What Is It?

Parkinson’s disease is the most common serious movement disorder and second most common neurodegenerative disorder in the world (the first being Alzheimer’s). The disease involves loss of neurons in the specific area of the brain that controls motor movement, the substantia nigra (Latin for “black substance”). Parts of the substantia nigra are darker due to high levels of melanin in these dopamine-producing neurons. The substantia nigra is part of the basal ganglia. Parkinson’s disease is caused by the gradual death of dopaminergic neurons in the substantia nigra. Many of these neurons are rich in dopamine, and the loss of these neurons means a loss of dopamine. Therefore, a loss of control of movement is the central theme in Parkinson’s.

Another finding in the brains of people with Parkinson’s is the presence of Lewy bodies in the remaining neurons. These Lewy bodies, or protein accumulations, have been associated with the destruction of brain cells in Parkinson’s and other neurodegenerative diseases.

What Causes It?

The cause of Parkinson’s disease is not entirely known. As with most conditions, the development of Parkinson’s is thought to be the result of a combination of genetic and environmental influences. Genetics plays a smaller role in the development of late-onset Parkinson’s, but in young-onset Parkinson’s, the genetic link has been found to be stronger.

The primary causes of Parkinson’s that are currently being studied include:

- Oxidative stress and free radicals
- Mitochondrial defect
- Excitotoxicity
- Inflammation

Oxidative stress occurs in the body when there are too many free radicals, which create different forms of oxygen-free radicals (superoxide, hydrogen peroxide, hydroxide). Free radicals are highly reactive molecules that cause damage when they are present in high amounts. When the body’s immune system and antioxidant levels are normal, free radicals are kept under control and do not cause cell damage. But, when there are too many free radicals, they injure cells and cause cell death leading to significant decreases in dopamine.

To make matters worse, free radicals can also be formed when dopamine is broken down. This can lead to the inability of proteosomes (cell trash processors) to remove intracellular protein wastes (Lewy bodies). This intracellular accumulation of waste further impairs the cell’s ability to make dopamine. In a healthy body (or brain), these free radicals are detoxified by glutathione. In a Parkinson’s patient, this does not happen in a normal way. Studies are underway looking at the effects of different antioxidants (including glutathione) in Parkinson’s patients.

Free radicals also play a role in the mitochondrial dysfunction in Parkinson’s patients. As a matter of fact, the free radicals may form as a result of this mitochondrial dysfunction. Mitochondria are the energy producers of the body, but they also regulate other processes including cell growth and cell death. Exactly how the mitochondria may lead to Parkinson’s is not entirely understood yet, but it is currently being studied.
Excitotoxicity is the process by which neurons are damaged and destroyed by glutamate and similar substances. Glutamate naturally occurs in the brain, but when it exists in high quantities, or when glutamate receptors are not functioning correctly, it can cause cell death in the brain. A high concentration of glutamate is present in the subthalamic nucleus, which is closely connected to the substantia nigra compacta, the area of the brain that is affected in those with Parkinson’s. Research is currently looking at new therapies that block glutamate receptors, and, therefore, protect dopamine cells.

Excitotoxicity has been implicated in other conditions such as autism, Alzheimer’s, multiple sclerosis and even stroke. Monosodium glutamate (MSG) has been shown to cause excitotoxicity in the brain in animal studies, and many caution against ingesting this ingredient, which is widespread in processed foods. Additionally, excitotoxicity may even occur with normal brain levels of glutamate.

Inflammation in the brain, or neuroinflammation, is another possible cause of Parkinson’s disease. In fact, inflammation may lead to a worsening of oxidative stress, mitochondrial dysfunction and cell death. Environmental exposures and chronic inflammatory conditions can potentially trigger this neuroinflammation. The underlying role inflammation plays in many disease processes is further illustrated here. Inflammation even reaches the brain and can produce devastating effects.

The main environmental risk factors that have been associated with Parkinson’s disease are:

- Pesticide exposure
- Rural living
- Farming
- Drinking well water
- Dairy consumption

Numerous studies have found an association between pesticide exposure and Parkinson’s disease. Some researchers believed that pesticide exposure was only a risk factor because it went along with rural living, farming and drinking well water. A recent study, however, has found that pesticide exposure is a risk factor even in people who did not live in rural areas, and who did not drink well water. Another study found that people who use pesticides in their homes are twice as likely to develop Parkinson’s disease as those people who are not exposed.

A particular pesticide group, the organochlorines, were found in higher concentrations in the blood of Parkinson’s patients. Organochlorines, such as DDT, are more tightly regulated today than in the past largely because they persist in the environment for many years. Organochlorines are still found in the environment, and so called “safer” organochlorines are still in use today. Important to mention, DDT is not banned in many other countries, so it may still be found on imported foods.

Manganese poisoning has also been linked to the development of Parkinson’s disease. Manganese exposure is most common in industries such as welding, mining and steel manufacturing. One substance, MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), was discovered as a direct cause of Parkinson’s disease in a small group of drug addicts who had ingested the chemical thinking that it was MPPP, a synthetic opioid that produces similar effects as heroin and morphine. Instead, the addicts developed symptoms of Parkinson’s. The similarity between MPTP and other chemicals in the environment supports the theory that there are environmental substances that contribute to the disease.

Diet may also play a role in the development of Parkinson’s. High intake of dairy products, especially in men, is associated with a greater risk of developing the disease. Men with the highest intakes had a 60 percent greater chance of developing Parkinson’s than men with the lowest intakes. This is an interesting link that is being further investigated.
What Are the Signs and Symptoms?

There are four main characteristics of Parkinson’s disease:  

• Rest tremor
• Rigidity
• Bradykinesia
• Postural instability

Rest tremor refers to shaking, usually of a limb, upon rest or when it is not being used. Rest tremor is the most common and easily recognized symptom of Parkinson’s. Rest tremors can also occur in the lips, chin or jaw. In Parkinson’s, tremor rarely occurs in the neck, head or with the voice. While most Parkinson’s patients experience rest tremor, up to 11 percent do not. This has been the cause of some confusion in patients, and even some doctors. The shaking of Parkinson’s is sometimes thought to be a universal symptom, and patients who do not exhibit tremors are sometimes misdiagnosed.

Rigidity is often described as stiffness, cramps, or aches and pains. This symptom often begins as shoulder stiffness, which can be misdiagnosed as arthritis, bursitis or rotator cuff injury. Rigidity may or may not occur in a Parkinson’s patient.

Bradykinesia is a slowness of movement. Most people with Parkinson’s have this symptom, and it can appear in a variety of ways. Bradykinesia can be mistaken for muscle weakness in the patient, but what is really occurring is a slowed movement. Some variations of bradykinesia include: difficulty with fine motor tasks, difficulty initiating movement, change in gait or walking (slow, shuffling, or dragging one leg), freeze attacks (sudden halt while walking), masked face (expressionless), micrographia (very small writing), and hypophonia (high-pitched voice).

Postural instability involves the loss of balance. In young-onset Parkinson’s, this symptom is usually absent for a number of years. As the disease progresses, and in many older adults with Parkinson’s, balance becomes increasingly difficult to maintain. Postural instability is a cause of falls in up to 38 percent of Parkinson’s patients.

Development of these motor symptoms in Parkinson’s usually begins on one side of the body. As the disease progresses, the other side is affected, but this may take many years. Other motor symptoms that may be seen in those with Parkinson’s are decreased blinking, limited upward gaze, difficulty speaking, drooling, and decrease in swallowing.

Non-motor symptoms are also common in Parkinson’s disease. These may include:

• Low blood pressure
• Erectile dysfunction
• Depression
• Anxiety
• Sleep disorders
• Hallucinations
• Impulsive behaviors such as: Binge eating
  Uncontrollable gambling
  Compulsive shopping

Some of the above listed symptoms have been linked to the medications that are prescribed for Parkinson’s. Hallucinations occur in about 30 percent of patients taking medication. Few doctors are aware that this side effect is so common, in part, because many patients do not want to admit that it exists for fear of being labeled as losing their mind. Uncontrollable gambling is another side effect that has received a lot of attention. People taking dopamine agonist drugs (drugs prescribed for Parkinson’s) have been found to be more likely to have a gambling compulsion.
Many people notice that their Parkinson’s symptoms worsen with stress. There is a physiologic reason for this. The body produces adrenaline, or epinephrine, to deal with stress. Adrenaline is formed from dopamine, however, which is the very chemical that is deficient in those with Parkinson’s. This highlights the importance of stress management in people with Parkinson’s.

Parkinson’s disease affects about one percent of adults over the age of 60. It is estimated to affect up to one million Americans, a number that will probably double in the next 15 to 20 years due to the aging of the American population.

**How Is It Diagnosed?**

By the time the symptoms of Parkinson’s are experienced, about 80 percent of the dopamine-producing cells and 50 percent of associated neurons are lost. Even when symptoms do appear, many patients are not properly diagnosed for up to five years. Some patients may even try to hide the symptoms, not realizing that they may be a part of a more serious problem.

The diagnosis of Parkinson’s disease involves a thorough medical history and physical examination. One reason for the difficulty in diagnosing Parkinson’s is that there is no definitive diagnostic test for the disease. The diagnosis is probable when two of the following three symptoms are present:

- Asymmetric resting tremor
- Asymmetric rigidity
- Asymmetric bradykinesia

The diagnosis is considered definite when the above criteria are met, and when the symptoms respond to Parkinson’s medication. However, a confirmation of diagnosis is only possibly during autopsy, as this is where the biological markers are verified.

Neuroimaging (MRI or CT scan) is not usually necessary in diagnosing Parkinson’s, but it can be useful in unusual cases. It is most helpful in ruling out other conditions that may present similar symptoms as Parkinson’s.

Due to the possibility that heavy metals and toxins may play a role in the development of Parkinson’s, urine, stool or hair-toxicity tests may be helpful to rule out this possibility. Another useful test may be the hepatic detox profile, which determines how well the liver is getting rid of toxins.

**What Are the Standard Medical Treatments?**

Treatment of Parkinson’s disease deals with symptom relief, as there is not yet a cure. Medication is not necessarily started upon diagnosis. If symptoms are not a bother to the patient, drug treatment can be postponed. The doctor should take patient preferences into account when initially prescribing medications.
The two most common medications for Parkinson’s treatment are levadopa, which is a dopamine precursor, and dopamine agonists. Levadopa is the most potent antiparkinson drug, and most patients will be on it at one time or another. Most patients will begin treatment with one of these two drugs. Younger patients will usually begin with a dopamine agonist and later add levadopa when more symptom control is needed. Older patients will usually begin with levadopa, which has less adverse effects. Hallucinations, especially in older patients, are one such adverse effect.

Interestingly, Parkinson’s is sometimes considered the opposite of schizophrenia. In schizophrenia, there is an overabundance of dopamine in the brain. Medications for schizophrenia work to decrease the dopamine levels in the brain. Side effects of these drugs can resemble Parkinson’s symptoms. The reverse of this effect is seen in Parkinson’s patients taking medication to increase dopamine in the brain. They experience hallucinations, which are common in schizophrenia.

One complication of long-term levadopa treatment is the on/off effect, also known as motor fluctuations or dyskinesia. This is when the drug begins to wear off, and Parkinson symptoms reappear. At first, these effects are predictable, and can be controlled with better timing of medications. But over time, the off periods fluctuate and become increasingly difficult to control.

One important consideration when taking levadopa is to avoid the drug-nutrient interaction of eating protein when taking the drug. The amino acids in protein compete with levadopa for absorption through the intestinal wall and through the blood brain barrier. This is why it is important to wait at least one hour after taking levadopa, or at least until the drug has taken effect, to eat protein. Likewise, it is recommended to wait two hours after eating protein before taking levadopa.

In young patients with tremor as the main symptom, anticholinergic drugs are sometimes used. These drugs are not usually recommended in older patients due to the many side effects. MAO-B inhibitors and amantadine are two other drug treatments that are associated with fewer side effects, but, when used alone, they do not usually provide adequate symptom control.

When symptoms become severe and no longer respond well to medication, the patient may be recommended for surgical therapy. Deep brain stimulation is the surgical treatment most common in Parkinson’s patients. Depending on the specific symptoms the patient experiences, different parts of the brain are stimulated by the placement of a metal electrode into the brain at the thalamus, globus pallidus or subthalamic nucleus site. The electrode is then connected to a battery source which stimulates the brain at a high frequency.

A recent study in the Journal of the American Medical Association found that deep brain stimulation was more effective than best medical practices (well-controlled medications and other therapies) in improving motor function and quality of life in Parkinson’s patients who experience motor complications and inadequate symptom control with medication. Deep brain stimulation was associated with an increased risk of serious adverse effects, however, so candidates should be made aware of these.

Other surgeries for Parkinson’s disease include pallidotomy, thalamotomy and gamma knife radiosurgery. These surgeries have not been shown to be as safe or effective as deep brain stimulation, however, and are not usually recommended.

Restorative treatment using stem cells has been studied, but, as of now, it has not been completely successful. This treatment involves implanting stem cells into the brain which can generate dopamine neurons. More research is going on in this area, and it may prove to be a promising option in the future.
There are several connections between gut issues and Parkinson’s disease (PD). In addition, established PD has been improved by fixing intestinal problems. One underlying principle involves gut dysbiosis, which upregulates (increases) pro-inflammatory cytokines from the GALT (gut-associated lymphoid tissue) and from the liver. These cytokines have systemic effects throughout the body and brain. When these inflammatory cytokines reach the brain, they can cause an excessive increase in brain nitric oxide production from the glial cells, and allow too much calcium to enter their neighboring neurons. This leads to mitochondrial apoptosis (cell death) and lowering of ATP (energy) levels. (Burn out the engines, and the cells slow down and may die.)

This could be especially dangerous in PD since the substantia nigra dopaminergic neurons are particularly vulnerable to energetic imbalances (i.e. low ATP). Optimum nitric oxide levels are needed for normal brain function, but excess nitric oxide levels create dangerous free radicals, especially peroxynitrite, that increase inflammation and damage. In PD, inflammation is a critical factor for cell degeneration.

Two other gut-brain connections in PD are H. pylori stomach infections and constipation. Studies have shown the role of H. pylori bacterium in cardiovascular diseases, neurodegenerative diseases, ocular diseases, and dermatological disorders. DNA damage and failure of antioxidant defenses come into play, especially in neurologic disorders like PD.

In a few well-documented cases of idiopathic (cause unknown) parkinsonism with weight loss and elevated IL-6 cytokines, treatment for H. pylori with triple antibiotic therapy resulted in reversal of weight loss and recovery from the neurologic disability associated with PD. In this paper, it was thought neuronal damage may be in response to a ubiquitous occult infection.

There is a very interesting connection between PD and the colon, especially in PD patients with chronic constipation. Clinical research has shown that PD and the enteric nervous system (ENS) of the intestinal tract have something in common—Lewy bodies, whose main component is phosphorylated alpha-synuclein. The colonoscopic biopsies in PD patients have found these inclusions in the ENS of the intestinal lining, and they are the same as the Lewy bodies found on autopsy in the brains of PD patients.

One theory is that iron and other toxic metals damage the DNA (in both substantia nigra of the brain and ENS of the intestines) and cause the production of damaged alpha-synuclein. This then builds up in the cells, damages the dopamine producing substantia nigra neurons, and causes both PD in the brain and marked decreased motility in the intestines, leading to severe constipation.

It is very important with PD to begin looking early since up to 70 percent of the substantia nigra dopamine producing neurons may be gone before patients have classic symptoms of PD (tremor and rigidity). Patients with abnormalities of smell, problems with constipation, and disordered REM sleep can be early signs of impending neurodegeneration including PD and Alzheimer disease, and should alert the physician to proceed with further testing. PET scanning of the brain is one of the best imaging techniques, to date, to show dopaminergic deficits in the substantia nigra pathway, which would be an early indicator of PD.

Since there are many interventions including: diet, exercise, specific supplementation, stress reduction, quality sleep, good elimination, detoxification and chelation of toxic metals (including excess iron) that may prevent progression of PD, it would be worth looking into early diagnostic testing and natural treatments that could prevent this condition from fully manifesting.
The inflammation and toxicity associated with Parkinson’s disease can be the result of long-term gut issues and toxin exposure that overburdens the body resulting in neurological damage. Many people with this condition have had constipation for their entire lives, so fixing digestive problems is important. Finding underlying gut imbalances with a comprehensive stool analysis (CSA) can be beneficial, as can toxicity testing, such as a heavy metal hair analysis.

“**The inflammation and toxicity associated with Parkinson’s disease can be the result of long-term gut issues and toxin exposure that overburdens the body resulting in neurological damage.**”

Another possible underlying factor in people with Parkinson’s disease is Lyme disease, which involves a bacterial infection when certain Borrelia bacteria is transmitted through a tick bite. This infection, when undiagnosed for a period of time, enters the central nervous system forming a cyst and creating neurological symptoms that are similar to Parkinson’s disease (also Alzheimer’s disease). Because the symptoms of Lyme disease resemble many other conditions, and because people may not even know that they were bitten by a tick, this condition often goes undetected. The Central Florida Research Laboratory, founded by Dr. Jo Anne Whitaker, M.D., specializes in a specific test that detects this disease.

If these tests are not possible, then following the Steps to Cleansing (see the Appendix), and the recommendations below and on the next page will help to build a healthy foundation that can help reduce inflammation and underlying digestive imbalance.

**Recommended testing**

- Comprehensive stool analysis (CSA) (See the Appendix.)
- Hair toxic element profile (See the Appendix.)

**Diet**

- Eat a low-protein breakfast and lunch and a high-protein dinner.
- Follow the Fiber 35 Eating Plan found in the Appendix of this book.
- Include plenty of fruits and vegetables, whole grains, nuts, fish and poultry in the diet to provide a balance of nutrients.
- Reduce or eliminate dairy consumption, as high dairy consumption can increase the risk of Parkinson’s disease, especially in men.
- To avoid toxins in foods, eat organic foods whenever possible.
- Reduce consumption of saturated fat and eliminate trans fats from the diet.

**Lifestyle**

- Regular physical activity can help to improve mood by releasing endorphins.
- If overweight, lose weight because body mass index (BMI) is associated with Parkinson’s disease risk.
- Reduce toxin exposure.
- Get adequate sleep because lack of sleep can adversely affect mood.

**Complementary Mind/Body Therapies**

- Stress reduction therapies such as yoga, biofeedback, massage, and meditation can be helpful to relieve stress.
- Acupuncture may be helpful for people with Parkinson’s.
- Colon hydrotherapy is beneficial to remove toxins.
<table>
<thead>
<tr>
<th><strong>Recommended Nutraceuticals</strong></th>
<th><strong>Dosage</strong></th>
<th><strong>Benefit</strong></th>
<th><strong>Comments</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Critical Phase</strong></td>
<td>Daily maintenance recommendations should also be taken during this phase unless otherwise indicated.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Probiotics</td>
<td>200 billion culture count daily for two weeks</td>
<td>Protects the intestinal lining, binds toxins, helps improve immune and digestive function.</td>
<td>Look for high amounts of bifidobacteria, the main bacteria in the colon.</td>
</tr>
<tr>
<td>Brain Support Formula</td>
<td>Use as directed</td>
<td>Helps to increase L-dopamine.</td>
<td>Look for ingredients such as L-tyrosine, velvet bean, antioxidants, and NADH.</td>
</tr>
<tr>
<td>Liver Detox</td>
<td>See Appendix</td>
<td>Encourages detoxification involving the liver.</td>
<td>Should contain milk thistle seed extract containing silymarin, phosphatidylcholine, selenium and herbs.</td>
</tr>
<tr>
<td><strong>Helpful</strong></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Multivitamin/mineral Formula</td>
<td>High potency</td>
<td>All vitamins and minerals support cardiovascular function.</td>
<td>Be sure vitamins are in their natural forms. Look for high amounts of B vitamins.</td>
</tr>
<tr>
<td><strong>Daily Maintenance</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Critical Liver Support Formula</td>
<td>Use as directed</td>
<td>Enhances liver detoxification.</td>
<td>Should include milk thistle seed extract containing silymarin, N-acetyl-cysteine, alpha lipoic acid, L-glutathione.</td>
</tr>
<tr>
<td>Probiotics</td>
<td>50 billion culture count daily after critical phase</td>
<td>Protects the intestinal lining, binds toxins, helps improve immune and digestive function.</td>
<td>Look for high amounts of bifidobacteria, the main bacteria in the colon.</td>
</tr>
<tr>
<td>Omega-3 Fatty Acids</td>
<td>At least 1-2 grams daily</td>
<td>Helps support healthy brain function.</td>
<td>Get a concentrated, enteric coated, high dose EPA/DHA formulation.</td>
</tr>
<tr>
<td>Fiber</td>
<td>4-5 grams twice daily</td>
<td>Helps bind toxins, produce healthy bacteria levels and good bowel elimination.</td>
<td>Constipation is common in people with Parkinson's.</td>
</tr>
<tr>
<td>Digestive Enzymes</td>
<td>1-2 capsules with meals</td>
<td>Helpful to fully digest foods and absorb nutrients important for brain function.</td>
<td>If stomach acid is low, use a formula with HCl.</td>
</tr>
<tr>
<td>Vitamin D₃</td>
<td>At least 1,000 to 2,000 iu daily</td>
<td>Helps heal leaky gut, decrease inflammation, increase overall health.</td>
<td>Vitamin D deficiency is common in people with Parkinson's.</td>
</tr>
</tbody>
</table>

See further explanation of supplements in the Appendix