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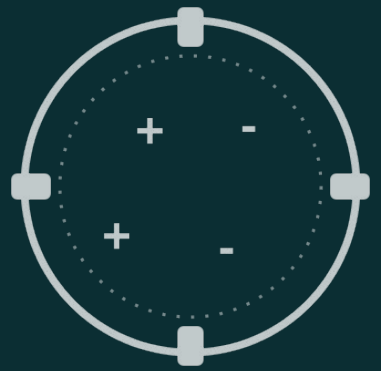
BIOELECTRIC MEDICINE · LYME & PTLDS SUPPORT

Building Resilience

The Bioelectric Case for Terrain Over Eradication

A deep dive into microcurrent, frequency, and C-fiber science — and why each supports immune function, mitochondrial health, cellular integrity, and the nervous system



The Frequency Therapist

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01

From Eradication to Resilience

Reframing the goal of Lyme & PTLDS support

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FRAMING

The Question Behind This Talk

Most approaches to Lyme disease ask: **“how do we kill the organism?”**

This talk asks a different question: **“how do we build a body the organism can’t destabilize?”**

- Persistent immune/antigen burden is common — the deciding factor in symptomatic vs. well-controlled outcomes is host resilience, not simply pathogen presence
- “Terrain” is measurable physiology: immune tone, mitochondrial output, cell membrane integrity, and neuroinflammatory load
- This talk goes deep on three mechanisms that support that terrain: microcurrent's cellular effects, frequency's influence on the nervous system, and the C-fiber network that connects them

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02

How Lyme Is Actually Treated

Two paradigms — and the case for a resilience-based strategy

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TREATMENT LANDSCAPE

Two Clinical Paradigms

Conventional Medicine

Mainstream Guideline Consensus

- Short-course oral antibiotics for early disease
- 2–4 weeks IV ceftriaxone for late neurologic disease or persistent arthritis
- Extending antibiotics beyond 8 weeks not shown to add benefit
- “Chronic Lyme disease” considered a poorly defined entity with insufficient evidence of ongoing active infection

Integrative Medicine

Integrative / Individualized Model

- Individualized treatment weighing persistent symptoms, coinfections, and immune dysfunction
- More flexibility on duration for patients who remain symptomatic
- Extended and repeat IV antibiotic courses, plus IV nutrient protocols
- Emphasizes clinical experience and patient response alongside trial data

Source: [illegible]

Why Resilience-Based Support Makes Sense

Supporting the body's own regulatory systems is a coherent, low-risk complementary strategy — alongside whatever medical management a person is already receiving.

- Repeated attempts to eradicate the organism carry documented risks and inconsistent symptom benefit in controlled trials
- Supporting immune regulation, mitochondrial function, cellular integrity, and nervous system tone addresses the systems that determine whether a person is symptomatic in the first place
- This is where bioelectric approaches — grounded in real neuroscience — offer a genuinely different lever

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03

What “Terrain” Means Physiologically

The measurable biology behind resilience in PTLDS

TERRAIN PHYSIOLOGY

Four Interlocking Mechanisms

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Immune Dysregulation

Overactive/aberrant immune activity, including microglial activation in the brain

Mitochondrial Bioenergetics

Reduced ATP output and metabolic inefficiency under sustained immune activation

Cellular Membrane & Oxidative Stress

Membrane damage and oxidative burden impairing normal cell signaling

Neuroinflammation

The downstream convergence point — fatigue, cognitive symptoms, pain sensitization

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Immune Dysregulation & Microglial Activation

- PET imaging using a marker of activated microglia (TSPO) found evidence consistent with overactive immune activity in patients with persistent symptoms following Lyme treatment
- This provides in vivo evidence that the brain's resident immune cells remain in an activated state well after the infection itself has been addressed
- This gives a clear, measurable target: calming and regulating immune signaling rather than only addressing the organism

TERRAIN PHYSIOLOGY

Mitochondrial Bioenergetics & Small Fiber Involvement

- Mitochondrial bioenergetic inefficiency is identified as a convergent mechanism across post-acute infection syndromes — including Lyme, long COVID, and chronic fatigue syndrome
- Post-treatment Lyme disease syndrome has been associated with small fiber neuropathy — measurable autonomic and sensory dysfunction detectable on skin biopsy and autonomic testing
- Small fiber neuropathy directly implicates C-fibers — the exact fiber class covered in depth later in this talk

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04

The Nervous System: A Foundation for Healing

Why regulation has to come before the body can tolerate treatment

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NERVOUS SYSTEM FOUNDATION

Chronic Illness Can Leave the Body “Stuck” in Protection

Mold / Lyme / inflammatory trigger

↓

Persistent inflammatory signaling

↓

Limbic + autonomic dysregulation

↓

Hypervigilance / sensitivity

↓

Poor tolerance to treatment

↓

More physiological stress

- In Toxic, physician Neil Nathan, MD devotes a full chapter to what he calls “rebooting the nervous system”
- His clinical model centers the limbic system and the autonomic/vagal system as key drivers of sensitivity in chronically ill, treatment-reactive patients
- The clinical question isn't only “what is driving the illness” — it's also whether the nervous system can currently tolerate the treatment being offered

KEY TAKEAWAY

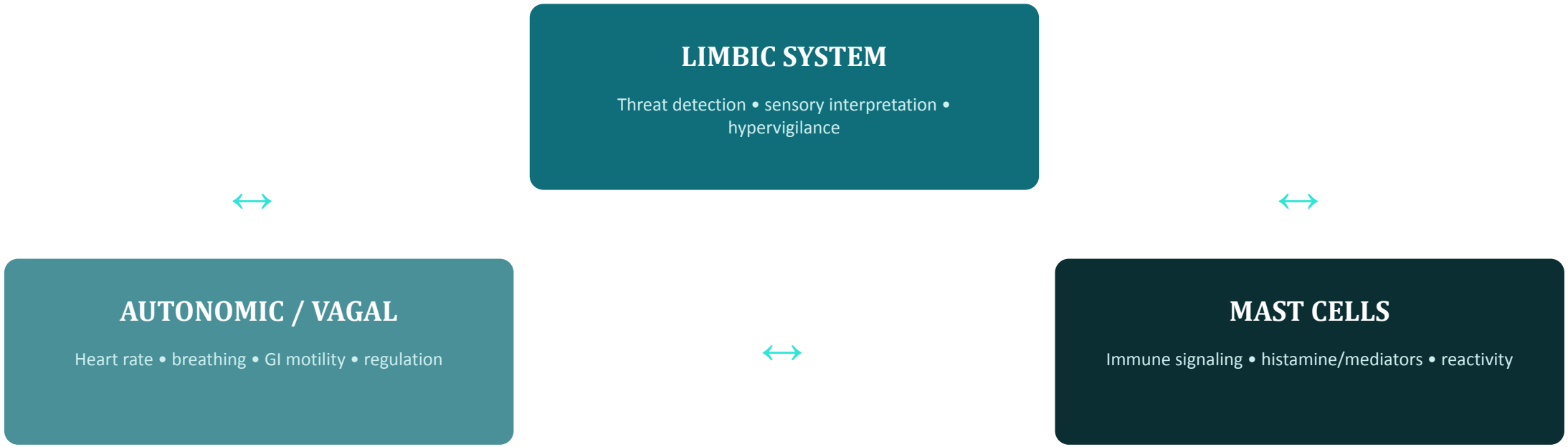
Help a reactive nervous system feel safe before asking it to do more.

Source: Dr. Neil Nathan

NERVOUS SYSTEM FOUNDATION

The Limbic–Vagal–Mast Cell Loop

Nathan's "trifecta of sensitivity" — three systems that regulate (or dysregulate) together



- These three systems continuously communicate — dysregulation in one amplifies the others
- In highly sensitized patients, Nathan's model holds that treating only one of the three often isn't enough to settle the system down

Source: Nathan, 4

NERVOUS SYSTEM FOUNDATION

Why Start With the BioModulator?

- Nathan's broader nervous-system framework includes gentle electrical neuromodulation as one tool for regulation
- He has described gentle electrical approaches like FSM as a way to help calm vagal nerve activity as part of a “reboot” of dysfunctional nervous-system patterns

Gentle transcutaneous electrical stimulation

↓

Peripheral sensory and nerve input

↓

Influence nervous-system processing

↓

Support a less reactive physiological state

↓

Improve the foundation for subsequent care

REGULATE → BUILD TOLERANCE → ADDRESS ROOT CAUSES

The BioModulator is not positioned as a “treatment” for Lyme disease, mold toxicity, or MCAS.

It is positioned as a tool for delivering noninvasive electrical input to the peripheral nervous system — supporting regulation as part of a broader strategy.

FDA-cleared indication (original Avazzia Tennant BioModulator): transcutaneous nerve stimulation for pain relief.

Sources: 4, 7, 8

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05

Bioelectric Medicine 101

The neuroscience of nerve signaling and immune tone

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BIOELECTRIC MEDICINE

The Cholinergic Anti-Inflammatory Pathway

The best-established mechanism linking nerve signaling to immune modulation

- Efferent vagal/cholinergic signaling releases acetylcholine, which binds the alpha-7 nicotinic receptor on immune cells to suppress inflammatory cytokine production
- This pathway prevents damaging cytokine release in sepsis, endotoxemia, ischemia/reperfusion injury, hemorrhagic shock, and arthritis
- First defined by Dr. Kevin Tracey and colleagues; now one of the most extensively studied neuro-immune circuits in medicine
- Confirmed clinically in rheumatoid arthritis and inflammatory bowel disease with non-invasive nerve stimulation

The Core Translatable Principle

**Peripheral nerve signaling is a legitimate, evidence-backed lever on systemic
immune tone — the scientific spine of everything that follows.**

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06

Deep Dive: Microcurrent Alone

Cellular voltage, mitochondrial ATP, calcium gating, and cellular signaling

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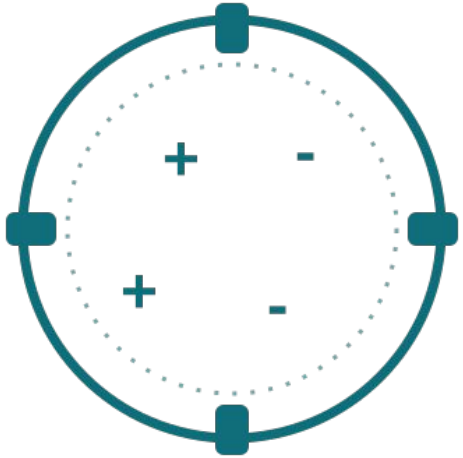
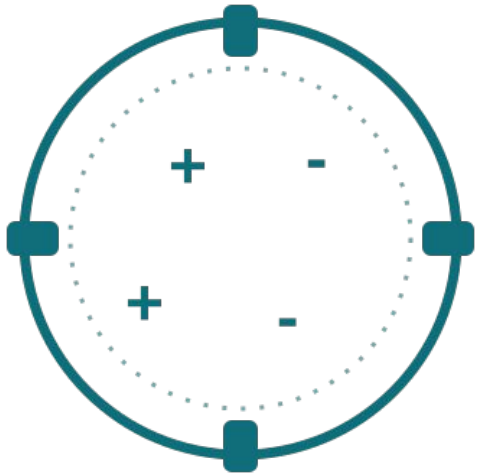
What Microcurrent Actually Is

- Microcurrent delivers electrical current in the microampere range (millionths of an amp) — below the sensory threshold, and roughly 1,000x lower than a standard TENS unit
- This range was chosen because it mirrors the body's own naturally occurring bioelectric currents — the same order of magnitude as the currents your cells already generate
- Every tissue layer in the body — skin, blood vessels, intestinal walls, even bone — maintains its own natural voltage gradient, on the order of millivolts, that drives these endogenous currents
- Because microcurrent operates in this same physiological range, it is theorized to work with the body's own electrical signaling rather than override it



Cellular Voltage: The Master Regulator

- Every cell in the body maintains a resting membrane potential — a voltage difference across its outer membrane created by the uneven distribution of ions (potassium, sodium, calcium, chloride)
- This voltage is not incidental — it is recognized as a key regulator of proliferation, differentiation, migration, and tissue regeneration
- Cells must pass through specific voltage states (depolarization and hyperpolarization) to progress through normal growth and repair cycles
- This is the physiological basis for why an external electrical input — like microcurrent — has a plausible route to influencing cell behavior: it is speaking the same voltage-based language the cell already uses to regulate itself



The Landmark ATP Study

Cheng et al., 1982

Clinical Orthopaedics and Related Research

Electrical stimulation at 50–1,000 microamps — the same range used in microcurrent therapy — increased ATP generation in tissue by 3-5x, alongside measurable increases in protein synthesis and membrane transport of amino acids.

This is one of the most cited findings in the bioelectric medicine field — and it directly targets the mitochondrial and cellular repair machinery.

3–5X

increase in ATP generation

3

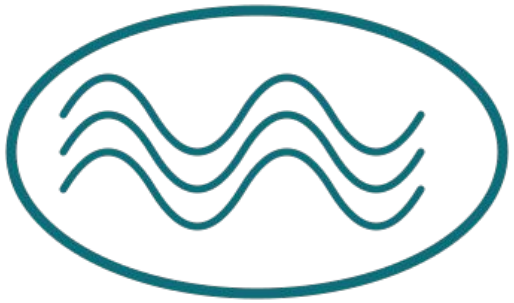
processes boosted:
ATP, protein synthesis,
membrane transport

Source: 2

MICROCURRENT DEEP DIVE

Direct Mitochondrial Support

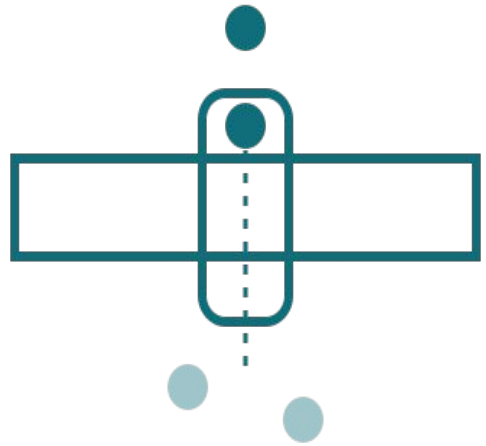
- A classic study went further, showing that an applied electric field can directly induce ATP synthesis within isolated mitochondria — even when normal respiration was chemically blocked
- The threshold for triggering ATP synthesis corresponded to an induced membrane potential of about 60–200 millivolts across the mitochondrial membrane — demonstrating that electrical energy can be converted directly into the chemical bond energy of ATP
- This is a direct mechanistic link between electrical input and the exact bioenergetic output (ATP) that is measurably reduced in post-acute infection syndromes like PTLDS
- AMPK — the cell's central energy sensor — is activated by the same calcium-channel signaling that microcurrent engages, further supporting mitochondrial biogenesis and energy metabolism



Sources: 2, 3

Calcium Gating & Cellular Signaling

- Calcium (Ca^{2+}) channels are voltage-sensitive gates in the cell membrane — they open in response to changes in the electrical field across the membrane, allowing calcium to flow into the cell
- Calcium influx is one of the most universal signaling events in biology — it activates enzymes, triggers gene expression, and regulates energy metabolism through its interconnection with AMPK
- Microcurrent stimulation has been shown to trigger MAPK signaling and TGF-β1 secretion — pathways directly involved in tissue repair and cellular communication
- This calcium-mediated signaling cascade is the mechanistic link between an external electrical input and downstream cellular repair processes



Protein Synthesis & Membrane Transport

- Beyond ATP, the same foundational research demonstrated increased protein synthesis and enhanced membrane transport of amino acids under microcurrent stimulation
- This matters directly for cellular membrane integrity — one of the four terrain targets identified earlier — since membrane repair depends on the cell's ability to synthesize new membrane proteins and transport building blocks across a damaged membrane
- Independent replication in other tissue types (skin, hair follicle papilla cells) shows the same pattern: microcurrent opens ion channels, increases Ca^{2+} influx, and upregulates ATP production and cell cycle progression
- This consistency across tissue types is what makes the mechanism credible as a general cellular principle, not a tissue-specific curiosity

MICROCURRENT DEEP DIVE

Summary: The Cellular Case

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Cellular Voltage	Microcurrent works within the body's own bioelectric range to influence membrane potential
Mitochondrial ATP	300–500% increases in ATP generation demonstrated; direct electric-field-driven ATP synthesis shown in isolated mitochondria
Calcium Signaling	Voltage-gated Ca^{2+} channels link microcurrent to MAPK/TGF-β1 repair signaling and AMPK energy regulation
Membrane & Protein Repair	Increased protein synthesis and membrane transport directly support cellular membrane integrity

WHY THIS MATTERS FOR LYME / PTLDS

This is a direct mechanistic match to the mitochondrial bioenergetic deficit and cellular membrane damage identified as core features of PTLDS.

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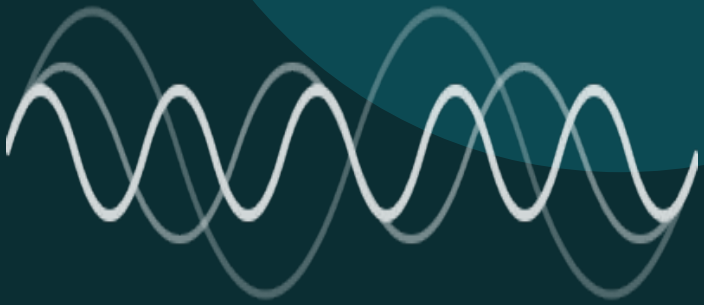
07



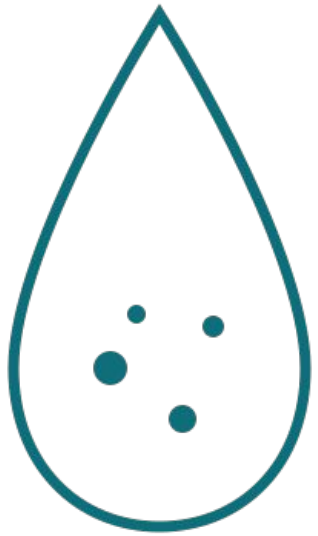
Frequency as a Bridge

From a clinical blood study to the nervous system's own rhythms

The Premise: Frequency and the Blood

McMakin, Gregory & Phillips (2005) — J Bodywork Mov Ther

- A retrospective clinical case series measured blood cytokine and hormone levels in fibromyalgia patients before and after specific-frequency microcurrent treatment
- Inflammatory markers IL-1, IL-6, and TNF-α, along with the pain-signaling neuropeptide substance P, dropped measurably following treatment
- Beta-endorphin and serum cortisol — the body's own pain-modulating and stress-regulating hormones — rose over the same treatment period
- 58% of patients (31 of 53) experienced resolution of fibromyalgia symptoms during the observation period



FREQUENCY

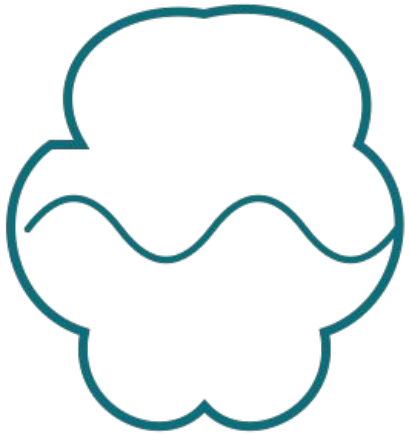
Why This Study Matters as a Premise

- This is a retrospective, uncontrolled clinical case series — it is not a randomized trial, and it deserves to be described accurately as what it is
- What makes it valuable as a premise: it is one of the few studies to measure actual blood chemistry — not just self-reported pain — before and after frequency-based treatment
- Measurable shifts in inflammatory cytokines and stress/pain hormones give a physiological signal, not just a subjective one, that specific frequency application can correspond with real changes in the body's chemistry
- This raises the natural next question: what does the broader literature say about frequency and physiology more generally? That's where we go next

FREQUENCY

The Brain's Natural Frequencies

Delta (0.5–4 Hz)	Deep, restorative sleep
Theta (4–8 Hz)	Deep relaxation, meditation, drowsiness
Alpha (8–13 Hz)	Calm, relaxed wakefulness
Beta (13–30 Hz)	Alert focus and active thinking
Gamma (30+ Hz)	High-level information processing



Source: Dr. Z

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FREQUENCY

Frequency & the Nervous System: What Research Shows

- Brainwave entrainment refers to the brain's electrical activity synchronizing toward the frequency of an external rhythmic stimulus — documented via EEG since the mid-20th century
- Controlled EEG studies of binaural beats (two slightly different tones, one per ear) show measurable changes in cortical activity — in one study, theta activity developed across the entire cortex within 10 minutes of a 6 Hz binaural beat
- Personalized alpha-range binaural beat protocols have shown sustained changes in frontal alpha rhythm activity in pilot studies, with effects observable after stimulation ended
- Regular use of binaural beats has been associated with reduced anxiety and improved quality-of-life measures in the existing (still-developing) research base

Source: Zaj, 2

FREQUENCY

Why This Matters for the Nervous System in PTLDS

WHY THIS MATTERS FOR LYME / PTLDS

Autonomic dysfunction and disrupted sleep/relaxation states are common in PTLDS. Frequency-based approaches that can measurably shift cortical activity toward calmer states (alpha/theta) offer a plausible, non-invasive way to support autonomic regulation and sleep architecture.

- Small fiber neuropathy in PTLDS affects the same autonomic nervous system that governs sleep, heart rate variability, and stress response
- Supporting the nervous system's ability to shift into calmer, more restorative frequency states is a reasonable complementary goal alongside immune and mitochondrial support
- This sets up the next question: beyond brainwave entrainment, how far does the evidence for “frequency and physiology” actually extend?

FREQUENCY

Beyond the Lab: The Schumann Resonance

- The Schumann resonance is a real, measured geophysical phenomenon — an extremely low-frequency electromagnetic resonance (~7.83 Hz) generated by global lightning activity resonating in the cavity between Earth's surface and the ionosphere
- Independent EEG research has found that spectral power in human cortical activity shows similarities to Schumann resonance frequencies, with replication across labs in different countries
- A more recent line of research proposes that cells and proteins may have evolved in the presence of this natural background frequency, potentially interacting with resting membrane potential and calcium signaling
- This remains an active, early-stage area of scientific interest — the physical phenomenon is well-established, but specific causal health claims are still being investigated, not yet settled

Source: The Frequency Therapist

FREQUENCY

Why This Matters for Lyme / PTLDS

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WHY THIS MATTERS FOR LYME / PTLDS

From the clinically-measured (McMakin's blood chemistry findings) to the well-studied (brainwave entrainment) to the early-stage (Schumann resonance) to the experiential (calming tone traditions), frequency-based approaches offer a graded set of tools for supporting nervous system regulation — directly relevant to the autonomic dysfunction seen in PTLDS.

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08

Deep Dive: C-Fibers

Not just pain fibers — the interface between the outside world and your inner physiology

Beyond Pain: What C-Fibers Really Are

- C-fibers are unmyelinated nerve fibers, 0.2–1.5 micrometers in diameter, with slow conduction velocities under 2 meters per second
- They are classically taught as pain (nociceptive) fibers — but neuroimmunological evidence shows they do far more: they monitor the body for immune challenges that don't even trigger a pain response
- Critically, C-fibers are not one-way sensors. They have both afferent function (carrying signals to the brain) and local efferent function — releasing their own signaling molecules directly into tissue
- This dual role is what makes them the body's true interface: sensing the internal and external environment, and actively shaping the tissue response to what they sense

Source: The Frequency Therapist

C-FIBER DEEP DIVE

The Interoceptive Interface

- Interoception is the nervous system's sense of the body's own internal state — and C-fibers (along with vagal afferents) are a primary channel for it
- Chemosensitive afferent nerves expressing the TRPV1 receptor don't just transmit pain — they play pivotal roles in initiating and modulating vascular, inflammatory, and immune reactions across multiple organ systems
- Signals from internal organs, carried via this interoceptive network, are now understood to influence emotion, cognition, and mood — meaning disrupted interoception has real implications for mental and physical wellbeing together
- This is the literal biological basis for “the interface between the outside world and our inner world” — C-fibers are where environmental and internal signals are translated into a coordinated bodily response

Sources: [3], [5]

C-Fibers and the Immune System

> *"C-fibres are also involved in monitoring the host for challenges that may not trigger a pain response but rather feed into the neuroimmune host defence system."*

- Direct experimental evidence shows brain-immune regulation occurring through C-fibers — central neural activation follows peripheral immune challenges, coordinated through intact C-fiber afferent and efferent pathways
- This means local immunity in the skin or tissue is not isolated — coordination of immune responses throughout the body requires intact C-fiber signaling
- This directly supports the broader cholinergic anti-inflammatory pathway discussed earlier: C-fibers are part of the same nervous-system-to-immune-system communication network

Neurogenic Inflammation: The Vascular Connection

- C-fiber nociceptors have a local efferent function: their peripheral endings release sensory neuropeptides directly into tissue — substance P, neurokinin A, and calcitonin gene-related peptide (CGRP)
- CGRP is the primary driver of vasodilation in this system; substance P drives increased vascular permeability and plasma protein extravasation
- Together, these neuropeptides directly regulate vascular tone and blood flow at the tissue level — this is neurogenic inflammation, a direct nerve-to-blood-vessel signaling pathway
- This means C-fiber activity has a direct, mechanistic influence on local circulation — relevant to tissue oxygenation, nutrient delivery, and the clearance of inflammatory byproducts

C-Fibers and Recovery

- The same neuropeptides that drive neurogenic vasodilation (CGRP, substance P) also play a role in the local tissue repair response — increased blood flow and vascular permeability are part of how injured or stressed tissue receives immune cells and repair signals
- Because C-fibers sprout and increase local density during inflammation, they become more, not less, involved in coordinating the tissue's response as recovery is needed
- Vagal C-fiber afferents specifically monitor visceral organ status and feed into homeostatic reflexes that support recovery from inflammatory insult
- This positions C-fiber signaling as a genuine participant in the recovery process — not merely a bystander reporting pain

C-FIBER DEEP DIVE

How the BioModulator May Reach This System

Tissue–Device Interaction → Neural Activation → Downstream Signaling

Stage 1: Tissue–Device Interaction

- Published studies using the BioModulator/BEST platform—particularly the Infinity waveform—describe an interactive, asymmetrical damped sinusoidal biphasic pulse whose electrical characteristics change in response to tissue characteristics such as skin impedance.
- Tissue impedance can vary with hydration, fluid distribution, electrolytes, tissue condition, and electrode contact

WHY THIS MATTERS FOR LYME / PTLDS

This is the physical starting point for any downstream effect — how the device's output actually meets the tissue matters before any nerve fiber is engaged.

Stage 2: Neural Activation — Which Fibers Respond

- Electrical fields alter membrane potential in sensory nerve endings; sufficient depolarization can initiate an action potential
- Potential fiber recruitment spans Aβ (large, fast, touch/pressure), Aδ (thinly myelinated, fast pain), and C fibers (unmyelinated, slow)
- Which fibers actually get recruited depends on waveform shape, pulse width, intensity/current density, frequency, electrode geometry, and the tissue itself — it is not a fixed, one-size-fits-all outcome
- This is why the BioModulator is better understood as an adaptive electrical neuromodulation system, not a device delivering one fixed frequency

Source: 2, 4

Stage 3: Downstream Signaling

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Peripheral Signaling

- CGRP
- Substance P
- Local vascular responses

Central Neuromodulation

- Endogenous opioids
- GABA
- Serotonin
- Noradrenaline

Cellular / Tissue Signaling

- ATP / metabolic activity (Cheng, rat skin)
- Cytokines: IL-1β, IL-6, TNF-α, IL-10
- Growth factors: VEGF, FGF-2, EGF, TGF-β1
- Fibroblast & endothelial activity

Electrical stimulation can influence physiology at multiple levels—including peripheral neural signaling, central sensory processing, and local cellular/tissue signaling—depending on the stimulation parameters and tissue being targeted.

Scientific boundary: these mediator changes are reported broadly across electrical-stimulation studies; the source systematic review itself notes the evidence is limited and heterogeneous, and none of it is BioModulator-specific.

Sources 2, 3

Summary: The Full Picture of C-Fiber Function

Interoception	The interface translating outside-world and internal signals into a coordinated response
Immune Coordination	Brain-immune regulation occurs directly through C-fiber afferent and efferent pathways
Vascular Tone	CGRP and substance P release directly regulates local blood flow and vessel permeability
Recovery Signaling	Local neuropeptide release participates in the tissue repair response, not just pain reporting

WHY THIS MATTERS FOR LYME / PTLDS

Small fiber neuropathy is a documented feature of PTLDS. Supporting C-fiber signaling directly addresses the immune coordination, vascular tone, and recovery pathways this same fiber class governs.

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09

Putting It Together

The full theoretical rationale, mapped to each terrain target

PUTTING IT TOGETHER

How the Three Pieces Connect

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Microcurrent

Directly supports mitochondrial ATP production, calcium-mediated cellular signaling, and membrane repair at the cellular level

Frequency

Extends that support to the nervous system — shifting autonomic and cortical states toward regulation and calm

C-Fibers + the BioModulator

The BioModulator delivers adaptive transcutaneous electrical stimulation to skin and superficial tissues where sensory afferents—including abundant C-fiber free nerve endings—are present. — If recruited, C fibers provide a plausible pathway through which electrical stimulation could influence nociceptive, vascular, neuropeptide, interoceptive, and neuroimmune signaling.

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PUTTING IT TOGETHER

Mapping the Rationale to Each Target

Target	Supportive Mechanism	Evidence Basis
Immune tone	Cholinergic anti-inflammatory pathway via C-fiber/vagal signaling	*Established physiology*
Mitochondrial function	Microampere electrical stimulation increased ATP concentrations approximately 3–5× in an experimental rat-skin model at specific current intensities.	*Clinically demonstrated*
Cellular membrane integrity	Microcurrent-driven protein synthesis & membrane transport	*Clinically demonstrated*
Neuroinflammation & nervous system	Frequency-based support for autonomic/cortical regulation; C-fiber vascular & immune coordination	*Mechanistic + early clinical*

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Closing Thought

Supporting the nervous system's own immune-regulatory and cellular-repair signaling is a scientifically coherent, low-risk part of building a body that can carry the burden of persistent immune activation more coherently.

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Questions & Discussion

Thank you

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LYME SUPPORT:
BEST PRACTICES

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Auricular Vagal Region

PRIMARY GOAL

Parasmpathetic / neuroimmune / central autonomic regulation

WHERE

Cymba conchae is the most physiologically defensible primary auricular target.

The concha/tragus can also contain vagal innervation, but innervation is more variable. Anatomical and neuroimaging evidence generally makes the **cymba conchae the cleaner target**.

WHY WE TARGET HERE

The external ear provides unusual superficial access to the:

Auricular branch of vagus nerve
↓
Nucleus tractus solitarius (NTS)
↓
brainstem autonomic networks
↓
autonomic + neuroimmune regulation

The NTS communicates with areas involved in autonomic and sensory regulation, including vagal motor nuclei and locus coeruleus-associated circuitry.

POTENTIAL DOWNSTREAM PHYSIOLOGY

Vagal afferent signaling
→ brainstem integration
→ autonomic regulation
→ neuroimmune signaling

The broader vagal inflammatory-reflex literature provides a plausible route through which vagal signaling can ultimately influence inflammatory cytokine production, although the exact human pathway is more complicated than the simplified “vagus → spleen” diagram often presented.

Best suited theoretically for:

Autonomic dysregulation • systemic inflammatory load • stress physiology • central sensitization/neuroimmune regulation

The Frequency Therapist

Sacral / Pelvic Region

PRIMARY GOAL

Pelvic autonomic + afferent regulation

WHERE

Sacral region — especially around S2–S4

WHY

Parasympathetic preganglionic neurons serving pelvic viscera originate from approximately **S2–S4**.
Sacral neuromodulation primarily works through modulation of **afferent pathways and spinal/supraspinal processing**, rather than simply electrically “turning on parasympathetics.”

Best suited theoretically for:

Pelvic autonomic dysfunction • bladder/bowel regulation • pelvic pain • distal GI/pelvic conditions

Important:

Use the sacrum as a secondary systemic immune/neuroinflammation target site.

For the objective of Nervous System regulation the Auricular vagal pathway has a considerably stronger physiological rationale.

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Upper Cervical + Suboccipital

PRIMARY GOAL

Reduce excessive afferent input entering central sensory networks

WHERE

Suboccipital tissues + upper cervical paraspinals

WHY WE TARGET HERE

The upper cervical spinal cord receives substantial sensory information from the head, neck, fascia, muscles and joints.
Upper cervical and trigeminal sensory pathways converge centrally.
Therefore, this region makes physiological sense when thinking about:
persistent peripheral input
↓
increased central sensory traffic
↓
central sensitization / altered pain processing
↓
potential autonomic consequences

WHAT WE ARE TRYING TO ACCOMPLISH

Not:
“electrically treating brain inflammation.”

Rather:
Modify peripheral afferent input entering neural networks involved in central sensory processing.

Best suited theoretically for:

Central sensitization • headache/craniofacial sensory load • neck-driven afferent load • autonomic dysregulation

The Frequency Therapist

Abdomen / Gut–Brain Interface

PRIMARY GOAL

Visceral afferent + enteric/autonomic regulation

WHY WE TREAT HERE

The gastrointestinal system is an enormous neuroimmune interface containing:

Enteric neurons
+
visceral sensory afferents
+
autonomic innervation
+
immune cells
+
microvasculature

These systems communicate bidirectionally with the CNS.

THE PHYSIOLOGICAL CONCEPT

Abdominal sensory stimulation
may influence
visceral/somatic afferent input
↓
spinal/central processing
↓
autonomic output

Important distinction

We would **not** say:
“Treating the abdomen stimulates the vagus nerve.”

Instead:
“We are introducing afferent electrical input into an anatomically important visceral/autonomic region.”

Best suited theoretically for:

Gut–brain dysregulation • visceral hypersensitivity • abdominal autonomic patterns • systemic inflammatory conditions with GI involvement

The Symptomatic / Inflamed Tissue

PRIMARY GOAL

Local sensory + tissue interaction

WHY TREAT THE PROBLEM AREA?

Because that's where you potentially have:

altered tissue electrical properties

+

sensitized nociceptors

+

local inflammatory mediators

+

altered vascular environment

+

abnormal sensory input

Inflammatory mediators can sensitize nociceptive afferents, including C fibers.

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