



soar

SOAR

*Parkinson's
Disease*

Reduce Risk. Improve Symptoms.

Eat Well. Move Well. Sleep Well. Soar On.

made with
Beacon

Table of Contents

1. Medical Disclaimer
2. Introduction
3. About The Author
4. What is Parkinson's Disease?
5. Eat Well - Causes
6. Move Well - Causes
7. Sleep Well - Causes
8. Soar On - Causes
9. Eat Well - Solutions
10. Move Well - Solutions
11. Sleep Well - Solutions
12. Soar On - Solutions



13. Conclusions

14. Tracking Lifestyle Habits

15. Set Your VISION

16. References

Medical Disclaimer

This ebook is for informational purposes only.

The information is a result of practice experience and research by the author. It is not intended as a substitute for the advice provided by your physician or other healthcare professional or any information contained on or in any product label or packaging. Do not use the information for diagnosing or treating a health problem or disease, or prescribing medication or other treatment. Always speak with your physician or other healthcare professional before taking any medication or nutritional, herbal or homeopathic supplement, or using any treatment for a health problem. If you have or suspect that you have a medical problem, contact your health care provider promptly. Do not disregard professional medical advice or delay in seeking professional advice because of something you have read in this ebook. Information provided here DOES NOT create a healthcare provider-patient relationship between you and the author. Information and statements regarding dietary supplements have not been evaluated by the Food and Drug Administration and are not intended to diagnose, treat, cure, or prevent any disease.

Introduction

Dr. Carolyn Dolan PT, DPT, Cert MDT, MSHN

The diagnosis of Parkinson's Disease can be daunting especially with the feeling of powerlessness. This book was designed to help you either as a patient or clinician or support person to understand how SIMPLE lifestyle strategies can have a HUGE impact on reducing your risk for developing Parkinson's Disease and even improve your symptoms with Parkinson's disease.

Key Subjects

After reading this book you will:

- Understand what Parkinson's Disease as a complex neurodegenerative process
- Be able to identify lifestyle factors that contribute to the onset Parkinson's Disease
- Be aware of the lifestyle habits which may reduce your risk of developing and improve symptoms of Parkinson's disease
- Learn how to make simple choices that will have a big impact on your mental and physical health

Parkinson's disease is near and dear to my heart as you will learn more later. As I have discovered, this otherwise very complicated disease can have some very simple solutions. Understanding those simple solutions allows us all to improve symptoms during rehabilitation and even reduce the risk of developing.



Dr. Carolyn Dolan, PT, DPT, Cert MDT, MSHN

About The Author

Dr. Carolyn Dolan PT, DPT, Cert MDT,
MSHN



In 2012, I started to take care of myself as I became dissatisfied with my health. I suffered from chronic sinus infections, chronic fatigue, chronic depression, chronic anxiety, and overweight. I started with exercise and then fell into the world of Paleo. I reversed my chronic conditions and lost weight. However, I gained more than the weight I lost. I started to see not only my own health improvements, but also that of my husband and children. I then saw results with patients in resolving debilitating conditions by coaching them about healthy lifestyle habits including nutritious food, movement, adequate rest and connection (to their body and community). You can learn more about my story in the book, **Soar Into Health**

My Education

I received my Bachelor of Science with Honors in Biological Systems Engineering from the University of California, Davis. My

Physical Therapy training began at the Graduate Program in Physical Therapy at the University of California San Francisco/ San Francisco State University where I received a Masters of Science in Physical Therapy. I then completed my Doctorate in Physical Therapy thru Mass General Hospital Institute of Health Professionals. I am also certified in the McKenzie Method of Mechanical Diagnosis and Therapy from The McKenzie Institute, USA. I have been practicing physical therapy in a variety of settings since 2001, specifically in outpatient orthopedics since 2007. I have recently completed Masters of Science in Holistic Nutrition to integrate into my physical therapy practice and coaching

My Mission

To inspire parents, patients and health care providers to address for the foundational lifestyle principles to optimize recovery, function and performance in the things they love to do.

What is Parkinson's Disease?

Parkinson's disease (PD) is the second most common neurodegenerative disorder behind Alzheimer's disease.¹ This statistic is projected to grow with our aging population.² The hallmark physical symptoms are tremor, bradykinesia (slow movement), rigidity (inability to move), loss of automatic movements, impaired balance, disturbed sleep, and monotone speech and/or writing changes.³ These symptoms are primarily a consequence of a loss of dopamine (a neurotransmitter involved in reward and motor control) production in the Substantia Nigra in the brain.¹ However, it is becoming increasingly clear there are other common and potentially equally disabling non-motor, multisystem signs and symptoms such as inflammation, pain, sleep difficulty, gastrointestinal dysfunction, depression and cognitive decline.⁴ Yet, even today the most well recognized motor signs and symptoms of PD are primarily focused on the loss of dopamine.

Dopamine is produced in the Substantia Nigra. The precursor to dopamine is L-Dopa. The methylation, a biochemical process adding a methyl (single carbon and 3-Hydrogens) group, of L-dopa results in dopamine, which is highly dependent on Vitamin B6 and B12.⁵ The process of methylation can be impaired by lack of intake of vitamins, but also genetic factors like polymorphic folate metabolic pathways genes like MTHFR actually impair the absorption of B12 and B6.⁶ This genetic polymorphism is related to many disease processes as it relates to decreased absorption of folate and other vitamins critical to the methylation pathway and production of critical neurotransmitters. This genetic polymorphism is frequently present in PD patients as is reduced concentration of B Vitamins.⁶ For example, another neurotransmitter, Gamma-aminobutyric acid (GABA) involved in the inhibition of the central nervous system has been linked to PD as well indicating a potential of multisystem neurodegeneration.⁷ Additionally, the poor inhibition (the inability to turn off unwanted neuronal firing of muscles afferents) could impart explain the rigidity and stiffness reported as symptoms of PD.

Currently PD is a clinical diagnosis based on criteria describing physical signs and symptoms as well as the response to dopamine medication levodopa.⁴ Other than autopsy, there is no other test to clinically confirm diagnosis. Although there is a move to develop biological markers using blood testing or imaging, there is yet to be an allopathic standard.⁸

Functional medicine testing that may be useful in supporting those with PD are:

- Inflammatory status (homocysteine levels and A1C)
- Fasting blood glucose (to evaluate metabolic health and insulin sensitivity)
- Nutrient status (specifically focused on antioxidant functions including B Vitamins and Vitamin D levels)
- Organic Acids Test (to evaluate metabolic health, Gastrointestinal function and mitochondrial energy production)
- Genetic testing (especially MTHFR gene as it relates to methylation pathways and production of Dopamine neurotransmitters)
- Intestinal Permeability Testing (looking for impaired barrier function of the intestines)
- Hair analysis (to evaluate toxic exposure and heavy metals and possible need for chelation therapies)

These tools are not diagnostic but can assist in supportive therapies that may potentially reduce the progression of PD.

Parkinson's disease is a challenging neurodegenerative disorder that is near and dear to my heart for two reasons. One, my mother, a PhD physical therapist, researcher and clinician, has spent many years of her career caring for patients with neurological impairments including Parkinson's disease. She focused on integrating evidence based neurological research on plasticity, exercise, balance, posture and cognitive exercise training in individual and group based settings. I have watched her manual efforts to aid in gait training patients to her own physical sacrifice despite advances in equipment such as the Alter GTM (a weight supported treadmill training system). Although her exercise sessions were beneficial, only those who altered their life style, exercised regularly at home and remained active were able to maintain their quality of life and independence. The taxing therapy sessions were insufficient to achieve lasting effect and dramatic improvement.

Second, my father-in-law suffers from this debilitating disease. I wrote a bit about him in my book *Soar Into Health*. In brief, during a chance encounter, I was

able to be in charge of his nutrition for 2 weeks. He was put on a strict Gluten-free and whole-nutrient rich food diet and a regimented exercise and activity plan monitored by a health aid. Although I have no objective measurement, all I have is my physical therapy experience to say, he made a dramatic turn around in that short time in terms of mental capacity, movement quality, and voice quality. To put it bluntly, in all my years as a physical therapist, I have never been able to exercise someone into this kind of improvement. As time continued unfortunately, the lifestyle changes I had initiated fell to the wayside and he continues now in a steady decline requiring increased assistance drinking sugary juice, eating refined grain products and the otherwise standard American diet. Given an “at risk” condition is a family history of PD⁹ makes this a particular interesting disease for me personally as I cannot change my husband’s genetics, yet we can effect how they are turned on with lifestyle (also called epigenetics).

Parkinsonism most commonly develops in individuals who are older. As the nervous system ages, there is a decrease in the release of dopamine, a critical neurotransmitter needed for learning. Progressive general cortical atrophy has also been documented in patients with PD.¹⁰ For example, in patients with PD, imaging studies report altered connectivity in the visual, sensorimotor and cerebellar networks as well as progressive cortical thinning with progressive severity.

The degenerative aging processes are accelerated when individuals become physically inactive, are sleep deprived, engage in only familiar habitual activities, lose balance confidence, are worried about falling, become socially isolated and are psychologically stressed. There is evidence that a healthy life style, balanced nutrition, good stress management, adequate sleep and regular exercise is associated with longer life and better health and this may even drive increased delivery of dopamine.¹¹

The real question still remains----what is the cause of PD? Is it rally a disease defined by a decrease in the secretion of dopamine or is it a case of multi system dysfunction? Is there a lifestyle strategy that will reduce the risk of developing PD despite genetics and how can lifestyle improve symptoms in the face of PD?

It turns out that the neurological message between nerve endings requires essential fats found in fish and seeds, phospholipids found in eggs and organ meats and amino acids which is the raw material of protein.⁵ Specifically, Dopamine is made from the essential amino acid Phenylalanine (meaning we can’t make it ourselves), which comes from beef, poultry, pork, fish, milk, yogurt,

eggs, cheese and whole grains.¹²

Beyond the obvious, potentially poor intake of the amino acid phenylalanine, there are other lifestyle factors that play a role in increasing the risk of developing PD. Specifically, oxidative stress and mitochondrial dysfunction have been well documented in post mortem patients with PD.^{13,14} This problem is associated with degeneration of the dopaminergic neuronal pathways that are so critical to movement. Why this is the case may become more apparent as we describe the lifestyle factors associated with developing PD.

We will proceed with breaking down the lifestyle risk factors as it relates to the SOAR principles of Eat Well, Move Well, Sleep Well and Soar On (purpose and connection).

As in most conditions, understanding the causes of disease brings the solution to reduction of risk and progression as well as reversal of symptoms.

“The solution is in the cause.”

– Dr. Carolyn Dolan, PT, DPT, Cert MDT

Eat Well - Causes

There are 3 main areas I would like to focus on in regards to eating as a potential causal source of developing Parkinson's disease:

- *Nutrient deficiency/excess*
- *The Gut*
- *Metabolic Disorders*

Nutrient deficiency/excess

A case controlled study comparing the dietary habits of patients with Parkinson's disease demonstrated that there was an increased carbohydrate intake and lower intake of anti-oxidants.¹⁵ Reduced antioxidant capacity in PD was associated with olfactory loss and cardiovascular dysautonomia, both early signs of PD.¹⁶ Glutathione depletion is associated with death of midbrain cells (e.g. the Substantia Nigra) which is an implication for PD development.¹⁷

Excess iron accumulations have been found in the mid brain of PD patients indicating the potential for excess being a potential causative factor.¹³ Yet, another study did not confirm a strong correlation between serum levels of trace elements (Iron, Zinc and Copper) as an effective approach to determine stage of disease in PD patients.¹⁸ Intestinal permeability (see below for more information) may explain the location of accumulation of Zinc and other elements and why blood testing may not be confirmative. Excess iron (from protein) and carbohydrate intake along with poor antioxidant and glutathione intake appear to be associated with PD.

The Gut

Intestinal permeability is a condition where the cellular lining of the small intestines becomes patent, impairing barrier function and exposing the immune system to undigested food particles, toxins and microbes that would otherwise not be present. A small study investigated intestinal permeability of 12 PD patients using the lactulose/mannitol ratio test. Intestinal permeability was

increased in a significant portion of PD patient even with minimal gastrointestinal symptoms.¹⁹ Although this may not be causative, disruption of the brain-gut axis has been associated with PD and this may be one way. I have discussed intestinal permeability [here](#).

Intestinal permeability allows for bacterial proteins to cross react with the immune system. Intestinal permeability may lead to dysbiosis and vice versa in patients with PD.²⁰ Even without confirmative causation, there is evidence that the gastrointestinal mucosa of PD patients is clearly different from healthy patients indicating a link between digestive dysfunction and PD disease process even if not entirely clear yet.²¹ Another review paper postulates that the disruption of the brain-gut-microbiome axis to being the initial steps in the cascade of neurodegeneration in PD.²²

Food causes of leaky gut/intestinal permeability are:

- Gluten/gliadin (protein in wheat) exposure^{23,24}
- Food additives²⁵
- Trans fats²⁶

Essentially, refined grain food products in a package containing additives and trans fats and even sugar potentially produce a leaky gut.

Metabolic Disorders

There is increasing evidence that patients with Type 2 Diabetes have an increased risk of developing PD. Historically insulin was thought to solely be involved in glucose and energy metabolism. However, it may have more disease promoting effects when dysregulated beyond diabetes. A study based on a health survey of individuals 30-79 found an increased rate of PD in those with a higher BMI. Interestingly, although high BMI is associated with increased PD risk, other symptoms of BMI like elevated blood pressure, total cholesterol and even plasma fasting glucose did *not* correlate with increased PD risk.²⁷

Interestingly, oxidative stress and mitochondrial dysfunction are hallmarks of PD. Causes of oxidative stress and mitochondrial dysfunction are other components of metabolic disorder like hyperglycemia, insulin resistance, hypertension, and inflammation and should be considered as risk factors for developing PD with or without obesity.²⁸

Other food sources that have been associated with PD are increased dairy products, which is a relative risk in men more than women.²⁹ Even protein intake can have disruptive effects particularly with those PD patients taking levodopa-carbidopa.²⁹ These are not causative associations, but there is concern that this relationship may be in part due to the quality of those food products in our food chain being a source of pesticide and xenobiotics that can contribute to disease risk as described below in the **Soar On – Causes** section.

Move Well - Causes

As I have described in my book, *Soar Into Health*, I place Vitamin D deficiency in this section because the best source of natural Vitamin D is full spectrum sunshine on your skin. Vitamin D becomes a movement strategy rather than a food strategy.

Vitamin D has a role of anti-inflammatory and regulation of calcium-mediated neuronal excitotoxicity, reduction of oxidative stress as well as a role in neurotransmitters³⁰ which is why it has become more well studied as it relates to PD. It has even been postulated to reduce the risk of denervation of midbrain dopamine neurons in PD.³¹ A recent review indicates that Vitamin D status may be protective and/or a prognostic factor in neurological disease.³⁰

In newly diagnosed PD patients, low circulating serum Vitamin D and sunlight exposure were significantly associated with an increased risk for developing PD.³² Sunlight exposure was the critical component associated with elevated serum Vitamin D over supplementation.³² A cohort study determined that Vitamin D insufficiency of <30.0 ng/mL was prevalent in PD patients.³³ A meta-analysis also made similar conclusions of low Vitamin D status associated with PD risk. Additionally, that outdoor work was related to a reduced risk of PD.³⁴

Interestingly, given the prominence of Vitamin D deficiency in the PD population there may also be a link to Vitamin D receptor gene being impaired. This impairment may be an indicator of PD risk (as well as other neurodegenerative disease).³⁵⁻³⁸

Lack of movement has been associated with obesity, oxidative stress and inflammation, all of which are associated with neurodegenerative disorders like PD.³⁹ This lack of movement is not necessarily a result of Vitamin D, but if you aren't moving outside it's likely you are not moving inside either.

Sleep Well - Causes

A risk factor for developing neurologic disorders like Alzheimer's, Multiple Sclerosis and even stroke have been associated with sleep loss (<7-8 hrs). Yet, in a review article and a prospective study of night shift work, it appears that night shift work and sleep loss may actually be protective to the development of Parkinson's disease. ^{40,41}

Sleep loss or night shift work may be protective against developing Parkinson's disease.

That seems difficult to grasp, especially since REM sleep in a healthy individual is important for the function of the dopaminergic receptor in substantia nigra neuronal activation (central function that is decreased in PD). ⁴² It's possible that the amount of sleep can be decreased without disrupting REM depending on the timing of the sleep. However, the amount of REM would still be decreased. Certainly this topic on sleep as a risk factor for PD development needs to be further researched.

Although sleep loss/night shift work may be protective to the development of PD, the research indicates that once PD is established, sleep problems are common. In addition to the disruption of the Rapid Eye Movement (REM) sleep typical in PD, there is also an increase in dyskinesia ⁴³, impairment of cognitive and neurotransmitter function ⁴⁴, and excessive daytime sleepiness ⁴⁵. Excessive night sleep in PD is associated with increased depression. ⁴⁵ The research in this area may seem conflictive, yet it is important to respect the importance of REM sleep as it relates to neurological health.

Soar On - Causes

As described in **Soar Into Health**, this principle is described as purpose and connection to your environment. This is also where I include excessive stress (including mechanical head trauma) as it promotes oxidative stress, which is a hallmark to PD and risk. Animal studies have confirmed associations with traumatic brain injury having similar biological mechanism as those associated with PD.⁴⁶ High profile cases like Muhammad Ali are examples of head trauma being a risk factor for development of PD.

Even environmental exposures have links to neurological diseases including PD. Glyphosate (the active ingredient in Round Up) has been linked to disruption in microbiome as well as magnesium deficiency which are associated with neurological diseases.⁴⁷ Even other pesticides (Paraquat, lindane), herbicides and other xenobiotics are neurotoxins and some have been found in the substantia nigra in PD post-mortem.^{9,29} The susceptibility to environmental toxins may be indicative to impaired detoxification of toxins.

Understand that PD is a multifaceted disease with multiple potential causative factors. The research continues to be elucidated on the topic yet seems that there isn't one thing alone that causes the disease to take hold. Understanding that helps to understand how to reverse progression of PD, reduce the risk of developing disease and even successfully live with PD.

"Don't count the days.

Make the days count"

-Muhammad Ali

Everyday we have an opportunity to make a choice. Each choice can either count towards health promotion or it can be a fight to avoid getting worse. The next section I will discuss 4 simple lifestyle habits that not only reduce the risk for developing PD, but each habit also has the potential to reduce and possibly reverse symptoms of PD.

Eat Well - Solutions

As described in previous sections, a primary cause of PD development is a reduction in antioxidant activity to reduce oxidative stress and decrease in mitochondrial health in the addition to detoxification of environmental toxins. Antioxidants (or antioxidant like compounds) reduce the mitochondrial decay and decrease cognitive decline in PD. ⁴⁸⁻⁵⁰

Antioxidants important in treating PD and reducing risk and the whole foods in which found listed in order of highest to lowest concentration are: ^{29,51}

Vitamin A	Vitamin B2 (Riboflavin)	Vitamin B6
Carrots	Crimini mushrooms	Spinach
Spinach	Calf's liver	Bell peppers
Kale	Spinach	Garlic
Parsley	Romaine Lettuce	Wild Tuna
Bell Peppers	Asparagus	Cauliflower
Romaine Lettuce	Swiss chard	Banana
Calf's Liver	Broccoli	Broccoli
Swiss chard	Collard greens	Celery
Sweet Potatoes	Venison	Asparagus
Cayenne pepper	Yogurt	Cabbage
Vitamin C	Vitamin E	Coenzyme Q 10
Bell Peppers	Sunflower seeds	Organ meats
Parsley	Swiss chard	Beef
Broccoli	Almonds	Sardines

Strawberries	Spinach	Mackerel
Cauliflower	Collard greens	Spinach
Lemon juice	Kale	Broccoli
Romaine Lettuce	Papaya	Cauliflower
Brussels sprouts	Olives	
Papaya	Bell peppers	
Kale	Brussels sprouts	
<i>Resveratrol</i> ⁴⁴		
Red grapes		
Peanut butter		
Dark Chocolate		
Blueberries		

Glutathione, another powerful antioxidant is particularly important in the substantia nigra of patients in the preclinical stages. Administration of reduced glutathione resulted in improved PD symptoms.²⁹ Whole food sources of glutathione are in many of the foods listed above including asparagus, potatoes, peppers, carrots, onion, broccoli, spinach, garlic as well as mushrooms and liver.

51

Many of the colorful vegetables and fruits contain the highest amounts nutrients that have antioxidant functions. They also contain other vital minerals and vitamins in complement that promote health as well as blood sugar regulation and supportive to the gut lining and microbiome. Additionally eating a whole-nutrient food means a natural avoidance of refined sugar foods that will also promote metabolic health. Seafood is also high in Omega 3 fats, which is neuroprotective and anti-inflammatory. Foods from these lists are what I provided my father in law for those weeks. This is not an all-inclusive list, but the idea of wholesome nutrient rich food from the earth may provide us the safest way to reduce our risk and progression of diseases.

Another food component that appears to have protective benefit is

caffeine/coffee (free of sweeteners of course) consumption of 1-3 cups/day.²⁹

Another eating strategy to consider to reduce your risk associated with metabolic disorders is Intermittent Fasting (IF) or extending the time between meals. Studies of IF have showed positive improvements in age related disorders especially in metabolic health. It also improves the cellular response to stress and even enhances mitochondrial health and DNA repair.⁵² A simple IF strategy to use is to extend the time between dinner and breakfast where a majority of the fasting time is spent sleeping. For example, setting up an 8-10 eating window and fast the rest of the time on fluids and rest. Clearly this is a useful strategy for adults even without disease. However, should you have PD then discussing with your doctor how to incorporate IF into your healing strategy is warranted. Many PD medications require to be taken with food and IF complicates when medications are time sensitive. ****An additional precaution is warranted for developing children, women of childbearing age and women whom are pregnant or nursing where fasting is not recommended and can have adrenal impacts for these populations.*

Simply put, whole colorful vegetables and fruit and adequate protein intake including seafood are supportive of metabolic health, gut health, antioxidant activity, mitochondrial health as well as detoxification pathways. IT would be important to consider organic foods whenever possible to reduce your detoxification burden and toxin exposure especially in the presence of PD. Consider feasting and fasting rather than snacking all day long. Eat when you are hungry. Stop when you are full and wait to eat again until the next meal or the next day.

Outside of lifestyle of nutrient dense eating free of toxins, you may want to consider a few supplements based on the presence of disease or in hopes of reducing your risk of development of PD. A multivitamin with methylated B Vitamins would be at the top of the list given the likelihood of MTHFR genetic mutation and nutrient deficiencies impairing detoxification pathways. Methylated B Vitamins should also benefit the as well as methylation needed to produce dopamine. In the presence of PD, other supplements should be considered carefully and specifically after further testing to guide specific needs.

Remove. Replace. Restore.

Remove refined food products devoid of antioxidant vitamins and minerals and excessive refined sugar.

Replace with bright colored vegetables, fruit and protein sources free of environmental toxins. Practice periods of feasting and fasting when healthy to promote mitochondrial health.

Restore neurological health and reduce your risk for developing PD and maybe reverses symptoms associated with PD.

Move Well - Solutions

Research supports the role of exercise in improving health for those with and without PD. Pair the exercise with a healthy antioxidant rich diet and the results and benefits will likely be far greater as I saw with my father-in-law. More specifically, physical activity and exercise attenuates neuroinflammation in neurological diseases. Current evidence supports exercise has disease-altering potential for PD by improving neuroimmune functions. This is critical, as excessive neuroinflammation will cause damage to neurons.^{39,53}

As I describe in **Soar Into Health**, the World Health Organization recommends for upwards of 60'/day of exercise for healthy individuals. Research on PD suggests that less than 60'/day still has positive impact. Aerobic exercise for PD patients in the form of cycling 60', 3 days a week for 8 weeks demonstrated improved olfaction (a symptom of PD) while non-exercisers had worsening olfaction.⁵⁴ A mouse model of PD demonstrated treadmill exercise alleviated motor deficits as well as improved mitochondrial dysfunction.⁵⁵

Other improvements in PD patients who participated in resistance training twice/week had improved sleep quality. As sleep quality improved knee extensor peak torque also trended to improve.⁵⁶ A recent literature review found research supporting that physical activity positively impacted measures of depression, apathy, fatigue, day time sleepiness, sleep quality and cognition.⁵⁷

With progression of severity of PD, movement becomes more challenging and may require assistance from a rehabilitation specialist. However, the key here is that regular movement is important, especially moving outside to facilitate the production of natural vitamin D. Along with exercise, vitamin D has the benefit of attenuating neuroinflammation in PD.⁵⁸

I often encourage folks to move whenever they can, often and safely. A simple start is to walk outside to sit in a chair in the sun with skin exposed for 10' and then walk back in. Have assistance as needed to be safe. Certainly, don't burn the skin. The effect of Vitamin D production AND exercise is combined.....seemingly

everything related to PD improves especially in the presence of an antioxidant rich diet. Obviously, if you live in an area where sunshine is unavailable at specific times of year, then consider Vitamin D supplementation. More on Vitamin D can be found [here](#).

Depending on a patient's mobility status, an unweighting gait system like the Alter G my mother works with can also be very useful for cardiovascular fitness while being supportive to avoid falls. These systems can even provide unweighting to facilitate jogging or running. However useful these systems are, they cannot replace the needed regular movement at home.

Balance problems and falls risk tend to be an issue for those with PD. Some specific exercises that address strength, flexibility and balance are very effective in reducing fall risk. I have clinically found that exercises incorporating trunk rotation are very useful at breaking the freezing pattern. Dancing can incorporate many of these concepts and provide much enjoyment for patients with PD. Rhythmic activities facilitate motor learning and coordination. However, when one thinks of dancing with PD, there is the concern about the lack of movement and coordination may be problematic for those with PD. However, recent research and case studies demonstrate immediate benefit to gait quality, speed and stabilization in the presence of rhythmic auditory stimulation (RAS), like music. Specifically, the neuronal connection between auditory and motor networks are improved with "groove" music that contains bass-frequency pulses. The presence of rhythmical musical beat during ambulation improves gait stability.⁵⁹

Watch a PD patient's response to music [here](#).

"The best exercise is the one that you will do."

- Grant Glass, PT

Any movement and exercise has the potential to benefit those with PD. The only challenge is finding the right activity that motivates a patient to continue to move.

Remove. Replace. Restore.

Remove sedentism.

Replace with everyday movement, walking counts. Move in the sunshine when possible and safely. Dance.

Restore neural health, positive well being...reduce symptoms associated with PD.

Sleep Well - Solutions

Although it appears that sleep loss and night shift work may be protective in developing PD, it is hardly conclusive and even goes against other research related to other neurological disease. For sure, if PD is present, then REM sleep is disrupted which affects cognitive functions.

How do we rectify these two things?

To begin, changing how one eats and moves improves sleep quality with or without PD. The actual amount of sleep needed likely varies and is individualized based on needs. Sleep is restorative. If you find yourself sleeping excessively or sleeping too little, a consultation with your physician is warranted to rule out sleep disorders, but especially PD.

With or without the diagnosis of PD and an otherwise healthy lifestyle of eat well and move well, then plan on 8-10 hours of sleep nightly. The amount of sleep is less important apparently as it relates to risk of PD, but I encourage waking naturally whenever possible and determining the amount of sleep needed based on whether or not you feel rested up waking. Back up your bedtime based on that so you wake consistently in the morning. If fatigue continues even with routine 9-10 hours then checking hormone levels is warranted.

Sleep quality is also based on exposure to the sun, a healthy diet to normalize the circadian rhythm. A few tips to improve circadian cycle(especially REM) for sleep optimization:

- Turn off screens 1-2 hours before bed.
- Keep your room dark(no lights, clocks or technology and black out curtains) and cool.
- Go to sleep when you are tired.
- Wake up to the morning sun whenever possible.
- Get out in the sunshine during the daytime, especially first upon waking.
- Get regular exercise.
- Use blue-blocking glasses if you need to look at screens often or f.lux on the

computer.

- Eat a whole foods based diet full of vegetables, fruits and protein.
- Eat dinner before 6PM.

Remove. Replace. Restore.

Remove ignoring your body's signals of fatigue.

Replace with regular 8-10 hours of sleep regularly in a dark-dark room. Get regular sunshine. Eat a nutrient rich diet.

Restore a normal circadian rhythm and optimal neural health and repair.

Soar On - Solutions

As described earlier, we discussed that excessive psychological and/or mechanical stress have negative effects on the nervous system. This drives neuroinflammation and creates risk for PD. Obviously, protecting the head from traumatic injury by using seat belts, helmets and avoiding risky behaviors is a huge step to reduce your risk.

Although I consider certain stress to actually be positive, if we ignore the cumulative effects then it begins to put us at risk. The mind-body connection has shown improvement in many conditions within the field of neurology.⁶⁰ One study related to mindfulness-based lifestyle training has shown a reduction in stress and anxiety in PD patients that lasts over 6 months.⁶⁰ This mindfulness program included:

- Education of the condition and management with lifestyle education
- Stress management in the form of group-support, meditation
- Spirituality to explore meaning and or purpose in life
- Exercise
- Nutrition promoting micronutrient rich diet
- Connectedness to professional and emotional support
- Environment to reduce toxin exposure

This particular mindfulness plan matches the **Soar Into Health** principles.

There is a published case of a 78-yo M with 16 years of a diagnosis with PD whom went into remission. The anecdotal case claims that the long-standing meditation practice was a primary contributing factor to reversal.⁶¹ There are clearly many things to consider in the case beyond meditative practice, but this research supports the positive benefits of meditation.

Maybe the most important benefits to mindfulness and meditation, is that it reduces gray matter atrophy⁶² and may in fact increase the gray matter density in PD.⁶³ Learning to meditate can be challenging, but there are some simple ways to incorporate more mindfulness into daily life. One easy way is to devote 5

minutes of deep diaphragmatic breathing twice daily. There are phone apps that have guided meditation of 10' like Headspace. Learning other yoga meditation techniques like Kundalini yoga can provide mindfulness, movement and connection in one activity. My personal favorite is Transcendental Meditation™ for its simplicity and ease. The most complicated part is the investment of time and money to learn. Simply standing barefoot in the natural sunshine amongst the trees can also count towards mindfulness. Prayer at church and even dancing can bring these concepts to life. There are many options here to promote, but truly this is a personal choice and must fit your personality.

Although I discussed potential toxic exposure and accumulation of metals as a potential association with PD risk, it is here that I will also include other means of addressing. I would encourage the use of safe detoxification strategies that support our natural process:

- Saunas
- Infrared Saunas
- Mineral detox baths
- Skin brushing
- Exercise
- Sleep

Further details on the importance of detoxification pathways can be found [here](#). Details of specific detoxification strategies can be found [here](#). If heavy metals are confirmed to be a problem then working closely with an appropriate health professional on natural chelation therapy is warranted.

Remove. Replace. Restore.

Remove ignorance of life stress impact on health.

Replace with connection to people in your life. Mindfulness and meditative practices regularly that fits your personality and life.

Restore and even improve the gray matter of your brain while reducing symptoms of PD.

Conclusions

Physical and mental dysfunction associated with the way a person or group live is considered a **lifestyle disease**. Lifestyle disease includes habits of how one eats, moves, sleeps and even connects to themselves and their environment. The neurodegenerative condition Parkinson's appears to have lifestyle implications for cause and treatment.

*The four foundation principles of **Soar Into Health** can help guide you to understand the causal lifestyle factors for PD. More important, the simple principles for lifestyle habits can help guide you towards reducing your risk, improving your function and overall enjoying your life with or without PD.*

Four Causes:

- Poor antioxidant and protein intake reducing detoxification and the ability to make neurotransmitters
- Lack of movement, especially in full spectrum sunshine resulting in Vitamin D deficiency and potentially obesity
- Disrupted sleep is an indicator of PD risk
- Excessive life stress resulting in increased oxidative stress and reduction connections

Four Solutions:

- Whole fruits, vegetables and protein intake improves detoxification and provides components for neurotransmitter production
- Exercise, especially in full sunshine, reduces symptoms of PD and improves Vitamin D status as well as learning and social interactions
- Adequate REM sleep improves neurological symptoms during the day
- Meditation decreases oxidative stress and even increases gray matter

Final Tip:

You didn't get sick overnight; don't expect to heal overnight.

Consistency in the foundational principles will reap the rewards over a lifetime.

Small changes can have a **BIG** impact.

What are you waiting for?

Eat Well. Move Well. Sleep Well. Soar On...even with PD.



soar

Tracking Lifestyle Habits

FORM

Set Your VISION

Rather than goal setting that will have an ending, I focus on the vision you have for yourself that can continue to grow without endpoint. This will help keep you focused on your daily choices to achieve your *vision*.

Make it personal.

Make it achievable.

Make it visible.

Make it real.

For example, I would like to hike trails as I age. Or I would like to keep my heart healthy to play with my grandkids.

Set Your VISION

References

1. Kohlstadt I, ed. *Advancing medicine with food and nutrients*. Second ed. Boca Raton, FL: Taylor and Francis Group, LLC; 2013.
2. Dorsey ER, Constantinescu R, Thompson JP, et al. Projected number of people with parkinson disease in the most populous nations, 2005 through 2030. *Neurology*. 2007;68(5):384-386.
3. www.mayoclinic.org.
4. Montgomery EB, Jr. Practice parameter: Diagnosis and prognosis of new onset parkinson disease (an evidence-based review): Report of the quality standards subcommittee of the american academy of neurology. *Neurology*. 2006;67(12):2266; author reply 2266.
5. Holford P. *New optimum nutrition for the mind*. . 2009.
6. Rozycka A, Jagodzinski PP, Kozubski W, Lianeri M, Dorszewska J. Homocysteine level and mechanisms of injury in parkinson's disease as related to MTHFR, MTR, and MTHFD1 genes polymorphisms and L-dopa treatment. *Curr Genomics*. 2013;14(8):534-542.
7. Blaszczyk JW. Parkinson's disease and neurodegeneration: GABA-collapse hypothesis. *Front Neurosci*. 2016;10:269.
8. www.pdf.org.
9. Schirinzi T, Martella G, D'Elia A, et al. Outlining a population "at risk" of parkinson's disease: Evidence from a case-control study. *Parkinsons Dis*. 2016;2016:10.1155/2016/9646057.
10. Guimaraes RP, Arci Santos MC, Dagher A, et al. Pattern of reduced functional connectivity and structural abnormalities in parkinson's disease: An exploratory study. *Front Neurol*. 2017;7:243.
11. Rapp SR, Luchsinger JA, Baker LD, et al. Effect of a long-term intensive lifestyle

intervention on cognitive function: Action for health in diabetes study. *J Am Geriatr Soc.* 2017.

12. umm.edu/health/medical/altmed/supplement/phenylalanine.

13. Filograna R, Beltramini M, Bubacco L, Bisaglia M. Anti-oxidants in parkinson's disease therapy: A critical point of view. *Curr Neuropharmacol.* 2016;14(3):260-271.

14. Onyango IG, Khan SM, Bennett JP, Jr. Mitochondria in the pathophysiology of alzheimer's and parkinson's diseases. *Front Biosci (Landmark Ed).* 2017;22:854-872.

15. Hellenbrand W, Boeing H, Robra BP, et al. Diet and parkinson's disease. II: A possible role for the past intake of specific nutrients. results from a self-administered food-frequency questionnaire in a case-control study. *Neurology.* 1996;47(3):644-650.

16. Campolo J, De Maria R, Cozzi L, et al. Antioxidant and inflammatory biomarkers for the identification of prodromal parkinson's disease. *J Neurol Sci.* 2016;370:167-172.

17. Canals S, Casarejos MJ, de Bernardo S, Rodriguez-Martin E, Mena MA. Glutathione depletion switches nitric oxide neurotrophic effects to cell death in midbrain cultures: Implications for parkinson's disease. *J Neurochem.* 2001;79(6):1183-1195.

18. Meamar R, Nikyar H, Dehghani L, Basiri K, Ghazvini MR. Assessing of plasma levels of iron, zinc and copper in iranian parkinson's disease. *Adv Biomed Res.* 2016;5:31-9175.178788. eCollection 2016.

19. Salat-Foix D, Tran K, Ranawaya R, Meddings J, Suchowersky O. Increased intestinal permeability and parkinson disease patients: Chicken or egg? *Can J Neurol Sci.* 2012;39(2):185-188.

20. Hasegawa S, Goto S, Tsuji H, et al. Intestinal dysbiosis and lowered serum lipopolysaccharide-binding protein in parkinson's disease. *PLoS One.* 2015;10(11):e0142164.

21. Clairembault T, Leclair-Visonneau L, Coron E, et al. Structural alterations of the intestinal epithelial barrier in parkinson's disease. *Acta Neuropathol Commun.* 2015;3:12-015-0196-0.

22. Mulak A, Bonaz B. Brain-gut-microbiota axis in parkinson's disease. *World J*

Gastroenterol. 2015;21(37):10609-10620.

23. Hollon J, Puppa EL, Greenwald B, Goldberg E, Guerrerio A, Fasano A. Effect of gliadin on permeability of intestinal biopsy explants from celiac disease patients and patients with non-celiac gluten sensitivity. *Nutrients.* 2015;7(3):1565-1576.

24. de Punder K, Pruimboom L. The dietary intake of wheat and other cereal grains and their role in inflammation. *Nutrients.* 2013;5(3):771-787.

25. Lerner A, Matthias T. Changes in intestinal tight junction permeability associated with industrial food additives explain the rising incidence of autoimmune disease. *Autoimmun Rev.* 2015;14(6):479-489.

26. Park MY, Kim MY, Seo YR, Kim JS, Sung MK. High-fat diet accelerates intestinal tumorigenesis through disrupting intestinal cell membrane integrity. *J Cancer Prev.* 2016;21(2):95-103.

27. Saaksjarvi K, Knekt P, Mannisto S, Lyytinen J, Heliovaara M. Prospective study on the components of metabolic syndrome and the incidence of parkinson's disease. *Parkinsonism Relat Disord.* 2015;21(10):1148-1155.

28. Zhang P, Tian B. Metabolic syndrome: An important risk factor for parkinson's disease. *Oxid Med Cell Longev.* 2014;2014:729194.

29. Gaby AM. *Nutritional medicine.* Concord, NH: Fritz Perlberg Publishing; 2011.

30. Mpandzou G, Ait Ben Haddou E, Regragui W, Benomar A, Yahyaoui M. Vitamin D deficiency and its role in neurological conditions: A review. *Rev Neurol (Paris).* 2016;172(2):109-122.

31. Orme RP, Middleditch C, Waite L, Fricker RA. The role of vitamin D(3) in the development and neuroprotection of midbrain dopamine neurons. *Vitam Horm.* 2016;100:273-297.

32. Wang J, Yang D, Yu Y, Shao G, Wang Q. Vitamin D and sunlight exposure in newly-diagnosed parkinson's disease. *Nutrients.* 2016;8(3):142.

33. Evatt ML, DeLong MR, Kumari M, et al. High prevalence of hypovitaminosis D status in patients with early parkinson disease. *Arch Neurol.* 2011;68(3):314-319.

34. Shen L, Ji HF. Associations between vitamin D status, supplementation, outdoor work and risk of parkinson's disease: A meta-analysis assessment. *Nutrients.* 2015;7(6):4817-4827.

35. Gatto NM, Paul KC, Sinsheimer JS, et al. Vitamin D receptor gene polymorphisms and cognitive decline in parkinson's disease. *J Neurol Sci.* 2016;370:100-106.
36. Kang SY, Park S, Oh E, et al. Vitamin D receptor polymorphisms and parkinson's disease in a korean population: Revisited. *Neurosci Lett.* 2016;628:230-235.
37. Gezen-Ak D, Alaylioglu M, Genc G, et al. GC and VDR SNPs and vitamin D levels in parkinson's disease: The relevance to clinical features. *Neuromolecular Med.* 2016.
38. Wang L, Maldonado L, Beecham GW, et al. DNA variants in CACNA1C modify parkinson disease risk only when vitamin D level is deficient. *Neurol Genet.* 2016;2(3):e72.
39. LaHue SC, Comella CL, Tanner CM. The best medicine? the influence of physical activity and inactivity on parkinson's disease. *Mov Disord.* 2016;31(10):1444-1454.
40. Palma JA, Urrestarazu E, Iriarte J. Sleep loss as risk factor for neurologic disorders: A review. *Sleep Med.* 2013;14(3):229-236.
41. Chen H, Schernhammer E, Schwarzschild MA, Ascherio A. A prospective study of night shift work, sleep duration, and risk of parkinson's disease. *Am J Epidemiol.* 2006;163(8):726-730.
42. Proenca MB, Dombrowski PA, Da Cunha C, Fischer L, Ferraz AC, Lima MM. Dopaminergic D2 receptor is a key player in the substantia nigra pars compacta neuronal activation mediated by REM sleep deprivation. *Neuropharmacology.* 2014;76 Pt A:118-126.
43. Galati S, Salvade A, Pace M, et al. Evidence of an association between sleep and levodopa-induced dyskinesia in an animal model of parkinson's disease. *Neurobiol Aging.* 2015;36(3):1577-1589.
44. Dos Santos AC, Castro MA, Jose EA, et al. REM sleep deprivation generates cognitive and neurochemical disruptions in the intranigral rotenone model of parkinson's disease. *J Neurosci Res.* 2013;91(11):1508-1516.
45. Ylikoski A, Martikainen K, Sieminski M, Partinen M. Sleeping difficulties and health-related quality of life in parkinson's disease. *Acta Neurol Scand.* 2016.

46. Impellizzeri D, Campolo M, Bruschetta G, et al. Traumatic brain injury leads to development of parkinson's disease related pathology in mice. *Front Neurosci*. 2016;10:458.
47. Samsel A, Seneff S. Glyphosate, pathways to modern diseases III: Manganese, neurological diseases, and associated pathologies. *Surg Neurol Int*. 2015;6:45-7806.153876. eCollection 2015.
48. Liu J, Ames BN. Reducing mitochondrial decay with mitochondrial nutrients to delay and treat cognitive dysfunction, alzheimer's disease, and parkinson's disease. *Nutr Neurosci*. 2005;8(2):67-89.
49. Przedborski S, Jackson-Lewis V, Fahn S. Antiparkinsonian therapies and brain mitochondrial complex I activity. *Mov Disord*. 1995;10(3):312-317.
50. Khan MM, Ahmad A, Ishrat T, et al. Resveratrol attenuates 6-hydroxydopamine-induced oxidative damage and dopamine depletion in rat model of parkinson's disease. *Brain Res*. 2010;1328:139-151.
51. Mateljan G. *The world's healthiest foods*. First ed. Seattle, Washington: George Mateljan Foundation; 2007.
52. Mattson MP, Longo VD, Harvie M. Impact of intermittent fasting on health and disease processes. *Ageing Res Rev*. 2016.
53. Spielman LJ, Little JP, Klegeris A. Physical activity and exercise attenuate neuroinflammation in neurological diseases. *Brain Res Bull*. 2016;125:19-29.
54. Rosenfeldt AB, Dey T, Alberts JL. Aerobic exercise preserves olfaction function in individuals with parkinson's disease. *Parkinsons Dis*. 2016;2016:9725089.
55. Koo JH, Cho JY, Lee UB. Treadmill exercise alleviates motor deficits and improves mitochondrial import machinery in an MPTP-induced mouse model of parkinson's disease. *Exp Gerontol*. 2017.
56. Silva-Batista C, Campos de Brito L, Corcos DM, et al. Resistance training improves sleep quality in subjects with moderate parkinson's disease. *J Strength Cond Res*. 2016.
57. Cusso ME, Donald KJ, Khoo TK. The impact of physical activity on non-motor symptoms in parkinson's disease: A systematic review. *Front Med (Lausanne)*. 2016;3:35.

58. Calvello R, Cianciulli A, Nicolardi G, et al. Vitamin D treatment attenuates neuroinflammation and dopaminergic neurodegeneration in an animal model of parkinson's disease, shifting M1 to M2 microglia responses. *J Neuroimmune Pharmacol.* 2016.
59. Hove MJ, Keller PE. Impaired movement timing in neurological disorders: Rehabilitation and treatment strategies. *Ann N Y Acad Sci.* 2015;1337:111-117.
60. Advocat J, Enticott J, Vandenberg B, Hasted C, Hester J, Russell G. The effects of a mindfulness-based lifestyle program for adults with parkinson's disease: A mixed methods, wait list controlled randomised control study. *BMC Neurol.* 2016;16:166-016-0685-1.
61. Smart K, Durso R, Morgan J, McNamara P. A potential case of remission of parkinson's disease. *J Complement Integr Med.* 2016;13(3):311-315.
62. Last N, Tufts E, Auger LE. The effects of meditation on grey matter atrophy and neurodegeneration: A systematic review. *J Alzheimers Dis.* 2017;56(1):275-286.
63. Pickut BA, Van Hecke W, Kerckhofs E, et al. Mindfulness based intervention in parkinson's disease leads to structural brain changes on MRI: A randomized controlled longitudinal trial. *Clin Neurol Neurosurg.* 2013;115(12):2419-2425.