

## REPORT

## Should Parkinson's patients take CoQ10?



Parkinson's disease is the second most common degenerative brain disorder. The percentage of the population afflicted by Parkinson's is on the rise.<sup>1</sup>

The symptoms of Parkinson's disease are attributed to a loss of cells in the substantia nigra region of the brain. These cells produce dopamine, a critical neurotransmitter responsible for motion control. As Parkinson's disease progresses, an accelerated rate of cell death occurs, resulting in even less dopamine being produced and the manifestation of more pronounced complications.

Today's treatments only address symptoms. These therapies don't protect against the underlying pathologies implicated in Parkinson's disease, namely the death of dopamine-producing neurons (brain cells). Drugs such as L-Dopa provide dopamine to the brain and temporarily alleviate symptoms. These drugs, however, do not slow the progression of the disease. At advanced stages, the drugs fail to work and complications become severe.

Researchers question why some people develop Parkinson's while others escape its clutches. One of the answers under consideration is that low coenzyme Q10 concentrations may predispose certain individuals to the disease. Coenzyme Q10 is naturally synthesized by the body and is a crucial component of the cell's energy cycle. As people age, they produce far less coenzyme Q10, which helps explain the heightened risk of Parkinson's with advancing age.<sup>2</sup>

In a year 2002 study, Yale scientists examined a group of healthy people ranging in age from 18 to 88 using a neuro-imaging technique. The researchers observed a steady decline in "striatal dopamine transporters," a marker of brain degeneration characteristic of Parkinson's disease. Comparing the youngest age people to the oldest, the number of striatal dopamine transporters was cut by 46%, or 6.6% per decade of life.<sup>3</sup>

The rate of loss of dopamine transporters is accelerated in Parkinson's patients, and this age-related factor may suggest why the disease progresses more rapidly in older patients. Some researchers are asking: Could a coenzyme Q10 deficiency be a starting point of this domino effect?

Will Parkinson's be cured in the near future?

Those suffering from Parkinson's have pinned their hopes on several potential therapies that might reverse the course of the disease. Stem cell therapy has received the most publicity because it offers the simple-to-understand approach of re-populating the brain with the very dopamine-producing cells that are destroyed by the disease. Findings from animal studies indicate that embryonic stem cells proliferate extensively and can generate dopamine-producing neurons suitable for possible cell replacement therapy for Parkinson's disease.<sup>4</sup>

Another potential treatment involves the surgical insertion of electrodes into the brain to stimulate nerve impulses in a region called the subthalamic nucleus. This new therapy is called "Deep Brain Stimulation." A study published in a year 2002 issue of the journal *Surgical Neurology* showed significant improvement in all motor function tests in 36 advanced Parkinson's patients. These patients were able to reduce their use of the drug L-Dopa by an average of 53%, with daily "off-times" reduced by 35%! Dyskinesia, which is an impairment of voluntary muscle movement, also markedly improved after "Deep Brain Stimulation" therapy.<sup>5,6</sup>

It might also be possible to treat Parkinson's disease by altering the genes in brain cells responsible for causing motor abnormalities. A year 2002 article published in the journal *Science* showed that a gene transfer technique could protect dopamine producing neurons and rescue parkinsonian behavioral abnormalities in rats. The scientists who conducted this research indicated that this gene transfer technology could be used to induce

#### How Coq10 Protects Brain Cells

Mitochondrial dysfunction is at the basis of many neurodegenerative diseases. The mitochondria are the "powerhouses" of cells, providing energy that fuel critical cellular functions. Aging and cumulative oxidative stress, however, impede the functioning of the mitochondria over time, resulting in dysfunction and demise.

therapeutic benefit.<sup>7</sup>

A clinical trial at the University of Kentucky is currently treating 10 Parkinson's patients with a bioengineered protein, called glial cell line-derived neurotrophic factor (GDNF), using a new drug-delivery method that sends the protein deep into the substantia nigra region of the brain where dopamine is produced. A constant supply of GDNF is administered by a pump implanted in the chest. So far, GDNF seems both to shield healthy brain cells from the disease and cause damaged cells to regenerate. According to University of Kentucky investigator Greg Gerhardt, after just a few months of testing, there is evidence of improvement in patients. In addition, British doctors reported in April 2002 that a similar trial in Bristol, England, improved muscle control of all five patients tested within a month of treatment.

These recent breakthroughs indicate a possible cure for Parkinson's disease in the foreseeable future. The question that Parkinson's patients ask today is: Can something be done to slow the progression of their disease? A growing number of researchers are looking at coenzyme Q10 as a potential treatment. Coenzyme Q10 acts as a critical energy carrier in mitochondrial electron transport. It also functions as an antioxidant to inhibit lipid peroxidation that kills dopamine producing neurons.<sup>8</sup> According to a study published in a year 2002 issue of *Neurochemistry Research*, scientists believe that coenzyme Q10 works by improving cellular respiration, preventing oxidative stress, and inhibiting neuronal cell death.

To date, various investigators have found that coenzyme Q10 may be useful as a neuroprotective agent for diseases marked by mitochondrial dysfunction. This includes ALS (Lou Gehrig's disease), Huntington's chorea, Friedreich's ataxia and Parkinson's disease. Coenzyme Q10 is presently being studied as a potential treatment for early Parkinson's disease, as well as in combination with another drug as a potential treatment for Huntington's disease.<sup>9</sup>

#### A promising approach to slow Parkinson's progression

A new approach to Parkinson's disease was presented at the 2002 annual meeting of the American Neurological Association (New York City, Oct. 13-16) and simultaneously published in the journal *Archives of Neurology*. Dr. Clifford Shults and colleagues at the University of California, San Diego showed that oral coenzyme Q10 can actually slow the progression of Parkinson's disease.<sup>10</sup>



While CoQ10 has been successfully tested in clinical trials for other neurological disorders including Huntington's Disease, this is the first human trial to test CoQ10 in a major neurodegenerative disease affecting millions of Americans. The multicenter study randomly assigned 80 people with early Parkinson's disease, who were not yet being treated, to either a placebo or coenzyme Q10 at dosages of 300, 600 or 1200 milligrams per day. All of the coenzyme Q10 dosages were safe and well-tolerated during the 16-month trial.

The patients in the study were assessed on the Unified Parkinson Disease Rating Scale

(UPDRS) to establish baseline scores for their basic motor skills, mental status, mood and behavior, and ability to perform daily living activities. Since the scale is designed to measure disease progression, lower UPDRS scores indicate better performance. Results showed that the UPDRS score increased by 11.99 for the placebo group, 8.81 for the 300-milligrams/day group, 10.82 for the 600-milligrams/day group, and 6.69 for the 1200-milligrams/day group.

Basically, these findings demonstrate that coenzyme Q10 supplementation at 1200 milligrams/day resulted in 44% less mental and physical disability than a placebo. In addition, reported Shults, "the greatest benefit was seen in activities of daily living: dressing, bathing, eating and walking." The patients in the 1200 milligrams/day group were better able to function, and maintained greater

Neuro-degenerative diseases involve early and accelerated nerve cell destruction. One of the primary causes of neuronal death is excitotoxicity, whereby the brain becomes over-sensitized to the neurotransmitter glutamate, whose role it is to send excitatory impulses.

Some scientists theorize that excitotoxicity can also result from a diminished energy level among neurons, which may lower their defenses against various neurotoxins and thus precipitate malfunctioning and death. Researchers now suggest that this bioenergy decline may be intimately involved with the progression of Parkinson's disease. Coenzyme Q10 seems to offer protection for neurons against the excitotoxicity of exposure to L-glutamate.\*

The region of the brain affected by Parkinson's disease is also afflicted by excessive oxidative stress, more than is evident elsewhere in the brain. This oxidative stress is especially damaging to neurons whose energy levels are already taxed by neurotoxicity and excitotoxicity. According to a study published in a year 2001 issue of *Neurotoxicology*,\* both neurotoxins and excitotoxins are "thought to involve free radical production, compromised mitochondrial activity and excessive lipid peroxidation." Logic would suggest then that restoring neuronal energy levels might boost these defenses and research has demonstrated that to be the case.

\*Mazzio E et al. Effect of antioxidants on L-glutamate and N-methyl-4-phenylpyridinium ion induced-neurotoxicity in PC12 cells. *Neurotoxicology* 2001 Apr;22 (2):283-8.

independence for a longer time.

Several years ago, the same research team (Shults et al.) found that coenzyme Q10 levels were much lower (35%) in the mitochondria from parkinsonian patients than in age- and sex-matched controls, and that these lower concentrations seemed to relate to diminished activity of enzyme complexes vital for mitochondrial function.<sup>11</sup> In that study, the authors 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 coenzyme Q10 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."

Continued on Page 2 of 2

[Back to the Magazine Forum](#)

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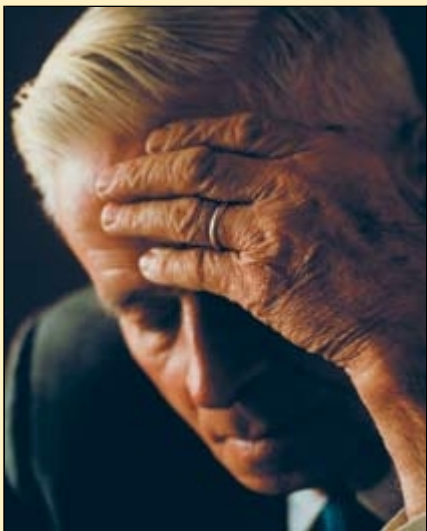
#### Confirmatory findings

Neurologist M. Flint Beal and colleagues at the Massachusetts General Hospital and Harvard Medical School has spent years proving that coenzyme Q10 has neuro-protective properties that may help diseases such as Parkinson's and Huntington's. Dr. Beal has a growing body of research to support his hypothesis.<sup>12</sup> Earlier research had established that patients with early, untreated Parkinson's disease have reduced activities of the electron transport complexes I and II/III in mitochondria from platelets, which seems to be attributable to the Parkinson's disease process.<sup>13</sup> For example, results indicated that complex II/III activity was reduced by 20% in Parkinson's disease compared with age-/sex-matched controls.

In a later study, oral supplementation with coenzyme Q10 in one-year old mice attenuated MPTP (1-methyl-4-phenyl-1,2,3,4-tetrahydropyridine) mediated neurotoxicity, which has been shown to cause a parkinsonian syndrome in test animals.<sup>14</sup> The researchers put four groups of one-year-old male mice on either a standard diet or a diet supplemented with coenzyme Q10 for five weeks. At four weeks, one of the standard diet groups and one of the supplemented groups were treated with MPTP. Striatal dopamine concentrations and dopaminergic axon density were reduced in both MPTP-treated groups, but they were much higher, 37% and 62%, respectively, in the group first treated with coenzyme Q10 and then MPTP compared to the group treated with MPTP alone.

#### The unpleasant symptoms of Parkinson's disease

Classic Parkinson's symptoms are characterized by tremors or shaking of one or both arms and sometimes of other muscles. Generally muscles are weak and rigid, movements slow and the face expressionless, also the voice becomes weak. Typically the walk is with slow, short, shuffling steps, the arms held stiffly at the sides and the trunk slightly bent forward; the patient may spontaneously break into a shuffling run.



Beal's team also demonstrated in middle-aged and old-aged rats, that coenzyme Q10 administration could restore levels of the nutrient to those of younger rats. The results showed that coenzyme Q10 levels rose by 10% to 40% in the mitochondria of the cerebral cortex region of the brain.<sup>14</sup> What many people don't understand is that increasing serum or tissue levels of a nutrient through oral administration doesn't necessarily translate into some measurable disease-fighting or anti-aging benefit. That's what made this study's findings so promising. They showed that two months of supplementation resulted not only in replenished coenzyme Q10 levels in the brain and the brain mitochondria, but also in a significant increase of 29% in mitochondrial energy expenditure in the brain. Moreover, topping up coenzyme Q10 levels helped counter the neurodegenerative effects of an experimental neurotoxin administered to the dopamine-producing (striatal) region of the brain in the test rats.

The same study also showed that coenzyme Q10 might help other neurodegenerative diseases, such as Huntington's and amyotrophic lateral sclerosis (ALS or Lou Gehrig's disease). The researchers found that the administration of coenzyme Q10 could restore concentrations, prevent nerve-cell degeneration, and extend survival in transgenic mice bred with a familial form of ALS genes.

Meanwhile, a pilot study showed that energy production in the central nervous system and muscle of Huntington's disease patients is impaired. But two or more months of coenzyme Q10 supplementation at 360 milligrams per day resulted in significant improvements in biochemical markers of energy production in 83% of patients.

Additional research findings suggest that coenzyme Q10 supplementation may also help various forms of ataxia, particularly cases that show a decreased level of coenzyme Q10 in their muscles. For example, UK scientists at the University of Oxford found marked improvements in mitochondrial defects among Friedreich's ataxia (FA) patients, which involve a deficiency of a mitochondrial protein called frataxin. Six months of antioxidant treatment with coenzyme Q10 (400 milligrams/day) and vitamin E (2100 IU/day) in 10 Friedreich's Ataxia patients resulted in a 178% increase in the cardiac phosphocreatine to ATP ratio and 139% increase in the maximum rate of skeletal muscle mitochondrial ATP production. These results were observed after only three months of treatment, and were sustained after the six month study was completed.<sup>15</sup>

In another example, a study of six patients with hereditary ataxia revealed baseline coenzyme Q10 levels that were 70% below normal.<sup>16</sup> However, following daily supplementation with coenzyme Q10, ranging from 300 milligrams to 3,000 milligrams, all of them had improved strength, ataxia and fewer seizures, and some were even able to walk.

After a year of supplementation, patient scores on an ataxia symptom scale improved by an average of 25%.

## Doctors remain cautious

Despite the hail of good news regarding the usefulness of coenzyme Q10 for Parkinson's and other neurodegenerative diseases, the mood among researchers is one of cautious optimism. For example, even Shults, lead author of the latest study, suggests that, before recommending coenzyme Q10 supplementation for Parkinson's disease patients, he would like to conduct a larger study, one that will more definitively determine coenzyme Q10's effects, especially in high doses. That upcoming study will likely involve about 400 patients taking doses of over 1200 milligrams per day for a longer study period.

Meanwhile, the National Institute of Neurological Disorders and Stroke, which helped fund the study, said that findings were "very promising," but called it premature to recommend coenzyme Q10 to Parkinson's patients until a larger trial is done. Scientists are also concerned about the purity of commercial CoQ10 supplements (i.e. how much coenzyme Q10 is actually in a product?), and what risks may exist as far as side effects, drug interactions and other contraindications. These scientists believe that more data is necessary in order to establish safe and effective doses of coenzyme Q10 for neurodegenerative diseases.

While the ideal or most effective dose cited in the latest study is 1200 milligrams daily, that's five times the upper dose consumed for prevention purposes. There is, however, strong historical data regarding the safety of congestive heart failure patients taking about 300 milligrams a day of coenzyme Q10 and those suffering from Huntington's disease using 600 mg/day.

## Why Parkinson's patients can't wait

Parkinson's disease is a progressive degenerative brain disorder that is currently incurable. Patients suffer increasing debilitating complications as the disease progresses. Today's therapies only mitigate the agonizing symptoms—they do not slow the rate of deterioration.

Even when treatments such as "Deep Brain Stimulation" become widely available, the benefits may only be partial. It is thus imperative for Parkinson's patients to protect as many dopamine-producing neurons as possible. The latest study indicates that high-dose coenzyme Q10 does just that.

For the first time in medical history, a human study has shown that the progression of Parkinson's disease can be slowed by 44% when patients consume 1200 mg of coenzyme Q10 a day.

While this is the first human study using coenzyme Q10 to treat Parkinson's disease, it was based on a large body of previously published research. For instance, a year 1998 study published in the Proceedings of the National Academy of Sciences<sup>17</sup> concluded that "CoQ10 can exert neuroprotective effects that might be useful in the treatment of neurodegenerative diseases."

Parkinson's patients don't have the option of waiting for mainstream medicine to reach a consensus about coenzyme Q10. There are, however, sensible approaches that Parkinson's patients can follow to reduce any possible risk of taking high-dose coenzyme Q10.



### The unpleasant symptoms of Parkinson's disease

Some studies have found that coenzyme Q10 may affect or be affected by other medications. For instance, findings suggest that coenzyme Q10 levels are depleted by various cholesterol-lowering drugs (statins)\* and antihypertensives (i.e. beta blockers).\*\* Those using these popular prescription drugs may need additional coenzyme Q10 because these drugs inhibit natural coenzyme Q10 synthesis in the body.

People taking certain prescription drugs may benefit from coenzyme Q10 because it reduces side effects, such as those side effects inflicted by timolol (a glaucoma medication).\*\*\* On the other hand, coenzyme Q10 supplementation may reduce the efficacy of certain drugs, such as warfarin (blood thinner),<sup>^</sup> and increase the half-life

For example, many healthy Life Extension members have been taking 300 mg a day and higher of coenzyme Q10 without encountering adverse effects. A Parkinson's patient may consider starting at 300 mg a day and then increasing to 600 mg two weeks later. If any prescription drugs are being taken, it is important to make sure this high dose of coenzyme Q10 will not create the need for a dosing adjustment. For instance, if Coumadin is being used, the weekly or bi-weekly coagulation blood tests that Coumadin patients are supposed to have can determine if additional Coumadin is needed.

After taking 600 mg a day of coenzyme Q10 for two weeks, a Parkinson's patient may want to increase the dose to 900 mg a day, and again make sure there is no prescription drug interference. After the 900mg/day dose has been established as being safe, the Parkinson's patient can increase the dose of coenzyme Q10 to

of others, such as enalapril (an old-line anti-hypertensive).^^ The evidence for interactions with warfarin (Coumadin) and enalapril is skimpy and is not based on any controlled study.

There are simple medical tests that can enable a physician to modulate the dosing of a drug like Coumadin or enalapril in the presence of high doses of coenzyme Q10. For instance, if an old-line anti-hypertensive drug like enalapril (Vasotec) is prescribed, it may be possible to take a lower dose of enalapril since coenzyme Q10 may prolong its effects in the body. Regular blood pressure monitoring can determine the optimal individual dose of enalapril.



Some reports indicate that coenzyme Q10 may reduce the anti-coagulant efficacy of Coumadin. In this case, all a physician has to do is continue the normally scheduled (weekly or bi-weekly) coagulation blood tests

(Prothrombin and INR) and increase the dose of Coumadin if coenzyme Q10 is blunting Coumadin's anti-coagulant effect. Those who are prescribed Coumadin have these blood coagulation tests done frequently because the dose of Coumadin often has to be adjusted to reflect changes occurring in the patient's body.

1200 mg a day. For maximum absorption into the bloodstream, always take coenzyme Q10 supplements with the fattiest meal of the day. Notify your doctors that you are taking this high dose of coenzyme Q10 so that they can monitor the effects of other drugs you are taking. It should be noted that while this cautious approach is prudent, Parkinson's patients participating in the most recent study were started at 1200 mg a day of CoQ10 with no significant side effects reported.

It is important to remember that coenzyme Q10 does not reverse Parkinson's disease, nor does it alleviate symptoms. The only effect that has been shown is that it slows disease progression. Parkinson's patients will still need to take medications to alleviate symptoms.

What has intrigued scientists the most is that coenzyme Q10 may help prevent common neurodegenerative diseases. If this turns out to be true, then healthy people may want to consume a higher dose of coenzyme Q10, perhaps as much as 300 mg a day.

Considering that initial studies from Japan showed as little as 30 mg a day of coenzyme Q10 was effective in the treatment of congestive heart failure, the fact that much higher doses do not produce side effects demonstrates the safety of this natural agent.

The body synthesizes abundant quantities of coenzyme Q10 in youth, but aging and the use of certain drugs (statins) causes a precipitous reduction in coenzyme Q10 production. This decline in coenzyme Q10 correlates with the many degenerative diseases that aging humans confront, including Parkinson's.

In response to this new study showing that 1200 mg a day of coenzyme Q10 slowed the progression of Parkinson's disease by 44%, commercial supplement companies are expected to bring out higher-potency CoQ10 capsules.

so important can be seen in a scenario of a patient taking Coumadin for one medical problem while at the same time using a high dose of coenzyme Q10 for related conditions. For instance, people who undergo artificial heart valve replacement often need Coumadin to prevent a blood clot forming on the valve, which can then travel up a carotid artery to cause a stroke. Many people needing valve replacement also have weakened heart muscles (congestive heart failure) and may require supplemental coenzyme Q10 to maintain cardiac output. Coenzyme Q10 enhances the energy-producing organelles called mitochondria to more effectively produce energy within heart muscle.

So for certain heart disease patients, Coumadin is prescribed to prevent abnormal clotting (thrombosis), while coenzyme Q10 is needed to maintain heart muscle output. Coenzyme Q10 also helps prevent oxidation of LDL cholesterol, which is felt to be part of the pathogenesis of vascular disease. These patients may safely benefit from Coumadin and coenzyme Q10 as long as their physician properly evaluates the blood tests and adjusts the Coumadin dose accordingly.

It should be pointed out that tens of thousands of Life Extension Foundation members have been taking coenzyme Q10 supplements over the past 20 years. There have been no reports of problems amongst Coumadin users. A physician who regularly prescribed Coumadin and coenzyme Q10 stated that he did not have to adjust the dose of Coumadin when adding coenzyme Q10. The only reason this issue is being raised now is that Parkinson's patients may be taking 1200 mg a day of coenzyme Q10. If these Parkinson's patients are taking Coumadin or enalapril, they should alert their physician in case the dose of Coumadin has to be increased or the enalapril decreased.

There is much debate about the interactions between dietary nutrients and prescription anti-coagulant medications (like Coumadin).<sup>^^</sup> For complete information on how to safely benefit from both anti-coagulant drugs and nutrients, refer to the new Thrombosis Prevention Protocol located under the "Health Concerns" section of the website [www.lef.org](http://www.lef.org) If you don't have computer access, call 1-800-544-4440 and you will be mailed a free copy of the updated Thrombosis Prevention Protocol.

\* Extra co-enzyme Q10 for statin-users? Treatment Update 2001 Jun;13(2):4-7.

\*\* Kishi T, et al. Bioenergetics in clinical medicine XV. Inhibition of coenzyme Q10-enzymes by clinically used adrenergic blockers of beta-receptors. Res Commun Chem Pathol Pharmacol 1977 May;17(1):157-64.

\*\*\* Spigset O. Reduced effect of warfarin caused by ubidecarenone. The Lancet. 1994;344:1372-1373.

^Takahashi N, Iwasaka T, Sugiura T, et al. Effect of coenzyme Q10 on hemodynamic response to ocular timolol. J Cardiovasc Pharmacol. 1989;14:462-468.

^^Danysz A, et al. Influence of coenzyme Q-10 on the hypotensive effects of enalapril and nitrendipine in spontaneously hypertensive rats. Pol J Pharmacol 1994 Sep-Oct;46(5):457-61.

## References

1. Ebadi M. et al. Ubiquinone (Coenzyme Q10) and mitochondria in oxidative stress of Parkinson's disease. Biol Signals Recept 2001 May-Aug;10(3-4): 224-53.
2. A Kalén, et al. Age-related changes in the lipid compositions of rat and human tissues. Lipids, July 1, 1989; 24(7): 579-84.
3. van Dyck CH, Age-related decline in dopamine transporters: analysis of striatal subregions, nonlinear effects and hemispheric asymmetries. Am J Geriatr Psychiatry 2002 Jan-Feb;10(1):36-43.
4. Kim JH, et al. Dopamine neurons derived from embryonic stem cells function in an animal model of Parkinson's disease. Nature, advance online publication, 2002, June 20, DOI: 10.1038/nature00900.
5. Vesper J, et al. Results of chronic subthalamic nucleus stimulation for Parkinson's disease: a 1-year follow-up study. Surg Neurol 2002 May;57(5):306-11; discussion 311-3.
6. Malhi GS, et al. Novel physical treatments for the management of neuropsychiatric disorders. J Psychosom Res 2002 Aug;53(2):709-19.
7. Luo J, et al. Subthalamic GAD gene therapy in a Parkinson's disease rat model. Science 2002 Oct 11;298(5592):425-9.
8. Albano CB, et al. Distribution of coenzyme Q homologues in brain. Neurochem Res 2002 May;27(5):359-68.
9. Beal MF. Coenzyme Q10 as a possible treatment for neurodegenerative diseases. Free Radic Res 2002 Apr;36(4):455-60.
10. Shults CW, 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.
11. Shults CW, et al. Coenzyme Q10 levels correlate with the activities of complexes I and II/III in mitochondria from parkinsonian and nonparkinsonian subjects. Ann Neurol 1997 Aug;42(2):261-4.
12. Shults CW, et al. A possible role of coenzyme Q10 in the etiology and treatment of Parkinson's disease. Biofactors 1999;9(2-4):267-72.
13. Haas RH, et al. Low platelet mitochondrial complex I and complex II/III activity in early untreated Parkinson's disease. Ann Neurol 1995 Jun;37(6):714-22.
14. Beal MF, et al. Coenzyme Q10 attenuates the 1-methyl-4-phenyl-1,2,3,4-tetrahydropyridine (MPTP) induced loss of striatal dopamine and dopaminergic axons in aged mice. Brain Res. 1998 Feb 2;783(1):109-14.
15. Lodi R, et al. Antioxidant treatment improves in vivo cardiac and skeletal muscle bioenergetics in patients with Friedreich's ataxia. Ann Neurol 2001 May;49(5):590-6.

^^Heck AM, et al. Potential interactions between alternative therapies and warfarin. Am J Health Syst Pharm 2000 Jul 1;57(13):1221-7; quiz 1228-30.

16. Musumeci O, et al. Familial cerebellar ataxia with muscle coenzyme Q10 deficiency. Neurology 2001 Apr 10;56(7):849-551.

17. Matthews RT, et al. 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.

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