Neurological Decline: Natural Strategies to Protect Against Dopamine Defects

by Chris D. Meletis, ND

Parkinson’s disease is a progressive neurological condition classified as a neurodegenerative disorder associated with a loss of dopamine production in the brain. Clinically, signs of Parkinson’s disease (PD) typically include rigidity, resting tremor, postural changes such as stooping and akinesia, defined as the absence or loss of voluntary motion.1

Pathologically, Parkinson’s disease arises from the loss of sufficient dopamine production in the portion of the brain called the substantia nigra. To understand Parkinson’s disease, it is necessary to have a brief understanding of where the dopamine-producing cells are located. The basal ganglia is a round mass in the center of the brain that includes the substantia nigra that contains dopaminergic (dopamine-producing) cells.2

The basal ganglia is located at the base of the cerebral cortex that helps control coordination and movement. Dopamine is a neurotransmitter responsible for controlling voluntary movement and coordination. The death of these dopaminergic cells is responsible for this loss of coordination and voluntary movement.2

In the majority of individuals with Parkinson’s disease the cause remains

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Vitamin D3: Its Critical Role in Overall Health

by Jason Barker, ND

We’ve come a long way in our understanding of the importance of vitamin D in health. It was only 40 years ago that scientists reasoned that vitamin D was necessary for bone health, and no other aspects of proper physiologic function. It was correctly assumed that a lack of vitamin D led to the bone condition osteomalacia, or “softening” of the bones in adults and presented as rickets (bowing of the legs) in children. And like much of the Recommended Daily Allowances (RDA) of the day, amounts of vitamins were based on prevention of outward disease conditions, rather than on optimal health. For instance, the RDA in 1963 for vitamin D was 400 IU for children and half that for adults—amounts shown to be just enough for prevention of osteomalacia and rickets.1

Only in the last several years has our understanding of the importance of vitamin D in other areas of health come to light. Vitamin D is made in our bodies after skin is exposed to sunlight. However, the latest medical dogma that sun exposure will cause skin cancer has caused many people to slather on the sunscreen when outdoors prior to receiving any sun exposure. This fact, along with busy work schedules that prevent us from emerging outdoors in the middle of the day, has created a widespread vitamin D deficiency linked to various forms of cancer, high blood pressure, poor blood sugar control and impaired immunity.

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unclear, although it is strongly associated with environmental toxins, such as long term exposure to industrial solvents, Rotenone, MPTP, and welding, in addition to excess oxidative damage and genetic causes.3

Studies conducted on Parkinson’s patients from 2001 through 2008 have concluded that PD may be caused by genetic susceptibility to neurotoxins. A 2008 study in the Faroe Islands concluded that “the high frequency of PD in the Faroes is most likely the result of interactions between multiple genetic and environmental factors, still to be identified.”1,4

However, the most powerful evidence that Parkinson’s disease is caused by environmental exposures and not hereditary factors comes from the studies showing that the degree of hydrocarbon solvent exposure during a person’s lifetime is a major risk factor for Parkinson’s. This began when groups of patients with Parkinson’s disease revealed a chronic history of hydrocarbon solvent exposure.3

Researchers then examined 990 Parkinson’s patients. Exposure to hydrocarbon solvents directly correlated to disease severity and inversely correlated to latency period. Nine blue collar occupations accounted for 91.1 percent of exposures. The conclusion of the study was that “Occupations involving the use of hydrocarbon solvents are a risk factor for earlier onset of symptoms of PD and more severe disease throughout its course. Hydrocarbon solvents may be involved in the etiopathogenesis of PD, which does not have a major genetic component.”

Hydrocarbon solvents cause damage to cells by a process called lipid peroxidation, which is cell membrane damage caused by free radicals attacking the fatty acid layers in the membranes. Numerous other environmental neurotoxins have been shown to work through free radical damage. Antioxidants and nutritional sup-

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Functions of the Substantia Nigra
- Controls Voluntary Movement
- Produces the Neurotransmitter Dopamine
- Regulates Mood

“Parkinson’s Disease may be caused by genetic susceptibility to neurotoxins.”

CoQ10-H2™

Coenzyme Q10 has been widely studied for its role in neurological health due to its ability to help restore impaired mitochondrial function. The 100 to 300 mitochondria in every cell produce the vast majority of free radicals as a byproduct of oxygen reduction. The brain is a high-fat content organ like the liver and has low antioxidant capacity. CoQ10 therefore becomes important because it provides protection from lipid peroxidation. Coenzyme Q10 is part of the mitochondrial respiratory chain and it also donates an electron to the mitochondrial inner membrane to terminate free radical reactions. CoQ10 helps to restore mitochondrial function, which declines with age along with CoQ10 serum levels in humans.

The first significant clinical trial of Coenzyme Q10 in early stage Parkinson’s patients was conducted in 2002. Subjects were given 300, 600 or 1,200 mg per day of CoQ10 with 300 mg of vitamin E. The CoQ10 and vitamin E were consumed for 16 months or to a point where levodopa standard therapy was required. The Unified Parkinson Disease Rating Scale (UPDRS) was used to record functional decline between visits. A significant positive association was observed between the CoQ10 dosage level and the mean change in the UPDRS score. The conclusion of the study was that coenzyme Q10 was well tolerated at all doses and was effective “in reducing the functional disability in patients in the early stages of Parkinson disease.”11

The first generation of supplemental coenzyme Q10 (CoQ10-Ox), which is the form used in the above study, although still absorbed by the human body to produce some benefits, isn’t nearly as effective at increasing blood levels as its newer, more biochemically active, second generation form, CoQ10-H2™.

Many of the clinical studies that have shown positive neurological effects used rather large doses of CoQ10. For example, a 16-month randomized, placebo-controlled pilot trial in 80 subjects with mild Parkinson’s disease found significant benefits for oral CoQ10 at 1,200 mg per day. At this dose, coenzyme Q10 appeared to slow functional deterioration.12 However, 1,200 mg per day is a substantial and expensive dose of this antioxidant. Achieving similar benefits with a much lower amount of CoQ10-H2™ would obviously be ideal.
Cognitive-Enhancing Effects of Select Natural Substances

Vitamin B12

High homocysteine levels are common in Parkinson’s patients. The use of levodopa treatment in Parkinson’s patients further depletes vitamin B12 and folate acid, two vitamins known to reduce homocysteine levels. Increased homocysteine levels accelerate dopaminergic cell death (dopamine-producing cells) in Parkinson’s patients through neurotoxic effects.13,14

Choline and DMAE (Dimethylaminoethanol)

Choline and dimethylaminoethanol (DMAE) are the dietary precursors of the brain neurotransmitter acetylcholine. Acetylcholine plays a vital role in the cortical cholinergic system. Abnormalities of the cortical cholinergic system contribute significantly to the type of dementia found in Parkinson’s disease and Alzheimer’s disease. The enzymes that manufacture acetylcholine from dietary choline or DMAE are extremely low in Parkinson’s disease. The use of choline and DMAE in brain cells whether the cause is from chemical or disease depletion.16-17

Pyroglutamic acid (L-Pyroglutamic acid)

Pyroglutamic acid is a natural amino acid that has shown brain cell protection in a variety of studies. It is present in large amounts in the human brain, blood and cerebrospinal fluid. Pyroglutamic acid has a number of cognitive-enhancing effects. Pyroglutamic acid stimulates memory and the ability to focus.

The primary function of pyroglutamic acid as a protector of brain cells (neurons) is to remove excess glutamate from the brain. It does so by regulation of sodium-dependent glutamate transport. In one key study, it stimulated sodium-dependent transport of glutamate by 46 percent. Other studies reviewing amino acid transport in the brain show pyroglutamic acid is a key transporter of all amino acids in and out of the brain and maintains total amino acid balance in that organ.18-19

Ginkgo Biloba

Ginkgo biloba is a well-known herbal memory enhancer and supplement known to improve brain circulation. Numerous human trials with ginkgo have demonstrated that it improves cognitive health.

The latest 2008 trial with ginkgo lasting 42 months involved 118 patients 85 years and older who showed no evidence of any cognitive decline. Using the standard Clinical Dementia Rating test (CDR), researchers demonstrated that ginkgo biloba extract reduced the progression of clinical dementia and protected against memory decline.20

Reviews have focused on the biochemical mechanisms by which ginkgo reduces or

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blocks neuronal (brain cell) death. Nuclear transcription factor (NFkappaB) is believed to be most responsible for brain cell death by causing brain cells to induce apoptosis, or cellular suicide. Ginkgo biloba blocks this pathway through its antioxidant properties.²¹

Vinpocetine

Vinpocetine is a compound isolated from the Vinca family of plants and has been proven to be a potent neuroprotective agent in humans and animals. Vinpocetine increases blood circulation and metabolism in the brain. Animal studies show that vinpocetine reduces the loss of neurons caused by decreased blood flow.²²

In three human trials with older persons, vinpocetine produced more improvement than placebo on global cognitive tests of memory, attention and concentration. The adults in the study had either poor brain circulation or dementia-related diseases, including Parkinson’s or Alzheimer’s disease.²²

Vinpocetine is a supplement of choice in Europe as a neuroprotective agent. Diseases of brain circulation are major contributors to all dementia-related diseases, including Parkinson’s and Alzheimer’s. In humans, vinpocetine works by increasing cerebral glucose uptake and supports glucose metabolism in regions of the brain where patients have had a stroke. Two week long supplementation with vinpocetine also increased cerebral blood flow in the thalamus, basal ganglia and visual cortex of the brain in both normal volunteers and in stroke victims.²³²⁴

Huperzine A

In animal models of Parkinson’s, huperzine A prevents the loss and degeneration of dopamine-producing neurons in the substantia nigra caused by two powerful neurotoxins, both of which have been detected in Parkinson’s brains. The substantia nigra is the exact area of the brain in humans where dopamine-producing cells are lost resulting in Parkinson’s.²⁵

In a trial of 104 patients with presenile and senile simple memory disorders, memory improved in 2 weeks in subjects using huperzine A.²⁶ In the same study, an additional 56 patients had multi-infarct dementia, a more serious form of dementia. All patients in this subgroup responded well to 4 weeks of huperzine A at a slightly higher dose.²⁶

Neuron Growth Factors

In previous articles in this newsletter, it was shown that when acetyl carnitine and acetyl carnitine arginate are combined together they dramatically stimulate nerve growth factor levels in the brain. In a study of mouse brain neurons, the presence of nerve growth factor protected the brain cells against glutamate toxicity and 59 percent of the brain cells survived the massive influx of glutamate as opposed to no survival in the brain cell control group.²⁷

Combining acetyl carnitine and acetyl carnitine arginate with uridine, ginkgo biloba and gotu kola can have an even more pronounced effect on neurological health.

Conclusion

One of the best approaches to ensuring optimal neurological health is to use a supplement that contains Vitamins B12, DMAE (Dimethylaminoethanol), choline, pyroglutamic acid, ginkgo biloba, vinpocetine and huperzine A. This approach can be made even more effective when combined with acetyl carnitine, acetyl carnitine arginate and other compounds that synergistically produce nerve growth factor and other brain growth factors necessary for regrowth of brain communication pathways and for protection of brain cells. Finally, adding CoQ10-H₂²⁸ can provide additional neurological support.

References

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The NHF warns that the draft bill’s provisions also give the FDA one-sided legal authority to recall supplements. The result? The FDA, on an unjustified whim, could decide to ban any number of nutritional supplements classified as “foods.” If passed and enacted into law in its current version, the legislation would no doubt lead to more FDA regulation of supplements, higher costs for supplement manufacturers both foreign and domestic (which obtain many of their raw ingredients abroad), and these higher costs would in turn be passed on to consumers of dietary supplements.

The nutritional supplement industry is already governed by a number of laws. The FDA Globalization Act conflicts with the consumer-safety requirements already mandated by the Dietary Supplement Health and Education Act (DSHEA). The FDA Globalization Act would make it even easier for the FDA to continue its past history of subverting DSHEA. The proposed bill also is at odds with the Dietary Supplement and Nonprescription Drug Consumer Protection Act (DSNDCPA), better known as the Adverse Events Reporting bill, which Congress passed into law in 2006. With these two laws already in place and with the recently enacted Good Manufacturing Practices, there is little need to include nutritional supplements in a bill that is essentially irrelevant to our industry. The anti-supplement congressmen and senators are simply using incidents unrelated to nutritional supplements to exert control over an industry that has an enviable safety record.

Please contact your congressional representative—especially Energy and Commerce Committee members—and ask them to amend this proposed bill to specifically exempt its application to dietary supplements. Together, we can stop the FDA from exerting even more control over your health choices and keep dietary supplements affordable and accessible to everyone. For more information, please visit the National Health Federation website, www.thenhf.com.

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