Dynamic Chiropractic

ANTI AGING / HEALTHY AGING

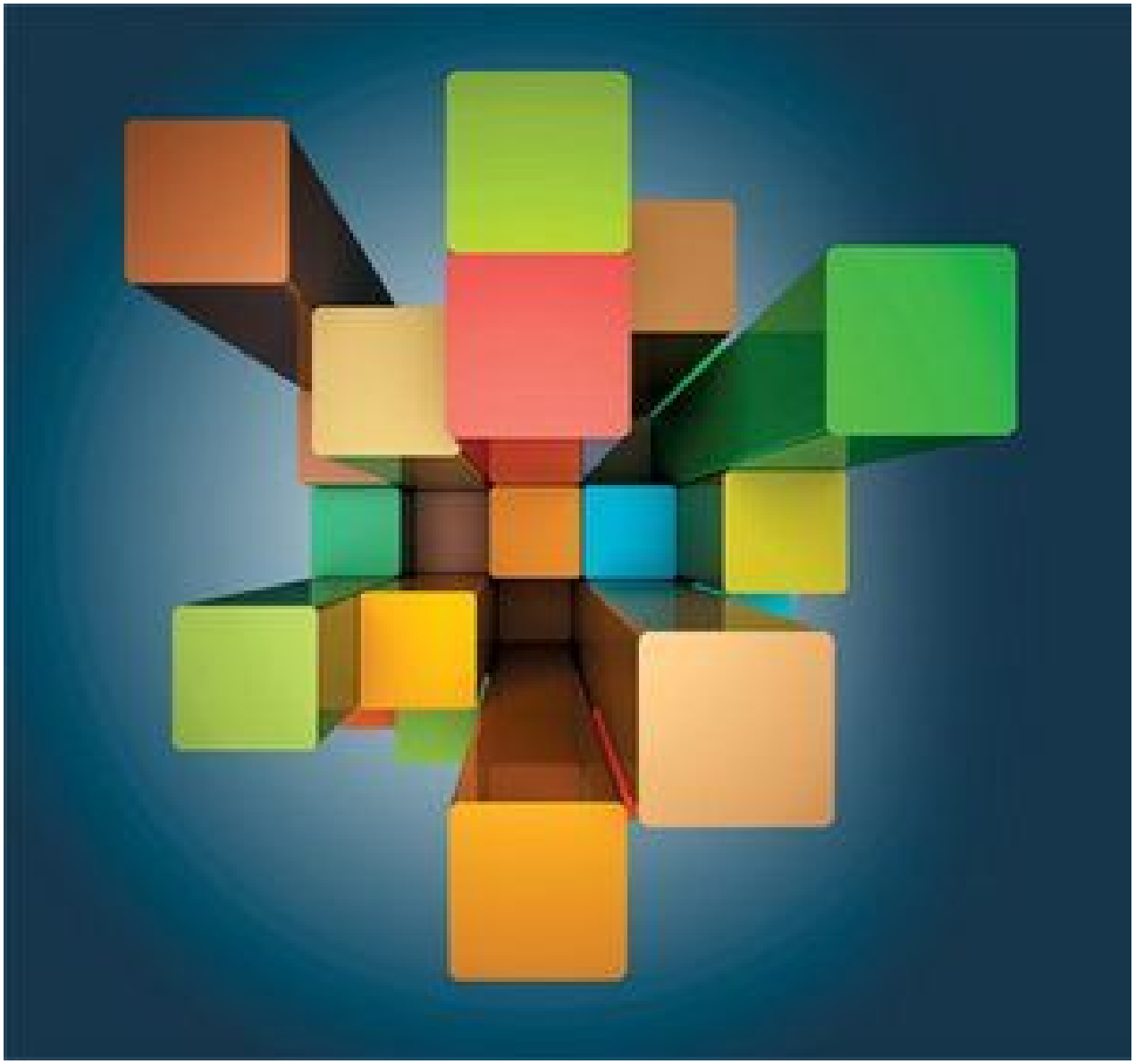
A Building Block of Healthy Aging

SUPPORTING BIOLOGIC ACTIVITY AS WE AGE WITH UBIQUINOL, THE ACTIVE FORM OF COQ_{10}.

Holly Lucille, ND, RN

Coenzyme Q_{10} has gained enormous attention in recent years, and with good reason —it's the Energizer Bunny of the cellular world. This essential quinine molecule is found in the mitochondria of every single cell in the body, where it plays a key role in energy production. CoQ_{10} not only assists in the production of adenosine triphosphate (ATP), but also scavenges free radicals.[1] To carry out these critical tasks, mitochondrial CoQ_{10} continuously cycles from ubiquinone, its ATP production state, to ubiquinol, its reduced active state.[2]

More than 4,000 published studies suggest that high CoQ_{10} levels are essential for optimal health — and this is especially true for the heart and brain. Since both of these organs require huge amounts of energy, supplementation can often help support their high biologic activity.[2-3] Research shows that CoQ_{10} supplementation can improve energy production and extend cell life by enhancing cellular mitochondrial levels of CoQ_{10}. In turn, this supports not only the heart and brain, but also periodontal, skin, reproductive, and immune health.[4-9] However, before you advise patients to add CoQ_{10} to their daily routine, be aware that there's a catch to taking this multitalented nutrient in supplemental form.



CoQ_{10}'s Critical Conversion

Creating ATP inside the mitochondria is quite complicated and involves a series of biochemical reactions. Since the body cannot store ATP, this multi-step process — known as the electron transport chain — ensures that this critical energy source is continually replaced.[1,10] Here's how it works: Ubiquinone contributes to ATP production by passing electrons from one enzyme complex to another, much like a bucket brigade.[3] During this process, ubiquinone is converted to its reduced active state, ubiquinol.

Surprisingly, our mitochondria are the most important cellular source of free radicals.[11] While most of the oxygen radicals generated by the mitochondria stay with its membrane folds, about 2 percent "escape" and create toxins that can threaten the health and survival of the entire cell.[11-13] Ubiquinol is able to neutralize these free radicals, both within the mitochondria and the cell membrane itself.[14]

Free radicals are harmful to all cells, but especially to cells with high biologic activity such as those in the heart and brain. Fortunately the highest concentration of CoQ_{10}, and subsequently ubiquinol,

is naturally found in the cells that make up the cardiovascular and nervous systems — sites where free radicals can inflict significant damage.[2-3, 14-15]

The Need to Supplement

Unfortunately, the body's ability to complete the conversion from ubiquinone to ubiquinol wanes as we age. Since this may contribute to premature aging, supplementation may be warranted. During a recent experiment, results of which appeared in the journal *Molecular Nutrition and Food Research,* and which used an animal model of aging, researchers found that mice given ubiquinol experienced significantly healthier aging than those taking a standard CoQ_{10} supplement.[16]

Certain conditions, including type 2 diabetes, congestive heart failure and hepatitis, have also been linked to lower serum levels of ubiquinol, as has the use of some cholesterol-lowering medications. What's more, it's estimated that 30-50 percent of people have a genetic single nucleotide polymorphism (SNP) known as NAD(P)H:quinone oxicloreductase (NQO1). Individuals with this SNP have a reduced ability to convert ubiquinone to ubiquinol efficiently.[17] Taken together, these factors clearly suggest that taking supplemental ubiquinol can benefit a large population of individuals.

Achieving optimal blood levels of CoQ_{10} can be challenging, however, since the nutrient is notoriously difficult to absorb.[19] Taking supplemental ubiquinol not only solves this dilemma, but also does so using smaller doses. This was clearly shown in a group of congestive heart failure patients struggling to achieve adequate plasma CoQ_{10} levels with supplemental CoQ_{10}, despite dosages of up to 900 mg/day.

During the study, conducted at the East Texas Medical Center and Trinity Mother Francis Hospital, all of the patients were switched from a standard CoQ_{10} supplement to an average daily dose of 580 mg of supplemental ubiquinol. Mean plasma CoQ_{10} levels increased from 1.6 microg/ml to 6.5 microg/ml. Mean ejection fraction also improved: from 22 percent to 39 percent. According to the researchers, ubiquinol dramatically improved absorption and plasma CoQ_{10} levels in patients with severe CHF. This, in turn, resulted in clinical improvement as well as improvement in the measurement of left ventricular function.[20]

The safety and bioavailability of ubiquinol have also been studied extensively over the past decade. Acute toxicity and safety studies, including genotoxicity, AMES, chromosome, and micronucleus testing, have confirmed its safety. During one placebo-controlled four-week study involving 15 healthy volunteers, daily doses of 150 mg and 300 mg were found to be safe and well-tolerated. A parallel study of 80 volunteers noted significant absorption of ubiquinol that resulted in dose-dependent, non-linear increases in serum concentrations over the course of four weeks.[21]

So, how much is enough? For patients over age 40 or anyone with chronic conditions linked to low CoQ_{10} levels, supplementing with 30-100 mg of ubiquinol daily is an exceptionally safe and effective way to raise serum levels. The generally recommend dose for those taking a statin drug or for patients with cardiovascular or neurological concerns is 100-200 mg daily, taken in divided doses.[2-3] Because ubiquinol is fat-soluble, supplements should be taken with a meal containing some fat.

References

1. Porth CM, Carroll EW. Mitochondria. In: Porth CM. *Pathophysiology: Concepts of Altered Health States. 6th Edition*. Philadelphia, Pa; Lippincott; 2004:8-9.
2. Coenzyme Q_{10} Monograph. *Altern Med Rev,* 2007;12:159-68.
3. Fleming T, ed. Coenzyme Q_{10}. In: *PDR® for Nutritional Supplements*. Montvale, NJ: Medical Economics Company; 2001:103-106.
4. Lee BJ, et al. CoQ_{10} supplementation reduces oxidative stress and increases antioxidant enzyme activity in patients with coronary artery disease. *Nutrition*, 2012;28(3):250-5.
5. Shetty RA, et al. Coenzyme Q(10) supplementation reverses age-related impairments in spatial learning and lowers protein oxidation. *Age* (Dordr), 2012 Nov 10 (epub ahead of print).
6. Hanioka T, et al. Effect of topical application of coenzyme Q_{10} on adult periodontitis. *Mol Aspects Med,* 1994;15 Suppl:s241-8.
7. Muta-Takada K, et al. Coenzyme Q_{10} protects against oxidative stress-induced cell death and enhances the synthesis of basement membrane components in dermal and epidermal cells. *Biofactors*, 2009;35(5):435-41.
8. Safarinejad MR. Efficacy of coenzyme Q_{10} on semen parameters, sperm function and reproductive hormones in infertile men. *J Urol*, 2009;182(1):237-48.
9. Gazdik F, et al. Biological properties of coenzyme Q_{10} and its effects on immunity. *Cas Lek Cesk*, 2003;142(7):390-3.
10. Guyton AC, Hall JE. Mitochondria. In: *Textbook of Medical Physiology. 9th Edition*. Philadelphia, Pa: WB Saunders; 2001:16-17.
11. Cadenas E. Mitochondrial free radical production and cell signaling. *Mol Aspects Med*, 2004;25:17-26.
12. Sohal RS, Forster MJ. Coenzyme Q, oxidative stress and aging. *Mitochondrion*, 2007;7:S103-11.
13. Turrens JF. Mitochondrial formation of reactive oxygen species. *J Physiol*, 2003;552:335-44.
14. Littarru GP, Tiano L. Bioenergetic and antioxidant properties of coenzyme Q_{10}: recent developments. *Mol Biotechnol*, 2007;37:31-7.
15. Langsjoen P H. Introduction to CoQ_{10}. Available at: http://www.grc.com/sr6dev/misc/coq10/coenzyme%20q10.pdf.
16. Schmelzer C, et al. Supplementation with the reduced form of coenzyme Q_{10} decelerates phenotypic characteristics of senescence and induces a peroxisome proliferator-activated receptor-alpha gene expression signature in SAMP1 mice. *Mol Nutr Food Res*, 2010;54(6):805-15.
17. Zhang J. Association of NAD(P)H: quinone oxidoreductase 1 (NQO1) C609T polymorphism in a German Caucasian and a northern Chinese population. *Carcinogenesis*, 2003;24:905-9.
18. Ueda T, et al. Method of stabilizing reduced coenzyme Q_{10}. Patent application number US 2005/0008630 A1. Jan. 13, 2005.
19. Bhagavan HN. Coenzyme Q_{10}: absorption, tissue uptake, metabolism and pharmacokinetics. *Fre Radic Res*, 2006;40(5):445-53.
20. Langsjoen PH. Supplemental ubiquinol in patients with advanced congestive heart failure. *Biofactors,* 2008;32(1-4):119-28.
21. Hosoe K, et al. Study on safety and bioavailability of ubiquinol (Kaneka QH) after single and 4-week multiple oral administration to healthy volunteers. *Regul Toxicol Pharmacol*, 2007;47:19-28.