REPORT

Alleviating Congestive Heart Failure with Coenzyme Q10

By Peter H. Langsjoen, MD, FACC

One of the most frequent causes of hospital admissions in older adults is the devastating condition known as congestive heart failure. Characterized by disabling symptoms of difficulty breathing, fatigue, and swelling of the extremities, congestive heart failure also increases the risk of early demise.

Fortunately, scientists have discovered that the mitochondrial energizer coenzyme Q10 (CoQ10) can offer powerful assistance to those challenged with congestive heart failure, improving the heart’s pumping ability and even reducing the need for medications.

Since CoQ10 levels are depleted by aging and statin medications and tend to be low in those with congestive heart failure, achieving optimal blood levels of CoQ10 may be an important strategy for safeguarding cardiac health.

WHAT IS COQ10?

It has been 50 years since Fred Crane discovered CoQ10 in 1957. Since that time, scientists have discovered what this extraordinary molecule is and what it does in settings of both health and disease. It is important to clarify that a coenzyme should not be confused with an enzyme (a protein that accelerates a biochemical reaction). A coenzyme is a simple molecule (many vitamins are coenzymes) that is essential for the normal function of specific enzyme systems in our cells. Coenzyme Q10 is the cofactor or coenzyme for three large enzyme systems that are essential for 90% of cellular energy production. Because the heart muscle uses more energy than any other tissue and normally has the highest concentration of CoQ10, it is very sensitive to CoQ10 deficiency.

Coenzyme Q10 is well established to be a clinically relevant first-line antioxidant in our defense system against excess oxidative stress. It is the only fat-soluble antioxidant that is synthesized by our body and is capable of regeneration back to its reduced or antioxidant form through normal cellular enzyme systems. Its location in the lipid mitochondrial membranes is particularly important, as mitochondria are the major site of free-radical production, and CoQ10 is an excellent free-radical scavenger.

COENZYME Q10 AND CONGESTIVE HEART FAILURE

In the late 1960s and early 1970s, it became clear that patients suffering from congestive heart failure had measurable deficiency of CoQ10 in both blood and heart muscle, and that the degree of deficiency correlated with the severity of heart failure. Much of this work was performed as a collaborative effort between Professor Karl Folkers, PhD, Gian Paolo Littarru, MD, and Denton Cooley, MD. In 1980, my father, cardiologist Per H. Langsjoen, MD, met with Karl Folkers, and together they performed the first human trial of CoQ10 in the treatment of congestive heart failure in the United States. I joined my father as a cardiology fellow in 1983 and after this favorable controlled study was published in 1985, we went on to evaluate the long-term efficacy of CoQ10 therapy in 126 patients with congestive heart failure.

Congestive heart failure is a condition in which there is weakening of heart muscle function so that fluid or congestion backs up and causes swelling or edema in the liver, lungs, the lining of the intestine, and the lower legs and feet. It was our initial concern that CoQ10 may have been
A high prevalence of skeletal muscle pain and weakness at 64% on initial visit was reduced to 6% in follow-up.
Fatigue decreased from 84% to 16%.

Shortness of breath went from 58% down to 12%.

Memory loss was reduced from 8% to 4%.

Peripheral neuropathy decreased from 10% to 2%.

There were no adverse effects from stopping statin drug therapy with no cases of heart attack or stroke during follow-up. Overall, there was an improvement in heart muscle function on discontinuation of statin therapy and addition of supplemental CoQ10. However, due to powerful propaganda surrounding both cholesterol and statin drug therapy, many patients and physicians are afraid to stop statin therapy.

**CHOLESTEROL AND HEART DISEASE**

Atherosclerosis remains a disease of unknown cause. Many factors more important than cholesterol—such as stress, smoking, hypertension, insulin resistance, high triglycerides, diabetes, and low testosterone (in men)—contribute to atherosclerosis and cardiovascular disease.17-19 Despite this, the theory that cholesterol is the dominant villain responsible for atherosclerosis has been promulgated for over 60 years, making the pharmaceutical industry’s anti-cholesterol campaign the most profitable medical myth of all time.

Statin drugs do show some benefit in reducing mortality in individuals with pre-existing coronary artery disease.20 This benefit occurs irrespective of cholesterol lowering and is likely secondary to their subtle anti-inflammatory or plaque-stabilizing effects.21 The vilification of cholesterol and the associated aggressive lowering of cholesterol blood levels has brought about increasingly severe CoQ10 deficiency in a large number of patients, making it absolutely critical to restore CoQ10 levels in these individuals.

**OPTIMAL COQ10 LEVELS IMPROVE HEART FAILURE**

In the early years of our experience with CoQ10 therapy, no one knew the therapeutic or ideal plasma level of CoQ10 for the treatment of heart failure. Over the course of 25 years, it has become clear that maximum improvement in heart function will not occur unless plasma levels are greater than 3.5 micrograms per milliliter (mcg/mL). By 2006, there were a total of 22 randomized, controlled trials involving a total of 1,605 patients evaluating the therapeutic benefit of supplemental CoQ10 in congestive heart failure.22-24 The majority of studies were favorable, showing significant improvement in heart muscle function. Furthermore, there have been 34 open-label trials involving 4,221 patients evaluating the clinical utility of CoQ10 in heart failure, and again clear benefits were observed without any adverse effect or drug interaction. Despite these studies, CoQ10 remains obscure to most physicians and is not routinely used in the treatment of congestive heart failure. This is in part due to the pervasive anti-nutrient bias in conventional medical practice, medical literature, and medical education.

Out of a total of 22 controlled trials of supplemental CoQ10 in congestive heart failure, only three have failed to show significant benefit. The first study by Permanetter et al. failed to measure plasma CoQ10 levels, such that there is no way to know if therapeutic CoQ10 levels were attained.25 The second trial by Watson et al. demonstrated a mean treatment plasma CoQ10 level of only 1.7 mcg/mL, with only two of the 30 patients having a plasma level greater than 2.0 mcg/mL.26 Finally, the third study by Khatta et al. demonstrated a mean treatment plasma CoQ10 level of 2.2 ± 1.2 mcg/mL, indicating that some patients on treatment had levels as low as 1.0 mcg/mL.27 Unfortunately, these last two trials with sub-therapeutic CoQ10 levels are the most frequently quoted as evidence for a lack of benefit for CoQ10 in heart failure.
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ABSORPTION OF COQ10

From the beginning of our experience with CoQ10 in heart failure, we have observed poor absorption of CoQ10 in patients with advanced congestive heart failure. This is extremely frustrating, because the patients who are the most ill and have the lowest plasma CoQ10 levels show minimal improvement because of their inability to absorb CoQ10. It has been our assumption that the fluid retention or edema in the intestine and liver in these critically ill patients has been responsible for this poor absorption. Up until approximately one year ago, all supplemental CoQ10 in the world has been in its oxidized or ubiquinone form. In this oxidized state, CoQ10 is stable, bright orange in color, and fat-soluble. It is this property of being fat-soluble that is responsible for the difficulty with CoQ10 absorption.

A BETTER FORM OF COQ10

After ingestion, immediately after CoQ10 crosses into the first cells lining the small intestine, it is enzymatically converted to its reduced, or ubiquinol, form. So, when we measure plasma CoQ10, approximately 98-99% of the CoQ10 is in this reduced ubiquinol form (see figure 3). Kaneka Corporation of Japan has succeeded in making a stable ubiquinol formulation that we have been carefully studying since October 2006. Ubiquinol is a white powder rather than orange and is less fat-soluble, making it better absorbed.

Our first patient to be treated with ubiquinol had severe heart failure with a 15% ejection fraction (a measurement of the amount of blood pumped out with each heartbeat, which is normally 60-65%). This gentleman had a sub-therapeutic plasma CoQ10 level of 2.2 mcg/mL on 450 mg per day of ubiquinone. His plasma level on 450 mg per day of ubiquinol increased dramatically to 8.5 mcg/mL and over the subsequent 10 months, his ejection fraction increased to 60% with a corresponding dramatic and perhaps life-saving clinical improvement (see figure 4). We now have a total of seven patients with advanced congestive heart failure and low plasma CoQ10 levels, despite taking as much as 600 mg per day of ubiquinone (the oxidized form of CoQ10) who have been changed over to the ubiquinol (the reduced form) formulation. Our preliminary observations have been very favorable, and it is my strong opinion that supplemental ubiquinol represents a major scientific advance in the 50-year history of CoQ10 research.

SUPPLEMENTING WITH COQ10

I would like to make a few practical comments based on 24 years of treating thousands of heart failure patients with supplemental CoQ10 in addition to standard prescription medications. We have seen no side effects and no drug interactions from supplemental CoQ10, but we have observed a gradual lessening of the requirement for many cardiac medications that occurs with an improvement in heart muscle function. For example, we have observed a significant decrease in the need for diuretics, because of a reduced tendency for fluid retention as heart function improves. Also, we have noted a gradual improvement in hypertension that occurs as heart function improves, which may require a gradual decrease in antihypertensive medications such as angiotensin-converting enzyme (ACE) inhibitors, a class of blood pressure-lowering medicines frequently used in heart failure patients. Patients with heart disease should be followed by their physicians, particularly when there are any changes in activity, diet, prescription drugs, or over-the-counter supplements.
I have treated hundreds of patients with CoQ10 and have never observed an interaction with warfarin (Coumadin®). There have been anecdotal reports that the combination may increase the risk of bleeding. Individuals who use warfarin should always consult a physician before using CoQ10.\textsuperscript{28,29}

**CHOOSING A COQ10 FORMULATION AND DOSAGE**

Most commercially available CoQ10 supplements comprise ubiquinone. Recommended daily dosages of this type of CoQ10 range from 100 mg to 600 mg.

The most advanced CoQ10 formulas now contain ubiquinol, the reduced form of CoQ10, which is definitely better absorbed into the bloodstream.\textsuperscript{30-32} Recommended daily dosages of ubiquinol range from 100 mg to 300 mg.

Because we know that CoQ10 levels tend to decrease with age and we live in a society that consumes very little food rich in CoQ10 (organ meats like heart, liver, and kidney), it makes sense to supplement with a modest amount of CoQ10 (ubiquinone or ubiquinol) beginning in middle age. Those who suffer from congestive heart failure or who use statin medications should aim to consume higher doses of CoQ10 (ubiquinone or ubiquinol).

**CONCLUSION**

Coenzyme Q10’s ability to fundamentally improve the production of energy and the antioxidant defense in every cell of the body has brought about many remarkable and unexpected improvements in all aspects of human health. This extraordinary molecule has dramatically changed my own practice of medicine and has brought joy to the treatment of previously devastating cardiovascular diseases.

If you have any questions on the scientific content of this article, please call a Life Extension Health Advisor at 1-800-226-2370.

**References**


9. Alsheikh-Ali AA, Maddukuri PV, Han H, Karas RH. Effect of the magnitude of lipid lowering on risk of elevated liver enzymes,


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