Parkinson’s Disease: An intelligent approach

- Rebecca Roentsch Montrone, BS
- Wondrous Roots, Inc.

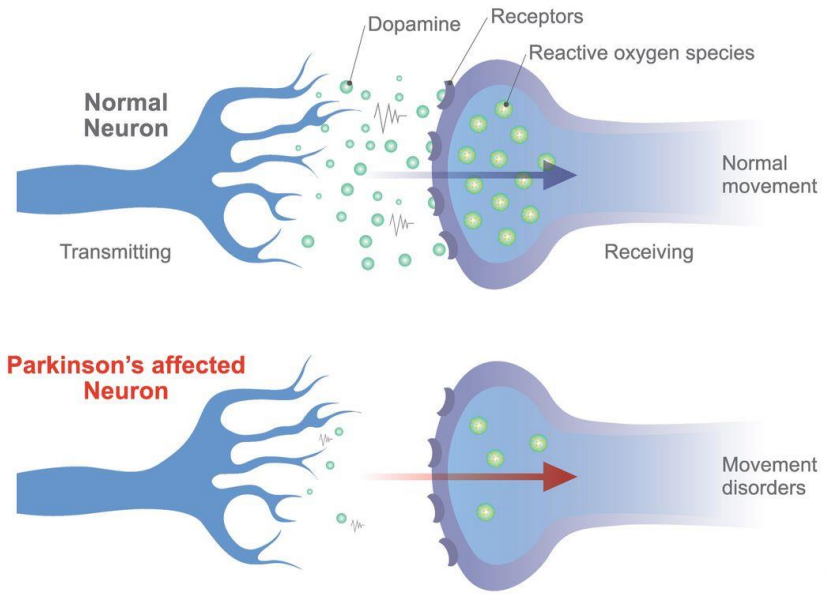
- 2024

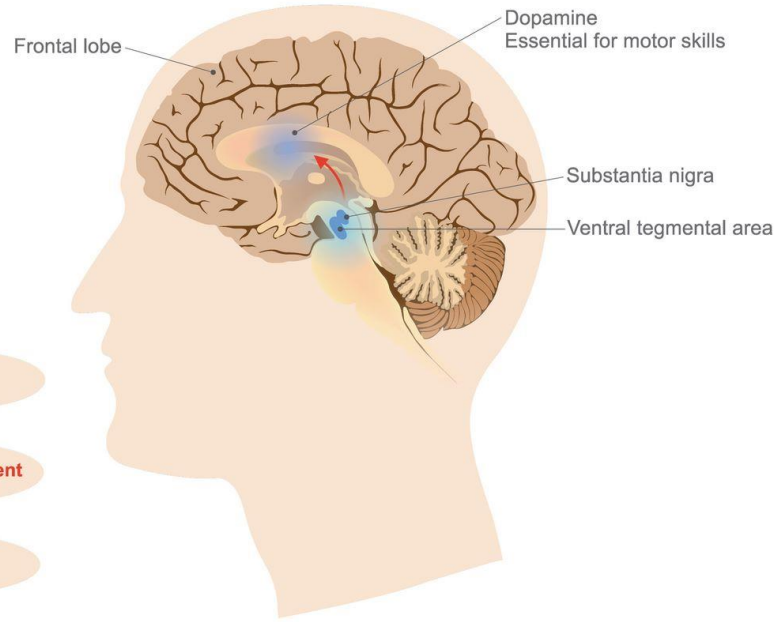
Parkinson’s Disease

Parkinson's disease (PD), or simply Parkinson's, is a long-term neurodegenerative disease of mainly the central nervous system that affects both the motor system and non-motor systems. The symptoms usually emerge slowly, and as the disease progresses, non-motor symptoms become more common. Usual symptoms are tremor, slowness of movement, rigidity, and difficulty with balance, collectively known as parkinsonism. Parkinson's disease dementia, falls and neuropsychiatric problems such as sleep abnormalities, psychosis, mood swings, or behavioral changes may arise in advanced stages.

Parkinson's disease

What's happening to the brain?

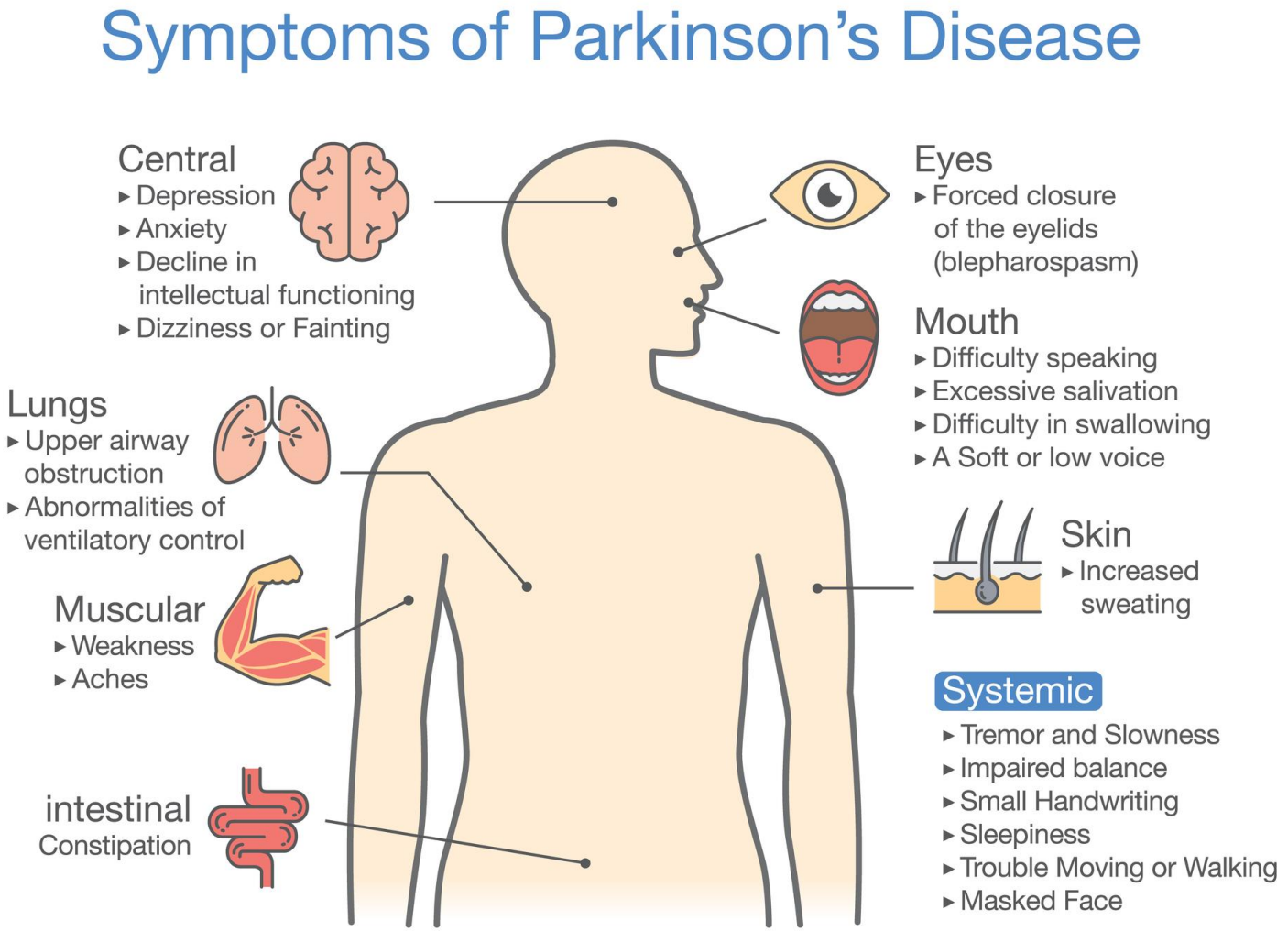




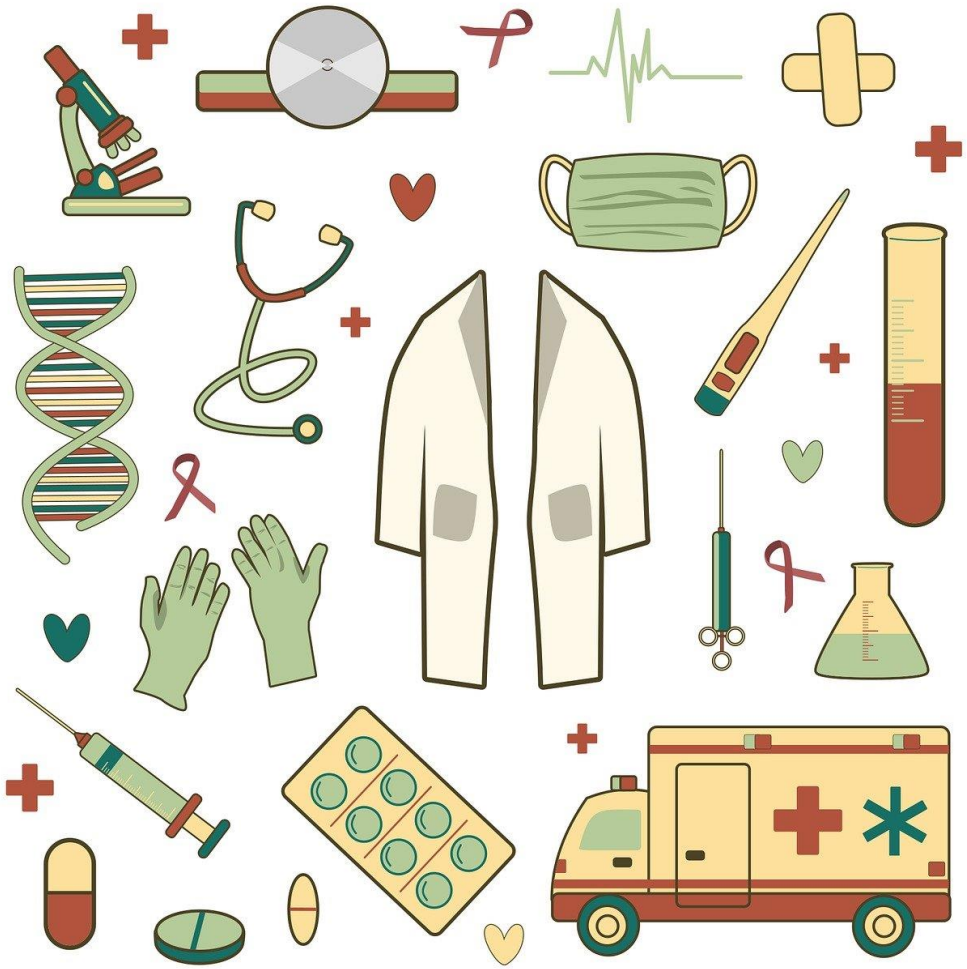
What goes missing?

Parkinson's disease (PD) is a complex, chronic, and neurodegenerative disorder, pathologically characterized predominately by a loss of substantia nigra pars compacta dopaminergic neurons, manifesting in functional and structural alterations throughout the brain. Pathological studies have shown that the cortical and subcortical regions are widely involved in PD pathology.

What are the effects?



How is Parkinson's disease diagnosed?



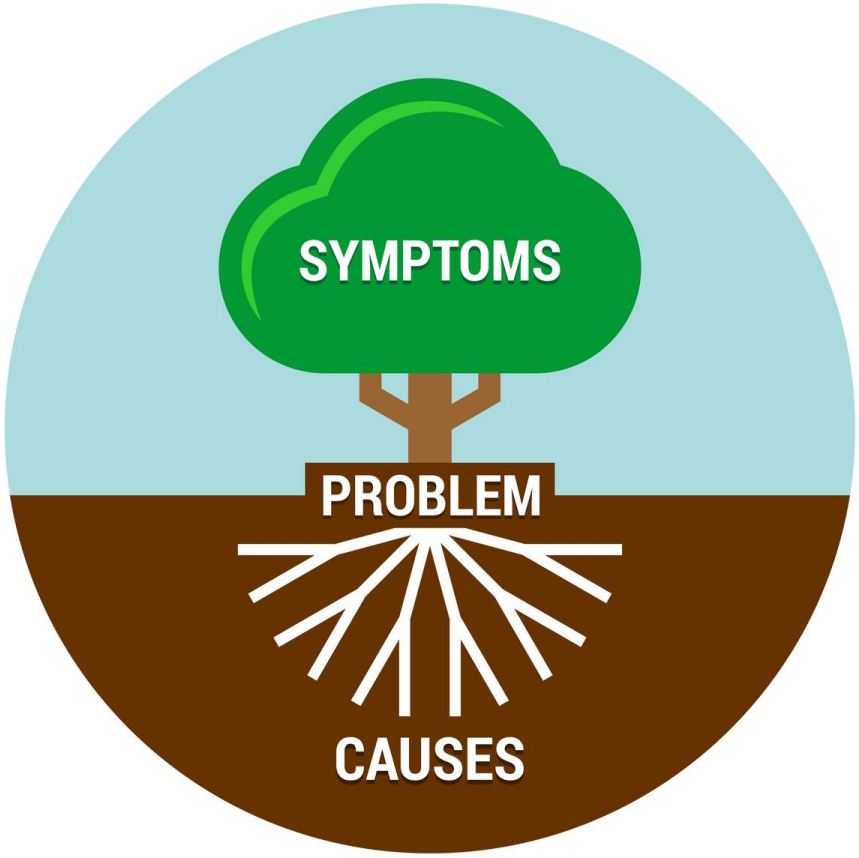
Parkinson's Diagnosis

- **Certain physical signs and symptoms** —These are the symptoms most often noticed by patients or their families:
  - Shaking or tremor: Called resting tremor, a trembling of a hand or foot that happens when the patient is at rest and typically stops when he or she is active or moving
  - Bradykinesia: Slowness of movement in the limbs, face, walking or overall body
  - Rigidity: Stiffness in the arms, legs or trunk
  - Posture instability: Trouble with balance and possible falls
- **Once the patient is at the doctor's office, the physician:**
  - Takes a medical history and does a physical examination.
  - Asks about current and past medications. Some medications may cause symptoms that mimic Parkinson's disease.
  - Performs a neurological examination, testing agility, muscle tone, gait and balance.

Testing

- There is no lab or imaging test that is recommended or definitive for Parkinson's disease. However, in 2011, the U.S. Food and Drug Administration approved an imaging scan called the DaTscan. This technique allows doctors to see detailed pictures of the brain's dopamine system.
- A DaTscan involves an injection of a small amount of a radioactive drug and a machine called a single-photon emission computed tomography (SPECT) scanner, similar to an MRI.
- The drug binds to dopamine transmitters in the brain, showing where in the brain dopaminergic neurons are. (Dopaminergic neurons are the source of dopamine in the brain; a loss of dopamine is what leads to Parkinson's.)
- The results of a DaTscan can't show that you have Parkinson's, but they can help your doctor confirm a diagnosis or rule out a Parkinson's mimic.

What causes Parkinson's disease?



A combination of genetic & environmental factors

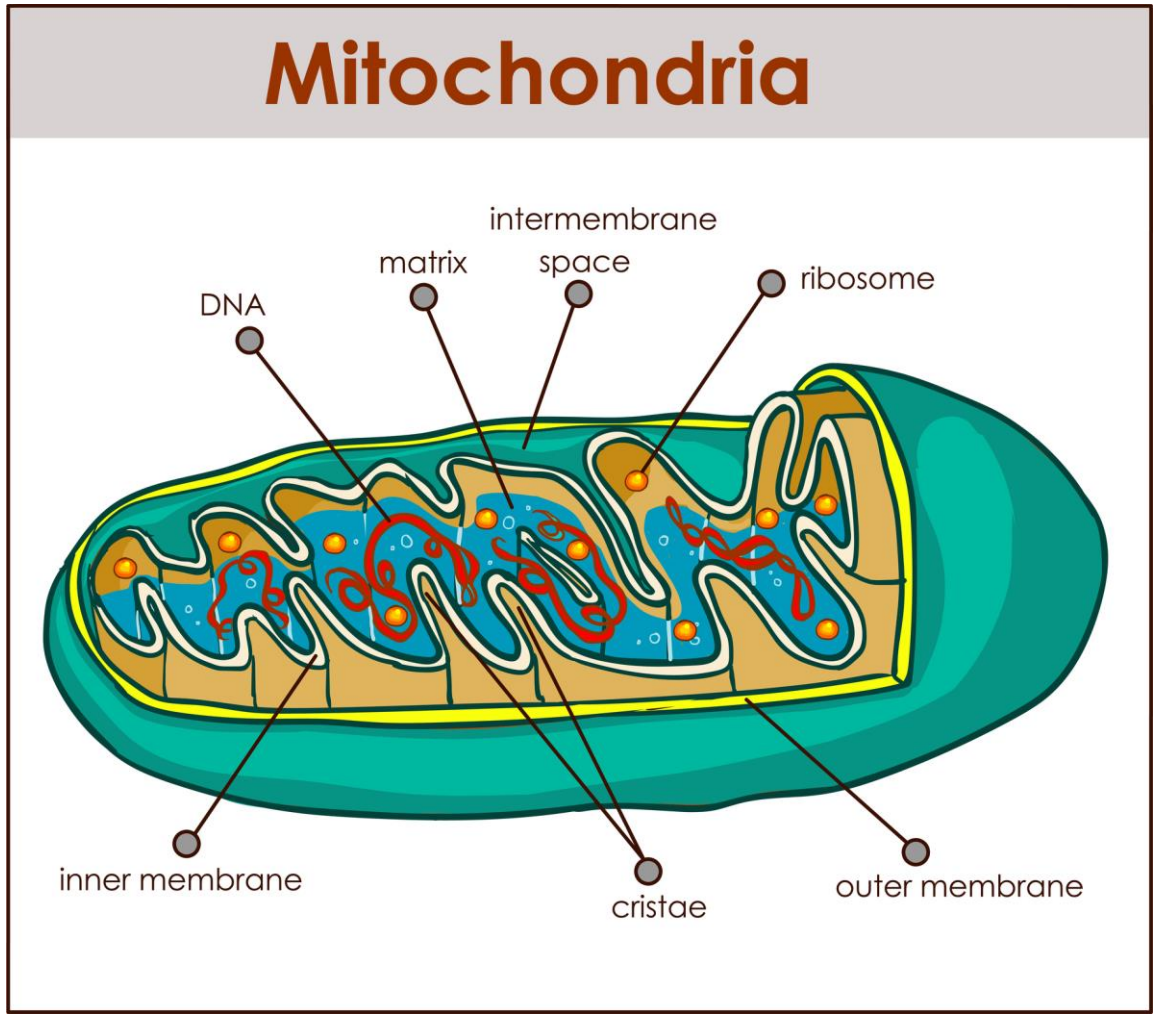
Scientists believe a combination of genetic and environmental factors are the cause of Parkinson’s disease (PD).

- **Pesticides:**

New research shows a link between use of two pesticides, **rotenone** and **paraquat**, and Parkinson's disease. People who used either pesticide developed Parkinson’s disease approximately 2.5 times more often than non-users.

Mitochondrial dysfunction and oxidative stress have long been implicated as pathophysiologic mechanisms underlying Parkinson's disease (PD. Genetic forms of PD associated with mutations in the alpha-synuclein, PARKIN, PINK1, or DJ-1 genes may involve these mechanisms. In experimental models, the pesticides **paraquat**, which causes **oxidative stress**, and **rotenone**, which inhibits **mitochondrial complex I**, both induce loss of nigral dopaminergic neurons and behavioral changes associated with human PD. Yet despite decades of laboratory study, neither pesticide has been definitively associated with PD in humans. Previous studies have reported associations with paraquat, but results are inconsistent and, in general, studies included few exposed cases; evidence concerning rotenone is sparse. **To assess the relevance of experimental results to human PD, we investigated the association of PD with use of pesticides linked to complex I inhibition or oxidative stress in a population with well-characterized pesticide exposure**.

Rotenone, Paraquat, and Parkinson's Disease | Environmental Health Perspectives | Vol. 119, No. 6 (nih.gov)



"Our study helps connect the dots between basic research and human populations. Rotenone and paraquat have been linked experimentally to pathophysiological mechanisms implicated in human PD. Groups of pesticides linked to the mechanisms of mitochondrial dysfunction or oxidative stress were also associated with PD in our study, thus extending experimental work to provide strong evidence that these mechanisms play a role in PD in humans. Importantly, the potential for exposure to many of these pesticides, including rotenone and paraquat, extends well beyond the occupational setting. Many persons with nonoccupational pesticide exposures may be unaware of the presence of pesticides in their environments (Centers for Disease Control and Prevention 2009). The potential for exposure to other toxicants with similar mechanisms is even greater. To continue the interplay between human and experimental studies, future mechanistic studies of these pesticides should model exposure conditions similar to those occurring in humans, including chronic low-dose exposure, exposure to multiple agents, and assessment of gene–exposure interactions. Such work could provide new insights into the pathogenesis and ultimately the prevention of PD."

Rotenone

Rotenone is a **naturally occurring** organic compound that has been used as a pesticide for several decades to control unwanted fish populations, in nurseries, and in organic farming.

Paraquat

Paraquat dichloride, or paraquat, is a highly toxic (poisonous) liquid herbicide (plant or weed killer). Commercial farmers may use paraquat to kill weeds or dry out certain crops, like cotton, before harvesting them.

But even more commonly found in our food and environment...

Glyphosate is the most widely-used herbicide in the world. As a non-selective herbicide, it kills most plants. Scientists now link glyphosate to a number of human health problems, from cancer and neurological diseases to endocrine disruption and birth defects.

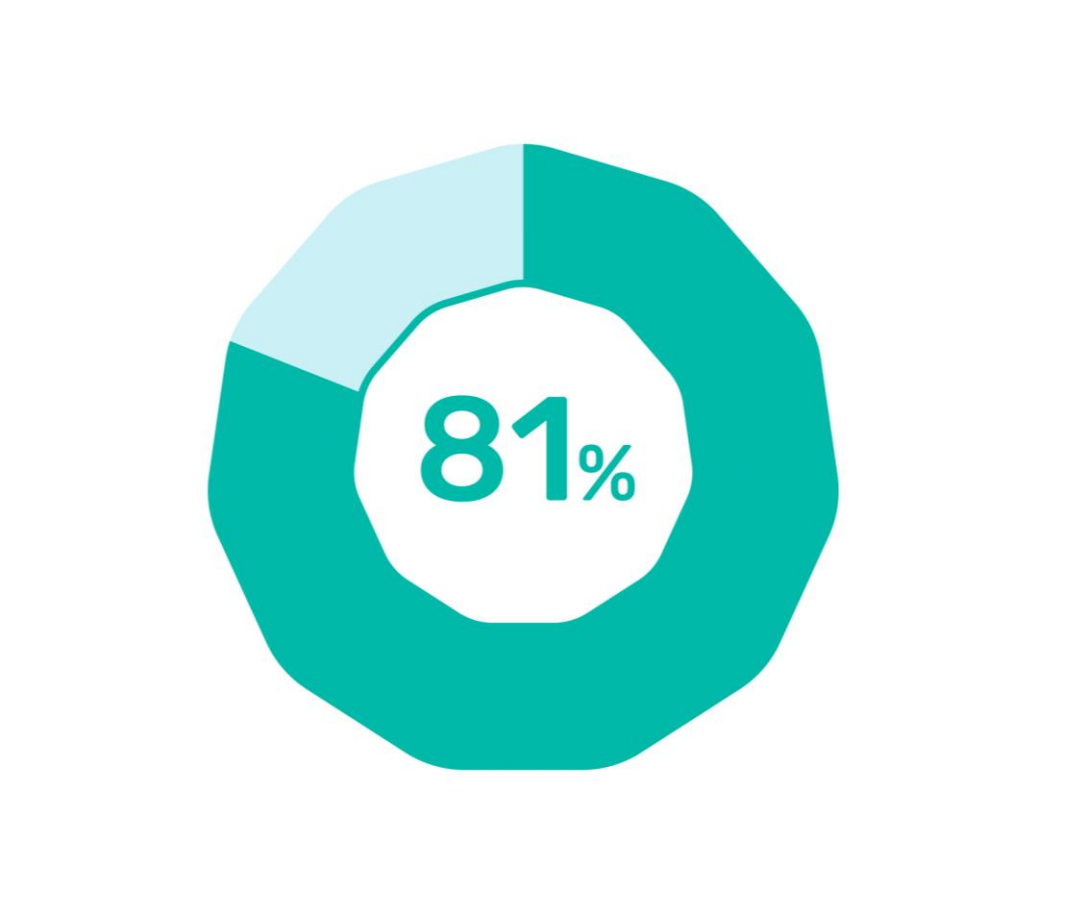
Toxic Effects of Glyphosate on the Nervous System

"Glyphosate, a non-selective systemic biocide with broad-spectrum activity, is the most widely used herbicide in the world. It can persist in the environment for days or months, and its intensive and large-scale use can constitute a major environmental and health problem. In this systematic review, we investigate the current state of our knowledge related to the effects of this pesticide on the nervous system of various animal species and humans. The information provided indicates that exposure to glyphosate or its commercial formulations induces several neurotoxic effects." Toxic Effects of Glyphosate on the Nervous System: A Systematic Review - PMC (nih.gov)

Parkinsonism Relating to Intoxication with Glyphosate

"We herein report the case of a 38-year-old man who developed parkinsonism 4 years after ingesting glyphosate. The patient presented with right-sided bradykinesia and cogwheel rigidity without autonomic symptoms. Magnetic resonance imaging of the brain and (123I)-metaiodobenzylguanidine myocardial scintigraphy were normal. A drastic response to levodopa and the presence of levodopa-induced dyskinesia without strong non-motor symptoms were seen in this patient. We considered that young-onset atypical parkinsonism was associated with a history of sublethal glyphosate ingestion. Epidemiologic investigations have shown that exposure to pesticides is a risk factor for Parkinson's disease (PD). Our findings support the notion that glyphosate exposure might be related to the onset of PD." Parkinsonism Relating to Intoxication with Glyphosate - PMC (nih.gov)

Who is vulnerable to chronic glyphosate poisoning?



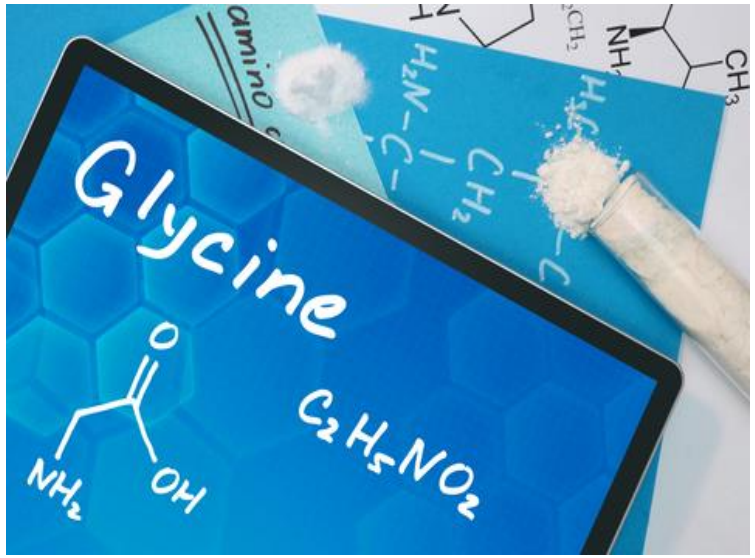
An estimated 81% of the U.S. population has had recent exposure to glyphosate, a chemical found in some weed killers, according to a new study, "Exposure to glyphosate in the United States: Data from the 2013–2014 National Health and Nutrition Examination Survey."

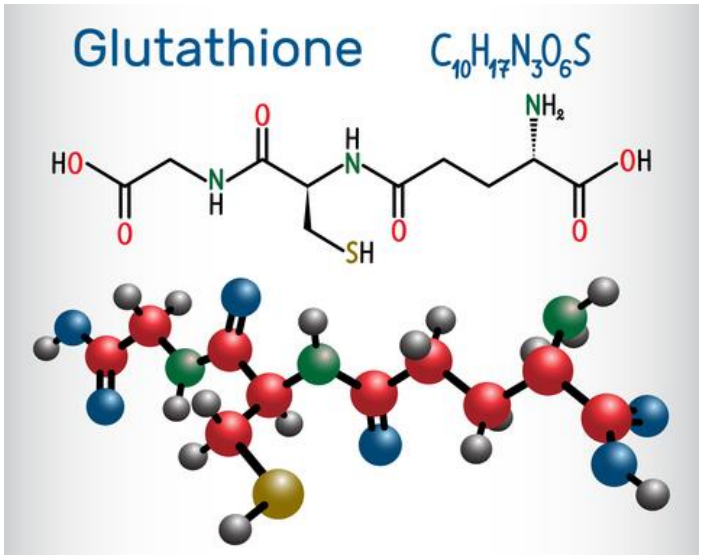
People can be exposed to glyphosate through diet, skin contact, and breathing in particles from the air. Fruits, fruit juices, vegetables, and cereals are possible sources of glyphosate, a widely used herbicide in the United States.

This is very cool!

Protect yourself against glyphosate poisoning...

GLYCINE: An amino acid needed to create glutathione — a powerful detoxifier and antioxidant that also protects the liver against toxicity. Interestingly, the body can mistake glyphosate for glycine during protein synthesis, tricking it into storing toxic glyphosate in tissues and organs. By supplementing with extra glycine, we can prevent glyphosate from being stored, enhance **GLUTATHIONE** activity, and help support healthy protein production.





[Glyphosate Toxicity Alert: How to Detox from America's #1 Weedkiller (draxe.com)]

A slap-it-silly approach…

As I often say, we must use a multi-pronged strategy whenever confronted with a daunting disease process, whatever it might be.

When it comes to Parkinson's disease, we must ask ourselves:

- What processes are occurring?
- Can they be stopped?
- If so, how?

A Proposed Anti-Parkinsonian Toolkit

“Phenomenal improvement in the psychic, physiological, and social functioning of 189 elderly subjects who had undergone procaine (Novocaine) therapy over a period of ten years. Patients bedridden with arthritis and rheumatism were mobile again, living normal lives; hypertension and angina pectoris vanished. Severely disoriented psychiatric patients recovered; memory, concentration and perception were restored; **extrapyramidal rigidity in Parkinson's disease diminished**. Hair growth was stimulated, re-pigmentation of gray hair occurred in some cases, flaccid senile skin regained its turgor and became tight and smooth, the subjects looked ten years younger. A notable reduction in mortality rates were also reported.” See source.

Gerovital GH3

Gerovital GH3 is one of my favorite nootropic tools I have been using myself and with many of my clients over many years. In the 1940's Dr. Ana Aslan wondered if injecting Novocaine into her patients with severe arthritis would make a difference in their arthritic pain. Not only was her hypothesis on target, she noticed remarkable anti-aging effects, from reversal of graying hair to normal color, restored hair growth from balding, a youthful effect on the skin, regulation of blood pressure whether too high or too low, resistance to an epidemic that swept through Europe, and a remarkable effect on mood and depression. For more info, see here.

“Daily consumption of methylene blue reduces attentional deficits and dopamine reduction in a 6-OHDA model of Parkinson's disease”

Daily consumption of methylene blue reduces attentional deficits and dopamine reduction in a 6-OHDA model of Parkinson's disease - PubMed (nih.gov)

Methylene Blue & Parkinson's

"MB significantly improved attentional performance in the five-choice task designed to measure selective and sustained attention. In conclusion, MB might be useful in improving some attentional function and preserving dopaminergic cells in this model. Future work should continue to study and optimize the abilities of MB for the treatment of PD." see here

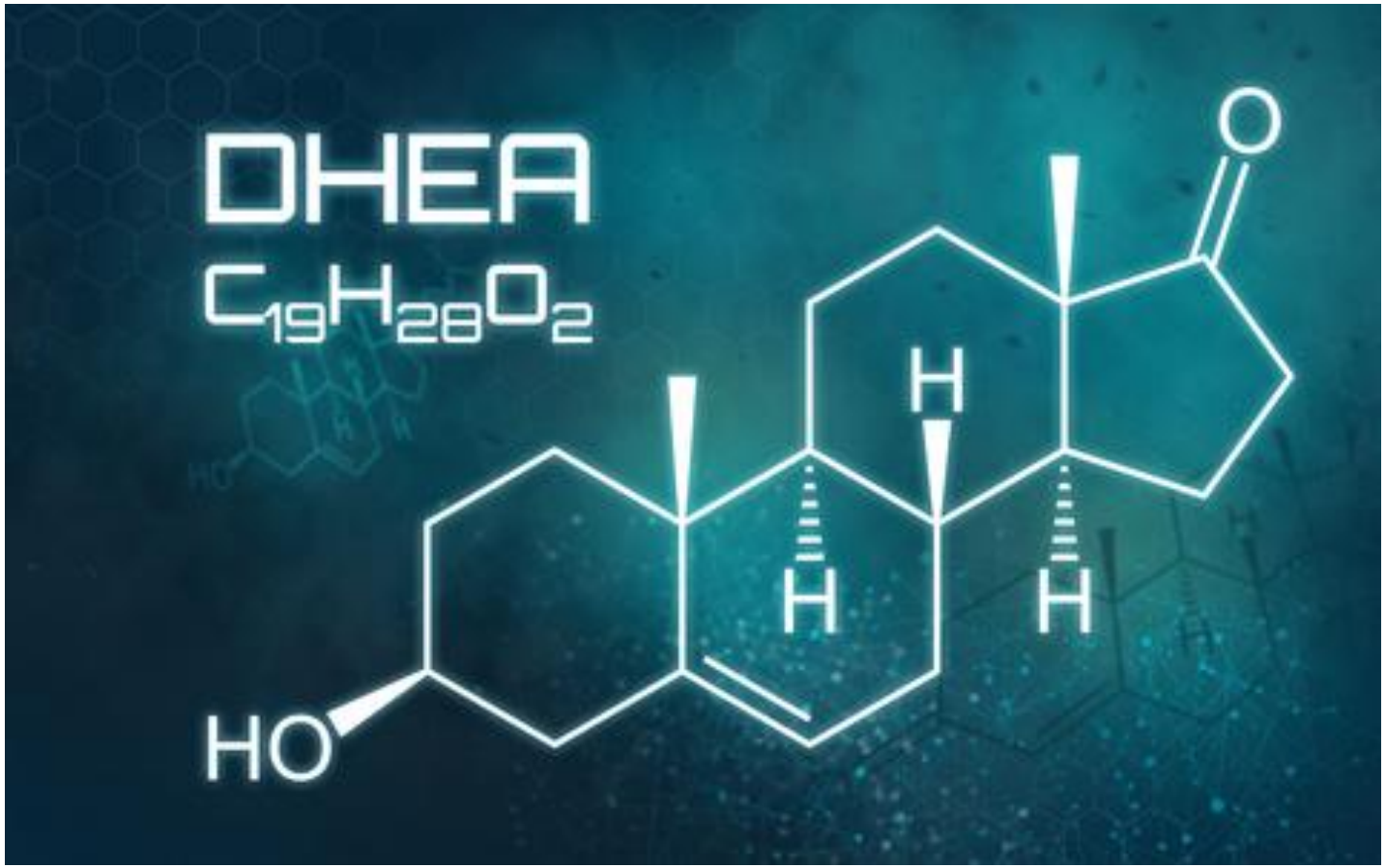
Very interesting in the study above is that they use rotenone to create Parkinson's in mice in order to experiment with Parkinson's disease. It was found impossible to induce Parkinson's in mice pretreated with methylene blue. I would extrapolate this information to see methylene blue as protective against the toxicity of pesticides, specifically rotenone.

"Long-term iodine deficiency appears linked to abnormalities in the **dopaminergic system** that include an increased number of dopamine receptors. It is argued that this raises susceptibility to **dopamine oxidation** which, in turn, causes deficiencies of the antioxidant enzymes Cu/Zn superoxide dismutase, **glutathione** peroxidase and catalase. Dopamine deficiency also leads to elevated cytotoxic glutamate levels. Implications of the iodine-dopachrome-glutamate hypothesis, for treatment of these three neurologic disorders, are then discussed. Possible interventions include the use of levodopa, vitamin B3, Coenzyme Q10, various antioxidants, amino acids, iodine and glutamate antagonists." Parkinson's Disease, Multiple Sclerosis and Amyotrophic Lateral Sclerosis:

Iodine & Parkinson's

"Not enough iodine in your diet negatively affects neurotransmitters in your brain. And can result in depression, brain fog, anxiety, learning and memory problems, and ultimately lead to neurodegenerative diseases like Alzheimer's and Parkinson's." Iodine - Nootropics Expert

DHEA



"Parkinson's disease (PD) is a neurodegenerative disorder characterized by a progressive loss of nigrostriatal dopaminergic neurons. Its treatment is symptomatic and shows limited efficacy. Dehydroepiandrosterone (DHEA) is a hormone produced in the brain. Several studies have reported beneficial effects of said steroid in experimental models of PD and various human diseases, but its potential for PD is inconclusive. Therefore, it is necessary to evaluate current evidence to determine the therapeutic potential of DHEA administration for PD since it could be an effective and low-cost treatment."
ane222f.pdf (medigraphic.com)

Effect of DHEA on recovery of muscle atrophy induced by Parkinson's disease. Choe MA , An GJ, Koo BS, Jeon S.

Purpose: The purpose of this study was to determine the effect of dehydroepiandrosterone (DHEA) on recovery of muscle atrophy induced by Parkinson's disease.

Results: The DHEA group showed significant increase in the number of tyrosine hydroxylase (TH) positive neurons in the lesioned side substantia nigra compared to the vehicle group.

Conclusion: Our results suggest that DHEA treatment recovers Parkinson's disease induced contralateral soleus muscle atrophy through Akt and ERK phosphorylation. Effect of DHEA on recovery of muscle atrophy induced by Parkinson's disease - PubMed (nih.gov)

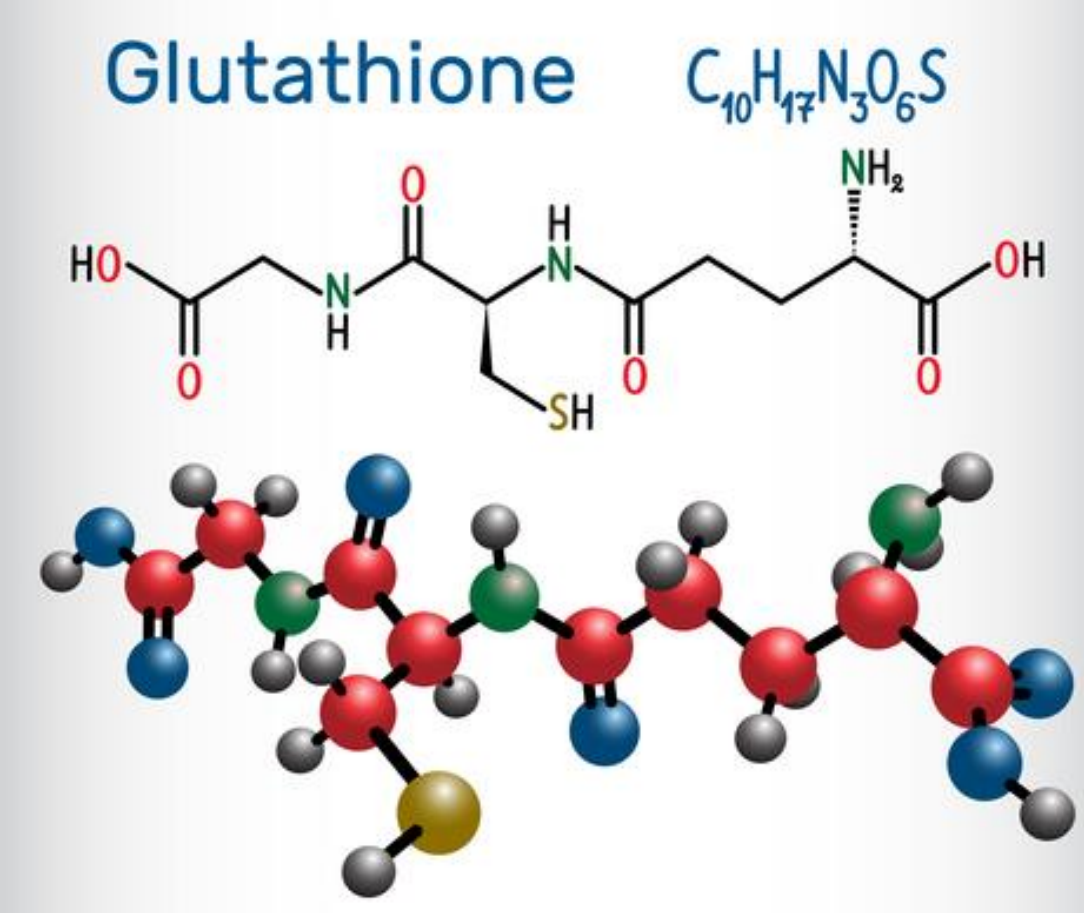
Sulforaphane

"The aim of this review is to summarize the experimental studies present on Pubmed that report the efficacy of SFN in the treatment of neurodegenerative disease, including Alzheimer's disease (AD), **Parkinson's disease (PD),** and multiple sclerosis (MS). Therefore, thanks to its beneficial effects, **SFN could be useful as a supplement to counteracting neurodegenerative diseases.**" [Efficacy of Sulforaphane in Neurodegenerative Diseases - PMC (nih.gov)]

Protective effect of sulforaphane against dopaminergic cell death

Abstract Parkinson's disease (PD) is a progressive neurodegenerative disorder with a selective loss of dopaminergic neurons in the substantia nigra. Evidence suggests oxidation of dopamine (DA) to DA quinone and consequent oxidative stress as a major factor contributing to this vulnerability. We have previously observed that exposure to or induction of NAD(P)H:quinone reductase (QR1), the enzyme that catalyzes the reduction of quinone, effectively protects DA cells. **Sulforaphane (SF) is a drug identified as a potent inducer of QR1 in various non-neuronal cells.** In the present study, we show that SF protects against compounds known to induce DA quinone production (6-hydroxydopamine and tetrahydrobiopterin) in DAergic cell lines CATH.a and SK-N-BE(2)C as well as in mesencephalic DAergic neurons. SF leads to attenuation of the increase in protein-bound quinone in tetrahydrobiopterin-treated cells, but this does not occur in cells that have been depleted of DA, suggesting involvement of DA quinone. SF pretreatment prevents membrane damage, DNA fragmentation, and accumulation of reactive oxygen species. SF causes increases in mRNA levels and enzymatic activity of QR1 in a dose-dependent manner. Taken together, these results indicate that SF causes induction of QR1 gene expression, removal of intracellular DA quinone, and protection against toxicity in DAergic cells. Thus, **this major isothiocyanate found in cruciferous vegetables may serve as a potential candidate for development of treatment and/or prevention of PD**. Protective effect of sulforaphane against dopaminergic cell death - PubMed (nih.gov)

Glutathione



Glutathione and Parkinson's disease: is this the elephant in the room?

Learn More

- Low GSH metabolism is linked to the pathophysiology of Parkinson's disease (PD).
- It is unclear if GSH deficiency is an etiological factor in PD or a consequence of it.
- In the future, external modulation of GSH levels may be used in the treatment of PD.
- More research is needed on active neuroprotective and anti-neuroinflammatory agents. Source

The glutathione system in Parkinson's disease and its progression

Lowered GSH metabolism and a low GSH/GSSG ratio following oxidative stress are associated with mitochondrial dysfunctions and constitute a critical factor in the neuroinflammatory and neurodegenerative processes accompanying PD. This review provides indirect evidence that GSH redox signaling is associated with the pathophysiology of PD. The results show that antioxidant approaches, including neuroprotective and anti-neuroinflammatory agents, which neutralize reactive oxygen species, may have therapeutic efficacy in the treatment of PD and its progression.
[The glutathione system in Parkinson's disease and its progression - PubMed (nih.gov)]

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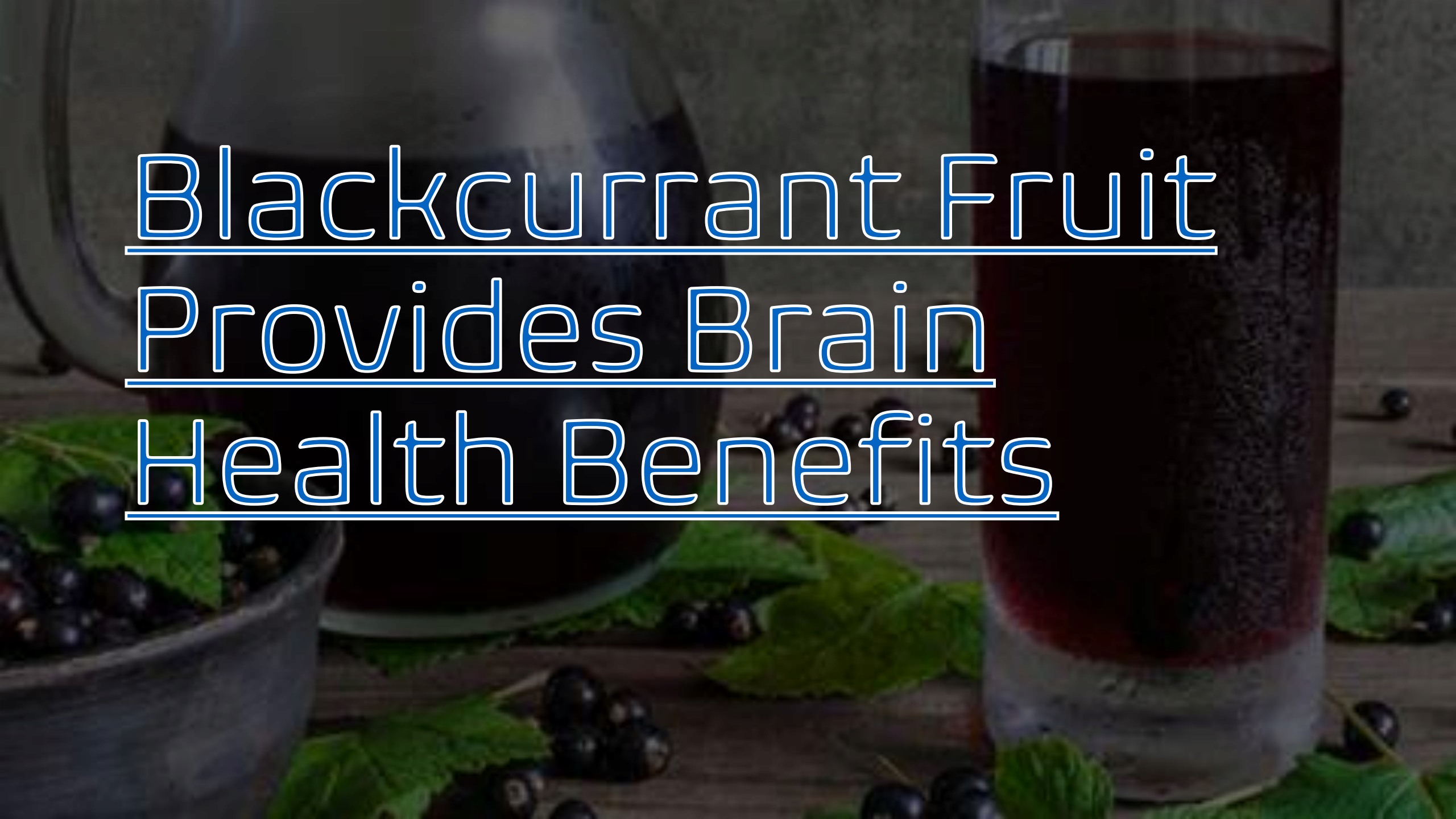
Glutathione--a review on its role and significance in Parkinson's disease

Abstract Parkinson's disease (PD) is the second most common neurodegenerative disease, affecting over a million people in the United States alone, and is characterized by rigidity, bradykinesia, resting tremor, and postural instability. Its main neuropathological feature is the loss of dopaminergic neurons of the substantia nigra pars compacta. However, the pathogenesis of this loss is not understood fully. **One of the earliest biochemical changes seen in PD is a reduction in the levels of total glutathione, a key cellular antioxidant.** Traditionally, it has been thought that this decrease in GSH levels is the consequence of increased oxidative stress, a process heavily implicated in PD pathogenesis. However, emerging evidence suggests that GSH depletion may itself play an active role in PD pathogenesis. **This review aims to explore the contribution of GSH depletion to PD pathogenesis.** Glutathione--a review on its role and significance in Parkinson's disease - PubMed (nih.gov)



Blackcurrant Fruit Provides Brain Health Benefits

Scientists from Plant & Food Research in New Zealand, in collaboration with Northumbria University in the United Kingdom, assessed the effects of blackcurrants on cognitive health and found that the fruit improved attention and regulated mood.

Supplementation of Blackcurrant Anthocyanins Increased Cyclic Glycine-Proline in the Cerebrospinal Fluid of Parkinson Patients: Potential Treatment to Improve Insulin-Like Growth Factor-1 Function

New Zealand blackcurrants have one of the highest recorded anthocyanin values of any fruit on the planet. Our 35% maximum-strength extract is a formulation containing Enzans®, key identifiers of the New Zealand blackcurrant bio-actives responsible for their scientifically-proven properties. Each capsule contains the equivalent of a generous handful of blackcurrants and represents outstanding value for money.

Magnesium & Parkinson's Disease

Magnesium (Mg) is essential for cell functions such as transport of calcium and potassium ions, and modulates signal transduction, energy metabolism, and cell proliferation. Several studies elucidated a reduced concentration of Mg in patients with Parkinson's disease.

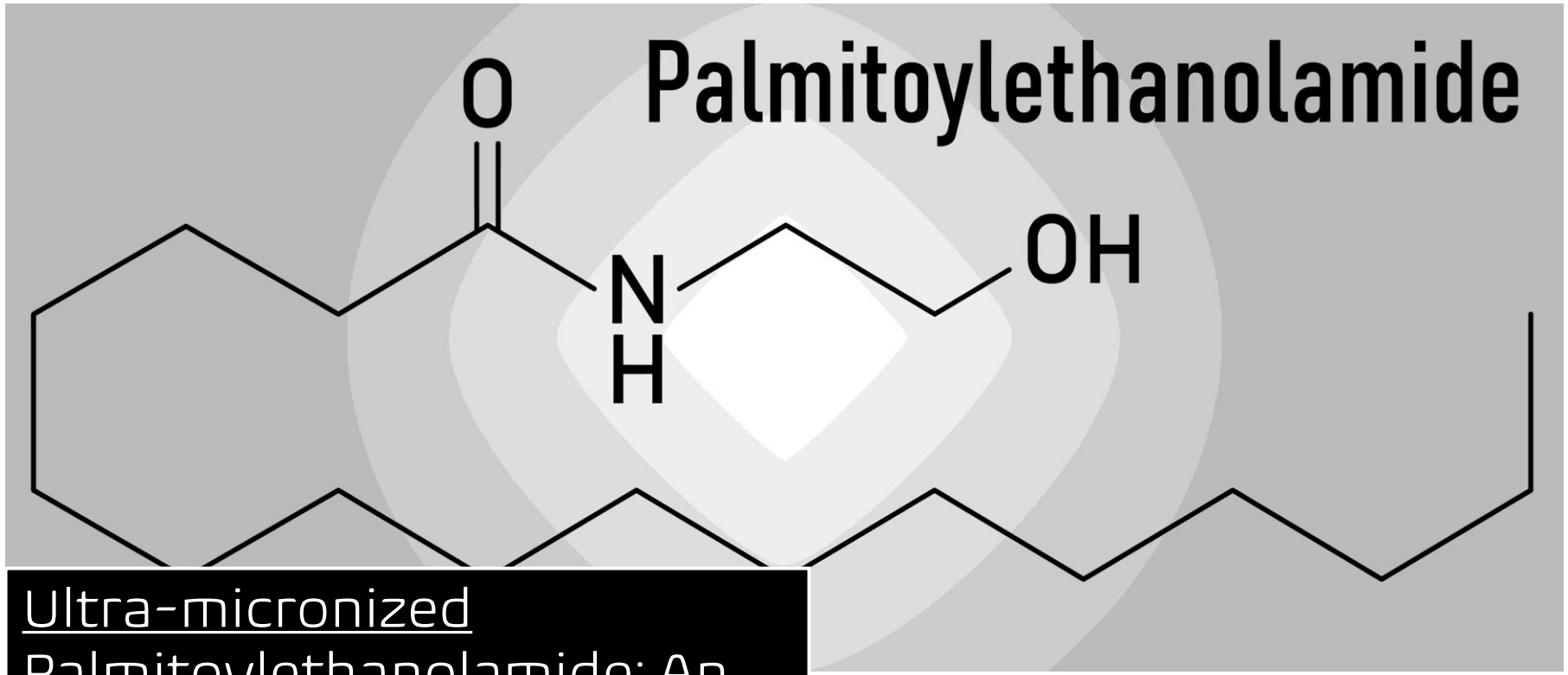
Treatment Of Magnesium-L-Threonate Elevates The Magnesium Level In The Cerebrospinal Fluid And Attenuates Motor Deficits And Dopamine Neuron Loss In A Mouse Model Of Parkinson's disease

- Magnesium in the CNS: recent advances and developments
- Magnesium and the Brain: A Focus on Neuroinflammation and Neurodegeneration
- Magnesium in Parkinson’s disease: an update in clinical and basic aspects
- The Role of Magnesium in Neurological Disorders
- The neuroprotective potential of magnesium in Parkinson's disease

Abstract: Magnesium (Mg) is involved in the regulation of metabolism and in the maintenance of the homeostasis of all the tissues, including the brain, where it harmonizes nerve signal transmission and preserves the integrity of the blood–brain barrier. Mg deficiency contributes to systemic low-grade inflammation, the common denominator of most diseases. In particular, neuroinflammation is the hallmark of neurodegenerative disorders. Starting from a rapid overview on the role of magnesium in the brain, this narrative review provides evidences linking the derangement of magnesium balance with multiple sclerosis, Alzheimer's, and Parkinson's diseases.

Coconut Oil & Parkinson’s Disease

Neuroprotective role of coconut oil for the prevention and treatment of Parkinson's disease: potential mechanisms of action



Ultra-micronized Palmitoylethanolamide: An Efficacious Adjuvant Therapy for Parkinson's Disease


NEUROGAN
+HEALTH
PEA PRO
ULTRA MICRONIZED
PALMITOYLETHANOLAMIDE
1500MG PER SERVING
NATURAL DISCOMFORT
POTENT ANTIOXIDANT
90 CAPSULES
45,000MG TOTAL
NET WT. 1.58 OZ (45G)


FLORA
Certified Organic
MCT Oil
Medium Chain Triglycerides
Ethically Sourced | Unflavored
100% from
Non-GMO Coconuts
14g per serving
Vegan + Gluten-Free + Keto-Friendly
USDA ORGANIC
DIETARY SUPPLEMENT
17 FL. OZ. (500 mL)
NON GMO Project
VERIFIED
nongmoproject.org

The B's have it!

Study suggests 2 vitamin B deficiencies may play a role in Parkinson's disease

Substantively Lowered Levels of Pantothenic Acid (Vitamin B5) in Several Regions of the Human Brain in Parkinson's Disease Dementia

Low Vitamin B12 and Parkinson Disease: Potential Link to Reduced Cholinergic Transmission and Severity of Disease –

Read here


B1

Neural Regen Res. 2016 Mar; 11(3): 406–407.
doi: 10.4103/1673-5374.179047

PMCID: PMC4828997
PMID: 27127471

An open-label pilot study with high-dose thiamine in Parkinson's disease

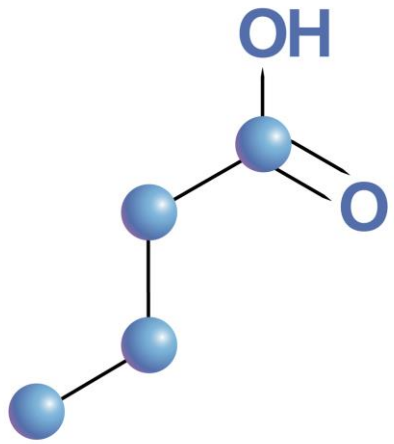
Antonio Costantini, M.D.* and Roberto Fancellu

The treatment with thiamine led to a significant improvement of PD symptoms: UPDRS part II improved from 12.5 ± 4.0 to 7.7 ± 3.5 ($P < 0.001$, *t*-test for paired data), motor UPDRS part III improved from 21.6 ± 4.8 to 11.8 ± 6.0 ($P < 0.00001$, *t*-test for paired data). The Hoehn and Yahr score (UPDRS part V), a disease stage measure**, significantly improved** from 3.0 ± 0.8 to 2.5 ± 0.6 ($P < 0.001$, *t*-test for paired data); also the Schwab and England functional score (UPDRS part VI) **significantly improved** from 69.0 ± 18.5 to 80.0 ± 12.5 ($P < 0.05$, *t*-test for paired data). **The clinical motor improvement was even higher during the second month of treatment**, after the increase of the dosage of thiamine associated with the increase of levodopa for the patients already in treatment with this drug, or after the beginning of the treatment with levodopa for the patients naïve for this therapy. The mean daily levodopa dose after two months was 515.0 ± 228.6 mg. **Some patients with a milder phenotype had a complete clinical recovery.** No patient experienced adverse events or discontinued the treatment.


SEALED FOR PROTECTION
Cardiovascular Research Ltd.
LIPOTHIAMINE
Dietary Supplement
60 Enteric Coated Tablets


SHORT
CHAIN
FATTY
ACIDS


BODYBIO
SODIUM BUTYRATE
A Short Chain Fatty Acid
+ Microbiome Support*
+ Improve Digestion*
+ Promotes Healthy DNA*
Dietary Supplement
100 Non-GMO Capsules



Butyric acid

Neuroprotective Effects of Sodium Butyrate by Restoring Gut Microbiota and Inhibiting TLR4 Signaling in Mice with MPTP-Induced Parkinson's Disease

Bacterial Butyrate in Parkinson's Disease Is Linked to Epigenetic Changes and Depressive Symptoms

Sodium butyrate exerts protective effect against Parkinson's disease in mice via stimulation of glucagon like peptide-1

Conclusions: Decreased levels of bacterially produced butyrate are related to epigenetic changes in leucocytes and neurons from PD patients and to the severity of their depressive symptoms. PD shares common butyrate-dependent epigenetic changes with certain GI and psychiatric disorders, which could be relevant for their epidemiological relation.

Parkinson's Disease: Potential Actions of Lithium by Targeting the WNT/β-Catenin Pathway, Oxidative Stress, Inflammation and Glutamatergic Pathway

7/24 by Buck Institute JULY 24, 2015. PRESS RELEASE

Low-dose lithium reduces side effects from most common treatment for Parkinson's disease

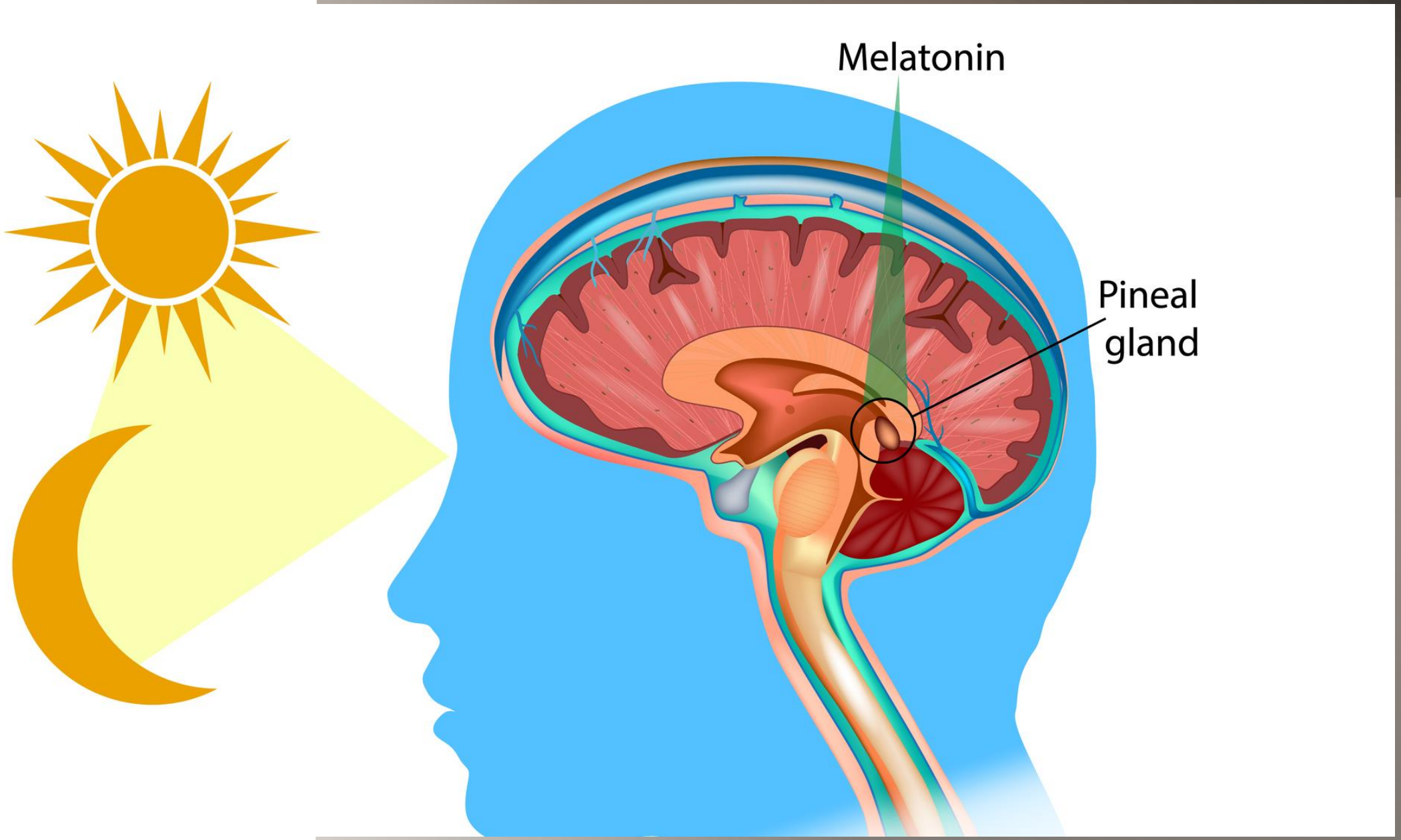
"Low-dose lithium reduced involuntary motor movements – the troubling side effect of the medication most commonly used to treat Parkinson's disease (PD) – in a mouse model of the condition that is diagnosed in about 60,000 Americans each year."

Lithium as a Nutrient

ABSTRACT In high doses, lithium acts as a drug, accompanied by potentially serious and debilitating side effects. In low doses, lithium acts as a nutrient required for B12 and folate transport and uptake, neuromodulation, and the function of many biochemical processes in both humans and animals. Studies since the 1970s have shown the ability of lithium to stimulate the proliferation of stem cells. Recent studies have described its ability to up-regulate neurotrophins such as brain-derived neurotrophic factor (BDNF) and nerve-growth factor (NGF), which are important in neuronal function, plasticity, and repair. With its newly described antioxidant and anti-inflammatory activity along with powerful neuroprotective effects, low-dose lithium therapy has largely unrealized potential to prevent or treat a wide-range of neurological disorders such as traumatic brain injury (TBI), Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis (ALS), chronic pain, mercury toxicity, depression/anxiety, alcoholism, and drug addiction.

Potential application of lithium in Parkinson's and other neurodegenerative diseases

Learn More


Melatonin
Pineal gland

Melatonin & Parkinson’s Disease

Mechanism of the beneficial effect of melatonin in experimental Parkinson's disease

Melatonin protects against neurobehavioral and mitochondrial deficits in a chronic mouse model of Parkinson's disease

Increased melatonin may play dual roles in the striata of a 6-hydroxydopamine model of Parkinson's disease

Nutrients. 2022 Aug; 14(15): 3240.
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PMCID: PMC9370710
PMID: 35956417

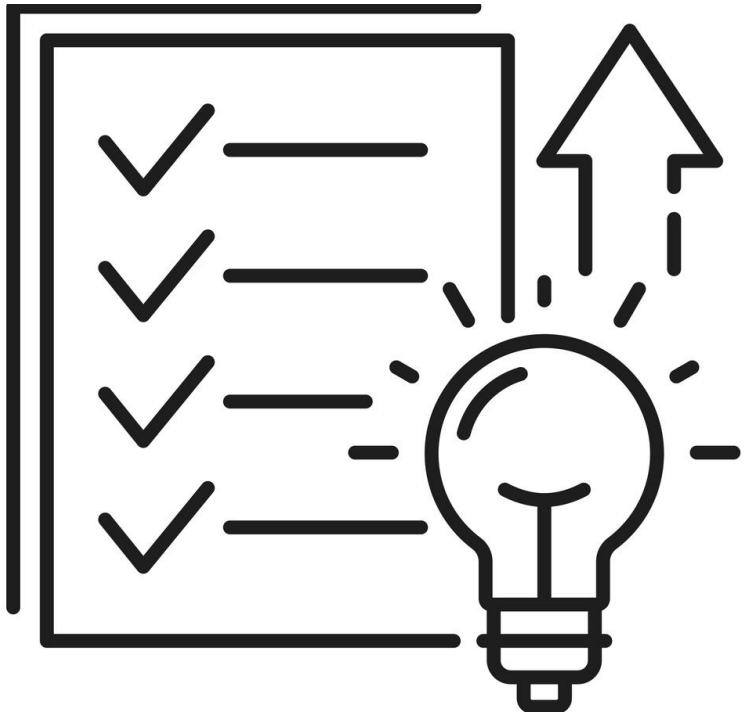
Parkinson's Disease and Sugar Intake—Reasons for and Consequences of a Still Unclear Craving

Julienne Haas,[1,*] Daniela Berg,[1] Anja Bosy-Westphal,[2] and Eva Schaeffer[1]

Abstract

Lately, studies have shown that patients with Parkinson's disease (PD) report a strong craving for sweets and consume significantly more fast-acting carbohydrates than healthy controls. Consuming food with a high-sugar content is assumed to lead to an increase in insulin concentration, which could positively influence dopamine concentration in the brain and unconsciously be used by patients as kind of "self-medication" to compensate for a lack of dopamine in PD. On the other hand, high-sugar intake could also lead to insulin resistance and diabetes, which is discussed as a causative factor for progressive neurodegeneration in PD. In this critical appraisal, we discuss the role of sugar intake and insulin on dopamine metabolism in patients with PD and how this could influence the potential neurodegeneration mediated by insulin resistance.

A Suggested Parkinson's Protocol



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"…and if the root be holy, so are the branches…"

EXAMPLE OF PARKINSON'S DAILY REGIMEN

IMPORTANT: This is an example of a typical protocol I use with clients with Parkinson's disease. Of course, I am always working with these clients individually and am very well aware of their medications, other health issues, etc., and so everything I recommend is centered within that framework.

It is important to know that **methylene blue** cannot be used by someone taking a prescribed antidepressant. When I work with someone who is and wants to use **methylene blue**, they can opt to wean off the medication and then use methylene blue. If you are reading this and are not my client, be aware of that.

Also, methylene blue is contraindicated in people with a G6PD enzyme deficiency. Here is information about the G6PD enzyme deficiency, what drugs/substances to avoid. To be certain, simply ask your doctor to test you for this before beginning methylene blue.

Other than that, there are no contraindications of any of the other things in the list with medications or detrimental to any other health condition. I have many of many of my clients on most of these nutrients for a number of reasons, and, with the exception of CURRANZ and the extra thiamine, I use every one of these myself. They are good for so many things when it comes to health.

Of note, I have not used extra thiamine with anyone yet, but this was brought to my attention in a book touting vitamin B1 as a possible cure for Parkinson's. The science makes sense, but the dosing is in question based on that work, which really individualizes it. I have added it into the protocol here in a form based on my reading is an appropriate form and in a dose that should be not too much or too little, especially when set within the framework of the synergy of the other things in the protocol. That book is found here.

WEE HOURS OF THE MORNING WHEN YOU BRIEFLY AWAKEN:

GEROVITAL GH3 – 2 tablets with water, no food or other liquid for one hour. Take 5 days off between bottles to remain sensitive to it. The procaine in this formula is destroyed by stomach acid, so it must be taken 2 hours minimum since last food and 1 hour before next food or any liquid other than water. It must be taken with plain, flat water, cold or room temp, not hot. The idea is to get it through the stomach into the small intestine before the enteric coating dissolves.

FIRST THING IN THE MORNING WHEN YOU GET UP:

INDIUMEASE 1 MG/DROP – start with 1 drop; place on the back of your tongue and swallow; over a week or so increase to 1 drop/50 lb bodyweight. Wait 15 minutes before eating or drinking (NO CREATINE SUPPLEMENTS WHEN USING INDIUM)

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BREAKFAST TIME:

MSM POWDER – start with 1/8 teaspoon – add to liquid or eat off the spoon; over a couple of weeks' time gradually increase to 1-3 heaping teaspoons.
METHYLENE BLUE – follow this instruction sheet to figure out your optimal dose and helpful information regarding how to take BE SURE TO READ & USE THIS
IODORAL 12.5 MG – start with 1 tablet every 4th day x 1 week, then 1tablet every other day x 1 week, then 1 tablet daily
SODIUM BUTYRATE – 1capsule
PURE ENCAPSULATIONS MICRONIZED DHEA 25 MG – 1 capsule for women, 2 for men
JARROW MAGMIND – 1 capsule
S-ACETYL-L-GLUTATHIONE 300 MG – 1 capsule
THORNE BASIC NUTRIENTS 2/DAY – 1 capsule
CURRANZ NEW ZEALAND BLACK CURRANT – 1 tablet
PURE ENCAPSULATIONS PANTOTHENIC ACID (VIT B5) 500 MG – 1 capsule
MICRONIZED PEA 600 MG – 1 capsule
SEEKING HEALTH ADENO B12 – 1 lozenge, dissolve under the tongue
CARDIOVASCULAR RESEARCH LIPOTHIAMINE B1 50 MG – 1 capsule

AFTER SUPPER:

METHYLENE BLUE – 20 DROPS - ADD TO SOME WATER AND DRINK
JARROW MAGMIND – 2 capsules
SODIUM BUTYRATE – 1capsule
S-ACETYL-L-GLUTATHIONE 300 MG – 1 capsule
THORNE BASIC NUTRIENTS 2/DAY – 1 capsule
CURRANZ NEW ZEALAND BLACK CURRANT – 1 tablet
PURE ENCAPSULATIONS PANTOTHENIC ACID (VIT B5) 500 MG – 1 capsule
MICRONIZED PEA 600 MG – 1 capsule
LITHIUM OROTATE 5 MG – capsule
MELATONIN POWDER 60 MG/LEVEL SCOOP – 1 to 3 scoops; add to a bite of soft food, mix, and eat (doesn't mix that well with liquid because it is very fine); you can also simply plop on your tongue and chase with water or other liquid. (The first time you purchase this, be sure to get the scoop, because it is metered instrument of measurement; each level scoop is exactly 60 mg. After that, to reorder, save your scoop and order without the scoop.)

I hope this presentation has been helpful, whether you have Parkinson's disease, know someone who does who might be interested in this information, or simply want to learn more about powerful, natural means of preventing disease and enjoying a long life of good health. Be well!

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