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REPORT

Continued from "Making Old Hearts Young Again"

Coenzyme Q10
It's in Your Thoughts

A new study is demonstrating potent neuro-protective benefits attributable to coenzyme Q10. With studies employing CoQ10 supplementation, the brain's mitochondrial energy expenditure increases, and dopamine-producing sections of the brain are protected. Survival time is even extended in cases of Lou Gehrig's disease.

Because heart cells have a high energy demand, initial clinical studies investigated the effect of coenzyme Q10 on cardiac mitochondrial function. Scientists are now looking at the effects of CoQ10 on another organ whose cells also require a high level of energy metabolism—the brain.

About 95% of cellular energy is produced from structures in the cells called mitochondria. The mitochondria have been described as the cells' "energy powerhouse," and the diseases of aging are increasingly being referred to as "mitochondrial disorders."

When coenzyme Q10 is taken orally, it is incorporated into the mitochondria of cells throughout the body where it facilitates and regulates the oxidation of fats and sugars into energy. One of the areas in which mitochondrial energy by CoQ10 is enhanced is the brain, offering attendant neuro-protective benefits.

Here are the highlights from a study just published in the *Proceedings of the National Academy of Sciences* (1998; 95)

- When coenzyme Q10 was administered to middle-age and old-age rats, the level of CoQ10 increased by 10% to 40% in the cerebral cortex region of the brain. This increase was sufficient to restore levels of CoQ10 to those seen in young animals.
- After only two months of CoQ10 supplementation, mitochondrial energy expenditure in the brain increased by 29%, compared with the group not getting CoQ10. The human equivalent dose of CoQ10 to achieve these results is 100 to 200 mg a day.
- When a neuro-toxin was administered, CoQ10 helped protect against damage to the striatal region of the brain where dopamine is produced.
- When CoQ10 was administered to rats genetically bred to develop amyotrophic lateral sclerosis (ALS, or Lou Gehrig's disease), a significant increase in survival time was observed.

The scientists concluded, "CoQ10 can exert neuro-protective effects that might be useful in the treatment of neuro-degenerative diseases."

This new study showed that short-term supplementation with moderate amounts of CoQ10 produced profound anti-aging effects in the brain. Previous studies have shown that CoQ10 may protect the brain via several mechanisms, including reduction in free radical generation and protection against glutamate-induced excitotoxicity. The study documented that orally supplemented CoQ10 specifically enhanced metabolic energy levels of brain cells. While this effect in the brain has been previously postulated, this new study provides hard evidence.

Based on the types of brain cell injury that CoQ10 protected against, the scientists suggested that it may be useful in the prevention or treatment of Huntington's disease and ALS. It was noted that while vitamin E delays the onset of ALS in mice, it does not increase survival time. CoQ10 was suggested as a more effective treatment strategy than vitamin E for neuro-degenerative disease because survival time was increased in mice treated with CoQ10.

In keeping with CoQ10's observed anti-aging and neuro-protective benefits, a report published in the *Annals of Neurology* (August 1997) identified a new mechanism showing that CoQ10 might be effective in the prevention and treatment of Parkinson's disease.

This study showed that the brain cells of Parkinson's patients have a specific impairment that causes the disruption of healthy mitochondrial function. It is known that mitochondrial disorder causes cells in the substantia nigra region of the brain to malfunction and die, thus creating a shortage of dopamine.

An interesting finding was that CoQ10 levels in Parkinson's patients were 35% lower than age-matched controls. This deficit of CoQ10 caused a significant reduction in the activity of enzyme complexes that are critical to the mitochondrial function of the brain cells affected by Parkinson's disease.

The ramifications of this study are significant. Parkinson's disease is becoming more prevalent as the human life span increases, and the new study confirms previous studies that Parkinson's disease may be related to CoQ10 deficiency. The scientists concluded, "The causes of Parkinson's disease are unknown, [but] evidence suggests that mitochondrial dysfunction and oxygen free radicals may be involved in its pathogenesis. The dual function of CoQ10 as a constituent of the mitochondrial electron transport chain and a potent antioxidant suggest that it has the potential to slow the progression of Parkinson's disease."

CoQ10 levels decrease with age. In fact, aged humans have only 50% of the CoQ10 that young adults have. Depletion is caused by reduced synthesis of CoQ10 in the body, along with increased oxidation of CoQ10 in the mitochondria. The inevitable conclusion might be that, if a CoQ10 deficit results in the inactivation of enzymes needed for mitochondrial energy production, supplementation with CoQ10 may preserve mitochondrial function.

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