypertension, especially for patients interested in non-pharmacological treatments or those with mild systolic BP increases (Karimi *et al.*, 2025).

Clinical evidence suggests that CoQ10 offers protection against cardiovascular diseases (CVDs), but its specific effects on diabetes-associated CVD risk require further scrutiny.

A recent meta-analysis of randomized controlled trials indicates that CoQ10 can lower total cholesterol and lipoprotein levels, enhancing endothelial health in individuals with diabetes and metabolic syndrome (Dludla *et al.*, 2020).

Another meta-analysis showed several randomized trials in patients with hypertension found larger drops: systolic pressure fell by about 11-17 mmHg, and diastolic by about 7-10 mmHg following CoQ10 treatment. These larger blood pressure reductions were typically observed in trials where CoQ10 was used along with standard antihypertensive treatments, or in patients with more severe hypertension (Rosenfeldt *et al.*, 2007).

For instance, in statin-treated diabetic patients (who often have impaired endothelial function), adding CoQ10 raised FMD by about 1.0% over 12 weeks. This is meaningful: even a modest improvement in FMD is associated with lower risk of vascular complications. In another study of people with moderate dyslipidemia (but no overt cardiovascular disease), supplementation with the reduced form of CoQ10 (ubiquinol) improved FMD by ~1.3% after eight weeks (Hamilton *et al.*, 2009).

In summary, CoQ10 offers several promising vascular benefits: it can lower systolic blood pressure, improve the function of the blood vessel lining, increase nitric oxide availability, and reduce oxidative stress. These effects, especially when combined with standard therapies, may help reduce the risk and progression of cardiovascular disease in people with hypertension or metabolic disorders.

Anti-inflammatory and lipid-modulating

Long-term inflammation lies at the heart of many cardiovascular diseases. In blood vessels and plaques, persistent activation of immune signals contributes to progression of damage, arterial stiffening, and plaque instability. Because of its dual role in supporting cellular energy and neutralizing free radicals, CoQ10 has been studied for its potential to dampen these inflammatory

processes and favorably influence lipids. CoQ10 may indirectly slow the growth of plaques and stabilize existing lesions. Inflammation recruits immune cells, degrades structural proteins, and promotes thinning of protective fibrous caps around plaques; reducing this burden can improve vascular health and lower the risk of rupture (Sifuentes-Franco *et al.*, 2022).

CoQ10 has been shown to reduce circulating levels of inflammatory messengers. In particular, studies have reported that supplementation can lower levels of tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), and C-reactive protein (CRP)—all molecules commonly elevated in atherosclerosis and cardiovascular disease. For example, a randomized trial carried out in patients with coronary artery disease found that taking CoQ10 for 12 weeks significantly decreased serum TNF- α and IL-6 compared to baseline (McRae, 2022).

Evidence indicates that patients with infections like influenza and COVID-19 have significantly lower endogenous CoQ10 levels compared to healthy individuals, correlating with inflammatory biomarkers. A computational study suggests that CoQ10 may act similarly to anti-inflammatory drugs such as methylprednisolone and embelin, which inhibit viral infections through various mechanisms and demonstrate activity against influenza and hepatitis B (Sifuentes-Franco *et al.*, 2022).

The nuclear component TNF- α and IL-6 gene expression are among the many proinflammatory cytokines that are up-regulated by the inducible transcription factor kappa B. 21 Regarding coenzyme Q10's mode of action, it has been established that it binds to peroxisome proliferator-activated receptor gamma (PPAR- γ), and that PPAR- γ inhibits NF κ B activation when coenzyme Q10 binds to this nuclear receptor protein. 7, 22, and 23 Thus, PPAR- γ -mediated coenzyme Q10 antagonism of NF κ B suppresses the expression of proinflammatory cytokines TNF- α and IL-6 (McRae, 2022).

Table 3. Table showing cardiovascular benefits of CoQ10

	Key aspect	Mechanism / role of CoQ10	Key findings	Key points	References
Heart failure	Energy deficiency in myocardium	Enhances ATP production; protects against oxidative damage	Improves cardiac function and reduces mortality (Q-SYMBIO trial)	Dual benefit: bioenergetics + antioxidant protection	(Mortensen <i>et al.</i> , 2014; Madmani <i>et al.</i> , 2014)
	Clinical evidence	Supports mitochondrial efficiency	Reduced major adverse cardiac events in 420 patients	One of the strongest RCTs supporting CoQ10	(Mortensen <i>et al.</i> , 2014)
	Statin interaction	Statins inhibit mevalonate pathway → ↓ CoQ10 synthesis	Supplementation may restore depleted levels	Important in patients on cholesterol-lowering drugs	(Claxton <i>et al.</i> , 2022)
Hypertension and Endothelial Function	Blood pressure reduction	Improves nitric oxide availability; reduces oxidative stress	↓ SBP (~4.77 mmHg avg; up to 11–17 mmHg in some trials)	More effective for systolic than diastolic BP	(Zhao <i>et al.</i> , 2022; Rosenfeldt <i>et al.</i> , 2007)
	Dose-response	U-shaped response	Optimal dose: 100–200 mg/day	Longer duration (>12 weeks) more effective	(Zhao <i>et al.</i> , 2022)
	Endothelial function	Improves flow-mediated dilation (FMD)	↑ FMD (~1–1.3%)	Indicates improved vascular health	(Hamilton <i>et al.</i> , 2009)
	Metabolic conditions	Improves lipid profile in diabetes	↓ cholesterol and lipoproteins	Benefits stronger in diabetics/dyslipidemia	(Dludla <i>et al.</i> , 2020)
Anti-inflammatory and Lipid Effects	Anti-inflammatory action	Inhibits NF-κB via PPAR-γ activation	↓ TNF-α, IL-6, CRP	Reduces chronic vascular inflammation	(McRae, 2022)
	Plaque stability	Reduces oxidative stress and immune activation	Slows plaque progression	May reduce risk of rupture	(Sifuentes-Franco <i>et al.</i> , 2022)
	Lipid protection	Prevents LDL oxidation	Protects against atherosclerosis	Early-stage plaque prevention	(Chiurchiù and Maccarrone, 2016)
	Statin-associated muscle symptoms	Restores CoQ10 levels	Mixed results: symptom relief in some studies	Evidence inconsistent across trials	(Šabovič, 2014; Hovland <i>et al.</i> , 2020)

Several randomized controlled trials and reviews have examined whether adding CoQ10 can mitigate these muscle side effects. Some meta-analyses found that CoQ10 supplementation improved symptoms such as muscle pain, cramps, fatigue, or weakness in statin users, even though it did not significantly lower creatine kinase (a marker of muscle damage) in the bloodstream. For instance, a pooled review of twelve trials showed significant reductions in muscle pain, cramping, and weakness with CoQ10 versus placebo, although no consistent changes in creatine kinase levels were seen (Šabovič, 2014).

However, not all evidence is conclusive. Some more recent meta-analyses report no clear benefit of CoQ10 over placebo in alleviating statin-related muscle symptoms. The contradictory findings may result

from differences in study design, duration, doses of CoQ10 used, or variability in how muscle symptoms were defined and measured (Hovland *et al.*, 2020).

Putting it all together, the combined anti-inflammatory and lipid-protective effects of CoQ10 make it a biologically plausible adjunct in cardiovascular care. By lowering TNF-α, IL-6, and CRP, it helps quiet chronic vascular inflammation—a root cause of many pathologies. By reducing LDL oxidation, it protects arteries from one of the initial steps in plaque formation.

And by potentially countering CoQ10 depletion from statins, it may help some patients continue lipid-lowering therapy with fewer side effects (Chiurchiù and Maccarrone, 2016).

Clinical Safety and Dosage

CoQ10 is generally very well tolerated, with only mild side effects reported in most clinical trials. Common complaints are limited to gastrointestinal issues like stomach upset, nausea, diarrhea or mild abdominal discomfort. Some people taking higher daily doses have reported mild increases in liver enzyme levels, occasional insomnia, or minor complaints like headache or dizziness. Importantly, serious adverse events are rare, even with long-term use (Hathcock and Shao, 2006).

Evidence of cardiovascular benefit from CoQ10 is strong across diverse populations and multiple clinical outcomes. Trials have found that CoQ10 enhances energy metabolism in heart muscle, supporting better ATP production, improves how well blood vessels dilate (endothelial function), reduces inflammation markers, and helps stabilize or slow the progression of atherosclerotic plaques. These effects have been observed in people with heart failure, hypertension, and other cardiovascular disorders (Zozina *et al.*, 2018).

Overall, given its strong safety profile and multiple mechanisms of action boosting heart cell energy, supporting the blood vessels, reducing oxidative stress and inflammation, and possibly helping stabilize plaques CoQ10 appears to be a potentially valuable adjunct in managing cardiovascular disease. What remains is further work to standardize dose regimens, understand which formulation works best under specific circumstances, and gather long-term data on outcomes to better inform clinical guidelines (Rabanal-Ruiz *et al.*, 2021).

Other Potential Applications

Coenzyme Q10 has been studied beyond heart disease, and several lines of research suggest it may have potential in other health areas, though the evidence is mixed and often preliminary.

In cancer research, CoQ10 plays a somewhat controversial role. On one hand, it's well known for its antioxidant qualities, which can protect cells from damage by reactive oxygen species (ROS). Since

oxidative stress contributes to DNA mutations and cancer initiation, CoQ10 might theoretically help prevent cancer. Some animal studies have shown that CoQ10 reduces early cancer markers, for example, it lowered numbers of aberrant crypt foci (precancerous lesions) in rat colons exposed to carcinogens, while also reducing overexpression of inflammatory or pro-tumor genes (Dabaghi *et al.*, 2024).

On the other hand, at higher doses or under certain conditions, CoQ10 may behave in a pro-oxidant way in cancer cells generating ROS to damage them and thus could potentially enhance the effects of some chemotherapy agents. For example, in vitro work combining CoQ10 or ubiquinone with the chemotherapy drug doxorubicin in breast cancer cell lines showed suppression of cancer cell growth, possibly via increases in ROS, lipid peroxidation, or reduced antioxidant enzyme activity in those cancer cells. Along with these cellular and animal studies, a systematic review has found that CoQ10 might help reduce some toxic side effects of cancer treatment (such as liver or heart toxicity), especially when anthracyclines are used—but the human trials so far are small and somewhat inconsistent (Dadali *et al.*, 2021).

In mitochondrial diseases, especially primary CoQ10 deficiency (genetic defects in the enzymes that produce CoQ10), CoQ10 can be directly beneficial. Primary CoQ10 deficiency is rare, and patients present with a wide variety of symptoms—encephalopathy, muscle weakness, kidney problems, cerebellar ataxia among them. In some of these genetic disorders, supplementation with CoQ10 or its more bioavailable form ubiquinol has improved mitochondrial respiratory chain activities and reduced neurological damage in animal models (Salviati *et al.*, 2017).

In dermatology and skin health, CoQ10 has been tested mostly in topical creams or lotions, and in small clinical/lab studies. In human keratinocytes and skin fibroblasts (cells of the skin), CoQ10 applied topically has been shown to reduce oxidative stress when skin is exposed to UV light, to reduce wrinkle formation or deepen fine lines (“crow’s feet”), and to improve mitochondrial

membrane potential, cell vitality, and intracellular antioxidants (Lopriore *et al.*, 2024).

To summarize, CoQ10 shows potential across many areas beyond cardiovascular disease. It may help in certain cancers (either protective or as part of treatment), improve insulin sensitivity and reduce inflammation in metabolic diseases, correct mitochondrial dysfunction in genetic disorders, slightly influence aging processes, and benefit skin health via topical or possibly systemic routes. But nearly everywhere, the pattern is that effects tend to be modest, often seen in small or short-term studies, and the benefits depend a lot on timing, formulation, dose, and individual patient context. Future work will need larger, longer trials, better standardization of form and dose, and clearer identification of which patient groups are most likely to benefit.

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