

# USANA Technical Bulletin

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## Coenzyme Q<sub>10</sub>

### Technical Background

- Coenzyme Q<sub>10</sub>, or CoQ<sub>10</sub>, is a coenzyme naturally synthesized by the body and found in every cell, with highest levels located in the heart, liver, kidneys, and pancreas.
- Coenzyme Q<sub>10</sub> plays an essential role in mitochondrial electron transport. As such, it is fundamental for energy production in human cells.
- Coenzyme Q<sub>10</sub> is also an antioxidant. Its ability to quench free radicals helps cell membranes and intracellular membranes maintain structural integrity and stability.<sup>1</sup> It further serves to reduce oxidation of low density lipoprotein (LDL) cholesterol.<sup>2</sup>
- Evidence suggests that the most important antioxidant activity of Coenzyme Q<sub>10</sub> involves regeneration of Vitamin E. Ubiquinol, the reduced form of CoQ<sub>10</sub>, may be responsible for the reduction of the Vitamin E phenoxyl radical.
- CoQ<sub>10</sub> supplementation has been used to treat and ameliorate many conditions. Some of the best-documented effects involve cases of heart failure and heart disease, hypertension, periodontal disease<sup>3</sup>, and recently Parkinson's disease<sup>4</sup> and chronic fatigue syndrome.<sup>5</sup>

### Sources and Recommended Intake

- CoQ<sub>10</sub> is synthesized in all cells of the body, but particularly in liver cells.
- The body's ability to synthesize CoQ<sub>10</sub> diminishes with age. Deficiencies may also result from reduced assimilation from dietary sources.<sup>6</sup>
- Additional CoQ<sub>10</sub> can be absorbed from food. Major sources of dietary CoQ<sub>10</sub> include meats, fish, and vegetable oils (particularly soybean, sesame, and grapeseed oils).<sup>6</sup> Vegetables are generally low in CoQ<sub>10</sub> (with the exception of spinach and broccoli).
- CoQ<sub>10</sub> supplements are available and safe. The compound is best absorbed by the body when taken with foods. The usual maintenance dose is 10-30 mg per day, although higher doses are used to treat heart and blood vessel disease.<sup>7</sup>

### Abstracts

*Langsjoen P, Willis R, Folkers K. Treatment of essential hypertension with coenzyme Q10. Mol Aspects Med 1994;15 Suppl:S265-72.* A total of 109 patients with symptomatic essential hypertension presenting to a private cardiology practice were observed after the addition of CoQ10 (average dose, 225 mg/day by mouth) to their existing antihypertensive drug regimen. In 80 per cent of patients, the diagnosis of essential hypertension was established for a year or more prior to starting CoQ10 (average 9.2 years). Only one patient was dropped from analysis due to noncompliance. The dosage of CoQ10 was not fixed and was adjusted according to clinical response and blood CoQ10 levels. Our aim was to attain blood levels greater than 2.0 micrograms/ml (average 3.02 micrograms/ml on CoQ10). Patients were followed closely with frequent clinic visits to record blood pressure and clinical status and make necessary adjustments in drug therapy. Echocardiograms were obtained at baseline in 88%

of patients and both at baseline and during treatment in 39% of patients. A definite and gradual improvement in functional status was observed with the concomitant need to gradually decrease antihypertensive drug therapy within the first one to six months. Thereafter, clinical status and cardiovascular drug requirements stabilized with a significantly improved systolic and diastolic blood pressure. Overall New York Heart Association (NYHA) functional class improved from a mean of 2.40 to 1.36 ( $P < 0.001$ ) and 51% of patients came completely off of between one and three antihypertensive drugs at an average of 4.4 months after starting CoQ10. Only 3% of patients required the addition of one antihypertensive drug. In the 9.4% of patients with echocardiograms both before and during treatment, we observed a highly significant improvement in left ventricular wall thickness and diastolic function.

## References

- <sup>1</sup> Somayajulu M, et al. Role of mitochondria in neuronal cell death induced by oxidative stress; neuroprotection by Coenzyme Q10. *Neurobiol Dis.* 2005 Apr;18(3):618-27.
- <sup>2</sup> Kagan VE, Nohl H, Quinn PJ. Coenzyme Q: Its role in scavenging and generation of radicals in membranes. In Cadenas E and Packer L, editors. *Handbook of Antioxidants.* New York:Marcel Dekker Inc 1996; p 174-6.
- <sup>3</sup> Littarru GP, Battino M, Folkers K. Clinical aspects of coenzyme Q: Improvement of cellular bioenergetics or antioxidant protection? In Cadenas E and Packer L, editors. *Handbook of Antioxidants.* New York:Marcel Dekker Inc 1996; p 203-39.
- <sup>4</sup> Ebadi M, Brown-Borg H, El Refaey H, Singh BB, Garrett S, Shavali S, Sharma SK. Metallothionein-mediated neuroprotection in genetically engineered mouse models of Parkinson's disease. *Brain Res Mol Brain Res.* 2005 Mar 24;134(1):67-75
- <sup>5</sup> Bentler SE, Hartz AJ, Kuhn EM. Prospective observational study of treatments for unexplained chronic fatigue. *JClin. Psychiatry.* 2005 May;66(5):625-32
- <sup>6</sup> Kishi T, Okamoto T, Kishi H et al. In Folkers K, Yamamura Y, editors. *Biochemical and Clinical Aspects of Coenzyme Q.* Vol 5. Amsterdam:Elsevier Science Publishers 1985; p 119-123.
- <sup>7</sup> Yamamura Y. In Lenaz G, editor. *Coenzyme Q.* John Wiley & Sons 1985; p 479.