Dynamic Chiropractic

VITAMINS / SUPPLEMENTS

Coenzyme Q10 Supplementation Improves Outcomes in Early Parkinson's Disease

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In the October edition of the *Archives of Neurology*, Clifford Shults, et al., presented the findings of a clinical trial that demonstrated that patients with early-stage Parkinson's disease, given coenzyme Q10 (CoQ10) supplementation for 16 months, showed significantly less impairment than placebo patients. The efficacy of treatment was readily apparent by the eighth month, and the study showed that patients given the highest dose of CoQ10 had the best overall results. The test doses of CoQ10 were 300, 600 and 1,200 mg per day. The side-effects of CoQ10 at these high doses were mostly mild and included back pain, headaches and dizziness. Researchers indicate that the administration of CoQ10 is aimed not only at symptomatic relief, but rather addresses the underlying biochemical disorders associated with the development of the disease.[1] The results indicate that followup research at perhaps even higher doses should proceed "pretty aggressively," said Bernard Ravina of the National Institute of Neurological Disorders and Stroke, which funded the study.[2]

In 1999, Drs. Shults and Richard Haas noted that Parkinson's disease patients had reduced activity in complex I of the electron transfer chain, in the area of the brain affected by Parkinson's (*substantia nigra*, but not other areas of the brain) and in their blood platelets. In the patients' blood platelets, the level of mitochondrial CoQ10 was approximately 141.8 ng/mg protein on average, compared to the age/sex-matched control group of those without the disease, whose average CoQ10 level was 216.3 ng/mg protein.

The electron transfer chain within the mitochondrial membrane is the region in which adenosine triphosphate (ATP) energy is produced from the aerobic metabolism of the macronutrients, carbohydrates, fat and protein. In oxidative phosphorylation, the hydrogen atoms stripped off from these macronutrients in the Krebs cycle are transferred to the cytochrome system within the mitochondrial membrane by nicotinamide adenine dinucleotide (NAD) and flavin adenine dinucleotide (FAD), derived from the B-vitamins niacin and riboflavin, respectively.

As the hydrogen electrons are shuttled down the cytochrome chain (similar to traveling down a staircase) from a high level of energy to lower levels of energy, the difference between each step is given off as free energy, which can be used to recouple adenosine diphosphate (ADP) with phosphate to form more ATP.

Like all other tissues, the *substantia nigra* of the brain requires an adequate supply of ATP energy to survive and function normally. Within the mitochondrial membrane, CoQ10 acts as a hydrogen electron shuttle that literally escorts the hydrogen electrons from one cytochrome to the next (from one step in the staircase to the next). It specifically functions as an electron acceptor for complex I and complex II within the mitochondrial membrane. As such, a decline in CoQ10 levels within the mitochondrial membrane results in a decreased ability to synthesize ATP, resulting in development of the type of cellular dysfunction and death found in Parkinson's disease.

Animal studies clearly show that CoQ10 supplementation protects mice from the development of

Parkinson's by the neurotoxin MPTP, which is known to produce the disease in the mouse model.[3]

A number of researchers indicate that a significant aspect of the genetic component of Parkinson's is an inability to synthesize adequate amounts of CoQ10, and that CoQ10 supplementation can compensate for this genetic defect and potentially halt the onset or further progression of the disease.

Shults, et al., previously showed that Parkinson's patients could absorb supplemental CoQ10 with a trend toward increasing complex I activity within the mitochondrial membrane and, in theory, reverse the underlying defect in the disease by facilitating more optimal synthesis of ATP energy within the mitochondria of the *substantia nigra*.[3] These previous investigations led to the clinical trial reported in the *Archives of Neurology*, the first placebo-controlled study to show that CoQ10 supplementation can halt the progression of early-stage Parkinson's in human subjects. However, this study involved only 80 subjects (40 in the CoQ10 group and 40 in the placebo group). The impressive finding of the study has paved the way for larger studies to follow that should more clearly establish the degree to which CoQ10 supplementation may be useful as a treatment for this disease, and possibly

as a preventive agent in high-risk populations (e.g., those displaying low levels of CoQ10 synthesis upon genetic testing of CoQ10 synthetic enzymes).[1,2]

It is also noteworthy that CoQ10 is a fat-soluble antioxidant, which scavenges free radicals within the mitochondrial membrane. Oxidative stress (free radical damage) has also been shown to contribute to the cellular dysfunction and cell death seen in Parkinson's. Animal studies demonstrate that CoQ10 supplementation can prevent the damage induced by various neurotoxins known to produce Parkinson's, amyotrophic lateral sclerosis (ALS) and Huntington's disease. This preventive effect has been shown to involve the antioxidant properties of CoQ10 acting as a vital fat-soluble free radical scavenger within the mitochondrial membrane - a place where a great deal of oxygen-free-radicals are created each moment of our lives during aerobic energy production.[3,4]

It is also compelling to note that Parkinson's disease primarily afflicts those over the age of 50, which correlates with the phase in life when most people experience an age-related decline in CoQ10 synthesis to some degree. This appears due to a genetically controlled program, which produces a decline in the activity of CoQ10 synthetic enzymes as we age. This age-related decline in CoQ10 synthesis has been associated with an increased risk of congestive heart failure (due to reduced ATP synthesis by the heart muscle), and some studies show that CoQ10 supplementation has been effective in reversing this condition in a significant number of congestive heart disease patients and in patients with other cardiomyopathies.[5-10] Many anti-aging experts suggest that all adults should take at least 60mg per day of CoQ10 as an anti-aging, disease prevention supplement after the age of 45. Support for this argument appears to be strengthened by the recent study involving Parkinson's patients, as the disease afflicts one million people, many over the age of 50, but the incidence of the disease in younger people is increasing at an alarming rate, according to the American Parkinson's Disease Association.[11]

(If you would like to receive a fully referenced nutrition management report for Parkinson's disease to help educate your patients on this subject, email your request to support@renaisante.com, or fax your request to 888-781-3030.)

References

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