CoQ10 Combats Congestive Heart Failure

Patients diagnosed with congestive heart failure have been shown to have low levels of CoQ10, which can increase their chance of death by 50% within five years. Using the proper form and dose of CoQ10 can reverse these underlying pathologies and promote cardiovascular health and longevity.

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Congestive heart failure is one of the most devastating forms of cardiovascular disease.[1]

More than 5.8 million people in the US are affected by congestive heart failure. If you're diagnosed with **congestive heart failure** today, you have a **50/50** chance of being dead within **five years**.[1]

Those aren't good odds. But researchers have determined that with the help of **CoQ10**, you can beat these odds.

Mainstream medicine treats congestive heart failure with a barrage of medications including beta blockers, ACE inhibitors, diuretics, digoxin, nitrates, aldosterone antagonists, anticoagulants, and glucose-lowering drugs. These drug combinations have added considerable years of life to patients with congestive heart failure.[2-7]

Overlooked by most cardiologists, however, are published clinical studies showing that CoQ10 can dramatically improve treatment outcomes when properly used in conjunction with conventional treatments.

A recent international, multicenter study of patients with moderate-to-severe **heart failure** demonstrated, by the most conservative analysis, a **50%** reduction in major cardiovascular events (strokes, heart attacks, etc.) and a **44%** reduction in cardiovascular deaths, in response to CoQ10 supplementation.[4]

This dramatic outcome validates earlier studies demonstrating the utility of CoQ10 in managing heart disease. It also points to the potential role CoQ10 plays in the ***prevention***, rather than treatment, of heart failure and other cardiovascular diseases.

WHAT IS CONGESTIVE HEART FAILURE?

Congestive heart failure is the inability of the heart to pump sufficient blood to meet the needs of all organs in the body, and is frequently the result of other, preventable factors such as high blood pressure, diabetes, and coronary heart disease (which causes heart attacks).[1]

Congestive heart failure results from a progressive weakening of the heart muscle, which is usually a result of insufficient production of ATP (adenosine triphosphate), the energy that fuels your heart.[34-36] In a healthy heart with ample energy in the form of ATP, the heart muscle is well-developed and thick, and it effortlessly pumps blood out of the left ventricle into the aorta and out into the body.[1]

But with inadequate ATP, which occurs from impaired energy transport, the robust heart muscle weakens and becomes flabby, resulting in relatively ineffective pumping action, so that blood pools in the heart.[1] We refer to this slowed and inefficient movement of blood in the heart as "congestive" heart failure. The major symptoms of congestive heart failure arise from this backup of blood in the weakened left ventricle.

CoQ10 Helps Prevent Heart Failure

CoQ10 has been shown to ***prevent*** underlying pathological disorders that produce heart failure. This includes

reducing atherosclerosis risk factors, improving endothelial function, and protecting against heart damage.[8,9] Here is a summary of the encouraging data supporting the role of CoQ10 in heart disease prevention:

CoQ10 Protects Against Arterial Occlusion

Atherosclerosis ("hardening of the arteries") underlies virtually all heart attacks, strokes, and other blood vessel diseases.[10-12] There are numerous risk factors that are associated with the onset of atherosclerosis, including LDL oxidation, chronic inflammation, elevated blood glucose, elevated lipid levels, and disordered growth factor signaling.[13-17] Published studies show that CoQ10 combats many of those risks. For example:

- Heart attack survivors who took **120 mg** a day of CoQ10 for one year reduced the rate of total cardiac events and nonfatal heart attacks by **45** and **46%**, respectively, compared to controls, while beneficial HDL cholesterol rose significantly.[8]
- When compared to patients on statins, those taking **60 mg** a day of CoQ10 favorably modified numerous atherosclerosis risk factors, including lipid profiles, platelet clumping, and oxidative stress.[18]
- In adults at intermediate risk for atherosclerosis who took a combination of CoQ10 (**120 mg** a day) and aged garlic extract (**1,200 mg** a day) for one year, atherosclerosis progression was **4-fold** lower compared with control subjects, while markers of atherosclerosis-promoting inflammation were significantly reduced.[19]
- CoQ10 helps prevent **low-density lipoprotein** (LDL) cholesterol from oxidizing and triggering arterial plaque formation.[20]

WHAT YOU NEED TO KNOW

CoQ10 Treats And Prevents Heart Disease

- Cardiovascular disease remains the leading killer of American adults.
- In particular, congestive heart failure, which can arise from hypertension, atherosclerosis, endothelial dysfunction, and heart attacks, prematurely sickens otherwise healthy people.
- Some forms of cardiovascular disease involve some degree of energy mismanagement at the cellular level, leaving heart and blood vessel cells weakened and incapable of proper function.
- CoQ10 is essential for transferring energy from food into ATP molecules, the universal cellular energy currency.
- Studies show that supplementation with CoQ10 augments heart and vascular function, improves clinical status, and prevents further damage from cardiovascular disease.
- CoQ10 may represent the single most vital supplement that everyone should take to sustain and support cardiovascular energy management.

CoQ10 Improves Endothelial Function

Dysfunction of the ultrathin cell layer lining arteries, the ***endothelium***, is a major early risk factor for the development of atherosclerosis and cardiovascular disease.[21,22] This ***endothelial dysfunction*** is especially prevalent in those with diabetes and/or lipid abnormalities.[23] Numerous studies have demonstrated that CoQ10 directly addresses multiple causes of endothelial dysfunction:

- In patients with diabetes, 12 weeks of supplementation with **200 mg** a day of CoQ10 significantly increased endothelial function in a major artery.[23]
- In a group of men with known endothelial dysfunction, CoQ10 supplementation improved endothelial function **significantly** compared with baseline.[21]
- In patients with mild-to-moderate heart failure, **300 mg** a day of CoQ10 improved endothelial function **38%**, an effect comparable with that of exercise training.[24]
- Blood vessel relaxation, a measure of endothelial function and blood flow, improved significantly in patients with known coronary artery disease at risk for heart attack who need optimal cardiac blood flow.[25]

NEW YORK HEART ASSOCIATION (NYHA) FUNCTIONAL CLASSIFICATION[81]

Class	Patient Symptoms
Class I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).
Class II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
Class III (Moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
Class IV (Severe)	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.

The New York Heart Association (NYHA) Functional Classification system has become the standard for measuring symptoms that affect functioning related to heart failure.[81]

CoQ10 Protects Against Heart Damage

Coronary artery disease typically refers to the consequences of atherosclerosis of the coronary arteries, which are the arteries that supply blood and nutrients to the heart muscle itself. Early disease may appear as painful and ominous ***angina*** (chest pain), while more advanced disease produces ***ischemia*** (lack of blood flow), and eventually ***infarction*** (death of heart muscle) in a classical heart attack.[26] Numerous studies have shown that in the event of a heart attack, ample levels of CoQ10 may mitigate the amount of damage:

- During open-heart surgery, when the heart is briefly stopped and ischemia is a major risk, CoQ10 protected heart muscle pumping and cardiac output.[27]
- In functioning hearts from old rats, pretreatment with CoQ10 improved recovery of heart function following stress.[28]
- In human atrial heart muscle tissue, CoQ10 treatment abolished the decreased ability to recover from ischemia seen in older hearts, producing a recovery pattern similar to that in younger hearts; this effect was shown to be related to improved mitochondrial energy efficiency in the treated tissue.[28,29]
- In humans undergoing elective heart surgery, patients treated with CoQ10 had lower levels of markers of heart damage, improved pumping action, and shorter hospital stays, compared with untreated controls.[28]

SYMPTOMS OF CONGESTIVE HEART FAILURE

When blood flow through the heart becomes congested, blood backs up throughout the body. The result is an accumulation of fluid that is squeezed out of the capillaries,[82] the tiniest of blood vessels found in all tissues.

The most evident symptoms of congestive heart failure reflect this process: As the lungs become heavy and fluid-filled, patients experience shortness of breath during normal, non-strenuous activities, and have trouble breathing when lying down; as fluid builds up in the abdomen and extremities, patients experience weight gain, with swelling of the feet, legs, ankles, or stomach.[1]

Most patients also report feeling generally tired or weak, as the heart becomes less and less able to meet the body's demand for oxygen and nutrients.

CoQ10 supplementation helps restore the heart's normal energy economy, allowing heart muscle to regain its youthful strength, to resume its robust pumping action, and to reduce symptoms and disability induced by congestive heart failure.

CoQ10 And Cellular Energy

Some forms of cardiovascular disease involve energy mismanagement at the cellular level, weakening heart and blood vessel cells and leaving them incapable of proper function.[30-33] This is especially true of congestive heart failure.

Despite its name, congestive heart failure is not a situation in which the heart stops beating. Rather, it results from a progressive weakening of the heart muscle, which is characterized by insufficient **ATP** (adenosine triphosphate) production.[34-36]

CoQ10 supplementation has repeatedly been shown to improve heart muscle function in patients with heart failure, supporting the scientific observation that heart failure is caused by a deficit in cellular energy.[37,38] This includes improvement of heart muscle movement, increased cardiac output (the amount of blood pumped per minute), ejection fraction (proportion of blood pumped out with each stroke), and other technical measures.[39-43]

But are you taking the right amount—and the right type—of CoQ10 to extend your life span? Chances are, the answer is no.

Even fairly low doses of CoQ10 have been shown to reduce the symptoms associated with congestive heart failure. For example, in a three-month open study, an average daily dose of **100 mg** a day of CoQ10 improved symptoms in a large majority of patients, including swelling, blueness (cyanosis), difficulty breathing, heart palpitations, sweating, insomnia, vertigo, and nighttime urination. In fact, **54%** of patients experienced improvement in at least three such symptoms.[44]

But those low doses can be deceiving because even though they improve symptoms, ultimately few meaningful benefits in terms of survival or improved functioning were shown at CoQ10 doses of **100 mg** a day.[44] This is especially true for people who already have *advanced* congestive heart failure (Class IV) because fluid build-up in the walls of their intestines reduces the amount of a given dose that can be absorbed into the bloodstream.[45]

A study published in *The Clinical Investigator* shows us the difference that even modest increases in dosing makes: It demonstrated that an increased dose of CoQ10 produced more than just symptom reduction—it kept patients out of the hospital.

For this study, when patients with moderate-to-severe (class III and IV) heart failure took **2 mg/kg** a day (about **190 mg** a day in an average-sized man, and about **150 mg** a day for an average woman) of CoQ10 for one year, it significantly reduced hospitalization rates by **38%**.[38] In the same study, symptoms caused by fluid backing up into the lungs (pulmonary edema, "cardiac asthma") were reduced by **61** and **51%**, respectively.

But even with those increased doses and beneficial results, we're still falling short of CoQ10's full life-extending potential. Changing long-term outcomes (like reducing mortality) clearly requires higher doses for longer periods.

FIGHT BACK AGAINST STATIN-INDUCED COQ10 DEFICIENCY

Mainstream medicine relies on drugs called ***statins*** to help lower cholesterol in an effort to prevent atherosclerotic heart disease. But a looming side effect of statins is that they deplete your heart muscle of CoQ10, in fact leaving you *more* vulnerable to congestive heart failure.[83,84]

Fortunately, as shown by recent studies, people taking statins can benefit from supplemental CoQ10 at **200** to **300 mg** a day.[84,85] Patients not only had improvements in CoQ10 levels and in natural free radical defense systems, but also had objective improvements in ejection fraction and in their NYHA functional classification as well.[84,85]

CoQ10 And Longevity

To achieve benefits measured by longer life spans, you need to increase the dose and amount of CoQ10 ***absorbed*** into the bloodstream.

Surprisingly few studies have bothered to measure blood levels of CoQ10 in patients, a basic step in gauging the effectiveness of a dosing program. What we do know is that leaders in the field have demonstrated that blood levels of more than **3.5 micrograms/mL** are required to reliably produce improvements in cardiac function.[24,46,47] Doses of standard CoQ10 (also called ***ubiquinone***) of at least **240 mg** a day may produce such elevations in blood levels, while lower doses rarely do so.[47,48]

A better way to achieve optimal blood levels of CoQ10 is to use a superior form of CoQ10 called ***ubiquinol***. In one particularly impressive study, patients with severe heart failure (average of class IV) had mean CoQ10 levels of just **1.6 micrograms/mL** even though they were taking **450 mg** a day of standard CoQ10. Once they changed to an average of **580 mg** a day of **ubiquinol**, their blood CoQ10 levels shot up to **6.5 micrograms/mL** and their mean ejection fraction improved **77.3%** from baseline. In addition, their **NYHA class** improved from a mean of **class IV** to a mean of **class II**, demonstrating substantial improvement in their ability to carry out tasks of daily living.[45]

Dramatic proof of the effectiveness of higher doses of standard CoQ10 for longer periods comes from the most recent large clinical trial, conducted by an international group of cardiologists.[49] In this study, patients with moderate-to-severe heart failure took either a placebo or **300 mg** a day of CoQ10 (in the lesser absorbable form called ubiquinone) for two years. Patient data was examined at 16 weeks (short term) and at the end of the study (long term). No meaningful changes were seen in any patients at the short-term data point (16 weeks) using the ubiquinone form of CoQ10.

In the same study, by the two-year mark, however, supplemented patients were half as likely to experience a major adverse cardiovascular event, compared with placebo recipients.[49] In addition, significantly more placebo patients died a **cardiovascular death** compared with supplemented subjects (**16** versus **9%**), while **deaths** from all causes were **18%** in placebo patients, and just **10%** in supplemented ones. The rate of heart failure-related **hospital stays** was also significantly lower in patients taking CoQ10 than in controls.

These studies indicate that making energy safely and abundantly available to heart muscle through CoQ10 supplementation at reasonable doses for a prolonged period is a powerful way to reduce the impact of congestive heart failure.

It is important to note here that in all studies, patients remained on their regular medications. This is important to emphasize as people with existing heart failure should use CoQ10 as a *long-term heart-strengthener* and not as a replacement for prescription medications.

UBIQUINOL: THE OPTIMAL FORM OF COQ10

Whenever tissues are deficient in CoQ10, their energy-providing mitochondria throughout the body suffer.[87] That's because CoQ10 is an essential component in the transfer of electrons, nature's tiniest unit of energy, from chemical bonds in food molecules to chemical bonds in the ATP molecules all tissues use as an immediate energy supply.[88,89] CoQ10 deficiency has been found in a number of age-related disorders, prominently including heart failure.[88,89]

Supplementing with CoQ10 has been found to be a highly effective means of increasing tissue CoQ10 activity, with improvements in function of heart muscle, brain cells, and other energy-intensive tissues. Increasingly, research is proving that not all forms of CoQ10 are the same; some are better absorbed than others. Better absorption means more benefit for you.[87]

There's evidence indicating that CoQ10 in the form of ***ubiquinol*** may be a better-absorbed, more readily available form of the coenzyme, compared with the more common ***ubiquinone***.[87] Ubiquinol is also the form of CoQ10 found naturally in the body, where it protects mitochondria and cell membranes.[90,91]

Research supports the idea that ubiquinol has a faster and more powerful effect—that it is more ***bioavailable***.[92] Animal studies have found higher tissue levels of CoQ10 when ubiquinol is the supplement used, and in one

study ubiquinol was the only form that could increase CoQ10 in brain mitochondria.[87] When CoQ10 was combined with the adaptogen shilajit, there was a **56%** increase in energy production in the brain.[93] The compounds in shilajit have been shown to stabilize CoQ10 in its ubiquinol form and help facilitate more efficient delivery of CoQ10 to the mitochondria.[93-96] And a human study showed that both a single oral dose of **150** or **300 mg** of ubiquinol and long-term administration of ubiquinol were rapidly absorbed, and no safety concerns or laboratory abnormalities were seen.[97]

Laboratory studies show that ubiquinol is highly effective in reducing the disastrous effects of shock induced by blood loss, a leading killer following major trauma.[98] The effect was attributed to ubiquinol's powerful ability to clean up products of oxidation and thereby decrease inflammatory changes.[99] Similarly, ubiquinol is the preferred supplement for use in certain forms of congestive heart failure.[88] And ubiquinol is showing great promise in a host of other conditions and health concerns for which oxidant damage is a major predisposing factor, such as male infertility due to weakened sperm,[100] blood markers of cardiovascular disease,[90] and autism in children.[101] There is also animal evidence supporting ubiquinol supplementation to prevent trauma-associated kidney damage.[102]

The beneficial effects of ubiquinol are so universal throughout the body that the compound is being explored for its effects on overall longevity. When age-accelerated mice (a model of human old age) were supplemented with high-dose ubiquinol (equivalent to about **1,680 mg** in humans), their performance on a treadmill was improved by more than **15%**, and they had a significant increase in their natural free radical defense systems, further adding to their protection against aging.[103] Revolutionary data published in mid-2014 showed that ubiquinol has a direct antiaging effect by supporting actions of the SIRT family of proteins that slow senescence through multiple biochemical activities.[104]

All of us face the age-accelerating effects of poor mitochondrial function, oxidant damage, and inflammation that arise from deficient CoQ10 levels. For those who seek the extra added benefits of greater bioavailability and enhanced expression of anti-aging genes, reduced CoQ10 in the form of ubiquinol may be the answer.

Potential Of CoQ10 In Noncardiac Disorders

Heart muscle and blood vessel cells are of course not the only tissues that require ample CoQ10 for efficient energy utilization. In reality, every cell in your body runs better when ample CoQ10 is available. This is especially true for the eyes, kidneys, and brain, which is why CoQ10 has shown such tremendous benefits for each of these organs.[50-53]

Promising studies have demonstrated that CoQ10 protects cells in the eye—specifically the energy-intense retina and the oxygen-exposed cornea.[54-63] These effects may prevent common causes of blindness in old age, such as macular degeneration, glaucoma, and cataracts.

The kidney, like the eye, heart, and blood vessels, is an organ with tremendous blood flow, high oxygen exposure, and a crucial need for maximum energy efficiency. Studies in humans and animals reveal powerful protective effects of CoQ10 on kidney tissue structure and function, potentially adding years to the lives of people who might otherwise succumb to kidney failure.[64-71]

The human brain is the body's largest consumer of oxygen and utilizer of energy. The major neurodegenerative diseases, including Alzheimer's, Parkinson's, ALS, and Huntington's, and many of their associated cognitive deficits, are beginning to show small signs of yielding to CoQ10 supplementation, at least in their earliest stages.[72-80]

THE HIGH COST OF HEART DISEASE

$818 billion … That's the estimated annual cost, in the United States alone, for total direct medical costs of cardiovascular disease by 2030.[86]

That represents a tripling, from about **$273 billion**, over a 20-year period, and a failure of the American health system to accomplish a major goal, which is the reduction of heart attacks and strokes by a million patients by 2017.[1]

During that time, real indirect costs from lost productivity will increase by **61%**, from **$172 billion** to **$276 billion**.[86] That's more than a **trillion dollars** a year in total.

With those kinds of figures, we can't afford to NOT get heart disease under control.

Studies show that supplementation with CoQ10 augments heart and vascular function, improves clinical status, and prevents further damage from cardiovascular disease.

CoQ10 may represent the single most vital supplement that everyone should take to sustain and support the cardiovascular energy management essential for a healthy heart.

Summary

Cardiovascular disease has many faces, but a central mechanism is ***loss of energy*** efficiency at the level of heart muscle and the coronary arteries that feed it.

CoQ10 is essential for transferring energy from food into ATP molecules, the universal cellular energy currency.

Studies show that CoQ10 levels are diminished in heart disease, particularly **congestive heart failure**. Supplementing with CoQ10 improves heart and vessel function in lab experiments, animal studies, and clinical trials.

If you suffer from existing heart disease, add a daily supplement of CoQ10, preferably the **ubiquinol** form, to your medication regimen after discussion with your doctor. If you are not yet a victim of overt cardiovascular disease, you are even better positioned to take advantage of CoQ10's preventive effects.

It is impossible to overstate the importance of CoQ10 supplementation in maintaining healthy bioenergetics in the heart, brain, kidney, eye, and other energy-intensive tissues. There is every reason to believe that regular CoQ10 supplementation will add to both your life span and your health span.

If you have any questions on the scientific content of this article, please call a **Life Extension**® Health Advisor at 1-866-864-3027.

USE OF UBIQUINOL IN CLINICAL STUDIES

Ubiquinol has now been shown to be important and effective in management of a number of chronic, age-related, oxidation-driven diseases, due to its potent effects in smoothing mitochondrial energy transfer and reducing the collateral damage to cells and tissues. For example:

- In diabetic retinopathy, a higher ratio of ubiquinol to ubiquinone was shown to be protective.[105]
- In diastolic heart failure, or heart failure with relatively normal pumping ability in the heart, ubiquinol is in growing use to improve patient outcomes and improve the function of the heart's left ventricle during its relaxation phase, when it recovers from energy-intensive contractions.[106]
- In men with impaired fertility due to weak or defective sperm, ubiquinol exerted favorable changes on sperm structure and motility, changes that favor fertility.[100]
- In cardiovascular diseases, an early marker of dangerous oxidative damage to heart cells is a rise in levels of a normally intracellular enzyme called GGT; ubiquinol was shown in a human study to reduce GGT activity, probably through complex effects on gene expression.[90]
- In autism, a childhood disorder thought to have roots in oxidative damage to brain tissue, ubiquinol supplementation improved communication with parents, verbal communication, game playing with other children, sleep, and food rejection, all common findings in autistic children.[101]

References

1. Available at: **http://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_heart_failure.htm**. Accessed December 21, 2014.
2. Thadani U, Ripley TL. Side effects of using nitrates to treat heart failure and the acute coronary syndromes, unstable angina and acute myocardial infarction. *Expert Opin Drug Saf*. 2007 Jul;6(4):385-96.

3. Andrey JL, Romero S, García-Egido A, et al. Mortality and morbidity of heart failure treated with digoxin. A propensity-matched study. *Int J Clin Pract*. 2011 Dec;65(12):1250-8.
4. Eurich DT, McAlister FA, Blackburn DF, et al. Benefits and harms of antidiabetic agents in patients with diabetes and heart failure: systematic review. *BMJ*. 2007 Sep 8;335(7618):497.
5. Tabrizchi R. Guidelines for choosing drugs in chronic heart failure. *Vasc Health Risk Manag*. 2005;1(3):171-2.
6. Miller AB. Aldosterone antagonism in heart failure. *Vasc Health Risk Manag*. 2007;3(5):605-9.
7. Hernandez AF, Liang L, Fonarow GC, et al. Associations between anticoagulation therapy and risks of mortality and readmission among patients with heart failure and atrial fibrillation. *Circ Cardiovasc Qual Outcomes*. 2014 Sep;7(5):670-9.
8. Singh RB, Neki NS, Kartikey K, et al. Effect of coenzyme Q10 on risk of atherosclerosis in patients with recent myocardial infarction. *Mol Cell Biochem*. 2003 Apr;246(1-2):75-82.
9. Gao L, Mao Q, Cao J, Wang Y, Zhou X, Fan L. Effects of coenzyme Q10 on vascular endothelial function in humans: a meta-analysis of randomized controlled trials. *Atherosclerosis*. 2012 Apr;221(2):311-6.
10. Marzilli M, Merz CN, Boden WE, et al. Obstructive coronary atherosclerosis and ischemic heart disease: an elusive link! *J Am Coll Cardiol* . 2012 Sep 11;60(11):951-6.
11. Pepine CJ, Douglas PS. Rethinking stable ischemic heart disease: is this the beginning of a new era? *J Am Coll Cardiol*. 2012 Sep 11;60(11):957-9.
12. Arenillas JF. Intracranial atherosclerosis: current concepts. *Stroke*. 2011 Jan;42(1 Suppl):S20-3.
13. Weber C, Noels H. Atherosclerosis: current pathogenesis and therapeutic options. *Nat Med*. 2011 Nov 7;17(11):1410-22.
14. Lusis AJ. Atherosclerosis. *Nature*. 2000 Sep 14; 407(6801): 233-41.
15. Aronson D, Rayfield EJ. How hyperglycemia promotes atherosclerosis: molecular mechanisms. *Cardiovasc Diabetol*. 2002 Apr 8;1:1.
16. Choy PC, Siow YL, Mymin D, O K. Lipids and atherosclerosis. *Biochem Cell Biol*. 2004 Feb;82(1):212-24.
17. Celletti FL, Waugh JM, Amabile PG, Brendolan A, Hilfiker PR, Dake MD. Vascular endothelial growth factor enhances atherosclerotic plaque progression. *Nat Med*. 2001 Apr;7(4):425-9.
18. Chapidze G, Kapanadze S, Dolidze N, Bachutashvili Z, Latsabidze N. Prevention of coronary atherosclerosis by the use of combination therapy with antioxidant coenzyme Q10 and statins. *Georgian Med News*. 2005 Jan (118):20-5.
19. Zeb I, Ahmadi N, Nasir K, et al. Aged garlic extract and coenzyme Q10 have favorable effect on inflammatory markers and coronary atherosclerosis progression: a randomized clinical trial. *J Cardiovasc Dis Res*. 2012 Jul;3(3):185-90.
20. Singh RB, Wander GS, Rastogi A, et al. Randomized, double-blind placebo-controlled trial of coenzyme Q10 in patients with acute myocardial infarction. *Cardiovasc Drugs Ther*. 1998 Sep;12(4):347-53.
21. Kuettner A, Pieper A, Koch J, Enzmann F, Schroeder S. Influence of coenzyme Q(10) and cerivastatin on the flow-mediated vasodilation of the brachial artery: results of the ENDOTACT study. *Int J Cardiol*. 2005 Feb 28;98(3):413-9.
22. Perez-Vizcaino F, Duarte J, Andriantsitohaina R. Endothelial function and cardiovascular disease: effects of quercetin and wine polyphenols. *Free Radic Res*. 2006 Oct;40(10):1054-65.
23. Watts GF, Playford DA, Croft KD, Ward NC, Mori TA, Burke V. Coenzyme Q(10) improves endothelial dysfunction of the brachial artery in Type II diabetes mellitus. *Diabetologia*. 2002 Mar;45(3):420-6.
24. Belardinelli R, Mucaj A, Lacalaprice F, et al. Coenzyme Q10 and exercise training in chronic heart failure. *Eur Heart J*. 2006 Nov;27(22):2675-81.
25. Tiano L, Belardinelli R, Carnevali P, Principi F, Seddaiu G, Littarru GP. Effect of coenzyme Q10 administration on endothelial function and extracellular superoxide dismutase in patients with ischaemic heart disease: a double-blind, randomized controlled study. *Eur Heart J*. 2007 Sep;28(18):2249-55.
26. Available at: **http://www.nhlbi.nih.gov/health/health-topics/topics/cad**. Accessed December 16, 2014.
27. Chen YF, Lin YT, Wu SC. Effectiveness of coenzyme Q10 on myocardial preservation during hypothermic cardioplegic arrest. *J Thorac Cardiovasc Surg*. 1994 Jan;107(1):242-7.
28. Rosenfeldt FL, Pepe S, Linnane A, et al. The effects of ageing on the response to cardiac surgery: protective

strategies for the ageing myocardium. *Biogerontology.* 2002;3(1-2):37-40.
29. Rosenfeldt F, Marasco S, Lyon W, et al. Coenzyme Q10 therapy before cardiac surgery improves mitochondrial function and in vitro contractility of myocardial tissue. *J Thorac Cardiovasc Surg.* 2005 Jan;129(1):25-32.
30. Kakinuma Y, Miyauchi T, Yuki K, Murakoshi N, Goto K, Yamaguchi I. Mitochondrial dysfunction of cardiomyocytes causing impairment of cellular energy metabolism induces apoptosis, and concomitant increase in cardiac endothelin-1 expression. *J Cardiovasc Pharmacol*. 2000 Nov;36(5 Suppl 1):S201-4.
31. Ventura-Clapier R, Garnier A, Veksler V. Energy metabolism in heart failure. *J Physiol*. 2004 Feb 15;555(Pt 1):1-13.
32. Huss JM, Kelly DP. Mitochondrial energy metabolism in heart failure: a question of balance. *J Clin Invest*. 2005 Mar;115(3):547-55.
33. Harvey PA, Leinwand LA. The cell biology of disease: cellular mechanisms of cardiomyopathy. *J Cell Biol*. 2011 Aug 8;194(3):355-65.
34. Sinatra ST. Metabolic cardiology: the missing link in cardiovascular disease. *Altern Ther Health Med.* 2009 Mar-Apr;15(2):48-50.
35. Sheeran FL, Pepe S. Energy deficiency in the failing heart: linking increased reactive oxygen species and disruption of oxidative phosphorylation rate. *Biochim Biophys Acta.* 2006 May-Jun;1757(5-6):543-52.
36. Baggio E, Gandini R, Plancher AC, Passeri M, Carmosino G. Italian multicenter study on the safety and efficacy of coenzyme Q10 as adjunctive therapy in heart failure. CoQ10 Drug Surveillance Investigators. *Mol Aspects Med.* 1994;15 Suppl:s287-94.
37. Mortensen SA, Vadhanavikit S, Muratsu K, Folkers K. Coenzyme Q10: clinical benefits with biochemical correlates suggesting a scientific breakthrough in the management of chronic heart failure. *Int J Tissue React.* 1990;12(3):155-62.
38. Morisco C, Trimarco B, Condorelli M. Effect of coenzyme Q10 therapy in patients with congestive heart failure: a long-term multicenter randomized study. *Clin Investig.* 1993;71(8 Suppl):S134-6.
39. Sacher HL, Sacher ML, Landau SW, et al. The clinical and hemodynamic effects of coenzyme Q10 in congestive cardiomyopathy. *Am J Ther.* 1997 Feb-Mar;4(2-3):66-72.
40. Munkholm H, Hansen HH, Rasmussen K. Coenzyme Q10 treatment in serious heart failure. *Biofactors.* 1999;9(2-4):285-9.
41. Molyneux SL, Florkowski CM, Richards AM, Lever M, Young JM, George PM. Coenzyme Q10; an adjunctive therapy for congestive heart failure? *N Z Med J.* 2009 Oct 30;122(1305):74-9.
42. Fotino AD, Thompson-Paul AM, Bazzano LA. Effect of coenzyme Q(1)(0) supplementation on heart failure: a meta-analysis. *Am J Clin Nutr.* 2013 Feb;97(2):268-75.
43. Belardinelli R, Mucaj A, Lacalaprice F, et al. Coenzyme Q10 improves contractility of dysfunctional myocardium in chronic heart failure. *Biofactors.* 2005;25(1-4):137-45.
44. Hofman-Bang C, Rehnqvist N, Swedberg K, Wiklund I, Astrom H. Coenzyme Q10 as an adjunctive in the treatment of chronic congestive heart failure. The Q10 Study Group. *J Card Fail.* 1995 Mar;1(2):101-7.
45. Langsjoen PH, Langsjoen AM. Supplemental ubiquinol in patients with advanced congestive heart failure. *Biofactors.* 2008;32(1-4):119-28.
46. Langsjoen PH, Langsjoen AM. Overview of the use of CoQ10 in cardiovascular disease. *Biofactors.* 1999;9(2-4):273-84.
47. Langsjoen PH. Lack of effect of coenzyme Q on left ventricular function in patients with congestive heart failure. *J Am Coll Cardiol.* 2000 Mar 1;35(3):816-7.
48. Zita C, Overvad K, Mortensen SA, Sindberg CD, Moesgaard S, Hunter DA. Serum coenzyme Q10 concentrations in healthy men supplemented with 30 mg or 100 mg coenzyme Q10 for two months in a randomised controlled study. *Biofactors.* 2003;18(1-4):185-93.
49. Mortensen SA, Rosenfeldt F, Kumar A, et al. The effect of coenzyme Q on morbidity and mortality in chronic heart failure: results from Q-SYMBIO: a randomized double-blind trial. *JACC Heart Fail.* 2014 Sep 25.
50. Noh YH, Kim KY, Shim MS, et al. Inhibition of oxidative stress by coenzyme Q10 increases mitochondrial mass and improves bioenergetic function in optic nerve head astrocytes. *Cell Death Dis*. 2013 Oct 3;4:e820.
51. Matthews RT, Yang L, Browne S, Baik M, Beal MF. Coenzyme Q10 administration increases brain

mitochondrial concentrations and exerts neuroprotective effects. *Proc Natl Acad Sci U S A*. 1998 Jul 21;95(15):8892-7.
52. Gazdíková K, Gvozdjáková A, Kucharská J, Spustová V, Braunová Z, Dzúrik R. Effect of coenzyme Q10 in patients with kidney diseases. *Cas Lek Cesk*. 2001 May 24;140(10):307-10.
53. Ishikawa A, Kawarazaki H, Ando K, Fujita M, Fujita T, Homma Y. Renal preservation effect of ubiquinol, the reduced form of coenzyme Q10. *Clin Exp Nephrol*. 2011 Feb;15(1):30-3.
54. Chen CC, Liou SW, Chen CC, et al. Coenzyme Q10 rescues ethanol-induced corneal fibroblast apoptosis through the inhibition of caspase-2 activation. *J Biol Chem.* 2013 Apr 26;288(17):11689-704.
55. Kernt M, Hirneiss C, Neubauer AS, Ulbig MW, Kampik A. Coenzyme Q10 prevents human lens epithelial cells from light-induced apoptotic cell death by reducing oxidative stress and stabilizing BAX / Bcl-2 ratio. *Acta Ophthalmol*. 2010 May;88(3):e78-86.
56. Lee D, Kim KY, Shim MS, et al. Coenzyme Q10 ameliorates oxidative stress and prevents mitochondrial alteration in ischemic retinal injury. *Apoptosis*. 2014 Apr;19(4):603-14.
57. Lee D, Shim MS, Kim KY, et al. Coenzyme Q10 inhibits glutamate excitotoxicity and oxidative stress-mediated mitochondrial alteration in a mouse model of glaucoma. *Invest Ophthalmol Vis Sci*. 2014 Feb;55(2):993-1005.
58. Lulli M, Witort E, Papucci L, et al. Coenzyme Q10 instilled as eye drops on the cornea reaches the retina and protects retinal layers from apoptosis in a mouse model of kainate-induced retinal damage. *Invest Ophthalmol Vis Sci*. 2012 Dec;53(13):8295-302.
59. Mencucci R, Favuzza E, Boccalini C, et al. CoQ10-containing eye drops prevent UVB-induced cornea cell damage and increase cornea wound healing by preserving mitochondrial function. *Invest Ophthalmol Vis Sci*. 2014 Oct 9;55(11):7266-71.
60. Nakajima Y, Inokuchi Y, Nishi M, Shimazawa M, Otsubo K, Hara H. Coenzyme Q10 protects retinal cells against oxidative stress in vitro and in vivo. *Brain Res*. 2008 Aug 21;1226:226-33.
61. Nucci C, Tartaglione R, Cerulli A, et al. Retinal damage caused by high intraocular pressure-induced transient ischemia is prevented by coenzyme Q10 in rat. *Int Rev Neurobiol*. 2007;82:397-406.
62. Qu J, Kaufman Y, Washington I. Coenzyme Q10 in the human retina. *Invest Ophthalmol Vis Sci*. 2009 Apr;50(4):1814-8.
63. Russo R, Cavaliere F, Rombola L, et al. Rational basis for the development of coenzyme Q10 as a neurotherapeutic agent for retinal protection. *Prog Brain Res*. 2008;173:575-82.
64. Carrasco J, Anglada FJ, Campos JP, Muntane J, Requena MJ, Padillo J. The protective role of coenzyme Q10 in renal injury associated with extracorporeal shockwave lithotripsy: a randomised, placebo-controlled clinical trial. *BJU Int*. 2014 Jun;113(6):942-50.
65. Gokbel H, Atalay H, Okudan N, Solak Y, Belviranli M, Turk S. Coenzyme Q10 and its relation with oxidant and antioxidant system markers in patients with end-stage renal disease. *Ren Fail*. 2011;33(7):677-81.
66. Ishikawa A, Homma Y. Beneficial effect of ubiquinol, the reduced form of coenzyme Q10, on cyclosporine nephrotoxicity. *Int Braz J Urol*. 2012 Mar-Apr;38(2):230-4; discussion 34.
67. Ishikawa A, Kawarazaki H, Ando K, Fujita M, Fujita T, Homma Y. Renal preservation effect of ubiquinol, the reduced form of coenzyme Q10. *Clin Exp Nephrol*. 2011 Feb;15(1):30-3.
68. Persson MF, Franzen S, Catrina SB, et al. Coenzyme Q10 prevents GDP-sensitive mitochondrial uncoupling, glomerular hyperfiltration and proteinuria in kidneys from db/db mice as a model of type 2 diabetes. *Diabetologia*. 2012 May;55(5):1535-43.
69. Saiki R, Lunceford AL, Shi Y, et al. Coenzyme Q10 supplementation rescues renal disease in Pdss2kd/kd mice with mutations in prenyl diphosphate synthase subunit 2. *Am J Physiol Renal Physiol*. 2008 Nov;295(5):F1535-44.
70. Sakata T, Furuya R, Shimazu T, Odamaki M, Ohkawa S, Kumagai H. Coenzyme Q10 administration suppresses both oxidative and antioxidative markers in hemodialysis patients. *Blood Purif*. 2008;26(4):371-8.
71. Triolo L, Lippa S, Oradei A, De Sole P, Mori R. Serum coenzyme Q10 in uremic patients on chronic hemodialysis. *Nephron*. 1994;66(2):153-6.
72. Levy G, Kaufmann P, Buchsbaum R, et al. A two-stage design for a phase II clinical trial of coenzyme Q10 in

ALS. *Neurology.* 2006 Mar 14;66(5):660-3.
73. Mischley LK, Allen J, Bradley R. Coenzyme Q10 deficiency in patients with Parkinson's disease. *J Neurol Sci.* 2012 Jul 15;318(1-2):72-5.
74. Muller T, Buttner T, Gholipour AF, Kuhn W. Coenzyme Q10 supplementation provides mild symptomatic benefit in patients with Parkinson's disease. *Neurosci Lett.* 2003 May 8;341(3):201-4.
75. Shults CW, Flint Beal M, Song D, Fontaine D. Pilot trial of high dosages of coenzyme Q10 in patients with Parkinson's disease. *Exp Neurol.* 2004 Aug;188(2):491-4.
76. Shults CW, Oakes D, Kieburtz K, et al. Effects of coenzyme Q10 in early Parkinson disease: evidence of slowing of the functional decline. *Arch Neurol.* 2002 Oct;59(10):1541-50.
77. Dumont M, Kipiani K, Yu F, et al. Coenzyme Q10 decreases amyloid pathology and improves behavior in a transgenic mouse model of Alzheimer's disease. *J Alzheimers Dis.* 2011;27(1):211-23.
78. Orsucci D, Mancuso M, Ienco EC, LoGerfo A, Siciliano G. Targeting mitochondrial dysfunction and neurodegeneration by means of coenzyme Q10 and its analogues. *Curr Med Chem.* 2011;18(26):4053-64.
79. Salama M, Yuan TF, Machado S, et al. Co-enzyme Q10 to treat neurological disorders: basic mechanisms, clinical outcomes, and future research direction. *CNS Neurol Disord Drug Targets.* 2013 Aug;12(5):641-64.
80. Shetty RA, Forster MJ, Sumien N. Coenzyme Q(10) supplementation reverses age-related impairments in spatial learning and lowers protein oxidation. *Age (Dordr).* 2013 Oct;35(5):1821-34.
81. The Criteria Committee of the New York Heart Association. *Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels*. 9th ed. Boston: Little, Brown & Co.; 1994.
82. Glaus T, Schellenberg S, Lang J. Cardiogenic and non cardiogenic pulmonary edema: pathomechanisms and causes. *Schweiz Arch Tierheilkd.* 2010 Jul;152(7):311-7.
83. Andalib S, Shayanfar A, Khorrami A, Maleki-Dijazi N, Garjani A. Atorvastatin reduces the myocardial content of coenzyme Q10 in isoproterenol-induced heart failure in rats. *Drug Res (Stuttg).* 2014 May;64(5):246-50.
84. Lee BJ, Tseng YF, Yen CH, Lin PT. Effects of coenzyme Q10 supplementation (300 mg/day) on antioxidation and anti-inflammation in coronary artery disease patients during statins therapy: a randomized, placebo-controlled trial. *Nutr J.* 2013;12(1):142.
85. Pourmoghaddas M, Rabbani M, Shahabi J, Garakyaraghi M, Khanjani R, Hedayat P. Combination of atorvastatin/coenzyme Q10 as adjunctive treatment in congestive heart failure: A double-blind randomized placebo-controlled clinical trial. *ARYA Atheroscler.* 2014 Jan;10(1):1-5.
86. Heidenreich PA, Trogdon JG, Khavjou OA, et al. Forecasting the future of cardiovascular disease in the United States: a policy statement from the American Heart Association. *Circulation.* 2011 Mar 1;123(8):933-44.
87. Garcia-Corzo L, Luna-Sanchez M, Doerrier C, et al. Ubiquinol-10 ameliorates mitochondrial encephalopathy associated with CoQ deficiency. *Biochim Biophys Acta.* 2014 Jul;1842(7):893-901.
88. Bates A, Shen Q, Hiebert JB, Thimmesch A, Pierce JD. Myocardial energetics and ubiquinol in diastolic heart failure. *Nurs Health Sci.* 2014 Dec;16(4):428-33.
89. Molyneux SL, Young JM, Florkowski CM, Lever M, George PM. Coenzyme Q10: is there a clinical role and a case for measurement? *Clin Biochem Rev*. 2008 May;29(2):71-82.
90. Onur S, Niklowitz P, Jacobs G, et al. Ubiquinol reduces gamma glutamyltransferase as a marker of oxidative stress in humans. *BMC Res Notes.* 2014;7:427.
91. Bhagavan HN, Chopra RK. Plasma coenzyme Q10 response to oral ingestion of coenzyme Q10 formulations. *Mitochondrion.* 2007 Jun;7 Suppl:S78-88.
92. Failla ML, Chitchumroonchokchai C, Aoki F. Increased bioavailability of ubiquinol compared to that of ubiquinone is due to more efficient micellarization during digestion and greater GSH-dependent uptake and basolateral secretion by Caco-2 cells. *J Agric Food Chem.* 2014 Jul 23;62(29):7174-82.
93. Bhattacharyya S, Pal D, Gupta AK, Ganguly P, Majumder UK, Ghosal S. Beneficial effect of processed shilajit on swimming exercise induced impaired energy status of mice. *Pharmacologyonline*. 2009;1:817-25.
94. Ghosal S. *Shilajit in Perspective*. Oxford, UK: Narosa Publishing House; 2006.
95. Islam A, Ghosh R, Banerjee D, Nath P, Mazumder U, Ghosal S. Biotransformation of 3-hydroxydibenzo-pyrone into 3,8 dihydroxydibenzo-pyrone and aminoacyl conjugates by Aspergillus niger isolated from native "shilajit." *Electro J Biotechno*. 2008 Jul 15;11(3):2-10.

96. Bhattacharyya S, Pal D, Banerjee D, et al. Shilajit dibenzo—pyrones: Mitochondria targeted antioxidants. *Pharmacologyonline*. 2009; 2:690-8.
97. Hosoe K, Kitano M, Kishida H, Kubo H, Fujii K, Kitahara M. Study on safety and bioavailability of ubiquinol (Kaneka QH) after single and 4-week multiple oral administration to healthy volunteers. *Regul Toxicol Pharmacol*. 2007 Feb;47(1):19-28.
98. Makley AT, Goodman MD, Friend LA, et al. Resuscitation with fresh whole blood ameliorates the inflammatory response after hemorrhagic shock. *J Trauma*. 2010 Feb;68(2):305-11.
99. Shen Q, Holloway N, Thimmesch A, Wood JG, Clancy RL, Pierce JD. Ubiquinol decreases hemorrhagic shock/resuscitation-induced microvascular inflammation in rat mesenteric microcirculation. *Physiol Rep*. 2014 Nov 1;2(11).
100. Cakiroglu B, Eyyupoglu SE, Gozukucuk R, Uyanik BS. Ubiquinol effect on sperm parameters in subfertile men who have astheno-teratozoospermia with normal sperm concentration. *Nephrourol Mon*. 2014 May;6(3):e16870.
101. Gvozdjakova A, Kucharska J, Ostatnikova D, Babinska K, Nakladal D, Crane FL. Ubiquinol improves symptoms in children with autism. *Oxid Med Cell Longev*. 2014;2014:798957.
102. Peerapanyasut W, Thamprasert K, Wongmekiat O. Ubiquinol supplementation protects against renal ischemia and reperfusion injury in rats. *Free Radic Res*. 2014 Feb;48(2):180-9.
103. Maruoka H, Fujii K, Inoue K, Kido S. Long-term effect of ubiquinol on exercise capacity and the oxidative stress regulation system in SAMP1 mice. *J Phys Ther Sci*. 2014 Mar;26(3):367-71.
104. Tian G, Sawashita J, Kubo H, et al. Ubiquinol-10 supplementation activates mitochondria functions to decelerate senescence in senescence-accelerated mice. *Antioxid Redox Signal*. 2014 Jun 1;20(16):2606-20.
105. Ates O, Bilen H, Keles S, et al. Plasma coenzyme Q10 levels in type 2 diabetic patients with retinopathy. *Int J Ophthalmol*. 2013;6(5):675-9.
106. Bates A, Shen Q, Hiebert JB, Thimmesch A, Pierce JD. Myocardial energetics and ubiquinol in diastolic heart failure. *Nurs Health Sci*. 2014 Dec;16(4):428-33.

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