

Dear Physician,

Many of you are aware of the work of David Perlmutter, MD, a Board Certified neurologist. Dr. Perlmutter has developed protocols, which have proven quite successful, for treating a variety of challenging neurological conditions.

Dr. Perlmutter's new book, *The Better Brain Book*, and *BrainRecovery.com* describes his treatment of brain disorders including multiple sclerosis, ALS, Alzheimer's disease, stroke, and Parkinson's disease.

**Wellness Pharmacy is referenced in *BrainRecovery.com* as well as *The Better Brain Book* as Dr. Perlmutter's sole source for injectable reduced L-Glutathione. Reduced L-Glutathione needs to be properly prepared and stored or it becomes oxidized in a matter of hours. Wellness has a patented method of preparation that enhances its stability to allow physicians to administer the product in near 100% reduced form.**

Sincerely,

The Staff of Wellness Pharmacy



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# New Advances in Parkinson's Disease

by David Perlmutter, MD

Board Certified Neurologist

From the book *BrainRecovery.com* (in print)

It has been estimated that in the United States alone more than 1 million people have Parkinson's disease, with more than 50,000 new cases being diagnosed each year. That translates to a prevalence of about 1-2 cases per 1000 individuals in the general population. This prevalence increases dramatically when looking at the over-55 population, approaching 1 in 100. The average age of onset is about 60 years, but it may be diagnosed as early as the mid 30's.<sup>1</sup> Perhaps because of some brain protective effects of female hormones, men are slightly more at risk than women.

Symptoms of Parkinson's disease vary from patient to patient but typically include tremor, rigidity, slowness of movement, and disturbances of posture. The tremor of the Parkinsonian patient is somewhat characteristic in that unlike other forms of tremor, it is worse at rest and may improve substantially when the affected limb is used. It is worse with stress and typically begins on one side, usually affecting the hand. Thereafter, the opposite hand may become involved as well as other parts of the body, including the legs, facial muscles and even the tongue.

The rigidity in Parkinson's may involve any of the major limbs. Typically there is increased tone throughout the range of motion of the involved joint.

Slowness of movement, technically known as *bradykinesia*, is another hallmark of the disease and can be one of the most incapacitating symptoms. Patients report difficulty in initiating movements and may have great difficulty in arising from a chair or starting to walk when standing. They may describe a sensation of feeling like they are wearing "cement boots," or that their feet are "magnetic." Facial expressions are reduced, and it may be difficult to begin speaking. The handwriting may become smaller and patients may find it difficult to turn over in bed.

As the disease progresses, the posture becomes affected with increased forward flexion at the waist and a tendency to stand and walk in a stooped position. As Dr. James Parkinson described in his original 1817 monograph:

"After a few more months the patient is found to be less strict in preserving the

upright posture: this being most observable whilst walking, but sometimes whilst sitting or standing. Sometime after the appearance of this symptom, and during its slow increase, one of the legs is discovered slightly to tremble, and is also found to suffer fatigue sooner than the leg of the other side: and in a few months this limb becomes agitated by similar tremblings, and suffers a similar loss of power."<sup>2</sup>

## The Glutathione Miracle

It has long been recognized that a fundamental abnormality in Parkinson's disease patients is the failure of a specific part of the brain, the *substantia nigra*, to produce an important brain chemical, the neurotransmitter *dopamine*. Focusing on this specific chemical flaw, the pharmaceutical industry has developed a wide array of medications to provide symptomatic relief.

In 1959 the first true therapeutic approach to treating the symptoms of Parkinson's disease was proposed, attempting to replace dopamine. This is the basis for the use of the dopamine derivative L-dopa (Sinemet®) in the treatment of Parkinson's disease symptoms today.<sup>3</sup> Indeed, even today L-dopa therapy remains the mainstay of treatment. Unfortunately, while L-dopa therapy may help to temporarily reduce the symptoms of Parkinson's disease, many scientific reports are now appearing in medical journals warning that L-dopa therapy may actually increase free radical production and thus speed up the progression of the illness, causing patients to worsen more quickly.<sup>4</sup>

With so much emphasis placed on L-dopa therapy, it is important to recognize that another vital brain chemical is also profoundly deficient in Parkinson's disease. This chemical, *glutathione*, is substantially reduced, virtually across the board, in Parkinson's patients. And yet, this deficiency seems to receive precious little attention.<sup>5</sup>

Glutathione is a critically important brain chemical. It is clearly one of the most important brain antioxidants. That is, glutathione helps to preserve brain tissue by preventing damage from *free radicals* - destructive chemicals formed by the normal processes of metabolism,

and as a normal response of the body to challenges by infectious agents or other stresses. In addition to quenching dangerous free radicals, glutathione also acts to recycle vitamin C and vitamin E, which, because of their antioxidant activity, also reduce free radicals in the brain.

So, with the understanding that glutathione is important for brain protection, and that this protection may be lacking in the brains of Parkinson's patients because of their glutathione deficiency, wouldn't it make sense to give glutathione to Parkinson's patients experimentally and observe their outcome? That's exactly what was done in a landmark study from the Department of Neurology, University of Sassari, Italy. In this research protocol Parkinson's patients received intravenous glutathione twice daily for 30 days. The subjects were then evaluated at one month intervals for up to six months. The published results indicated "all patients improved significantly after glutathione therapy, with a 42% decline in disability. Once glutathione was stopped the therapeutic effect lasted 2-4 months." Further, the researchers indicated "...glutathione has symptomatic efficacy and possibly retards the progression of the disease."<sup>6</sup>

It is unclear exactly why this study has remained almost completely unrecognized. In the United States, the use of L-dopa, or other drugs designed to mimic it, remains the standard of care. And yet, this Italian study demonstrated that providing glutathione, a substance naturally occurring in the brain, provided Parkinson's patients substantial benefit.

Glutathione as such, cannot be patented. So it cannot be owned exclusively by any particular pharmaceutical company and therefore won't find its way to the highly influential advertising sections of the medical journals. And yet, quite simply we know that the brains of Parkinson's patients are profoundly deficient in this important chemical, with clinical research supporting its incredible effectiveness.

We began administering intravenous glutathione in late 1998. The effectiveness of this brain antioxidant in

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Parkinson's disease is nothing short of miraculous. Certainly, its administration is more complicated than simply "taking a pill," but on the other hand, there are essentially no reported side effects. In addition, while our Parkinson's patients are now realizing profound improvements with respect to reduction of rigidity, increased mobility, improved ability to speak, less depression, and decreased tremor, glutathione has the added benefit of protecting the brain from free radical damage, thus *slowing* the progression of the underlying illness. This contrasts vividly with the simplistic approach of only treating symptoms while potentially worsening the underlying disease.

Following even a single dosage of intravenous glutathione, many of the symptoms of Parkinson's disease are rapidly improved, often in as little as 15 minutes. Injections are typically repeated from 3 times a week to as often as daily.

Here is an example of a typical response to glutathione therapy in a patient with moderately advanced Parkinson's disease:

Dear Dr. Perlmutter:

This letter is to advise you of the progress of my husband's response to the glutathione therapy started two weeks ago.

As you know (HS), now 72 years old, had been diagnosed with Parkinson's disease five years ago, starting with a tremor in his right hand. The disease progressed rapidly, impairing his walking ability, balance, and reducing his voice volume and clarity. Most recently, his inability to walk had made it necessary for him to use a wheelchair when leaving the house. At home, he has used a walker for the past two years.

His prescribed medications have included Sinemet®, Mirapex®, and Tasmartol® over the past years, the effects of which have diminished.

Almost immediately after your first treatment of glutathione IV two weeks ago, there was a marked improvement in his facial expression, his voice volume, and ability to walk and turn. He started with 400 mg., 3 times a week. The effective period of time after injection has increased from one hour to almost the whole day. When we visited your office last he received 600 mg., and his ability to walk almost normally lasted the full day and part of the next.

He also reports that he has a general feeling of wellbeing after each treatment. And he is now taking 400 units of

glutathione IV once a day, together with the supplements you have prescribed.

We are thrilled to report that he has not used the wheelchair for the past two days and is able to take full strides with his arm linked in mine. His facial expression is animated and his voice volume has increased.

In addition, we have cut back on his intake of Sinemet® and stopped the Tasmartol®. In the past, without the Sinemet®, he had been unable to walk - his legs practically frozen. With the glutathione therapy, he can walk with a reduced intake of Sinemet®.

We feel our prayers have been answered, that there is something positive that can be done to fight and arrest this dreadful disease now. We cannot thank you enough for the hope you have given us and we will keep you informed as to his progress until the next office visit.

Most consider Parkinson's disease to be an affliction only of the elderly. But we are now seeing patients in their 50's, 40's, and even 30's, with regularity. Here is a report from a 57-year-old plastic surgeon and former marathon runner:

Dear David:

This is a followup since my visit with you in April of 1999. As you will recall, my symptoms were those of micrographia (small handwriting), drooling, exhaustion, tremor, inanimate facies, poor voice projection and modulation, and depression. Your diagnosis was that of Parkinson's disease. I was started on glutathione 400 mg, three times a week, and I was instructed to take vitamins D, E, and B12 in addition to my rather extensive vitamin and herbal supplementation program.

I received my first dose of glutathione in your office that day and within two hours I felt like a new person. I was more animate and expressive almost immediately. Over the next few weeks, my voice became stronger, I felt less tired and my tremor almost disappeared. More slowly, my writing has improved; it's not perfect (never was) but at least with effort and slowing down, I can write legibly now. I still tend to drool some but even that is much improved. My energy is not totally back to normal but I am working a full schedule as a plastic surgeon with a very busy practice. My depression is gone and I have my sense of humor back.

When I was originally diagnosed at Duke, I was given a prescription for Sinemet® and advised to get my affairs in order. Your approach has kept me off

this medication, almost restored me to normal, and more importantly, has given me hope that we may slow the progression of my disease if not halt it altogether.

I want you to know how very much I appreciate your care, your caring, and your pioneering efforts.

Thank you

There are several factors that explain why glutathione is so beneficial in Parkinson's disease. First, glutathione has the unique ability to make certain areas of the brain more sensitive to dopamine, so that even though dopamine is decreased, it nevertheless becomes more effective.<sup>7</sup> The concept of enhancing cellular receptor sensitivity has become quite familiar in medicine today. In diabetes for example, before actually administering insulin, physicians often begin therapy by prescribing the drug *metformin*, which acts by enhancing the sensitivity of cells to whatever insulin is still being produced.

In addition, as mentioned above, glutathione has profound antioxidant activity - protecting the brain from free radical damage. But an even more intriguing benefit of glutathione lies not in the brain but in an area of the body far beyond the scope of typical neurology.

### The Liver Connection

Glutathione is one of the most important components of the liver's detoxification system. It has long been recognized that most Parkinson's patients manifest flaws in their ability to detoxify various chemicals to which they are exposed. This is the obvious explanation as to why Parkinson's disease is so much more prevalent in individuals with a history of occupational exposure to agricultural pesticides or various other toxic chemicals.<sup>8</sup> While not every person exposed to pesticides or other toxins develops Parkinson's disease, those unfortunate few who harbor an inherited flaw in their detoxification pathways are at far greater risk to the brain damaging effects of a wide variety of toxins as we described in 1997.<sup>9</sup>

Giving glutathione is one of the most effective techniques for enhancing liver and brain detoxification. The nutritional supplement N-acetyl-cysteine, (often abbreviated NAC), enhances the body's

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production of glutathione and thus aids the detoxification process. Other nutritional supplements which enhance glutathione and thus aid in detoxification include vitamins E and C, alpha lipoic acid, and the herb *silymarin*.

*UltraClear Plus™* a nutritional supplement designed by Dr. Jeffery S. Bland, has been extensively evaluated in clinical studies and has been found to significantly enhance liver detoxification.<sup>10</sup> We use this product as first line therapy when we identify individuals with abnormalities of detoxification.

Enhancing liver detoxification can have a dramatic effect on the manifestations of Parkinson's disease as exemplified by the following case history:

B.K. is a 40-year-old male who, in 1989 at the age of 34, began experiencing a tremor of the right hand. This was associated with micrographia (small handwriting) and the subsequent development of a right leg tremor. Over the next several years he developed slowness of movement, a reduction of facial expression, and a prominent loss of arm swinging when walking. He was placed on a sustained release preparation of L-dopa (Sinemet CR®) which produced a definite improvement of his symptoms.

When evaluated on 10/10/95, his medications included sustained release L-dopa (Sinemet CR® 25/100) three times each day, standard release L-dopa (Sinemet 25/100) twice each day, selegiline (Eldepryl®) 5 mg twice each day, and bromocriptine (Parlodel®) 5 mg twice a day. As with many of our patients, a videotape recording was made to document his clinical status.

His past medical history revealed that he had lived directly adjacent to a large commercial pesticide-using farm for the first twelve years of his life, and he recalled how he and his friends would follow the pesticide spraying tractors through the corn fields for fun.

On 02/06/96, the patient began a two-week nutritional program designed to improve liver detoxification. After the initial two weeks the patient reported, "My medications are working better and I have much less rigidity and tremor." These findings were confirmed on the physical examination. Videotape recording was made prior to and subsequent to the treatment protocol, and a significant improvement was also

noted in fluidity of movement, facial expression, and arm movement with walking. Perhaps even more impressive was the fact that these improvements persisted even after the medications were markedly reduced.

At followup examination *three years* after the initial detoxification he demonstrated continued improvement of clinical symptoms compared to his initial videotaped exam, and he remains on a reduced schedule of medications.

Evaluating an individual's detoxification status is easily accomplished using a very simple test, the *Hepatic Detoxification Profile* available from the Great Smokies Diagnostic Laboratory in Asheville, North Carolina (see below). The test involves the oral administration of several over-the-counter challenge substances. Subsequently, saliva, urine and blood are collected and analyzed to determine how these substances are metabolized. The results provide an extremely comprehensive picture of the various liver detoxification pathways, allowing the treating physician to design a specific interventional program to improve liver function.

Finally, keep in mind that certain drugs can reduce liver detoxification function. Acetaminophen, a drug commonly used for pain and fever, can actually reduce liver glutathione and should therefore be avoided.<sup>11</sup> It is found in a large number of over-the-counter and prescription medications so pay close attention to labels.

### Cellular Activation

During the 1990's, the so-called "Decade of the Brain," scientists learned that the fundamental flaw not allowing certain brain cells to produce dopamine in the Parkinson's patient is a deficiency in the actual energetics of these cells. It is as if these cells, while still alive, are simply unable to produce the energy needed for normal activity. Incredibly, the most widely prescribed medication for Parkinson's disease, L-dopa (Sinemet®), has been shown to actually lead to further compromise of the brain's ability to produce energy.<sup>12</sup> This further reduces the production of dopamine, leading to worsening of the disease.

With a formal understanding of the biochemistry of energy production, researchers have explored a variety of interventions designed to "jump start" these lethargic cells, often with dramatic results. And best of all, most of the research has involved non-

pharmacological products. The most promising of these cellular activators are NADH, CoQ10, and phosphatidylserine.

### NADH (Nicotinamide Adenine Dinucleotide)

NADH is an enzyme which has a pivotal role in energy production in all living cells, and particularly in brain cells. The amount of energy a cell can produce is directly related to NADH availability. Since Parkinson's disease represents a failure of cellular energy production, it's reasonable that researchers would take a look at NADH as a potential therapeutic agent.

Pioneering work published by Dr. Jörg Birkmayer in 1993 revealed just how potent NADH can be as part of a comprehensive program for the Parkinson's patient. Of 885 patients who received NADH in his study, an astounding 80% showed "moderate to excellent improvements in their disability."<sup>13</sup> This shouldn't come as a surprise given NADH's profound effectiveness in other neurological disorders including Alzheimer's disease.

### Coenzyme Q10 (CoQ10)

The other important player in energy production is CoQ10. Like NADH, CoQ10 is also present in all living cells where it too plays a critical role in cellular energy production. Energy deficiencies in specific parts of the brain can produce inadequate production of important brain chemicals. And, according to Dr. M. Flint Beal at the Massachusetts General Hospital, Parkinson's patients demonstrate a profound deficiency of coenzyme Q10 which may explain why their brains produce an inadequate supply of dopamine. Interestingly, Dr. Beal's research revealed that not only was coenzyme Q10 deficient in Parkinson's patients, but in their spouses as well - although to a lesser extent (see figure 1.1).<sup>14</sup> This unexpected finding lends further support to the concept that Parkinson's disease may in some way be related to some extrinsic environmental factor.

The encouraging news from Dr. Beal's research is that orally administered CoQ10 is readily absorbed, well tolerated, and measurably increases cellular energy production. These qualities, coupled with its profound antioxidant properties, likely explain why the therapeutic potential of CoQ10 in Parkinson's disease is now the subject of intensive research at major medical institutions all across the country.

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excessive free radical production coupled with deficient antioxidant defenses. This is not a new concept. The research exploring both the role of free radicals and the protective effects of antioxidants in diseases like Parkinson's goes back at least 2 decades. In a 1988 report entitled "Case-control study of early life dietary factors in Parkinson's disease" published

Finally, recognizing the importance of coenzyme Q10 makes it critical to identify any factors which may lower its availability. Unfortunately, two of the most commonly prescribed cholesterol lowering drugs, pravastatin (Pravachol®) and lovastatin (Mevacor®), dramatically lower serum coenzyme Q10 levels.<sup>16</sup>

### Coenzyme Q10 in Parkinson's Patients<sup>14</sup>

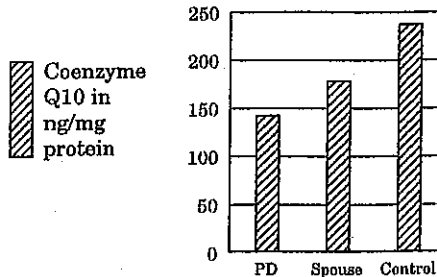


Figure 1.1. Relative levels of coenzyme Q10 in Parkinson's disease (PD) patients, their spouses, and controls.

### Phosphatidylserine

Phosphatidylserine is one of the key components of *neuronal membranes* – the site where brain cells both receive and transmit chemical messages. Enhancing chemical transmission is of obvious importance in Parkinson's disease, an illness in which the fundamental abnormality is a flaw in the ability of neurons to communicate chemically because of a deficiency of dopamine. Even in the face of this deficiency, increasing phosphatidyl-serine may enhance the effectiveness of what little dopamine remains – helping to preserve brain function.

The energy-producing mitochondria also rely upon a healthy membrane to carry out the function of energy production. Like the cellular membrane, the mitochondrial membrane requires adequate phosphatidylserine to maintain normal function.

It has been estimated that as many as 30% of Parkinson's disease patients suffer from a progressive decline not only in motor function, but in cognitive ability as well. At times the dementia associated with the disease is more debilitating than the common problems of tremor, rigidity and balance disorders. This further supports the inclusion of phosphatidylserine in treating Parkinson's disease since research supports its profound therapeutic potential in dementia. This was confirmed in a 1991 article in the journal *Neurology* in which

researchers from Stanford University demonstrated a marked improvement on performance tests related to memory and learning in a group of 149 memory-impaired patients treated with phosphatidyl-serine for 12 weeks.<sup>17</sup>

### Antioxidant Protection

As in other neurodegenerative diseases, antioxidants have an important role in protecting the brain in Parkinson's disease – a disease characterized by

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in *Archives of Neurology*, researchers discovered that simple dietary sources of vitamin E *profoundly* reduced the risk of Parkinson's disease. Compared to controls, those who consumed diets rich in nuts had a risk of Parkinson's disease only 39% of controls. Consumers of seed based salad dressings had a risk only 30% of normal, while consumption of plums was associated with a risk reduced to an incredible 24% of the average population.<sup>18</sup>

Retrospective epidemiological studies like these have prompted research to determine if administering antioxidants could slow the progression of disease in those already diagnosed. Dr. Stanley Fahn, one of the country's most highly respected neurologists and chairman of the Department of Neurology at Columbia University College of Physicians and Surgeons, evaluated the effectiveness of vitamins E and C in a large group of Parkinson's patients over several years. At the beginning of the study, none of the patients was debilitated enough to need the standard Parkinson's drug, L-dopa (Sinemet®). The time until patients required L-dopa therapy was extended an incredible 2.2 years in those taking these simple nonprescription vitamins.<sup>19</sup>

These results clearly indicate the power of antioxidants to slow the progression of Parkinson's disease. Shouldn't this information be provided to all Parkinson's patients and their families? Unfortunately, vitamin and nutritional information is not typically conveyed on the prescription pad – the ultimate coin of medical commerce.

Below is a descriptive list of powerful brain antioxidants, key players in the BrainRecovery.com protocol for Parkinson's disease.

### Alpha Lipoic Acid

The subject of intensive study in the neurodegenerative diseases, alpha lipoic acid not only serves as an extremely powerful antioxidant in and of itself, but in addition, it regenerates vitamins C and E as well as glutathione. But unlike glutathione, which isn't useful when given orally, alpha lipoic acid is readily absorbed from the gut and has the unique ability to cross the blood-brain barrier and enter the central nervous system.

Yet another quality of alpha lipoic acid is its ability to serve as a metal chelator. That means it can bind to a variety of

potentially toxic metals in the body, including cadmium and free iron, and enhance their excretion. This is an important function since these metals may increase the formation of damaging free radicals and research demonstrates substantially increased concentrations of iron in the brains of Parkinson's patients.<sup>20</sup>

### Vitamin E

Since cell membranes and the brain in general contain large amounts of fat, vitamin E, a "fat-soluble" vitamin is particularly important in protecting the nervous system. This important brain antioxidant remains the focus of worldwide scientific evaluation for its therapeutic potential in Parkinson's disease, Alzheimer's disease and various other neurodegenerative conditions. Since Dr. Fahn's original publication in 1989, countless other researchers have confirmed the antioxidant power of this inexpensive nutritional supplement.

When buying vitamin E, always read the label carefully to ensure you are getting *d-alpha tocopherol*, not *dl-alpha tocopherol*, since the latter is synthetic and far less biologically active. Also, always refrigerate vitamin E and all other oil-based nutritional supplements to preserve their potency.

### N-acetyl-cysteine (NAC)

The critical role of glutathione in the development and progression of Parkinson's disease cannot be overemphasized. While glutathione cannot be administered orally since it is readily digested to its constituent amino acids, the good news is that the nutritional supplement N-acetyl-cysteine directly encourages brain glutathione production. This activity is enhanced in the presence of adequate vitamin C and vitamin E. In addition, NAC itself is a potent antioxidant and has been shown to specifically reduce the formation of the free radical *nitric oxide*, which has been implicated as having a causative role in Parkinson's disease, Alzheimer's disease, and several other neurodegenerative disorders.<sup>21</sup> Consideration of the brain damaging effects of nitric oxide is particularly timely in view of the popularity of the drug Viagra® which works by enhancing nitric oxide production.

### Acetyl-L-carnitine

Like coenzyme Q10 and NADH, acetyl-L-carnitine enhances energy production in damaged neurons. But in addition, it is one of the most important

and specific antioxidants in the BrainRecovery.com protocol for Parkinson's disease. In a fascinating study reported in 1995, researchers demonstrated the ability of acetyl-L-carnitine to completely prevent parkinsonism in laboratory animals. When laboratory animals are exposed to the brain toxin MPTP, they immediately develop full-blown parkinsonism as a consequence of enhanced production of destructive free radicals specifically in the brain area that produces dopamine. Pre-treating the animals with acetyl-L-carnitine prior to MPTP exposure offered complete protection – none of the animals developed parkinsonism. This study affirmed the potency of acetyl-L-carnitine as an antioxidant specifically useful in Parkinson's disease.<sup>22</sup>

### Vitamin D

Although widely recognized for its role in maintaining healthy bones, vitamin D has recently become the subject of scientific interest since it too has been found to be an important antioxidant, possibly even more potent than vitamin E. In a recent (1997) study reported in the journal *Neurology*, Japanese researchers found surprisingly low levels of vitamin D in the blood of the 71 Parkinson's patients they evaluated. Not recognizing the important antioxidant, and therefore brain-protecting activity of vitamin D, these researchers simply concluded that because of their low vitamin D levels, Parkinson's disease patients are at higher risk for osteoporosis.<sup>23,24</sup>

### Ginkgo biloba

While widely known for its effectiveness in Alzheimer's disease, Ginkgo biloba must be included in this protocol as it too has profound brain antioxidant activity. Like acetyl-L-carnitine, Ginkgo biloba can also protect laboratory animals against the Parkinson's producing effect of the neurotoxin MPTP.<sup>25</sup> While humans are not typically exposed to this toxin, the idea that Parkinson's disease may be related to some other agent(s) is obviously supported by the profound increased incidence of the disease in those exposed to herbicides and other chemical agents as described above. Further, since dementia frequently complicates Parkinson's disease, inclusion of Ginkgo in the Parkinson's program makes sense, as this herb has been shown in extensive worldwide studies to enhance and preserve cognitive performance.

## Vitamin C

Rounding out the list of antioxidants for Parkinson's disease is vitamin C. Having proven itself to be effective in slowing the progression of this disease in Dr. Fahn's original research, vitamin C, like vitamin E, became the focus of extensive research not only in Parkinson's, but in other progressive brain disorders as well. Its specific utility in Parkinson's disease was emphasized in a study also performed by Dr. Fahn and colleagues in which it was found that vitamin C helped preserve the energy-producing capacity of the mitochondria – an abnormality actually made worse by the administration of L-dopa, the most widely prescribed drug for Parkinson's disease in the country.<sup>28</sup>

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## BrainRecovery.com – Parkinson's Protocol

## Intravenous Glutathione

Our protocol for using glutathione is relatively simple. Glutathione is inexpensive and easily obtained (see below). We use liquid glutathione, not reconstituted powder. It should be administered, at least initially, by a qualified healthcare practitioner as follows:

1. Dilute the appropriate dosage of glutathione liquid in 10 cc of sterile normal saline. Usually, vials contain 200mg, but read the label.
2. This solution is then injected through a 21-gauge butterfly catheter intravenously over a 15 to 20 minute period of time.
3. Alternatively, many patients choose to have intravenous access ports inserted. This allows frequent glutathione administration without repeated needle sticks.
4. Treatment begins at 600mg glutathione 3 times a week and may be increased to daily injections of up to 800mg depending on results. Alternatively, a schedule of 1000mg glutathione 3 times a week may be utilized.
5. An instructional videotape demonstrating glutathione administration with case reports is available at [www.BrainRecovery.com](http://www.BrainRecovery.com) or by calling: 800-530-1982

Injectable glutathione is available from: Wellness Health and Pharmaceuticals, 2800 South 18th Street, Birmingham, Alabama 35209 USA, 800-227-2627, Fax 800-369-0302.

Cellular energizers	daily dose
Coenzyme Q10 .....	120 mg
NADH .....	5 mg (twice)
Phosphatidylserine .....	50 mg

## Antioxidants

Vitamin E .....	1200 IU
Vitamin C .....	800 mg
Alpha lipoic acid .....	80 mg
Vitamin D .....	400 IU
N-acetyl-cysteine .....	400 mg
Acetyl-L-carnitine .....	400 mg
Ginkgo biloba .....	60 mg

## Note:

In Parkinson's patients less than 65 years of age check liver detoxification by performing Hepatic Detoxification Profile available from Great Smokies Diagnostic Laboratory, 63 Zillicoa Street, Asheville, North Carolina 28801-9801 USA, 800-522-4762.

If hepatic detoxification abnormalities are detected :

UltraClear Plus™ ..... 2 scoops twice daily  
Silymarin ..... 200 mg – twice daily

After 3 weeks discontinue UltraClear Plus™, continue silymarin and begin the standard BrainRecovery.com Parkinson's Protocol described above.

UltraClear Plus™ must be ordered by a physician and is available from Metagenics, 4403 Vineland Road, B-10, Orlando, Florida 32811 USA, 800-647-6100.

## Resources

*Living With Parkinson's Disease*, Kathleen E. Biziere, Matthias C. Kurth, Demos Vermande; ISBN: 1888799102. Published 1997

*Parkinson's: A Personal Story of Acceptance*, Sandi Gordon, Lee W. Tempel, Branden Publishing Co; ISBN: 0828319499; Published 1992

*Parkinson's Disease: The Complete Guide for Patients and Caregivers*, A. N. Lieberman (Editor), Frank L. Williams, Fireside; ISBN: 0671768190; Published 1993

*Understanding Parkinson's Disease: A Self Help Guide*, David L. Cram, Addicuc Books; ISBN: 0671768190; Published 1993

## References

1. Nutt, John G. In: Porter R.G. (ed), *100 Maxims in Neurology - (2) Parkinson's Disease*. St. Louis: Mosby Year Book, 1992; 1
2. Parkinson's, James, *An Essay on the Shaking Palsy*. London: Whittingham and Rowland, 1817; 4
3. Carlsson, A. The occurrence, distribution and physiological role of catecholamines in the nervous system. *Pharmacol Rev* 11:490-493, 1959

## Parkinson's Disease

4. Graham, D.G. Oxidative pathways for catecholamines in the genesis of neuromelanin and cytotoxic quinones. *Mol Pharmacol* 14:633-43, 1978
5. Perry, T.L., Godin, D.V., Hanson, S.: Parkinson's disease: A disorder due to nigral glutathione deficiency? *Neurosci Lett* 33: 305-310, 1982
6. Sechi, G., Deledda, M.G., Bua, G., et al., Reduced glutathione in the treatment of early Parkinson's disease. *Prog Neuropsychopharmacol Biol Psychiatry* 20(7): 1159-70, 1996
7. Bains, J. S., Shaw, C.A.: Neurodegenerative disorders in humans: the role of glutathione in oxidative stress-mediated neuronal death. *Brain Res Rev* 25 (3): 335-58, 1997
8. Tanner, C.M., Liver Abnormalities in Parkinson's disease. *Geriatrics* 46 (1): 60-63, 1991
9. Perlmutter, D., New Perspectives in Parkinson's Disease. *Thunsend Letter for Doctors* 162: 48-50, 1997
10. Bland, J.S., and Bralley, J.A., Nutritional Upregulation of Hepatic Detoxification Enzymes. *Journal of Applied Nutrition* 44 (3&4): 5-15, 1992
11. Vendemiale, G., Grattagliano, I., Altomare, E., et al., Effect of acetaminophen on hepatic glutathione compartmentation and mitochondrial energy metabolism in the rat. *Biochem Pharmacol* 25:52 (8): 1147-54, 1996
12. Przedborski, S., Jackson-Lewis, V., Muthane, U., et al., Chronic levodopa administration alters mitochondrial respiratory chain activity. *Ann Neurol* 34 (5): 715-23, 1993
13. Birkmayer, J.G. D., et al, Nicotinamide Adenine Dinucleotide (NADH) – A New Therapeutic Approach to Parkinson's Disease: Comparison of Oral and Parenteral Application. *Acta Neurol Scand* 87 (146): 32-35 1993
14. Schults, C.W., Haas, R.H., Passov, D., Beal, M.F., Coenzyme Q10 Levels Correlate with the Activities of Complexes I and II/III in Mitochondria from Parkinsonian and Nonparkinsonian Subjects. *Ann Neurol* 42:261-264, 1997
15. Shults, C.W., Beal, M.F., Fontaine, K. et al., Absorption, tolerability and effects on mitochondrial activity of oral coenzyme Q10 in parkinsonian patients. *Neurology* 60: 793-796, 1998
16. Mortensen, S.A., Leth, A., Agner, E., Dose-related decrease of serum coenzyme Q10 during treatment with HMG-CoA reductase inhibitors. *Mol Aspects of Med* 18(Suppl.) S137-44, 1997
17. Crook, T.H., Tinklenberg, J., Yasavage, J., et al., Effects of phosphatidylserine in age-associated memory impairment. *Neurology* 41:844-49, 1991
18. Golbe, L.I., Farrell, T.M., David, P.H., Case-control study of early life dietary factors in Parkinson's disease. *Arch Neurol* 45(12): 1350-3, 1988
19. Fahn, S., The endogenous toxin theory of the etiology of Parkinson's disease and a pilot trial of high-dose antioxidants in an attempt to slow the progression of the illness. *Ann N Y Acad Sci* 570:186-96, 1989
20. Olanow, C.W., Attempts to obtain neuroprotection in Parkinson's disease. *Neurology* 49 (Suppl 1) S26-S33, 1997
21. Pahan, K., Shelkh, G.S., Nmbodiri, A.M.S., et al., N-acetyl cysteine inhibits induction of NO production by endotoxin or cytokine stimulated rat peritoneal macrophages, C6 glial cells and astrocytes. *Free Radical Biology and Medicine* 24(1):39-48, 1998
22. Steffen, V., Santiago, M., de la Cruz, C.P., et al, Effect of intraventricular injection of 1-methyl-4-phenylpyridinium protection by acetyl-L-carnitine. *Human Exp Toxicol* 14:865-871, 1995
23. Sato, Y., Kikuyama, M., Oizumi, K., High prevalence of vitamin D deficiency and reduced bone mass in Parkinson's disease. *Neurology* 49(5): 1273-78, 1997
24. Sardar, S., Chakraborty, A., Chatterjee, M., Comparative effectiveness of vitamin D3 and dietary vitamin E on peroxidation of lipids and enzymes of the hepatic antioxidant system in Sprague-Dawley rats. *Int J Vitam Nutr Res* 66(1):39-46, 1996
25. Wu, W.R., Zhu, X.Z., Involvement of monoamine oxidase inhibition in neuroprotective and neurorestorative effects of Ginkgo biloba extract against MPTP- induced nigrostriatal dopaminergic toxicity in C57 mice. *Life Sci* 65(2): 157-64, 1999
26. Przedborski, S., Jackson-Lewis, V., Muthane, U., et al., Chronic levodopa administration alters cerebral mitochondrial respiratory chain activity. *Ann Neurol* 34 (5): 715-23, 1993