The Frequency Therapist

Senergy® Wellness

ONE MECHANISM.
FOUR DISEASES.

Glutamate Excitotoxicity as a Unified Theory of Neurodegeneration

ALS · Multiple Sclerosis · Parkinson's Disease · Peripheral Neuropathy

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⚠ IMPORTANT DISCLAIMER

This presentation is for educational purposes only.

The information presented does not constitute medical advice and is not intended to diagnose, treat, cure, or prevent any disease.

Discussion of the Tennant BioModulator® is based on physiological principles and published peer-reviewed science. No claims are made that the BioModulator® treats or cures any of the conditions discussed.

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All cited research reflects the scientific literature available at time of preparation.

PART 1

The Core Theory

Understanding Glutamate Excitotoxicity

Why One Mechanism Produces Four Different Diseases

Glutamate: Essential Messenger → Deadly Toxin

✓ NORMAL GLUTAMATE

Glutamate is the brain's primary excitatory neurotransmitter — responsible for:

- Learning & memory formation
- Controlling voluntary movement
- Sensory processing
- Nearly all fast signal transmission

It is released, activates the next neuron briefly, then is immediately cleared back into astrocytes (support cells). The whole process takes milliseconds.

✗ EXCITOTOXICITY

When glutamate accumulates beyond control, it overstimulates NMDA receptors, flooding neurons with calcium:

- Calcium overload activates destructive enzymes
- Mitochondria collapse, producing toxic ROS
- Cell membranes rupture
- Neurons die — and they release more glutamate as they die

This self-amplifying death spiral is the core mechanism shared by ALS, MS, Parkinson's, and neuropathy.

The Excitotoxic Death Cascade: Step by Step

1 Glutamate Accumulates

Too much glutamate in the synapse — not cleared fast enough by astrocyte transporters (EAAT2)

2 NMDA Receptors Overactivated

NMDA receptors are overwhelmed — calcium and sodium flood into the neuron uncontrollably

3 Calcium Overload

Excess Ca^{2+} activates destructive enzymes: proteases, phospholipases, endonucleases — shredding the cell from inside

4 Mitochondria Collapse

Calcium floods mitochondria, opens the death pore (mPTP), collapsing ATP production and releasing cytochrome c

5 ROS Storm

Reactive oxygen species (free radicals) explode, damaging DNA, proteins, and membranes across the whole cell

6 Cell Death → More Glutamate

Dying neurons release their stored glutamate, poisoning neighboring cells. The death spiral spreads.

Voltage & Cellular Energy — The True Root

Normal Cell Voltage

Healthy neurons maintain –70mV. This voltage drives ion channels and the Na+/K+ ATPase pump that removes glutamate from synapses. Voltage IS the glutamate clearance system.

Voltage Drop = Glutamate Buildup

When cellular voltage falls, the Na+/K+ ATPase pump slows or stops. Glutamate is no longer cleared. The excitotoxic cascade begins not from 'too much glutamate' but from 'too little energy to remove it'.

Mitochondria Are the Voltage Source

ATP from mitochondrial complex I–V powers every ion pump. Toxins (rotenone, TCE, mycotoxins), nutritional deficits (CoQ10, B vitamins), and oxidative stress impair complex I — the most vulnerable point in all four diseases.

Restoring Voltage = Restoring Protection

This is the foundational rationale of both the BioModulator® (restoring cellular voltage via adaptive microcurrent) and Proper Supplementation (restoring biochemical substrates that produce voltage at the mitochondrial level).

Levin, M. (2014). Molecular bioelectricity. Molecular Biology of the Cell, 25, 3835.

How Excitotoxicity Drives Each Disease

ALS

CENTRAL MECHANISM

The only FDA-approved ALS drug (riluzole) works by blocking glutamate release and restoring astrocyte transporter EAAT2. ALS astrocytes lose up to 60% of their EAAT2 — causing runaway glutamate accumulation that kills motor neurons.

Parkinson's

MAJOR DRIVER

Dopamine normally modulates glutamate activity in the basal ganglia. When dopamine neurons die, glutamate circuits become hyperactive and uncontrolled — accelerating further neurodegeneration in a vicious cycle.

Multiple Sclerosis

KEY AMPLIFIER

MS inflammation causes glutamate release from microglia and damaged oligodendrocytes. Demyelinated axons are hypersensitive to excitotoxic damage. Glutamate drives axon degeneration — the cause of permanent disability.

Neuropathy

PERIPHERAL DRIVER

Peripheral NMDA and AMPA receptors on sensory nerves are overactivated in painful neuropathy. Hyperactive C fiber discharge creates a peripheral excitotoxic loop — causing burning pain, sensitization, and progressive small fiber loss.

The Unified Theory

Same weapon. Different target. Same root cause.

Glutamate excitotoxicity preferentially attacks the most metabolically demanding, least protected neural zones. The zone that fails depends on genetics, toxic exposures, injuries, nutritional status, and lifetime voltage history.

ALS

Zone:

Motor Cortex
& Anterior Horn

Motor Neurons

MS

Zone:

White Matter
Myelin

Oligodendrocytes

Parkinson's

Zone:

Substantia Nigra
Basal Ganglia

Dopamine Neurons

Neuropathy

Zone:

DRG (Dorsal Root Ganglia)
&
Small Fibers

C & Aδ(A Delta) Fibers

Lewerenz, J. & Maher, P. (2015). Chronic Glutamate Toxicity in Neurodegenerative Diseases. Frontiers in Neuroscience, 9, 469.

Deep Dive:
8 Root Causes of Glutamate Excitotoxicity

Why does the system break down in the first place?

Sugar → Excitotoxicity: 4 Mechanisms

High sugar doesn't just cause diabetes — it directly fuels the glutamate death cascade in the brain.

A — High Glucose Disrupts Glutamate Receptors

Long-term exposure to high blood glucose alters AMPA and NMDA receptor expression in neurons, making them progressively more sensitive to glutamate's toxic effects. Neurons exposed to chronic hyperglycemia lose the molecular brakes that normally prevent overactivation.

B — Insulin Resistance Makes Neurons Defenseless

Insulin normally protects neurons against glutamate toxicity. Chronic high sugar → insulin resistance → neurons lose this protection. Research shows hyperinsulinemia directly increases vulnerability to glutamate-induced excitotoxicity in cortical neurons.

C — Sugar Drives Neuroinflammation via Cytokines

High-sugar diets trigger systemic inflammation (NF-κB, TNF-α, IL-6). These cytokines cross into the brain, activate microglia, suppress astrocyte EAAT2 transporter expression, and can cause microglia to release glutamate directly.

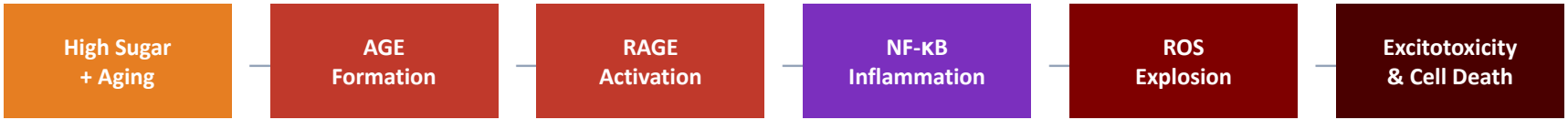
D — Advanced Glycation End Products (AGEs)

Sugar reacts with proteins to form AGEs. AGEs bind RAGE receptors on neurons → NF-κB activation → ROS explosion → neuroinflammation → mitochondrial failure. AGEs accumulate preferentially in glutamatergic neurons — the exact cells most vulnerable to excitotoxicity.

Advanced Glycation End Products (AGEs): Sugar's Brain Damage

What Are AGEs?
AGEs form when sugar molecules bond non-enzymatically to proteins and lipids — a process called glycation. They accumulate with age and accelerate dramatically with chronic high-sugar diets. They are the molecular link between diabetes and neurodegeneration.

The AGE-RAGE Cascade → Neurodegeneration



Key research finding: AGEs accumulate preferentially in glutamatergic pyramidal neurons in cerebral cortical layers most prone to neurodegeneration — meaning the cells most targeted by AGEs are the exact cells most vulnerable to excitotoxicity.

AGEs also: Cross-link tau proteins → neurofibrillary tangles · Promote amyloid-beta aggregation · Damage BBB endothelial cells · Directly inhibit mitochondrial function

The Good News
AGE formation is dramatically slowed by blood sugar control, reduced processed food intake, and antioxidants (vitamin C, E, NAC). Cooking method matters: boiling and steaming create far fewer AGEs than grilling and frying.

Astrocyte Failure & Mitochondrial Collapse: The Core Machinery

Astrocyte Failure: The Broken Janitor

Astrocytes clear ~90% of all synaptic glutamate via EAAT1 and EAAT2 transporter proteins. When these fail, glutamate accumulates no matter how little is released.

Why astrocytes fail:

- Neuroinflammation (TNF-α) downregulates EAAT2
- Oxidative stress damages transporter proteins
- ATP depletion — pumps need energy to run
- Reactive astrogliosis — astrocytes shift from clearing glutamate to releasing it

In ALS, EAAT2 drops by 60% in motor regions. Riluzole works by restoring it.

Mitochondrial Collapse: No ATP = No Defense

Mitochondria are the ultimate protection against excitotoxicity. They buffer calcium and power the glutamate transport pumps. When they fail, both protections disappear simultaneously.

The vicious cycle:

- Glutamate overload floods mitochondria with Ca^{2+}
- mPTP opens → ATP production collapses
- No ATP → EAAT2 pumps stop → more glutamate builds
- More Ca^{2+} floods in → cycle accelerates

Mitochondria fail faster when ATP is already depleted by: sugar crashes, pesticides, B-vitamin deficiency, oxidative stress, and aging.

Chronic Stress, Cortisol & the Gut–Brain Highway

Chronic Stress → Glutamate Overload

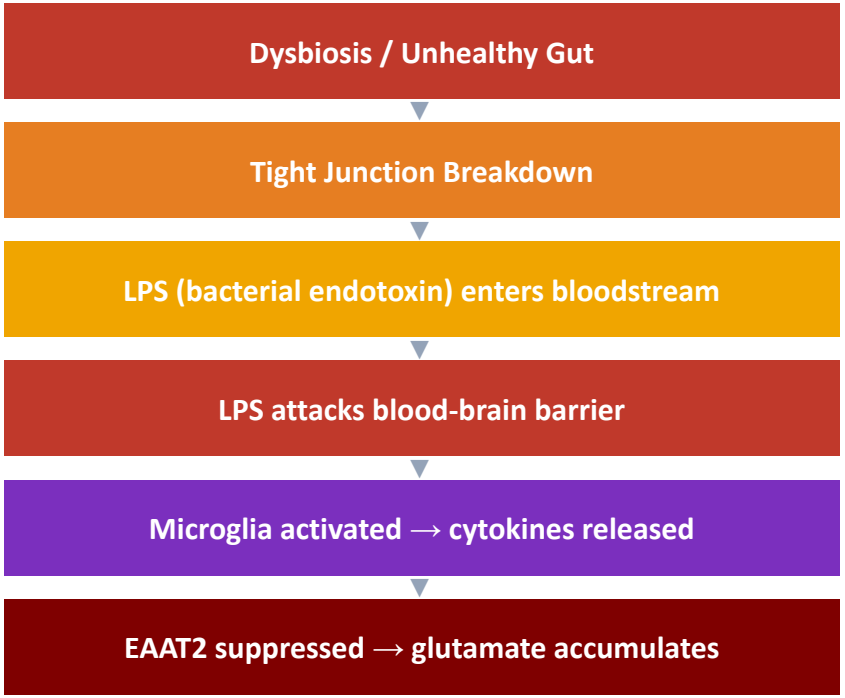
Chronic stress → elevated cortisol → impairs astrocyte glutamate clearance → glutamate spills over to extrasynaptic NMDA receptors (the pro-death receptors) → chronic stress may decrease glutamate-glutamine cycling, reducing glutamate metabolism → neurodegeneration accelerates.

Key insight: Synaptic NMDA receptors promote survival. Extrasynaptic NMDA receptors trigger cell death. Stress specifically pushes glutamate to the deadly location.

Alcohol, Heavy Metals & GABA Deficit

Alcohol damages astrocytes permanently, upregulates NMDA receptors, and depletes GABA. Heavy metals (mercury, lead) poison glutamate transporters and block the magnesium NMDA receptor site. B2/B6 deficiency impairs glutamate-to-GABA conversion — removing the inhibitory brakes that balance excitatory signaling.

The Leaky Gut → Leaky Brain Pathway



What causes leaky gut? The same factors: chronic sugar, processed food, stress, antibiotics, alcohol, NSAIDs.

Common Upstream Vulnerabilities — All Four Zones

These factors weaken ALL FOUR zones simultaneously

Mold / Mycotoxins

Inhibit mitochondria, destroy GLT-1 transporters, produce white matter lesions

Gut Dysbiosis

LPS neuroinflammation, BMAA production, vagal pathway seeding

Mitochondrial Dysfunction

Every vulnerable zone runs high metabolic demand — all four are hit

Glymphatic Failure

Poor sleep = glutamate & protein aggregate CNS accumulation overnight

Neuroinflammation

Microglia release glutamate and downregulate transporters system-wide

Nutritional Depletion

B vitamins, Mg, Zn, CoQ10, omega-3s required for every protective mechanism

Chronic Stress / HPA

Cortisol impairs neurogenesis, degrades BBB, promotes excitotoxicity

Toxin Bioaccumulation

Lipophilic toxins concentrate in lipid-rich neural tissue over decades

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Senergy's Tennant BioModulator®: Supporting Neurological Health

How microcurrent therapy addresses excitotoxicity at the physiological level

What the BioModulator Supports: 5 Core Mechanisms

The BioModulator addresses excitotoxicity through the same physiological systems it targets: ATP production, neuroinflammation, and C fiber signaling.

1

ATP Production +500%

Microcurrents in the 10–500 µA range stimulate cellular ATP production by up to 500% (Cheng et al., 1982). This directly powers the EAAT2 glutamate transporter pumps that keep excitotoxicity at bay.

2

Neuroinflammation Modulation

Microcurrent therapy modulates MAPK signaling pathways — reducing microglial activation, suppressing Iba1 and GFAP expression, and lowering TNF-α and IL-6 that destroy astrocyte transporter function.

3

Cellular Voltage Restoration

Restores membrane potential to optimal range. Cells with restored voltage regulate ion channels normally — reducing pathological NMDA receptor calcium influx.

4

Endorphin & Neuropeptide Release

Stimulates natural release of endorphins, neuropeptides, and nitric oxide. NO promotes vasodilation — improving blood flow to ischemic nerve tissue starved of oxygen and ATP.

5

Adaptive Biofeedback Loop

The device detects tissue electrical response and modifies every subsequent signal. This cybernetic loop between device and living tissue optimizes delivery — distinguishing it from simple TENS devices.

BioModulator Application Across All Four Conditions

ALS

- ATP boost powers surviving motor neuron energy metabolism and axonal transport
- Neuroinflammation reduction decreases microglial attack on remaining neurons
- C fiber pathways remain intact in ALS — provide a functional signaling route to motor circuits
- Muscle fiber preservation: neuromuscular stimulation slows denervation atrophy

Multiple Sclerosis

- ATP restoration in demyelinated tissue helps metabolically stressed axons survive longer
- MAPK pathway modulation shifts microglia from pro-inflammatory to protective state
- C fiber pain modulation normalizes aberrant firing driving MS neuropathic pain
- Parasympathetic shift (measurable via HRV) supports immune regulation in autoimmune disease

Parkinson's

- ATP production supports dopamine neuron mitochondria — the primary failure point in PD
- FDA-listed for Parkinson's disease symptom relief (tremor, rigidity, pain)
- C fiber afferent stimulation engages thalamic circuits — non-invasive circuit modulation
- Non-motor symptom support: autonomic regulation via parasympathetic activation

Neuropathy

- Strongest direct evidence: FDA-cleared for diabetic neuropathy
- Peripheral nerve regeneration: microcurrent promotes ~1mm/day axon regrowth in animal studies
- C fiber normalization: quiets hyperactive pain fibers while reactivating hypoactive sensory fibers
- Nitric oxide release improves microvascular perfusion to ischemic peripheral nerves

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C Fiber Activation: The Physiological Mechanism

Why C fiber targeting is the key to the BioModulator's neurological benefit

What Are C Fibers — and Why Do They Matter?

C Fibers: The Body's Primary Signal Network

C fibers are small, unmyelinated nerve fibers — the most abundant fiber type in the peripheral nervous system. They carry:

- Pain and temperature signals (nociception)
- Autonomic signals (heart rate, digestion, vascular tone)
- Inflammatory chemical signals
- Afferent signals to the spinal cord, thalamus, and cortex

Unlike A-delta fibers (fast, myelinated), C fibers conduct slowly but have enormous reach and connect to regulatory centers throughout the CNS.

Critical fact for ALS and Parkinson's: C fibers remain largely intact even as motor neurons die. They provide a functional signaling highway into the central nervous system when the motor output system is failing.

Fiber Types Compared

Type	Speed	Myelin	Carries
Aα	70–120 m/s	Yes (thick)	Motor, proprioception
Aβ	30–70 m/s	Yes	Touch, pressure
Aδ	5–30 m/s	Thin	Sharp pain, cold
C fibers	**0.5–2 m/s**	**None**	**Slow pain, temp, autonomic**

How the BioModulator Targets C Fibers: The Physics

The Key Discovery: Waveform Shape Determines Which Fibers Are Activated

Research published in Frontiers in Pain Research (2025) confirmed that the shape of the electrical pulse — not just its frequency — determines whether A-delta or C fibers are preferentially stimulated. The BioModulator's proprietary waveforms are engineered around this principle.

Standard TENS / Rectangular Pulse

- 500 µs short rectangular pulses
- Preferentially activates A-delta fibers (fast, myelinated)
- Produces immediate but short-lived analgesia
- Does not engage the deep autonomic C fiber network
- No adaptive biofeedback — same signal regardless of tissue response

Result: Surface pain blocking only. Misses the C fiber regulatory system entirely.

BioModulator: Half-Sine / Frequency-Specific

- 25 ms half-sine slow depolarizing pulses
- Preferentially activates unmyelinated C fibers
- Engages ascending pain inhibition pathways
- Reaches autonomic regulatory networks
- Adaptive biofeedback adjusts every subsequent pulse

Result: Deep modulation of autonomic, pain, and neuroinflammatory circuits.

What C Fiber Activation Does Physiologically

When the BioModulator activates C fibers, these signals travel afferently (inward) through a cascade of physiological effects:

1

Ascending to Thalamus & Cortex

C fiber signals travel via the spinothalamic tract to the thalamus, hypothalamus, and sensorimotor cortex — activating the brain's own pain suppression and autonomic regulation centers.

2

Descending Pain Inhibition (Gate Control)

Activating C fibers engages descending inhibitory pathways from the periaqueductal gray (PAG) and rostral ventromedial medulla — the brain's own pain-off switch. This provides sustained analgesia via endogenous opioid release.

3

Autonomic Nervous System Shift

C fiber afferents are primary regulators of the autonomic nervous system. BioModulator stimulation measurably shifts heart rate variability (HRV) toward parasympathetic dominance — the anti-inflammatory, restorative state.

4

Basal Ganglia Circuit Modulation (Parkinson's)

Ascending C fiber signals reach thalamic relay circuits that feed into the basal ganglia loop. This may interrupt pathological oscillatory firing patterns in Parkinson's — the same circuits targeted by deep brain stimulation, but non-invasively.

5

Neuroinflammation Suppression via Reflex Arcs

Afferent C fiber stimulation activates anti-inflammatory reflex arcs including the cholinergic anti-inflammatory pathway (CAP) — reducing systemic and central neuroinflammation by activating vagal nerve-mediated immune suppression.

6

Motor Circuit Access in ALS

In ALS, motor output is failing — but sensory C fiber input is preserved. BioModulator stimulation uses this intact afferent highway to activate sensorimotor integration circuits, potentially maintaining motor circuit connectivity longer.

The Complete Chain: BioModulator → C Fibers → Reduced Excitotoxicity

The Unified Mechanism

The BioModulator addresses glutamate excitotoxicity not by directly blocking glutamate receptors, but by restoring the physiological systems that the body uses to regulate glutamate — making it a systems-level intervention rather than a single-target drug.

Track 1: C Fiber Pathway → Neuroinflammation Suppression → Glutamate Clearance

BioModulator Microcurrent → C Fiber Activation → Afferent Signaling CNS → Parasympathetic Dominance → Neuroinflam-mation ↓ → EAAT2 Restored → Glutamate Cleared → Excitotoxicity Reduced

Track 2: ATP Pathway → Membrane Voltage → NMDA Receptor Protection

BioModulator Microcurrent → +500% ATP → Ion Pumps Powered → Membrane Voltage ↑ → NMDA Channel Mg^{2+} Block ↑ → Ca^{2+} Influx Reduced → Mitochondria Protected → Excitotoxicity Reduced

Why This Matters for Neurological Disease

Drugs like riluzole address excitotoxicity at one point: blocking glutamate release or restoring EAAT2. The BioModulator works simultaneously across the entire excitotoxic cascade — boosting ATP to power transport pumps, reducing neuroinflammation that destroys those pumps, restoring cellular voltage that governs NMDA receptor sensitivity, and using C fiber pathways to engage the body's own anti-inflammatory and pain-suppression systems. It supports the system rather than targeting a single molecule.

Key Takeaways

1. Glutamate excitotoxicity is a shared final death pathway in ALS, MS, Parkinson's disease, and neuropathy — not a side effect, but a primary driver of neuronal death.

2. The root causes are upstream and lifestyle-modifiable: high sugar/AGEs, astrocyte failure, mitochondrial dysfunction, chronic stress, leaky gut, heavy metals, alcohol, and B vitamin deficiency.

3. Sugar causes excitotoxic vulnerability through 4 mechanisms: receptor disruption, insulin resistance, neuroinflammation, and AGE-RAGE signaling. Diet is neurological medicine.

4. Mitigation is multi-target: magnesium, NAC, omega-3, B vitamins, gut healing, and exercise each address different points in the excitotoxic cascade with solid mechanistic evidence.

5. The BioModulator addresses glutamate excitotoxicity at a systems level: boosting ATP (powers glutamate transport), reducing neuroinflammation (protects EAAT2), and restoring cellular voltage (governs NMDA sensitivity).

6. C fiber activation is the bridge: intact in ALS and Parkinson's even as motor neurons die, C fibers carry afferent signals that suppress neuroinflammation, shift autonomic balance, and engage pain-inhibition circuits — a non-invasive systemic intervention.

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PART 2

ALS

Amyotrophic Lateral Sclerosis

Motor Neuron Disease & the Glutamate Connection

ALS: What Is It?

ALS (Lou Gehrig's Disease) is a progressive fatal neurodegenerative disease destroying both upper motor neurons (motor cortex) and lower motor neurons (spinal cord and brainstem), eliminating all voluntary muscle control. It affects speaking, swallowing, walking, and ultimately breathing.

- 'Amyotrophic' = muscle wasting from loss of nerve supply
- 'Lateral' = damage in lateral columns of the spinal cord
- Both upper (cortex → spinal cord) and lower (spinal cord → muscle) motor neurons progressively die
- Respiratory failure is the primary cause of death — typically 2–5 years from diagnosis
- No cure; riluzole (reduces glutamate release) is primary pharmacological therapy — confirming the glutamate mechanism
- 90–95% of cases sporadic — no identified family history

~32,000

Americans living with ALS

5,000+

new U.S. diagnoses per year

2–5 yrs

median survival from diagnosis

90–95%

of cases sporadic — no family history

National ALS Registry (2023). CDC. | Mehta, P. et al. (2023). Prevalence of ALS. ALS & Frontotemporal Degeneration, 24(1–2), 108–118.

ALS: The Glutamate Mechanism

GLT-1 Transporter Shutdown

In ALS, astrocytes progressively lose GLT-1 glutamate transporter expression — the primary mechanism for clearing glutamate from synapses. The cleanup system shuts down. Riluzole's mechanism of action (reducing glutamate release) confirms this is central to disease (Rothstein et al., 1995).

Calcium-Permeable AMPA Receptors

Motor neurons lack the GluR2 subunit that normally blocks calcium entry through AMPA receptors. Every glutamate stimulus floods calcium in unrestricted. Most neurons in the brain have this protection — motor neurons do not, making them uniquely vulnerable (Kawahara et al., 2004).

Zero Calcium Buffering

Calbindin and parvalbumin absorb excess calcium in most neurons. Motor neurons have virtually none — there is no safety net. Calcium accumulates rapidly after every excitatory signal. This single structural factor may be the primary explanation for their selective destruction.

Corticospinal Glutamate Overdrive

Upper motor neurons continuously pour glutamate onto lower motor neurons via the corticospinal tract. This represents an enormous continuous excitatory load — lower motor neurons are always operating near their calcium threshold, even in health, with no margin for error.

Bensimon, G. et al. (1994). Riluzole in ALS. NEJM, 330, 585–591. | Rothstein, J.D. et al. (1995). Loss of glial GLT-1 in ALS. Annals of Neurology, 38, 73–84.

ALS: Voltage & Cellular Energy Connection

Why Voltage Matters

Motor neurons are the largest and most metabolically demanding neurons in the body. They require enormous, continuous ATP production to maintain membrane potential and fire action potentials along axons that may exceed one meter in length.

When cellular voltage drops — from toxin exposure, mitochondrial impairment, or nutritional deficit — the Na+/K+ ATPase pump that clears glutamate from motor neuron synapses begins to fail. Glutamate accumulates. The cascade begins.

This is not fundamentally a 'glutamate disease.' It is a voltage and energy failure that manifests through glutamate accumulation.

Mitochondrial complex I dysfunction is found consistently in ALS motor neurons — explaining why rotenone, BMAA, trichothecenes, and heavy metals (all complex I inhibitors) are ALS environmental risk factors.

Restoring cellular voltage and mitochondrial function is the foundational supportive approach.

-70mV

Healthy motor neuron resting potential

ATP

Must have for basic cellular function

1m+

Longest motor neuron axon length

Grosskreutz, J. et al. (2010). Calcium dysregulation in ALS. Cell Calcium, 47, 165–174. .

ALS: Why Motor Neurons Are Vulnerable

Enormous Metabolic Demand

Betz cells are among the largest neurons in the CNS. Greater size = greater metabolic demand = greater vulnerability when energy supply is disrupted. They run near maximum mitochondrial output continuously at baseline.

No Calcium Buffers

Calbindin and parvalbumin act as calcium sponges in most neurons. Motor neurons have virtually none — calcium accumulates rapidly. This single factor may explain their selective vulnerability in ALS (Alexianu et al., 1994).

Longest Axons in the Body

Motor axons can exceed 1 meter. Mitochondria must be physically transported this entire distance. Transport failure starves distal portions of energy and severs trophic feedback from the target muscle.

No GluR2 Calcium Gate

Motor neurons lack the GluR2 subunit of AMPA receptors that blocks calcium entry in other neurons. Their AMPA receptors are calcium-permeable by design — manageable normally, lethal under excitotoxic conditions.

Sparse GLT-1 Baseline

Astrocytes in motor regions express lower GLT-1 levels than other brain areas even before disease. The glutamate cleanup system starts already underpowered — leaving minimal reserve when disease begins.

Trophic Factor Dependence

Motor neurons depend on retrograde neurotrophic signals (BDNF, GDNF, IGF-1) from target muscles. As disease advances and muscles weaken, this survival signal is lost — accelerating degeneration in a destructive feedback loop.

Kawahara, Y. et al. (2004). RNA editing and death of motor neurons. Nature, 427, 801. | Alexianu, M.E. et al. (1994). Calcium-binding proteins in ALS. Annals of Neurology.

ALS: What Weakens the Motor Neuron Zone

Heavy Metals

Mercury directly inhibits GLT-1 glutamate transporters — the exact ALS mechanism. Lead disrupts neurofilament phosphorylation. Cadmium impairs SOD1 (most-studied ALS gene product). Arsenic impairs mitochondrial complex I/III. Sources: dental amalgam, contaminated fish, occupational exposure (Roos et al., 2012).

BMAA — Cyanobacterial Neurotoxin

Beta-methylamino-L-alanine is a false glutamate receptor substrate causing chronic NMDA overactivation and protein misfolding. Found in cyanobacteria blooms and in dysbiotic gut microbiomes. Linked to ALS clusters in Guam, coastal New England, and golf courses (Cox et al., 2016).

Mycotoxins — Mold Exposure

Ochratoxin A (Aspergillus/Penicillium) crosses the BBB and accumulates in motor cortex, inhibiting protein synthesis in the highest-demand neurons. Trichothecenes (Stachybotrys) directly inhibit mitochondrial protein synthesis. Sources: water-damaged buildings, moldy food and grain.

Military Service & Elite Athletics

Consistently among the strongest ALS risk factors. Combined heavy metal exposure, pesticides, TBI microtrauma, extreme exertion, and infection all prime motor neurons for failure. Elite soccer and American football athletes show elevated risk — pesticide-treated fields plus repetitive head trauma (Horner et al., 2008).

Glymphatic Failure / Chronic Poor Sleep

The glymphatic system flushes glutamate and misfolded proteins from motor cortex during deep sleep via CSF circulation. Chronic sleep deprivation impairs this overnight cleanup — glutamate and TDP-43 aggregates accumulate progressively. Lateral sleep position improves glymphatic flow (Iliff et al., 2012).

Gut Dysbiosis, LPS & Cervical Compression

Dysbiotic LPS crosses a leaky gut, compromises the BBB, and activates motor cortex microglia. Separately, chronic cervical stenosis creates ischemic mechanical stress at the anterior horn — priming lower motor neurons for degeneration before any systemic disease begins.

Roos, P.M. et al. (2012). Metal concentrations in ALS. PLOS ONE. | Cox, P.A. et al. (2016). BMAA and ALS. Neurology. | Iliff, J.J. et al. (2012). Paravascular pathway. Science Translational Medicine.

ALS: Integrative & Lifestyle Support

Evidence-informed lifestyle approaches addressing underlying vulnerability factors — for educational discussion only.

Sleep Optimization — Glymphatic Repair

7–9 hrs minimum. Lateral sleep position improves glymphatic flow. Avoid alcohol and blue light 2 hrs before bed. Treat sleep apnea — it devastates overnight clearance of glutamate and TDP-43 from motor cortex.

Structured Exercise — Trophic Support

Moderate aerobic exercise upregulates BDNF and GDNF — primary motor neuron survival factors. Avoid exhaustive overtraining (increases ROS). Resistance + aerobic combination preferred. Aquatic therapy reduces loading while maintaining afferent input.

Vagal Activation & Stress Reduction

Chronic cortisol accelerates motor neuron vulnerability. Meditation, breathwork, cold exposure, and humming activate vagal tone. Reduces HPA-driven excitotoxicity and supports the parasympathetic state essential for neural repair.

Anti-Inflammatory Nutrition

Mediterranean-pattern diet. Eliminate processed seed oils (omega-6 excess). Increase omega-3 EPA/DHA (3–4g daily). Colorful vegetables support Nrf2 antioxidant pathways. Ketogenic diet may reduce glutamate excitotoxicity via altered brain fuel.

Environmental Toxin Assessment

Hair/urine heavy metal testing. Biological dentist assessment for amalgam burden. ERMI mold testing of home and workplace. Assess agricultural pesticide exposure. Filter water for BMAA, heavy metals, and chlorination byproducts.

Cervical & Structural Assessment

Evaluate for cervical stenosis — anterior horn ischemia reduction is direct motor neuron support. Fascial release to improve tissue perfusion. Chiropractic or osteopathic evaluation of mechanical stress along motor pathways.

ALS: Supplement Suggestions

MyCell® Stabilized Micelle Technology — ultra-fine water-dispersible micelles for near-complete cellular absorption. Educational purposes only.

MITACELL WELLNESS available on Senergy.us

CoQ10

Mitochondrial complex I–III support. Motor neurons need maximum mitochondrial output. MyCell® CoQ10 achieves near-complete absorption vs. conventional CoQ10's 3–8% bioavailability. Addresses energy deficit directly.

Glutathione

Primary antioxidant defense against motor neuron ROS burden from continuous firing. MyCell® glutathione bypasses GI degradation — conventional oral glutathione is largely destroyed before absorption.

Curcumin

NF-κB inhibitor reducing microglial activation. Also supports GLT-1 glutamate transporter expression in astrocytes. MyCell® achieves dramatically higher plasma levels than standard formulations.

NMN Spermidine

NMN restores NAD+ for mitochondrial repair. Spermidine induces autophagy, clearing misfolded TDP-43 and SOD1 aggregates that drive ALS progression at the molecular level.

Frankincense

Boswellic acids cross the BBB and reduce motor cortex neuroinflammation via leukotriene synthesis inhibition — directly reducing microglial M1 activation and associated glutamate release.

C3 Cellular Repair

Vitamin C + Curcumin + Frankincense in MyCell® delivery. Addresses antioxidant defense, neuroinflammation, and cellular repair simultaneously — foundational combination for high-demand motor neuron support.

**Not evaluated by FDA. Not intended to diagnose, treat, cure, or prevent any disease. Consult your healthcare provider.*

ALS: Biomodulator® — Physiological Rationale

Educational Note: The following discusses physiological mechanisms only. The BioModulator® is not claimed to treat, cure, or prevent any neurological disease.

Inhibitory Peptide Release

C fiber stimulation triggers endogenous opioids, galanin, and neuropeptide Y — inhibiting presynaptic glutamate release at dorsal horn and supraspinal levels, reducing the excitatory drive ascending toward motor cortex (Yaksh, 1989).

GABAergic Restoration

Adaptive afferent input engages inhibitory GABAergic interneurons progressively lost in ALS. Restoring GABAergic tone directly counterbalances the glutamate excess driving motor neuron death. Interneuronal loss is increasingly recognized in ALS pathophysiology (Turner & Kiernan, 2012).

Neurotrophic Factor Upregulation

Peripheral afferent stimulation upregulates BDNF and GDNF — the precise trophic factors motor neurons progressively lose as ALS advances. Trophic support delays motor neuron degeneration in preclinical models (Henderson et al., 1994).

Adaptive Anti-Windup Mechanism

Fixed-frequency stimulation risks driving neurons into sustained depolarization — worsening excitotoxicity. Every BioModulator® impulse adapts to tissue response, avoiding repetitive firing that causes glutamate windup in motor circuits. It reads and responds — never just drives.

Yaksh, T.L. (1989). Behavioral and autonomic correlates. Pain. | Henderson, C.E. et al. (1994). GDNF as neurotrophic factor. Science, 266, 1062–1064.

ALS: Key C Fiber Stimulation Zones

Zones based on proximity to motor neuron pathways, corticospinal tract access, and trophic feedback circuits via ascending afferent neuroanatomy.

1. **Cervical Spine (C3–C7)**
   Upper motor neuron pathway & corticospinal modulation
2. **Trapezius / Scalenes**
   Vagal nerve proximity — cholinergic anti-inflammatory
3. **Thoracic Paraspinals (T1–T6)**
   Sympathetic chain — reduces excitatory tone to spinal cord
4. **Lumbar Spine (L1–L4)**
   Lower motor neuron zone — anterior horn C fiber access
5. **Forearm / Hand**
   Distal motor nerve territory — trophic signal feedback
6. **Lower Leg / Foot**
   Longest motor axon territory — restores distal trophic loop

PART 3

Multiple Sclerosis

White Matter Disease & the Inflammatory Glutamate Loop

When the Immune System Becomes the Excitotoxic Source

MS: What Is It?

Multiple Sclerosis is a chronic autoimmune disease in which the immune system attacks myelin — the protective sheath produced by oligodendrocytes that insulates nerve fibers and enables rapid electrical signal conduction throughout the CNS. Without myelin, signals slow, fail, or misfire.

- Myelin is produced by oligodendrocytes — the cells MS preferentially destroys
- Without myelin, nerve impulses slow 50–90% — causing the neurological symptoms of MS
- Relapsing-remitting MS: inflammatory attacks cause episodic relapses with partial recovery
- Progressive MS: axonal loss becomes permanent and recovery capacity is exhausted
- Glutamate excitotoxicity from immune cell glutamate release is now central to understanding oligodendrocyte death
- Women affected 3× more than men — hormonal and immune factors intersect

~1M

Americans living with MS

3:1

Women to men diagnosis ratio

20–40

Typical age of onset

2.8M

People affected worldwide

National MS Society (2023). | Wallin, M.T. et al. (2019). Prevalence of MS in the United States. Neurology. | Matute, C. et al. (2007). Glutamate-mediated damage. Glia, 55.

MS: The Glutamate Mechanism

Immune Cell Glutamate Release

Activated microglia and infiltrating macrophages/T cells release large amounts of glutamate directly into white matter as part of the inflammatory attack — far beyond what GLT-1 transporters can clear within plaques. In MS, inflammation IS the glutamate source.

Oligodendrocyte AMPA Sensitivity

Oligodendrocytes express dense AMPA and kainate receptors with very poor calcium buffering. When glutamate floods in, calcium overwhelms them and they die — stripping axons of both insulation and lactate energy supply (Káradóttir et al., 2005).

Reverse Na/Ca Exchanger

Denuded axons become chronically depolarized without myelin. This drives the Na/Ca exchanger in reverse — the axon imports calcium directly from outside, independent of any receptor. Self-loading excitotoxic calcium entry continuing indefinitely.

GLT-1 Downregulation in Plaques

Astrocytes within MS plaques progressively lose GLT-1 expression — exactly as in ALS. Glutamate accumulates in inflammatory lesions with no clearance mechanism. Immune attack and transporter failure compound each other in a sustained loop.

Srinivasan, R. et al. (2016). Glutamate transporter GLAST in white matter. PLOS ONE. | Káradóttir, R. et al. (2005). NMDA receptors in oligodendrocytes. Nature, 438, 1162.

MS: Voltage & Cellular Energy Connection

Why Voltage Matters

Myelin is not simply insulation — it is an active metabolic support structure. Oligodendrocytes provide axons with lactate, the primary energy substrate for sustaining action potential firing over long myelinated segments.

When oligodendrocytes die and myelin is stripped, axons lose both electrical insulation AND metabolic support simultaneously. Denuded axons redistribute sodium channels abnormally, leading to persistent membrane depolarization — a state of chronically reduced cellular voltage.

Depolarized axons operate like a dying battery. Every sodium channel leak requires extra ATP to correct. The cell cannot maintain ionic homeostasis. Calcium enters through reversed exchangers and activated NMDA receptors simultaneously.

In progressive MS, this energy crisis becomes the dominant driver of axon loss — even without new inflammatory attacks. Restoring cellular voltage and mitochondrial energy production addresses the mechanism of progressive disability.

50–90%

Signal slowing
in denuded axons

ATP

Required
for myelin repair

50×

Metabolic cost increase
in denuded axons

Nave, K.A. (2010). Myelination and axonal integrity. Nature, 468, 244. | Trapp, B.D. & Stys, P.K. (2009). Virtual hypoxia and necrosis. Lancet Neurology.

MS: Why Oligodendrocytes Are Vulnerable

Extreme Metabolic Demand

Each oligodendrocyte simultaneously myelinates up to 50 axon segments — among the most metabolically active cells in the CNS. Running near maximum mitochondrial output at all times; any energy disruption is immediately life-threatening to the cell.

High Iron + Low Glutathione

Oligodendrocytes require high iron for myelin synthesis — but iron under oxidative stress catalyzes the Fenton reaction. They also have the lowest glutathione levels in the CNS relative to their oxidative burden. Iron-rich and defense-poor.

Dense AMPA Receptor Expression

High densities of calcium-permeable AMPA and kainate receptors with minimal calcium buffering. When immune cells flood white matter with glutamate, oligodendrocytes experience fatal calcium overload before neighboring neurons are even affected.

Limited Regenerative Capacity

Mature oligodendrocytes cannot regenerate. Remyelination depends on precursor cells (OPCs) that become progressively exhausted over the course of disease — each relapse causes slightly less complete recovery than the last.

Sparse White Matter Vascularity

White matter has proportionally less vascularity than gray matter — slower oxygen delivery, slower metabolite clearance, and less robust response to metabolic stress in the exact region most affected by MS plaques.

Gap Junction Injury Spread

White matter astrocytes are connected by gap junctions. During inflammation, injury signals — calcium waves, ROS — spread through this network and can amplify damage across large white matter regions far from the primary plaque.

Káradóttir, R. et al. (2005). NMDA receptors in oligodendrocytes. Nature, 438. | Irvine, K.A. & Blakemore, W.F. (2008). Remyelination protects axons. Brain.

MS: What Weakens the White Matter Zone

EBV — Molecular Mimicry

A landmark military cohort study found EBV seroconversion preceded MS in virtually all cases. EBV proteins structurally resemble myelin basic protein — once the immune system learns to attack EBV, it attacks myelin as a bystander. The single most replicated MS environmental risk factor (Bjornevik et al., 2022, Science).

Vitamin D Deficiency

Vitamin D3 directly promotes oligodendrocyte survival and myelin gene expression beyond immune modulation. The north-latitude MS gradient maps almost perfectly to sunlight availability. Deficiency during pregnancy and childhood creates developmental vulnerability that persists into adult life (Munger et al., 2006, JAMA).

Mycotoxins — Satratoxins, Trichothecenes

Trichothecenes and satratoxins from Stachybotrys produce white matter MRI lesions radiographically indistinguishable from MS plaques. Geographic overlap between high indoor mold prevalence and high MS prevalence regions is striking and largely unexplored in clinical practice.

Thyroid Disruption — T3 Deprivation

T3 (triiodothyronine) is one of the most potent oligodendrocyte survival and maturation signals known. Environmental thyroid disruptors — PFASs, brominated flame retardants, perchlorate in water — chronically deprive oligodendrocytes of this essential survival signal.

Gut Dysbiosis & Intestinal Permeability

Loss of Akkermansia muciniphila and Lactobacillus strains depletes short-chain fatty acids that maintain BBB integrity. Autoreactive T cells escape the gut and access white matter. Gut microbiome composition differs significantly between MS patients and healthy controls (Berer et al., 2011, Nature).

Melatonin Deficiency & Omega-6 Excess

Melatonin has direct oligodendrocyte protective effects — MS relapse rates cluster in spring/summer (lowest melatonin). Excess dietary omega-6 (seed oils) provides inflammatory lipid substrate for myelin synthesis. Myelin quality reflects the dietary lipid environment over months.

Bjornevik, K. et al. (2022). EBV and multiple sclerosis. Science, 375, 296–301. | Berer, K. et al. (2011). Gut microbiota and MS. Nature, 479, 538–541.

MS: Integrative & Lifestyle Support

Evidence-informed lifestyle approaches addressing underlying vulnerability factors — for educational discussion only.

Vitamin D3 + K2 Optimization

Target 25-OH-D levels 60–80 ng/mL under physician supervision. D3 promotes oligodendrocyte survival and immune tolerance. K2 (MK-7) prevents soft tissue calcification from higher D3 dosing. Test every 6 months.

Omega-3 — Myelin Lipid Quality

EPA and DHA provide anti-inflammatory myelin lipid substrate. Target 3–4g EPA+DHA daily. Replace seed oils (omega-6 excess) with olive oil and animal fats. Myelin quality reflects dietary lipid composition over months.

Melatonin & Light Hygiene

Melatonin has direct oligodendrocyte protective effects. Block blue light after 8pm. Blackout curtains. Low-dose melatonin (0.5–1mg) 60 min before sleep. Directly addresses the spring/summer MS relapse cluster pattern.

Gut Microbiome Restoration

Prioritize Akkermansia muciniphila and Lactobacillus via fermented foods, prebiotic fiber, and targeted probiotics. Test for intestinal permeability. Address SIBO if present. The gut-brain axis is central to MS immune dysregulation.

Mold & Environmental Assessment

ERMI mold testing of home and workplace. Remediation of water-damaged materials. HEPA air filtration. Binders under physician guidance for mycotoxin reduction. Consider mycotoxin urine testing (Great Plains or RealTime Labs).

Thyroid & Hormonal Optimization

Full thyroid panel including Free T3 — ensure T3 is in upper quartile for oligodendrocyte support. Test for PFAS and halide burden. Estrogen and progesterone optimization in women — both are directly myelinoprotective.

MS: Supplement Suggestions

MyCell® Stabilized Micelle Technology — ultra-fine water-dispersible micelles for near-complete cellular absorption. Educational purposes only.

MITACELL WELLNESS USA — senergy.us

Vitamin C (CoVitC)

High-dose vitamin C via MyCell® supports BBB integrity, reduces inflammatory cytokine production, and supports collagen in myelin support structures. Far superior absorption to conventional ascorbic acid which saturates at 200mg.

Curcumin

NF-κB inhibition reduces immune-cell glutamate release in MS plaques. Also promotes OPC survival. MyCell® achieves 185× higher plasma levels than conventional curcumin — the difference between active and inactive at target white matter tissue.

Glutathione

Oligodendrocytes have the lowest CNS glutathione levels. MyCell® glutathione reaches these cells directly — addressing the fundamental antioxidant deficit that makes oligodendrocytes so vulnerable to immune-generated reactive oxygen species.

CoQ10

Mitochondrial support for denuded axons operating at 50× normal energy cost. MyCell® CoQ10 addresses the energy crisis of progressive MS — the primary driver of irreversible disability beyond the inflammatory phase.

Frankincense

Boswellic acids cross the BBB and modulate T cell activity, specifically inhibiting leukotriene synthesis involved in the autoimmune myelin attack. Clinical studies show benefit in MS-related cerebral edema and neuroinflammation.

Reishi (Ganoderma)

Powerful immune modulator supporting Treg activity that maintains immune tolerance to myelin antigens. Also supports gut barrier integrity — addressing the leaky gut that allows autoreactive T cells to access white matter.

**Not evaluated by FDA. Not intended to diagnose, treat, cure, or prevent any disease. Consult your healthcare provider.*

MS: Biomodulator® — Physiological Rationale

Educational Note: The following discusses physiological mechanisms only. The BioModulator® is not claimed to treat, cure, or prevent any neurological disease.

Microglial M1→M2 Modulation

Normalized afferent C fiber signaling can shift microglia from pro-inflammatory M1 to homeostatic M2 phenotype — reducing the primary source of glutamate release within MS plaques. Microglial state is highly sensitive to afferent signaling tone (Hanisch & Kettenmann, 2007).

Cholinergic Anti-Inflammatory Path

C fiber stimulation activates the vagal cholinergic anti-inflammatory pathway — reducing TNF-alpha, IL-1beta, and IL-6, the cytokines driving GLT-1 downregulation and sustained inflammatory glutamate release in white matter (Tracey, 2002).

Spasticity Circuit Normalization

MS-related spasticity involves hyperactive excitatory circuits. Adaptive C fiber stimulation engages GABAergic interneurons and dorsal horn inhibitory pathways — addressing spasticity through excitatory/inhibitory rebalancing rather than pharmacological blockade.

Remyelination Trophic Support

BDNF and NGF support oligodendrocyte precursor (OPC) survival and differentiation into mature myelinating cells. Afferent stimulation-associated trophic factor upregulation may support the remyelination capacity that progressive MS progressively exhausts (Cohen et al., 2014).

Hanisch, U.K. & Kettenmann, H. (2007). Microglia. Nature Neuroscience. | Tracey, K.J. (2002). The inflammatory reflex. Nature, 420, 853.

MS: Key C Fiber Stimulation Zones

Zones selected for vagal pathway proximity, sympathetic chain access, and ascending afferent pathways relevant to neuroinflammation and immune modulation.

1. **Occiput / Cervical Base**
   Direct vagal access — cholinergic anti-inflammatory reflex
2. **Anterior Neck (Vagus)**
   Primary route of vagal anti-inflammatory pathway activation
3. **Upper Thoracic (T1–T4)**
   Sympathetic outflow to immune organs — thymus and spleen
4. **Thoracolumbar Junction**
   Autonomic rebalancing — parasympathetic tone restoration
5. **Forearm / Radial Nerve**
   Peripheral afferent input engaging anti-inflammatory pathways
6. **Plantar Foot**
   High C fiber density — powerful ascending modulatory input

PART 4

Parkinson's Disease

Dopamine Circuit Failure & the Self-Amplifying Loop

Why the Substantia Nigra Falls First

Parkinson's: What Is It?

Parkinson's disease is a progressive neurodegenerative disorder caused by selective loss of dopaminergic neurons in the substantia nigra pars compacta — a midbrain region essential for smooth, coordinated voluntary movement. It is the second most common neurodegenerative disease worldwide.

- Cardinal motor symptoms: resting tremor, rigidity, bradykinesia (slowness), postural instability
- Non-motor prodrome: anosmia (smell loss), constipation, REM sleep behavior disorder — appear years before motor onset
- Hallmark pathology: alpha-synuclein aggregates (Lewy bodies) in and around dopamine neurons
- Dopamine modulates basal ganglia circuits — without it, a runaway glutamate loop destroys remaining neurons
- By diagnosis, 60–70% of nigral dopamine neurons are already gone
- The gut-brain axis is now considered the likely disease origin (Braak et al., 2003)

1M+

Americans with Parkinson's disease

90,000

new U.S. diagnoses per year

60–70%

neurons lost before diagnosis

2×

global prevalence increase since 1990

Parkinson's Foundation (2023). Statistics. | Dorsey, E.R. et al. (2018). The Parkinson pandemic. Journal of Parkinson's Disease, 8, S3–S8.

Parkinson's: The Glutamate Mechanism

STN Disinhibition — The Trigger

Dopamine normally suppresses the subthalamic nucleus (STN) via the indirect basal ganglia pathway. Without dopamine, STN is released from inhibition and fires pathologically — flooding the globus pallidus and remaining nigral neurons with glutamate. DBS of the STN works precisely because it interrupts this.

The Self-Amplifying Loop

Glutamate from hyperactive STN kills more nigral dopamine neurons → fewer dopamine neurons → more STN disinhibition → more glutamate → more death. Each cycle accelerates the next. This explains Parkinson's progressively accelerating natural history (Blandini et al., 1996).

L-Type Calcium Channel Pacemaking

Nigral neurons are autonomous pacemakers firing using L-type Cav1.3 calcium channels without any synaptic input, creating chronic calcium cycling and oxidative stress. Isradipine (an L-type Ca channel blocker) is in clinical trials for PD based on this exact mechanism (Chan et al., 2007).

Alpha-Synuclein & Complex I Failure

As nigral neurons accumulate alpha-synuclein aggregates (Lewy bodies), mitochondrial complex I is progressively inhibited — the same target as rotenone and MPTP, two compounds that reliably produce PD in models. Complex I failure starves pacemaker neurons of their survival energy.

Blandini, F. et al. (1996). Glutamate and Parkinson's disease. Molecular Neurobiology, 12, 73–94. | Chan, C.S. et al. (2007). Rejuvenation protects neurons. Nature, 447, 1081.

Parkinson's: Voltage & Cellular Energy Connection

Why Voltage Matters

The substantia nigra pars compacta may be the most voltage-stressed tissue in the nervous system. Dopamine neurons are autonomous pacemakers — they fire continuously using L-type calcium channels without waiting for synaptic input. They never rest.

This pacemaking requires continuous reliable ATP production. When mitochondrial complex I is impaired — by pesticides, solvents, mycotoxins, or alpha-synuclein aggregates — cellular voltage drops.

As voltage falls, the autonomous pacemaker rhythm becomes erratic. Calcium influx becomes dysregulated. The cell's ability to maintain membrane potential fails. Glutamate from the disinhibited STN then exploits this already-compromised neuron.

Dopamine synthesis itself requires high-energy biochemistry. Low cellular voltage impairs dopamine production — accelerating the cascade. The nigra loses its voltage supply and its product simultaneously.

Restoring mitochondrial function, reducing iron-mediated ROS, and supporting complex I activity are foundational supportive approaches.

100%

of nigral pacemakers
run Ca channels continuously

10×

higher ROS production
vs. other brain regions

−55mV

nigral neuron resting
potential (borderline)

Chan, C.S. et al. (2007). 'Rejuvenation' protects neurons. Nature, 447, 1081–1086. | Schapira, A.H.V. (1994). Mitochondrial dysfunction in PD. Movement Disorders.

Parkinson's: What Weakens the Nigral Zone

Rotenone, Paraquat & Agricultural Pesticides

Rotenone inhibits mitochondrial complex I — the precise nigral vulnerability — and perfectly replicates PD pathology in animal models. Paraquat is structurally similar to MPTP, a proven PD-causing toxin. Combined paraquat + maneb exposure multiplies PD risk dramatically. Rural living and well water are consistent risk factors (Tanner et al., 2011).

Trichloroethylene (TCE) & Industrial Solvents

TCE has remarkable specificity for nigral neurons — metabolizing to trichloroacetic acid which inhibits complex I. Military bases with TCE-contaminated groundwater show statistically elevated PD clusters. Dry cleaning, degreasing, and aerospace industry exposure represent a largely unrecognized PD driver (Goldman et al., 2023, Annals of Neurology).

Gut-Brain Axis — The Braak Hypothesis

Alpha-synuclein misfolding begins in the enteric nervous system — triggered by gut dysbiosis, H. pylori, and mycotoxins including gliotoxin (Aspergillus). Misfolded protein travels retrogradely up the vagus nerve to the brainstem and substantia nigra years before motor symptoms appear. The gut may be PD's true origin (Braak et al., 2003).

Head Trauma & Neuroinflammation

Single severe TBI or repeated subconcussive impacts trigger alpha-synuclein release and sustained neuroinflammation in nigral pathways. Muhammad Ali's Parkinson's following career-long head impacts is the iconic clinical example. The nigra's vulnerability to trauma-driven neuroinflammation is well established.

Iron Accumulation & Lowest Glutathione

The substantia nigra has the highest brain iron concentration AND the lowest glutathione antioxidant levels of any brain region. Iron catalyzes the Fenton reaction generating hydroxyl radicals. Additional iron from diet, chronic inflammation, or hemochromatosis pushes this into catastrophic oxidative territory.

Olfactory Route & REM Sleep Disorder

The olfactory bulb connects directly to nigral circuits via short pathways, bypassing the BBB entirely. Loss of smell is PD's earliest symptom — potentially marking the actual entry route for environmental initiating factors. REM sleep behavior disorder is the strongest prodromal PD marker, appearing decades before motor symptoms.

Tanner, C.M. et al. (2011). Rotenone, paraquat and Parkinson's. Environmental Health Perspectives, 119, 866–872. | Goldman, S.M. (2023). TCE and Parkinson's. Annals of Neurology.

Parkinson's: Integrative & Lifestyle Support

Evidence-informed lifestyle approaches addressing underlying vulnerability factors — for educational discussion only.

Gut Microbiome Restoration

Address the disease's likely origin. Restore Akkermansia, Lactobacillus, Bifidobacterium. Test for H. pylori and SIBO. Treat intestinal permeability. High-fiber prebiotic diet. Consider comprehensive stool analysis targeting ENS health.

Olfactory & Sinus Health

Given the olfactory BBB bypass route, nasal mold colonization and sinus inflammation may deliver initiating insults directly to nigral circuits. Nasal rinses, anti-fungal protocols where indicated, HEPA filtration in sleep environment.

Iron & Oxidative Load Reduction

Reduce dietary iron excess. Test ferritin (optimal 50–80 ng/mL). Avoid cast iron cooking if iron-replete. Lactoferrin chelates gut iron before absorption. High-polyphenol diet reduces Fenton reaction-mediated ROS in the nigra.

Circadian Rhythm Restoration

Dopamine synthesis is clock-dependent. Consistent sleep/wake cycles, morning bright light exposure (10,000 lux, 20 min), evening darkness. Night shift work and chronic jet lag are independent PD risk factors.

Vigorous Exercise — Neuroprotection

The strongest evidence-based supportive intervention in PD. High-intensity aerobic exercise upregulates GDNF, supports dopaminergic pathway integrity, and reduces STN hyperactivity. Boxing, dance, cycling, and treadmill programs show documented clinical benefit.

TCE & Pesticide Exposure Assessment

Detailed occupational and residential history for TCE, organochlorines, paraquat. Urine pesticide metabolite testing. Well water assessment in agricultural areas. Far-infrared sauna (3×/week) supports lipophilic toxin elimination via sweat.

Parkinson's: MitaCell Supplement Protocol

MyCell® Stabilized Micelle Technology — ultra-fine water-dispersible micelles for near-complete cellular absorption. Educational purposes only.

🔬 **MITACELL WELLNESS USA — mitacellusa.com**

CoQ10

Mitochondrial complex I support — the precise failure point in nigral neurons. MyCell® CoQ10 achieves superior absorption vs. conventional forms. Addresses energy deficit driving pacemaker vulnerability and dopamine synthesis impairment.

Glutathione

Substantia nigra has the lowest glutathione in the brain. MyCell® delivers directly to nigral cells, restoring antioxidant defense depleted by dopamine metabolism, iron catalysis, and environmental toxins. Potentially the most critical nigral intervention.

NMN Spermidine

NMN restores NAD+ for mitochondrial repair and complex I function. Spermidine induces autophagy — clearing alpha-synuclein aggregates (Lewy bodies) that drive dopamine neuron death. Addresses pathological protein accumulation directly.

Curcumin

Inhibits alpha-synuclein aggregation and disaggregates pre-formed fibrils in laboratory studies. Also reduces STN-driven neuroinflammation via NF-κB inhibition. MyCell® achieves plasma levels that conventional formulations cannot reach.

Ginger

Gingerols support dopamine neuron survival in oxidative stress models. Also supports gut motility — addressing the constipation and ENS dysfunction that may represent PD's origin point. Dual peripheral and central relevance.

Reishi (Ganoderma)

Modulates the neuroinflammatory environment in the substantia nigra, supports mitochondrial biogenesis, and exhibits neuroprotective effects on dopaminergic neurons. Powerfully supports gut microbiome diversity — addressing the ENS disease origin.

**Not evaluated by FDA. Not intended to diagnose, treat, cure, or prevent any disease. Consult your healthcare provider.*

Parkinson's: Biomodulator® — Physiological Rationale

Educational Note: The following discusses physiological mechanisms only. The BioModulator® is not claimed to treat, cure, or prevent any neurological disease.

Basal Ganglia Circuit Modulation

STN hyperactivity is driven by loss of inhibitory dopaminergic control. C fiber afferent stimulation engages GABAergic pathways that ascend to modulate basal ganglia excitability — the same circuit targeted by DBS, but via the body's own afferent signaling infrastructure (Takakusaki, 2017).

Autonomic Normalization

PD involves significant autonomic dysfunction — constipation, orthostatic hypotension, dysautonomia. C fiber stimulation shifts the ANS toward parasympathetic dominance, reducing the sympathetically-maintained excitatory states that amplify excitotoxic damage to vulnerable nigral neurons.

GDNF & Dopamine Pathway Support

Afferent stimulation supports the nigrostriatal pathway through BDNF and GDNF upregulation. GDNF is the primary trophic survival signal for dopamine neurons and is consistently depleted in PD. Direct infusion of GDNF is among PD's most promising experimental interventions (Gill et al., 2003).

Vagal Modulation of Gut-Brain Axis

Given the Braak hypothesis — PD originating in the enteric nervous system — C fiber stimulation of vagal afferent pathways represents a direct interface with the disease's proposed origin. Normalized vagal tone may influence enteric alpha-synuclein dynamics and gut-to-brain transmission.

Takakusaki, K. (2017). Functional neuroanatomy for posture and gait. Journal of Movement Disorders. | Gill, S.S. et al. (2003). GDNF in Parkinson disease. Nature Medicine, 9, 589.

Parkinson's: Key C Fiber Stimulation Zones

Zones selected for vagal pathway proximity, sympathetic chain access, ENS modulation potential, and basal ganglia circuit influence via ascending afferent pathways.

1. **Occipital / Suboccipital**
   Brainstem access — modulates dopaminergic projection pathways
2. **Anterior Neck / Vagus**
   Vagal afferent — gut-brain axis modulation, anti-inflammatory
3. **Thoracic Spine (T6–T10)**
   Celiac ganglion influence — gut autonomic normalization, ENS
4. **Lumbar Spine (L1–L3)**
   Lower sympathetic chain — reduces excitatory tone to STN
5. **Abdominal Region**
   Enteric nervous system access — direct gut-brain axis interface
6. **Plantar Foot Reflex Points**
   Powerful reflex zone — engages GDNF trophic pathways ascending

Senergy® Wellness

PART 5

Peripheral Neuropathy

Small Fiber Damage & Metabolic Excitotoxicity

Why C Fibers Are Both the Patient and the Portal

Peripheral Neuropathy: What Is It?

Peripheral neuropathy describes damage to the peripheral nervous system — the vast network of nerves outside the brain and spinal cord. Most commonly affects small sensory fibers (C fibers and Aδ fibers) first, causing burning pain, numbness, and temperature loss typically beginning in the feet.

- Dorsal root ganglia (DRG) — clusters of sensory neuron cell bodies — are the primary site of pathology
- C fibers and Aδ fibers affected first: burning, tingling, temperature loss — length-dependent, feet first
- Large fiber involvement follows: vibration, proprioception, gait instability
- Dorsal horn central sensitization develops secondarily — amplifying peripheral damage into a CNS-level problem
- DRG sits OUTSIDE the blood-brain barrier — directly accessible to everything in circulation
- Causes: diabetes, autoimmune, chemotherapy, infections, toxins, idiopathic in 30% of cases

20M+
Americans with peripheral neuropathy

50%
of diabetics develop neuropathy

30%
of cases remain idiopathic

~$10B
annual U.S. healthcare cost

NIDDK (2023). Diabetic Neuropathy Statistics. NIH. | Callaghan, B.C. et al. (2012). Diabetic neuropathy. Lancet Neurology, 11, 521–534.

Neuropathy: The Glutamate Mechanism

DRG Metabolic Depolarization

Energy failure impairs Na+/K+ ATPase ion pumps — DRG neurons become chronically partially depolarized. This perpetually activates voltage-gated calcium channels and NMDA receptors without any specific receptor stimulus. Calcium accumulates from continuous low-level activation.

Satellite Glial Cell Failure

Satellite glial cells surrounding DRG neurons normally buffer glutamate and regulate the ionic environment at the soma. In neuropathy they become dysfunctional — removing the local excitotoxic buffer precisely at the cell body level, the origin of the entire afferent fiber.

Central Sensitization Cascade

Continuous C fiber barrage from injured peripheral nerves drives structural changes in the dorsal horn: inhibitory interneurons die, microglia activate, NMDA receptors upregulate. The CNS amplifies the peripheral problem. Clinically: pain that outlasts any original injury (Woolf, 2011).

AGE Accumulation & Microvascular

Advanced glycation end products crosslink and damage endoneurial capillaries — reducing oxygen and nutrient delivery to exactly the small fibers most dependent on local microvascular perfusion. A siege — cutting off supply lines to the most vulnerable outposts first.

Woolf, C.J. (2011). Central sensitization. Pain, 152, S2–S15. | Fernyhough, P. (2015). Mitochondrial dysfunction in diabetic neuropathy. Current Diabetes Reports.

Neuropathy: Voltage & Cellular Energy Connection

Why Voltage Matters

The peripheral nervous system outside the BBB is the most electrically exposed neural tissue in the body. DRG neurons and their small fiber axons depend on precisely maintained membrane voltage to regulate ion flux, sustain signal conduction, and prevent calcium accumulation.

In metabolic neuropathy, the first casualty is the endoneurial microvasculature — tiny capillaries supplying oxygen and glucose to peripheral nerves. When these vessels are damaged by AGEs, inflammation, or ischemia, DRG neurons become energy-deprived.

Energy deprivation means the Na+/K+ ATPase pump — requiring 30–40% of the neuron's total ATP just to maintain resting membrane potential — slows down. Sodium accumulates inside the cell. Membrane voltage falls. The cell chronically depolarizes.

This is structurally destructive. Chronically depolarized small fibers have elevated intracellular calcium, impaired axonal transport, and reduced neurotrophic factor responsiveness. They are consuming themselves at the cellular level while sending pain signals to the CNS.

Restoring voltage to DRG neurons through improved microvascular function, mitochondrial support, and adaptive C fiber normalization addresses the root of the disease process.

30–40%

of DRG ATP used just to maintain resting potential

Outside

DRG location relative to blood-brain barrier

1m+

small fiber length from DRG to foot

Ferrannini, E. (2015). Mitochondrial dysfunction in diabetic neuropathy. Current Diabetes Reports. Woolf, C. J., & Salter, M. W. (2000). Neuronal plasticity. Science, 288.

Neuropathy: Why Small Fibers Are Vulnerable

Outside the Blood-Brain Barrier

DRG neurons sit outside the BBB — directly exposed to glucose, toxins, inflammatory mediators, heavy metals, mycotoxins, and pathogens. No meaningful protective filter between the bloodstream and DRG neurons.

Highest Surface-to-Volume Ratio

Unmyelinated C fibers have the highest membrane-to-cytoplasm ratio of any neural structure. More membrane = more ion channels = higher metabolic cost per unit of cytoplasm. Energetically the most expensive fibers to run.

Longest Axons — Distal Starvation

Length-dependent neuropathy begins in feet because the most distal portions of the longest axons are furthest from metabolic support at the DRG cell body. Axonal transport failure hits the foot first — the classic 'stocking glove' distribution.

Satellite Glial Cell Dependency

DRG neurons depend on surrounding satellite glial cells for glutamate buffering, trophic factor supply, and ionic environment regulation. In metabolic neuropathy, satellite cells lose their protective function first — removing the primary local support structure.

Endoneurial Microvascular Fragility

Endoneurial capillaries supplying peripheral nerve fibers are among the first vessels damaged in metabolic disease and ischemia. Small fibers with no energy reserve receive inadequate oxygen and glucose before any symptoms appear.

Dorsal Horn Secondary Destruction

Under sustained C fiber barrage, dorsal horn inhibitory interneurons progressively die, microglia activate, and NMDA receptors upregulate. The nervous system becomes hypersensitive — the original peripheral problem creates a permanent central amplification problem.

Smith, A.G. & Singleton, J.R. (2013). Obesity and hyperlipidemia as neuropathy risk factors. Journal of Diabetes Complications. | Woolf, C.J. (2011). Central sensitization. Pain.

Neuropathy: What Weakens the Small Fiber Zone

Insulin Resistance & Pre-Diabetes

AGE accumulation, endoneurial microvascular injury, and DRG oxidative stress begin years before blood sugar meets diabetic criteria. Small fiber neuropathy may be the earliest detectable consequence of metabolic syndrome — appearing before any standard lab abnormality. Skin punch biopsy shows reduced intraepidermal nerve fiber density in metabolic syndrome (Smith & Singleton, 2013).

B12 Deficiency — Metformin, PPIs, Veganism

Methylcobalamin (active B12) is essential for myelin synthesis and DRG mitochondrial function. Metformin (the most prescribed diabetes drug), PPIs, and plant-based diets all deplete B12 — creating a cruel iatrogenic loop where treatment of the underlying condition accelerates its most common complication.

Lyme Disease, Long COVID & Post-Infectious

Borrelia burgdorferi has direct DRG tropism — accessible because of the open blood-nerve barrier. Post-COVID small fiber neuropathy from spike protein/immune complex deposition is increasingly documented on skin punch biopsy and may represent an emerging epidemic affecting millions (Oaklander et al., 2022).

Chemotherapy — Cisplatin, Paclitaxel, Vincristine

These agents are direct DRG neurotoxins accumulating precisely because DRG neurons lack BBB protection. CIPN affects 30–40% of chemotherapy patients and often persists for years post-treatment. The same BBB vulnerability that makes DRG accessible also makes them uniquely vulnerable to oncological agents.

Mycotoxins — Ochratoxin A & Fumonisins

Ochratoxin A accumulates in peripheral nerve and kidney tissue. Fumonisin B1 disrupts ceramide metabolism in Schwann cells — ceramide is structurally essential for myelin of small myelinated fibers. Mycotoxin-associated neuropathy is almost never considered clinically despite strong biological plausibility.

Fascial Restriction & Compartment Pressure

Chronically elevated tissue pressure within fascial compartments reduces endoneurial perfusion to small fibers — a sustained ischemic, metabolically-compromised microenvironment that may perpetuate cases resistant to metabolic management. Rarely discussed in neuropathy literature but mechanistically compelling.

Oaklander, A.L. et al. (2022). Peripheral neuropathy in prolonged long COVID. Neurology Neuroimmunology. | Callaghan, B.C. et al. (2012). Diabetic neuropathy. Lancet Neurology.

Neuropathy: Integrative & Lifestyle Support

Evidence-informed lifestyle approaches addressing underlying vulnerability factors — for educational discussion only.

Metabolic Reversal — Root Cause

Time-restricted eating (16:8) and low-glycemic diet to reduce AGE burden. Continuous glucose monitoring to identify postprandial spikes. Metformin users must supplement methylcobalamin B12 aggressively. Target HbA1c below 5.7% for neuropathy reversal potential.

Methylcobalamin B12 — Critical

Not cyanocobalamin — the neurologically active methylcobalamin form. Japanese trials showed 500mcg methylcobalamin 3× daily improved nerve conduction in diabetic neuropathy. B12 injection superior to oral in severe deficiency. Rule out malabsorption.

Alpha Lipoic Acid — DRG Mitochondria

R-ALA (300–600mg daily) reduces AGE burden, supports DRG mitochondrial function, and reduces oxidative stress in peripheral nerves. Both fat and water-soluble — penetrates both DRG cell bodies and peripheral nerve myelin. Strongest evidence base of any supplement in diabetic neuropathy.

Fascial Release & Structural Work

Manual therapy targeting fascial compartments affecting nerve territories. Particularly relevant in tarsal tunnel, carpal tunnel, and thoracic outlet patterns. Reduces endoneurial compartment pressure, restoring microvascular perfusion to ischemic small fiber territories.

Infectious & Toxin Assessment

Lyme serology (Western blot, not just ELISA). Mycotoxin urine testing (Great Plains, RealTime Labs). Heavy metal testing. Full thyroid panel — hypothyroidism directly causes peripheral neuropathy via Schwann cell dysfunction. Fasting insulin to identify insulin resistance years before diabetic range.

Temperature Contrast & Circulation

Alternating warm-cold foot baths improve endoneurial microvascular reactivity. Far-infrared sauna (3×/week) supports toxin elimination via sweat and improves peripheral circulation. Discuss contraindications with healthcare provider.

Neuropathy: MitaCell Supplement Protocol

MyCell® Stabilized Micelle Technology — ultra-fine water-dispersible micelles for near-complete cellular absorption. Educational purposes only.

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CoQ10

Endoneurial mitochondrial support — the Na+/K+ ATPase pump maintaining DRG membrane voltage requires continuous ATP. MyCell® CoQ10 delivers to peripheral nerve mitochondria with far greater efficiency than conventional formulations, addressing voltage failure at the cellular level.

Glutathione

DRG neurons outside the BBB are bathed in oxidative stress from AGEs, inflammatory mediators, and ischemia. MyCell® glutathione reaches peripheral nerve tissue directly — restoring the antioxidant defense that satellite glial cells can no longer provide in diseased tissue.

Curcumin

Reduces the neuroinflammatory environment at DRG level, inhibits AGE formation, and supports Schwann cell function via NF-κB pathway modulation. Also reduces dorsal horn central sensitization. MyCell® delivery essential for achieving therapeutic peripheral nerve levels.

Ginger

Gingerols and shogaols reduce endoneurial inflammation and improve peripheral microvascular circulation. Also supports the gut microbiome — addressing post-infectious neuropathy and systemic pro-inflammatory states damaging small fibers.

NMN Spermidine

NMN restores NAD+ in DRG neurons — critical for axonal transport and mitochondrial energy in the longest axons in the body. Spermidine supports autophagy, clearing damaged mitochondria in peripheral nerve that accumulate in metabolic neuropathy.

C3 Cellular Repair

Vitamin C + Curcumin + Frankincense in MyCell® delivery. Vitamin C supports collagen in endoneurial connective tissue and perineurial barriers. Frankincense reduces leukotriene-mediated inflammation contributing to endoneurial ischemia. Foundational cellular repair for peripheral nerve tissue.

**Not evaluated by FDA. Not intended to diagnose, treat, cure, or prevent any disease. Consult your healthcare provider.*

Neuropathy: Biomodulator® — Physiological Rationale

Educational Note: The following discusses physiological mechanisms only. The BioModulator® is not claimed to treat, cure, or prevent any neurological disease.

Direct C Fiber Interface

The primary injured tissue in small fiber neuropathy IS the C fiber — the exact fiber through which the BioModulator® delivers adaptive stimulation. The device reads and responds to the same fibers that are dysfunctional. Adaptive biofeedback potentially normalizes membrane potential oscillations at the exact site of injury.

Dorsal Horn Normalization

C fiber stimulation directly modulates the dorsal horn — the site of the central sensitization loop that transforms peripheral small fiber damage into a CNS-level amplification problem. GABAergic interneuron engagement and inhibitory peptide release interrupt the windup cycle (Woolf & Salter, 2000).

Trophic Factor Restoration at DRG

NGF and NT-3 (neurotrophin-3) are essential survival factors for small sensory fibers progressively depleted in neuropathy. Afferent stimulation-associated trophic factor upregulation directly addresses the survival signal deficit at the primary site of injury (Apfel, 2002).

Anti-Windup in Sensitized Tissue

Fixed-frequency stimulation in sensitized neuropathic tissue risks reinforcing the windup pattern — driving already hyperexcitable C fibers into greater excitation. The BioModulator®'s adaptive response, changing every impulse based on tissue feedback, is precisely suited to chronically excitable tissue.

Woolf, C.J. & Salter, M.W. (2000). Neuronal plasticity: increasing the gain in pain. Science, 288, 1765–1769. | Apfel, S.C. (2002). NGF for diabetic neuropathy. Current Pain and Headache Reports.

Neuropathy: Key C Fiber Stimulation Zones

C fibers stimulated at distal zones work retrogradely toward DRG cell bodies, while spinal zone stimulation addresses the DRG soma directly — a dual approach to normalizing the entire dysfunctional afferent unit.

1. **Plantar Foot / Toes**
   Highest small fiber density — direct access to distal C fiber terminals
2. **Lower Leg / Ankle**
   Sural nerve, tibial nerve — primary neuropathy territory
3. **Hand / Fingers**
   Distal upper extremity small fibers — glove-pattern neuropathy zone
4. **Lumbar Spine (L4–S1)**
   DRG cell body access — stimulation nearest to neuropathy origin
5. **Sacral Region (S2–S4)**
   Parasympathetic outflow — autonomic neuropathy support
6. **Cervical Spine (C5–C8)**
   Upper extremity DRG — for glove-distribution neuropathy

PART 6

The Synthesis

Unified Mechanisms & the Path Forward

How Voltage, Glutamate, and Adaptive Stimulation Connect

Mechanism Relevance Across All Four Diseases

How adaptive C fiber stimulation interfaces with each condition's specific pathophysiology

BioModulator® Mechanism	ALS	MS	Parkinson's	Neuropathy
Glutamate Release Inhibition	●●●	●●	●●	●●●
GABAergic Restoration	●●●	●●	●●●	●●●
Autonomic Rebalancing	●●	●●	●●●	●●●
Microglial Modulation	●●●	●●●	●●●	●●
Neurotrophic Factor Support	●●●	●●	●●	●●●
Dorsal Horn Normalization	●●	●●	●	●●●
Anti-Windup Protection	●●●	●●●	●●●	●●●
Vagal / Autonomic Pathway	●●	●●	●●●	●●
Voltage Restoration	●●●	●●●	●●●	●●●

● = some relevance ●● = moderate relevance ●●● = high relevance Based on published peer-reviewed neurophysiology

MitaCell Wellness USA

🔬 MyCell® Stabilized Micelle Technology — mitacellusa.com

The most bioavailable nutraceutical delivery system available — designed for real cellular impact, not label impressiveness.

Why Absorption Is the Problem

Standard supplements — even 'premium' liposomal formulations — are largely degraded in the stomach. In neurologically compromised tissue with impaired microvascular delivery, cells receive far less than the label suggests. Most supplements are designed for labels, not biology.

MyCell® Difference

Every MitaCell active ingredient is transformed into ultra-fine, fully water-dispersible stabilized micelles that bypass digestive degradation and enter the bloodstream with near-complete efficiency. Smaller doses with dramatically greater potency at the cellular level.

Why It Matters for These Diseases

All four conditions involve mitochondrial dysfunction, oxidative stress, and neuroinflammation — the precise targets of MitaCell's product line. CoQ10, glutathione, curcumin, and NMN delivered at therapeutic cellular concentrations may support the voltage and antioxidant environment that protects vulnerable neural zones.

Medical Direction

MitaCell is led by Dr. Natalie Greenberg, ND — trained at Columbia, UC San Diego, and Bastyr — with personal experience overcoming mold toxicity and Lyme disease. The product line reflects clinical understanding of the exact conditions discussed in this presentation.

**These statements have not been evaluated by the FDA. MitaCell products are not intended to diagnose, treat, cure, or prevent any disease. Always consult a licensed healthcare provider.*

The Unified Summary

"ALS, Parkinson's, MS, and Peripheral Neuropathy are not separate diseases. They are different expressions of a nervous system under excitotoxic stress — each manifesting in the zone that was most voltage-depleted, most toxin-loaded, and most metabolically vulnerable in that individual."

ALS: Motor neurons fall when GLT-1 fails, calcium-permeable AMPA receptors are flooded, and mitochondrial energy can no longer sustain glutamate clearance

MS: Oligodendrocytes are destroyed by immune-cell glutamate release within inflammatory plaques — compounded by voltage loss in denuded axons

Parkinson's: Dopamine neurons are consumed by the self-amplifying STN glutamate loop after complex I failure reduces pacemaker voltage below survival threshold

Neuropathy: Small fibers are metabolically depolarized by endoneurial vascular failure — driving local excitotoxicity and dorsal horn central sensitization

Lewerenz, J. & Maher, P. (2015). Chronic Glutamate Toxicity in Neurodegenerative Diseases. Frontiers in Neuroscience, 9, 469.

QUESTIONS & DISCUSSION

"The body's ability to heal is far greater than we have been taught to believe."

— Danielle Palmer, CNMT

Website: thefrequencytherapist.com

Devices & Training: senergy.us

Supplements: senergy.us

Instagram: @thefrequencytherapist

This presentation is for educational purposes only. The Tennant BioModulator® is an FDA-accepted Class II medical device indicated for symptomatic pain relief. MitaCell products are not intended to diagnose, treat, cure, or prevent any disease. No claims are made that any product or device discussed treats or cures any neurological disease. All physiological discussion is based on peer-reviewed scientific literature presented in an educational context. Always consult a licensed healthcare provider.