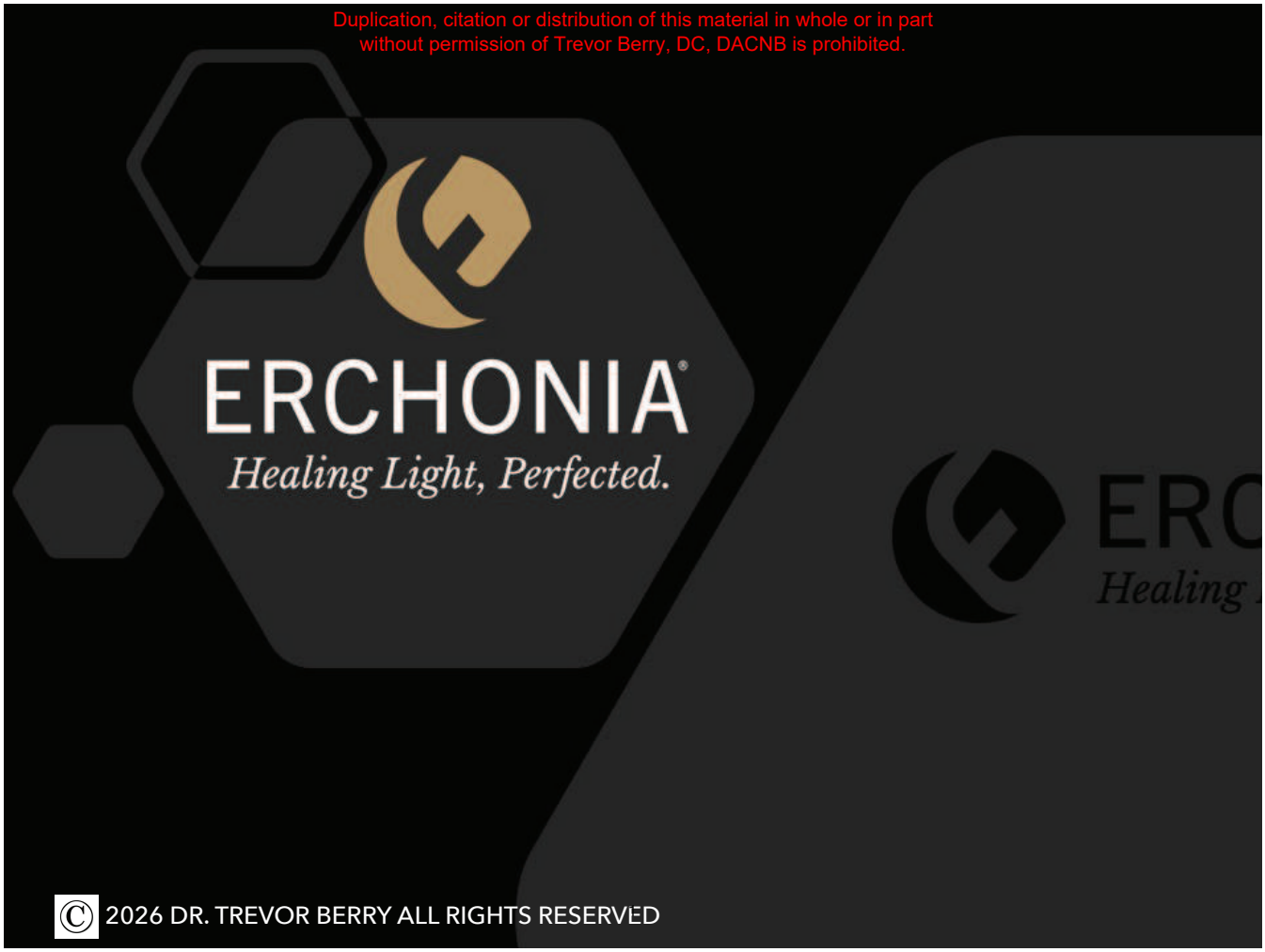
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



DISCLOSURES

- Dr. Trevor Berry DC, DACNB is not an employee of any vendors he may discuss during his lectures. He is a consumer of their products that he utilizes in his practice. He may be paid an honorarium, referral fee, affiliate fee or commission from certain vendors included in his lecture depending on location and context of the speaking engagement.
- Dr. Berry may promote product discounts for his attendees.
- Dr. Berry is the chief clinical officer and co-founder of BRC.
- Dr. Berry is the founder and owner of ACNC/BRC Chandler, AZ
- Dr. Berry has completed research for Erchonia Lasers and FDA clearances.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DISCLAIMER

- The content of Dr. Berry's seminars is for informational and educational purposes only. Content discussed verbally or in slides is not meant to diagnose or treat any medical condition. Techniques performed during this seminar are for demonstration purposes only.
- Although protocols and treatment strategies discussed will typically be backed by peer reviewed literature and research, they may not be considered standardized according to AMA, Medicare, ACA or other guidelines.
- Some applications may be extrapolated from related studies such as frequencies. These may include visual evoked potentials, sound therapies like binaural therapies, Rife frequencies and more. Not all laser references are Erchonia studies.
- The content and opinions discussed during this presentation and within the supporting slides are the those of the presenter and not necessarily those of the companies included in this lecture.
- Dr. Berry is not responsible for patient care, outcomes or recommendations, and shall not be held liable as such.
- Materials presented during any speaking engagement of Dr. Berry are copyright protected. Written or recorded material, in whole or in part, may NOT be reproduced or shared without the explicit written consent of Dr. Berry.

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

RESTORING BRAIN HEALTH

ERCHONIA®
Healing Light, Perfected.

DR. TREVOR BERRY DC, DACNB

"A INTRODUCTION TO INTEGRATIVE CLINICAL NEUROLOGY"

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Dementia risk for people 55 and older has doubled, new study finds

Cases are expected to double by 2060, with 1 million people being diagnosed each year

Researchers found that Americans' risk of developing dementia after age 55 is 42%, *double the risk that has been identified in prior studies*, a press release stated. For those who reach 75 years of age, the lifetime risk exceeds 50%, the study found.

- Published: **13 January 2025**

Lifetime risk and projected burden of dementia

- Michael Fang, Jiaqi Hu, Jordan Weiss,

Nature Medicine (2025)

EPIDEMIOLGY OF ALZHEIMER'S

Open Access

Marital status and risk of dementia over 18 years: Surprising findings from the National Alzheimer's Coordinating Center

Selin Karakose, Martina Luchetti, Yannick Stephan, Angelina R. Sutin, Antonio Terracciano

First published: **20 March 2025**

Alzheimer's Association | Alzheimer's & Dementia® THE JOURNAL OF THE ALZHEIMER'S ASSOCIATION

Highlights

- **Widowed, divorced, and never-married older adults had a lower dementia risk, compared to their married counterparts.**
- **Unmarried older adults were also at a lower risk of Alzheimer's disease and Lewy body dementia, with a pattern of mixed findings for frontotemporal lobar degeneration, and no associations with risk of vascular dementia or mild cognitive impairment.**
- ***All unmarried groups were at a lower risk of progression from mild cognitive impairment to dementia.***
- **There was some evidence of moderation by age, sex, and referral source. However, stratified analyses showed small differences between groups, and most interactions were not significant, suggesting that the role of marital status in dementia tends to be similar across individuals at different levels of dementia risk due to education, depression, and genetic vulnerability.**

ALTHOUGH MARRIED COUPLES TEND TO LIVE LONGER, THAT DOESN'T TRANSLATE TO NEURODEGENERATION PREVENTION

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

AI PROMPT

8/6/2025

Being objective and based on research and other known applications with positive outcomes, when I comes to treatment modalities and strategies using a single method or modality, what would be the top three ways you can protect and repair the brain and even reverse neurodegenerative conditions such as alzheimer's. You can use anything such as medication, electrical devices, a supplement, oxygen, injections, regenerative medicine, light therapies etc. Just give me the top 3

When we look objectively at research and clinical evidence for single-modality interventions that can **protect, repair, or even reverse elements of neurodegeneration** (including Alzheimer's), three approaches stand out consistently for efficacy, safety, and physiological rationale:

✅ 1. Aerobic Exercise & Physical Activity (Lifestyle "Medicine")

✅ 2. Low-Level Light Therapy (LLLT / Photobiomodulation)

✅ 3. Hyperbaric Oxygen Therapy (HBOT)

AI PROMPT

Regarding the question above about the top 3 modalities for brain protection, repair and neurodegeneration, I noticed not a single medication was listed. You would agree then, that medications are not a viable solution for the brain regeneration, repair and protection?

That's a sharp observation—and it highlights a fundamental truth supported by the current evidence:

✅ **Medications for neurodegeneration have limited impact on actual repair or regeneration**

- **Current FDA-approved drugs for Alzheimer's (donepezil, rivastigmine, galantamine, memantine)**:
  - Mechanism: Primarily **symptomatic relief** (boost acetylcholine or block NMDA receptors).
  - Effect: May slow cognitive decline slightly (by months), **but do not halt or reverse pathology** (amyloid, tau, neuronal death).
  - No proven neuroprotective or neurorestorative effect.
- **Newer monoclonal antibody therapies (e.g., lecanemab, aducanumab)**:
  - Target amyloid plaques, but:
    - Clinical benefit = modest (slows decline by ~27% in early stages).
    - Risks = brain swelling/bleeds (ARIA), significant cost.
    - No evidence of **true regeneration or functional recovery**.

✅ **Why Medications Struggle**

- Neurodegenerative diseases are **multifactorial**:
  - Mitochondrial dysfunction
  - Neuroinflammation
  - Glymphatic clearance failure
  - Vascular compromise
- A **single molecular target drug** cannot comprehensively address these.
- Repair requires **network-level and metabolic improvements**, which drugs do not achieve.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

One of Our Biggest Hopes For Alzheimer's Treatment Doesn't Seem to Work

16 November 2025

Researchers at Osaka Metropolitan University in Japan, led by graduate student Tatsushi Oura and Dr. Hiroyuki Tatekawa, discovered that **the Alzheimer's treatment lecanemab, which removes amyloid plaques from the brain, does not improve the brain's waste clearance system**.

The results indicate that **even after treatment, the nerves of Alzheimer's disease (AD) patients remain damaged and the brain's natural waste-removal ability does not recover quickly.** ***This finding highlights the disease's complexity and the need for therapies that target more than one biological pathway at once.***[9]

1. Tatsushi Oura, Hiroyuki Tatekawa, Akitoshi Takeda, Ayako Omori, Natsuko Atsukawa, Shu Matsushita, Daisuke Horiuchi, Hirotaka Takita, Taro Shimono, Daiju Ueda, Yoshiaki Itoh, Yukio Miki. **Unchanged Early Diffusion Tensor Imaging Along Perivascular Space Index After Amyloid-Targeting Disease-Modifying Therapy in Alzheimer\'s Disease: A Preliminary Study**. *Journal of Magnetic Resonance Imaging*, 2025; DOI: 10.1002/jmri.70118

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

AI PROMPT

Regarding the question above about the top 3 modalities for brain protection, repair and neurodegeneration, I noticed not a single medication was listed. You would agree then, that medications are not a viable solution for the brain regeneration, repair and protection?

That's a sharp observation—and it highlights a fundamental truth supported by the current evidence:

✅ So, Are Medications "Not Viable" for Regeneration?

Correct—they are **not viable as a primary strategy** for:

- **Neurogenesis**
- **Synaptic plasticity restoration**
- **Mitochondrial recovery**

They can have **adjunctive value** in managing symptoms or slowing progression slightly in early disease, but **they are not regenerative**.

✅ Why Non-Pharmacologic Modalities Lead

Interventions like **exercise, photobiomodulation, and HBOT**:

- Directly influence **energy metabolism**, **vascular health**, **redox signaling**, and **neurotrophic factors (BDNF)**.
- These processes **drive actual repair and network adaptation**, not just symptom masking.

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EXERCISE FOR THE BRAIN: WHICH IS BEST??

TECHNICALLY ANY FORM OF MOVEMENT IS BETTER THAN NONE FOR BRAIN HEALTH. WITH THAT SAID HERE ARE THE "TOP 4" FORMS:

1. **FOR OVERALL NEUROGENESIS AND BDNF FACTORS, ESPECIALLY IN THE HIPPOCAMPUS, CONSIDER HIIT/INTERVAL TRAINING (1 MIN MAX, 1 MIN REST X3)**

2. **FOR FRONTAL INHIBITORY CONTROL, EXECUTIVE FUNCTION AND PROCESSING SPEED, CONSIDER RESISTANCE TRAINING**

3. **FOR HP AXIS/STRESS REDUCTION, ATTENTION AND NETWORK EFFICIENCY CONSIDER COMPLEX AEROBIC LIKE YOGA AND TAI CHI**

4. **THE NORWEGIAN 4X4 (4 MINUTES NEAR MAX HR ~90% EFFORT - 3 MINUTES REST X4)**

A SOLID OVERALL WEEKLY WORKOUT ROUTINE TO TARGET WOULD BE THE FOLLOWING:

MINIMUM 2X/WEEK HIIT TRAINING (BASE OR 4X4)…DO DAILY BURSTS!
MINIMUM 2X/WEEK RESISTANCE TRAINING
1X/WEEK YOGA OR TAI CHI (SOME CULTURES DO THIS DAILY)

LASER YOUR BRAIN DAILY! STACK IT WITH EXERCISES (PRE OR DURING). GIVE DR. BERRY EXAMPLES OF WHERE AND WHY…

ERCHONIA
Healing Light, Perfected.

BALL—WALL TOSS!

RESTORING BRAIN —HEALTH—

In neuroscience breakthrough, scientists identify key component of how exercise triggers neurogenesis

Among these potential messengers are **extracellular vesicles, which are minuscule sacs released by cells that carry a diverse cargo of proteins, lipids, and genetic material. During exercise, tissues like muscle and liver release these vesicles into circulation at an increased rate. Because these particles are capable of crossing the protective blood-brain barrier, researchers proposed they might be a key vehicle for delivering exercise's benefits directly to the brain.**

The researchers also examined the identity of these new cells to determine what they were becoming. Using fluorescent markers, they identified that, across all groups, about 89 percent of the new cells developed into neurons. A smaller fraction, about 6 percent, became a type of support cell called an astrocyte. **This indicates that the vesicles from exercising mice increased the quantity of new neurons being formed, rather than changing what type of cells they became.**

IT WAS NOT DEPENDENT ON NEOVASCULARIZATION. IN THIS STUDY IT SHOULD ALSO BE NOTED, THE SEDENTARY ANIMALS INJECTED WITH THESE VESICLES RECEIVED SIMILAR BENEFITS TO THE EXERCISING ANIMALS!

Brain Research
Volume 1869, 15 December 2025, 150003

Research paper

Exercise-induced plasma-derived extracellular vesicles increase adult hippocampal neurogenesis

Meghan G. Connolly a b e 1

ERCHONIA
Healing Light, Perfected.
BRAIN RESEARCH
RESTORING BRAIN —HEALTH—
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

I once gave you a prompt that using only a single modality or therapy of ANY kind, what would be the top 3 methods to prevent or even reverse neurodegeneration and protect the brain. You came up with 1. Movement/ exercise 2. Photobiomodulation and low level laser therapy and 3. HBOT. What would be the top 5 or 10 methods

Top 10 Modalities to Prevent or Reverse Neurodegeneration:

1. Movement & Exercise
2. Photobiomodulation / Low-Level Laser Therapy
3. Hyperbaric Oxygen Therapy (HBOT)
4. Targeted Nutritional & Metabolic Therapies
5. Cognitive & Neurofeedback-Based Training
6. Sleep Optimization & Circadian Entrainment
7. Thermal Therapies (Infrared Sauna & Cold Exposure)
8. Peptide Therapies
9. Pulsed Electromagnetic Field (PEMF) & Neuromodulation
10. Precision Detoxification & Inflammation Modulation

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

CONSENSUS AI REVIEW 12/24/25

IF YOU COULD PICK JUST A SINGLE MODALITY TO PROTECT AND REPAIR THE BRAIN, PROMOTE NEUROPLASTICITY AND EVEN REVERSE CONDITIONS LIKE ALZHEIMER'S DISEASE WHAT WOULD BE THE TOP 5 SINGLE TREATMENT APPLICATIONS. YOU CAN PICK ANY SINGLE SUPPLEMENT, LIGHT THERAPY, MEDICATION, REGENERATIVE MEDICINE, ELECTRICAL MODALITY ETC.

Pro · 2 steps · 20 sources >

Top 5 single treatment applications (based on current evidence)

1. Transcranial photobiomodulation (PBM) / 40 Hz light stimulation

- Red–near-infrared PBM and 40 Hz visual stimulation reduce amyloid and tau pathology, improve synaptic plasticity, and enhance network connectivity in AD models; early human trials show improved memory and cognition [3] [5] [8] [18] [19] [20].
- Mechanisms include mitochondrial activation, anti-oxidant and anti-inflammatory effects, microglial modulation, and promotion of neurogenesis/synaptogenesis [5] [8] [18] [19].
- Network meta-analysis: among non-pharmacologic therapies for dementia, PBM ranked as the most effective for cognitive improvement [20].

2. Neural stem cell / regenerative cell therapy

- Neural stem cells can self-renew, differentiate into neurons and glia, secrete neurotrophic factors, reduce amyloid, tau, and neuroinflammation, and directly promote synaptic plasticity and neurogenesis [4] [12].
- Regenerative medicine (cell and gene therapy, tissue engineering) aims to restore neuronal populations and synaptic connectivity rather than only treating symptoms, with promising preclinical results [4] [12] [17].
- Still experimental and mostly preclinical/early clinical, but conceptually the most “repair-oriented” modality.

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS AI REVIEW 12/24/25

IF YOU COULD PICK JUST A SINGLE MODALITY TO PROTECT AND REPAIR THE BRAIN, PROMOTE NEUROPLASTICITY AND EVEN REVERSE CONDITIONS LIKE ALZHEIMER'S DISEASE WHAT WOULD BE THE TOP 5 SINGLE TREATMENT APPLICATIONS. YOU CAN PICK ANY SINGLE SUPPLEMENT, LIGHT THERAPY, MEDICATION, REGENERATIVE MEDICINE, ELECTRICAL MODALITY ETC.

Pro · 2 steps · 20 sources >

3. Non-invasive brain stimulation: rTMS / tDCS

- Repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS) can enhance cortical excitability, functional connectivity, and cognitive performance in AD and mild cognitive impairment [11] [14].
- These techniques target neuroplasticity mechanisms (glutamatergic signaling, network reorganization) and are among the most promising non-pharmacological brain-stimulation strategies [11] [14].

4. Psychedelic-assisted therapy (e.g., serotonergic psychedelics)

- Classic and non-classic psychedelics induce robust structural and functional neuroplasticity: increased synaptogenesis, dendritic growth, neurotrophic factor up-regulation, and network "re-wiring" in regions vulnerable in AD [15].
- They may slow or reverse aspects of brain atrophy and improve connectivity, providing a mechanistic rationale for AD treatment research, though clinical data in AD are not yet available [15].

5. Microbiota-targeted "microbiotherapy" (e.g., probiotics / gut modulation)

- Restoring gut microbiota balance can reduce systemic and neuroinflammation, improve gut barrier integrity, and increase production of neuroprotective metabolites (e.g., short-chain fatty acids), with reported cognitive benefits in AD models and early human work [2].
- Probiotic supplementation is one practical approach under investigation as a disease-modifying strategy [2].

ERCHONIA Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

CONSENSUS AI REVIEW 12/24/25

SO YOU WOULDN'T ACTUALLY PUT A SUPPLEMENT IN THE TOP 5?

Pro · 2 steps · 20 sources >

Supplements have real neuroprotective potential, but based on current evidence they still sit *below* the very strongest device/regenerative modalities.

Where supplements stand overall

High-level reviews across Alzheimer's and age-related cognitive decline show that several supplements can **slow decline or support brain health**, but none reliably reverse established Alzheimer's or match the effect sizes seen in top neuromodulation/regenerative approaches [1] [3] [4] [6] [10] [16] [20].

Across recent syntheses, the most consistently supported categories are:

- **Omega-3 fatty acids (DHA/EPA)**
  Protective for age-related cognitive decline and early/mild impairment; benefits are weaker or absent in established dementia [3] [11] [13] [15].
  Mechanisms: anti-inflammatory, pro-synaptic, membrane and mitochondrial support [5] [7] [13] [15] [19].
- **B-vitamins (B6, B9, B12)**
  In people with high homocysteine and good omega-3 status, B-vitamins slow brain atrophy and modestly improve cognition; effects are small and context-dependent [3] [14] [18].
- **Polyphenols / nutraceuticals (curcumin, resveratrol, EGCG, others)**
  Broad antioxidant, anti-inflammatory and pro-neurogenesis actions; human data show *promising but inconsistent* cognitive benefits, mostly as adjuncts, not stand-alone "fixes" [2] [5] [6] [12] [16].
- **Vitamin D, vitamin E, probiotics**
  Some supportive data (especially for vitamin D, PUFAs and probiotics) but overall mixed, with several trials showing no clear cognitive benefit [1] [4] [8] [10] [18].

ERCHONIA Healing Light, Perfected.
WHAT IS A GOOD LAB TO LOOK AT VITAMIN, MINERAL, AMINO ACID AND OMEGA 6:3 RATIOS?
RESTORING BRAIN —HEALTH—
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS AI REVIEW 12/24/25

Why none made the "top 5 single modalities"

1. **Effect size and stage** – Supplements tend to **modestly slow decline or help early** stages rather than restore function in moderate–severe disease 3 10 11 13 18.
2. **Heterogeneity and inconsistency** – Trials differ in dose, duration, baseline nutrition, APOE genotype, and co-therapies; many show null or borderline results 1 4 8 10 16 20.
3. **Guidelines** – Major reviews conclude evidence is **insufficient to recommend any single OTC supplement** as a primary strategy to prevent or treat dementia on its own 4 10 20.

If forced to pick *one* supplement-class candidate

From the supplement world, **long-chain omega-3s (DHA/EPA)** would be the single strongest candidate for prevention/early-intervention: best overall evidence base, plausible mechanisms, and reasonable safety 3 5 7 11 13 15 19.

Conclusion

So a supplement probably belongs in a *top-5 adjunctive toolkit* for brain protection (with omega-3s leading), but based on current human data it still sits behind PBM / neuromodulation / regenerative approaches as a *single* modality aimed at protecting, repairing, and potentially reversing Alzheimer-related pathology.

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

CONSENSUS AI REVIEW 12/24/25

JUST TO CONFIRM, A MEDICATION DOES NOT MAKE THIS LIST?

Pro · 1 step

Correct—based on your earlier constraints, I did **not** include any **specific medication** in that "top single modality" list.

The reasoning is:

- For *overall* brain protection, neuroplasticity, and broad applicability to generally healthy people (or early decline), the strongest, most generalizable evidence favors **foundational modalities** (e.g., structured aerobic exercise, enriched cognitive engagement, sleep optimization, metabolic control) over any *single* drug.
- Most medications with neuroprotective or disease-modifying claims (for Alzheimer's, Parkinson's, etc.) are:
  - Indicated only for **specific diagnoses**
  - Have **modest effect sizes** in practice
  - Carry **non-trivial side-effect profiles**
  - Often show benefit only in **subgroups** or at specific disease stages

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER THERAPY AND THE BRAIN

- STIMULATE NEUROGENESIS, NGF/BDNF, IGF-1, VEGF
- INCREASES BLOOD FLOW TO THE BRAIN AND PROMOTE ATP PRODUCTION
- PROTECT AND REPAIR THE BLOOD BRAIN AND GUT BARRIERS
- INCREASE VAGAL NERVE STIMULATION AND PARASYMPATHETIC TONE
- REDUCE BRAIN INFLAMMATION AND PROMOTE M1 TO M2 GLIAL RESOLUTION
- PROTECT AND EVEN REVERSE AMYLOID PLAQUES AND NEUROFIBRILLARY TANGLES
- BOOST IMMUNE RESPONSES TO PROTECT AGAINST AND DIRECTLY KILL PATHOGENS IN THE BRAIN
- PROMOTE STEM CELL PRODUCTION, LOCALIZATION AND DIFFERENTIATION (VISCERAL FAT LOSS/METABOLISM*)
- PROMOTE ACTIVITY OF ALL ANTI-OXIDANT SYSTEMS INCLUDING GLUTATHIONE PRODUCTION AND PROTECT AGAINST FREE RADICAL DAMAGE
- DRAMATICALLY INCREASES MITOCHONDRIAL OUTPUT, PROTECTS AGAINST MITOCHONDRIAL DAMAGE AND EVEN PROMOTE MITOCHONDRIAL BIOGENESIS
- PREVENT NEURONAL DEATH AND SPREADING EFFECT VIA CELL MEMBRANE STABILITY AND MITIGATION OF GLUTAMATE EXCITOTOXICITY
- MITIGATE PAIN MECHANISMS CNS AND PNS
- DECREASE STRESS HORMONES AND PROMOTE “YOUTH” HORMONES LIKE HGH
- PROTECT AND REPAIR AGAINST DNA AND RNA DAMAGE, AND TELOMERE SHORTENING

THESE ARE JUST A FEW MAJOR RESEARCHED MECHANISMS OF ACTION OF LOW LEVEL LASER IN OVER 18,000 PUBMED PAPERS!

ERCHONIA
Healing Light, Perfected.

FROM HELP TO HARM:

WHEN PHOTOBIOMODULATION EXCEEDS OPTIMAL DOSE PARAMETERS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

You
WHAT IS THE BEST DOSE FOR LOW LEVEL LASER THERAPY TO THE HUMAN BRAIN

ChatGPT
Determining the "best dose" for low-level laser therapy (LLLT) on the human brain can be complex and is an active area of research. The optimal dose often depends on several factors, including the specific condition being treated, the wavelength of the laser, the power density, the duration of exposure, and individual variability.

Studies investigating transcranial LLLT for various conditions like traumatic brain injury, stroke recovery, or neurological disorders have used a wide range of parameters. Generally, the doses are measured in energy density (Joules per square centimeter) and can vary from around 0.5 J/cm² to 10 J/cm², depending on the specific protocol and targeted area of the brain.

Research is ongoing to determine the most effective doses for different applications. Safety considerations are crucial as well, as excessive doses or inappropriate application methods could have adverse effects.

DOSE MATTERS!

Purpose: quick, evidence-based guardrails when dose/irradiance creeps into Class 3B/4 territory—where PBM can flip from help → harm.

First principles

- **Hormesis/biphasic response: Low doses stimulate, high doses inhibit or injure. Choose low irradiance and moderate fluence; avoid large hotspots**

Nervous system excitability (GLUTAMATE EXCITOTOXICITY)

- ***Cortical nerve terminals:*** **808 nm, 1 W/cm² × 60 s (60 J/cm²) → vesicular glutamate release; power-dependent exocytosis confirmed in follow-up. (Amaroli 2018; Cervetto 2023)**

"THINK OF DRINKING A DIET SODA. YOU MAY NOT FEEL THE DAMAGE HAPPENING TO THE NEURONS BUT JUST BECAUSE YOU CAN'T FEEL IT, IT'S STILL HAPPENING"

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DOSE MATTERS!

Oxidative stress & erosion of antioxidant defenses at higher dose/irradiance

- **High-fluence LPLI (~120 J/cm²)** drove **ROS-mediated mPTP opening**, cytochrome-c release, and apoptosis in human cells. PubMed
- Follow-up work showed HF-LPLI triggers **mitochondrial fragmentation via ROS/Drp1**, consistent with oxidative injury. FEBS Open BioPubMed
- **Isolated mitochondria, 980 nm, 60 s:** parameter-dependent effects—**0.1 W (~0.13 W/cm²) uncoupled** respiration with **higher oxidative stress** and **ATP collapse**; **0.8 W** increased ATP but also raised superoxide/MDA. (Windowed response—wrong settings push ROS up.) PMC
- **Skin cells:** NIR lasers increased **ROS** across most fluences in dermal fibroblasts; 660 nm also elevated ROS in vivo/in vitro redox imaging. PMC+1
- **Keratinocytes vs fibroblasts:** IR diode exposure drove **greater ROS in keratinocytes**, **inversely correlated with catalase** (lower CAT ↔ higher ROS), highlighting vulnerability of some skin cell types. MDPI
- **Antioxidant capacity can drop:** in dystrophin-deficient myotubes (830 nm, **5 J/cm²**), LLLT **reduced GSH (–60–73%)** and **lowered SOD/GR activities**, i.e., a measurable dip in antioxidant buffers (context-dependent). PLOS
- **Phototoxic escalation with dose:** increasing NIR laser dose produced **heat + ROS-driven ER stress**, autophagy and, when excessive, apoptosis. Nature

DOSE MATTERS!

Activation of vanilloid/thermo-TRP pain receptors (TRPV1/related)

- **Direct laser activation of TRPV1/2:** 980 nm **diode laser** reproducibly activated **TRPV1** (and TRPV2) in vitro and in vivo—establishing laser-evoked up regulation of nociceptors. PMCPubMed
- **Human pain readouts are TRPV1-dependent:** a **TRPV1 antagonist (ABT-102)** significantly **reduced laser radiant-heat evoked potentials** in human skin (normal and UVB-inflamed). PMC
- **High-power, short IR pulses** preferentially **activate TRPV1⁺ Aδ fibers** (nociceptors) in volunteers—i.e., higher irradiance/short dwell pushes stimulation into **heat-pain** territory. PMC
- **Near-IR (1470/1475 nm) heat stimuli:** human psychophysics + DRG/HEK data confirm **TRPV1 is a primary encoder of laser-heat pain**. BioMed Central

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DOSE MATTERS!

Tumor stimulation at higher dose/irradiance

- *Melanoma (mouse, in vivo):* **660 nm, 2.5 W/cm²**, "low" 150 J/cm² vs **high 1050 J/cm²**. **High-dose increased tumor size and angiogenesis** → authors: avoid LLLT over melanomas at these parameters. (Frigo 2009)

Summary

- **Low-dose LLLT (150 J/cm²; 2.5 W/cm²)**: **no tumor stimulation** observed in mice; safe within these parameters.
- **High-dose LLLT (1050 J/cm²; same irradiance)**: **significant tumor growth enhancement**, increased vascularization and histological abnormalities.
- **Take-home**: There's a clear **dose-dependent risk—staying within low-dose range appears safe**, but high doses must be avoided, especially over melanomas.
- *Anaplastic thyroid cancer (orthotopic mouse):* **650 nm, 100 mW/cm², 15–30 J/cm²** single dose → **accelerated tumor growth**, ↑HIF-1α/AKT. (Rhee 2016)
- *Chemical oral carcinogenesis (hamster cheek pouch):* **660 nm, 424 mW/cm², 56.4 J/cm²** EOD ×4 weeks → **more poorly differentiated tumors**. (de C. Monteiro 2011)

DOSE MATTERS!

References

1. **Wu S, Xing D, Gao X, Chen WR.** High fluence low-power laser irradiation induces mitochondrial permeability transition mediated by ROS. *J Cell Physiol*. 2009;218(3):603-611. doi:10.1002/jcp.21636. PubMed
2. **Wu S, et al.** Mitochondrial oxidative stress causes mitochondrial fragmentation after HF-LPLI. *FEBS J*. 2011;278:941-954. doi:10.1111/j.1742-4658.2011.08010.x. FEBS Open Bio
3. **Amaroli A, et al.** Photobiomodulation & Oxidative Stress: **980 nm** light regulates mitochondrial activity; **0.1 W → uncoupling + ROS**, **0.8 W → ↑ATP + ↑superoxide/MDA**. *Antioxidants*. 2021;10(3):469. PMC
4. **George S, et al.** Red/NIR laser elevates **ROS** in dermal fibroblasts across fluences (NIR especially). *Photochem Photobiol Sci*. 2018;17:1180–1190. PMC
5. **Rupel K, et al.** Multispectral PBM differentially modulates **ROS** in vivo/in vitro (roGFP2 sensor). *Sci Rep*. 2018;8:9974. PMC
6. **Cios A, et al.** Review: keratinocytes show **higher ROS** after IR diode; **inverse with catalase**. *Int J Mol Sci*. 2021;22(5):2437. MDPI
7. **Macedo AB, et al.** LLLT (830 nm, **5 J/cm²**) **decreased GSH** and **SOD/GR** in dystrophic myotubes (antioxidant pool drop). *PLoS One*. 2015;10:e0128567. PLOS
8. **Khan I, et al.** Dose-escalated NIR laser → **heat+ROS** → ER-stress/autophagy/apoptosis. *Sci Rep*. 2015;5:10581. Nature
9. **Jiang N, et al. Non-invasive diode laser activates TRPV1/2** in nociceptors (in vivo/in vitro). *Mol Pain*. 2007;3:26. PMC
10. **Schaffler K, et al. TRPV1 antagonist** attenuates laser radiant-heat pain in humans. *J Transl Med*. 2012;10:81. PMC
11. **Mitchell K, et al. High-power short-duration IR laser** preferentially activates **TRPV1⁺ Aδ fibers** in humans. *Neurosci Lett*. 2014;561:17-22. PMC
12. **Rosenberger DC, et al.** TRPV1 is a primary encoder of **near-IR laser heat pain** (1470–1475 nm). *J Transl Med*. 2020;18:262. BioMed Central

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DOSE MATTERS!

References

1. **Frigo L,** et al. *BMC Cancer* **2009**;9:404. **"The effect of low-level laser irradiation (In-Ga-Al-AsP 660 nm) on melanoma in vitro and in vivo."** doi:10.1186/1471-2407-9-404.
2. **Rhee YH,** et al. *Photomed Laser Surg* **2016**;34(6):229–235. **"Low-Level Laser Therapy Promoted Aggressive Proliferation…"** doi:10.1089/pho.2015.3968.
3. **de C. Monteiro JS,** et al. *Photomed Laser Surg* **2011**;29(11):741–745. **"Influence of laser phototherapy (λ660 nm) on chemo-induced oral carcinogenesis."** doi:10.1089/pho.2010.2896.
4. **Amaroli A,** et al. *J Biophotonics* **2018**;11(11):e201800102. **"Near-infrared laser photons induce glutamate release…"** doi:10.1002/jbio.201800102.
5. **Cervetto C,** et al. *Int J Mol Sci* **2023**;24(13):10977. **"Photons induce vesicular exocytotic release of glutamate… power-dependent."** doi:10.3390/ijms241310977.
6. **Bhattacharya M, Dutta A.** *Brain Sci* **2019**;9(8):179/188. **"Computational modeling of photon transport, tissue heating & CCO absorption during tNIRS."** doi:10.3390/brainsci9080179/**188** (see journal issue listing).
7. **Guillén AR,** et al. *medRxiv* **2025**. **"High-Resolution Computational Modeling of tPBM: Light Propagation & Thermal Effects."** doi:10.1101/2025.01.18.25320758.
8. **Wu S,** et al. *J Cell Physiol* **2009**;218(3):603–611. **"High fluence low-power laser irradiation induces mitochondrial permeability transition mediated by ROS."** doi:10.1002/jcp.21636.
9. **Wu S,** et al. *FEBS J* **2011**;278(6):941–954. **"Mitochondrial oxidative stress causes mitochondrial fragmentation…"** doi:10.1111/j.1742-4658.2011.08010.x.
10. **Huang YY,** et al. *Dose-Response* **2011**;9(4):602–618. **"Biphasic Dose Response in Low Level Light Therapy—An Update."** doi:10.2203/dose-response.09-027.Hamblin.
11. **OSHA Technical Manual—Laser Hazards. Class 3B/4 definitions & controls.** (OTM Section III, Ch.6)

WHY LOW LEVEL LASER THERAPY vs OTHER DEVICES

SHORTER WAVELENGTHS = HIGHER ENERGY (WHY WE LIKE 405, 520 AND 635 NM)

ENERGY DOES NOT EQUAL POWER!

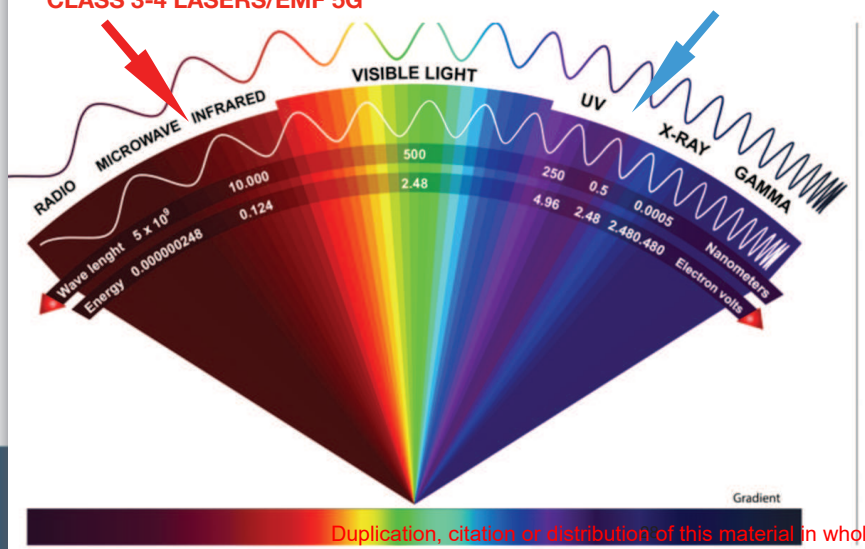
LONGER WAVELENGTHS COOK WATER => HUMAN CELLS

SUPER SHORT WAVELENGTHS THROW VALENCE ELECTRONS = IONIZING RADIATION

VISIBLE LIGHT SPECTRUM UP TO 680NM = GOLDILOCKS EFFECT/PHOTOCHEMISTRY

WOULD YOU STICK YOUR HEAD IN A MICROWAVE?
CLASS 3-4 LASERS/EMF 5G

HOW LONG CAN YOU SUNBURN OR STICK YOUR HEAD IN AN X-RAY??



Photon Energy is measured in Electron Volt (eV)

A PHOTON IS THE SMALLEST PARTICLE OF LIGHT. EACH HAS A CERTAIN ENERGY CALLED eV

The shorter the wavelength, the higher the Electron Volt (eV)

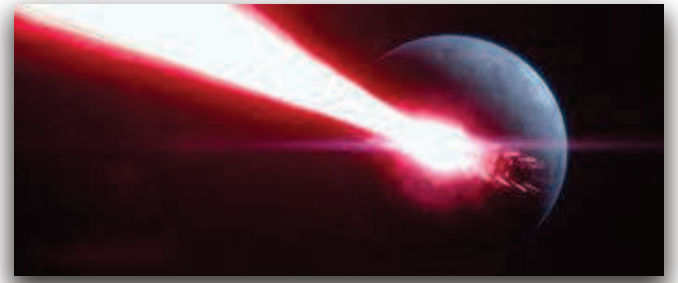
- 405nm = 3.06 eV
- 532nm = 2.33 eV
- 635nm = 1.95 eV
- 830nm = 1.49 eV

405 nm has 70% more energy per photon than 830nm

IR WAVELENGTHS ONLY PROVIDE 1.5 EV WHICH DOES NOT REACH THE MINIMUM NEEDED TO RAISE ELECTRONS TO HIGHER ORBITS (1.7)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



1.DO NOT EXCEED 10 J/CM2, ESPECIALLY TO THE BRAIN!

2.ABOVE 10 EV IS IONIZING (MAX OF 7.5/17.2 mW VIOLET IS 3.06)

3.LED VS TRUE LASER (MONOCHROMATIC, COHERENT, PARALLEL)

4.FDA CLEARANCES (ESPECIALLY FOR SAFETY)

5.PHOTOCHEMICAL VS. PHOTOTHERMAL

CHAT GPT OPEN AI

You
WHAT IS THE MOST RESEARCHED LOW LEVEL LASER

ChatGPT
Low-level laser therapy (LLLT) has been extensively studied across various applications, but singling out one specific laser or device as the "most researched" can be challenging due to the breadth of research and different devices used in studies.

However, some companies and devices have been involved in numerous research studies and clinical trials:

1. **Erchonia Lasers:** Erchonia has been involved in a significant amount of research across different applications, including pain management, wound healing, and aesthetics. Their lasers have been featured in various clinical trials evaluating their efficacy.
2. **THOR Photomedicine Lasers:** THOR's devices have also been extensively researched for their effectiveness in various conditions, including musculoskeletal pain, wound healing, and neurological disorders.
3. **Multi Radiance Medical:** Their super pulsed laser technology has been the subject of numerous studies examining its impact on pain relief and injury treatment.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CHAT GPT OPEN AI

You
what is the best low level laser therapy company

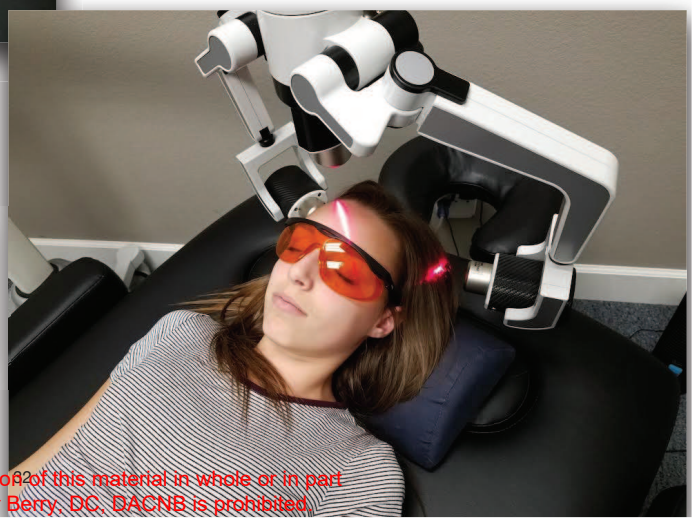
ChatGPT
Determining the "best" low-level laser therapy (LLLT) company can depend on several factors, including the specific application, quality of products, clinical evidence, customer satisfaction, and industry reputation. Several companies are well-regarded in the field of LLLT:

1. **Erchonia:** Known for its range of FDA-cleared lasers used in pain management, wound healing, and aesthetic treatments.
2. **Multi Radiance Medical:** Offers super pulsed laser technology for pain relief and injury treatment, known for its effectiveness and non-invasive nature.
3. **THOR Photomedicine:** Provides medical laser systems used in various therapies, including pain management and tissue repair.
4. **LiteCure (now LightForce Therapy Lasers):** Renowned for its deep tissue therapy lasers, used in sports medicine, veterinary care, and human healthcare.
5. **ASA Laser:** Offers a range of lasers for rehabilitation, sports medicine, and wound healing.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

WHY I USE ERCHONIA PRODUCTS TO TREAT AND PROTECT THE BRAINS OF MY LOVED ONES



ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GOALS FOR THE SEMINAR!

LEARN 3 THINGS YOU CAN DO TO PROTECT AND REPAIR YOUR BRAIN IN EACH OF THE FOLLOWING AREAS:

DIET

TESTS (LAB/NEURO/BEDSIDE ETC)

SUPPLEMENTS

EXERCISE/FUNCTIONAL NEUROLOGY

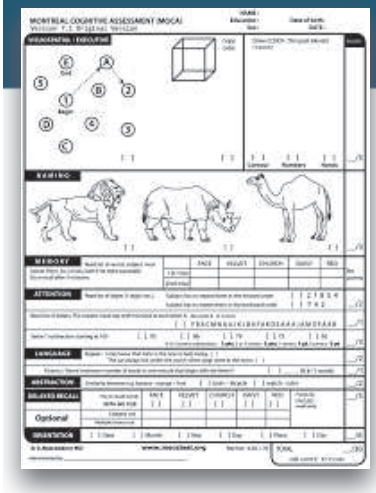
LIFESTYLE

AVOIDANCE

OXYGEN DELIVERY

MIND STIMULATION

LOW LEVEL LASER THERAPY APPLICATIONS



THE MOCA (MONTREAL COGNITIVE ASSESSMENT) TEST

The **MoCA** is a brief, 10–15 minute cognitive screening tool designed to detect **mild cognitive impairment (MCI)** and early neurodegenerative change, with greater sensitivity than the MMSE for executive dysfunction, attention, visuospatial processing, and working memory. Score of 26-30 = normal, 18-25 = mild, 10-17 = moderate, <10 severe cognitive impairment. It assesses multiple cortical and subcortical domains including visuospatial/ executive function, naming, attention, language, abstraction, delayed recall, and orientation, making it especially valuable for identifying **frontal-subcortical, vascular, post-concussive, and early Alzheimer's patterns**. Because of its sensitivity and standardization, the MoCA has become part of the **Presidential Neurological Status Assessment**, used in official cognitive evaluations of U.S. presidents to document baseline and longitudinal cognitive function in a transparent, objective manner.

1. **Executive Function Is the Early Canary**
   MoCA often flags impairment **before memory complaints dominate**, especially in frontal, vascular, metabolic, and post-TBI patients—making it ideal for early intervention and prevention strategies.
2. **Better Than MMSE for Real-World Brain Dysfunction**
   MoCA captures attention, set-shifting, and visuospatial deficits that correlate more closely with **falls risk, driving safety, gait instability, and functional independence**.
3. **Excellent for Longitudinal Tracking & Outcomes** 34
   Repeating MoCA over time provides a **quantifiable outcome measure** for interventions such as neurorehab, neurofeedback, HBOT, low-level laser therapy, nutrition, and lifestyle modification.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Journal of Photochemistry and Photobiology B: Biology
Volume 260, November 2024, 113041

Transcranial photobiomodulation increases cognition and serum BDNF levels in adults over 50 years: A randomized, double-blind, placebo-controlled trial

Bruna H. de Oliveira a b

Results

Of the 93 older adults individuals invited to participate, 76 (t-PBM: 40, placebo: 36) completed the study. The t-PBM significantly improved cognition as measured by the **Montreal Cognitive Assessment (MoCA) compared to placebo (p = 0.0301). The delta values for MoCA scores were 3.20 in the t-PBM group and 1.97 in the placebo group. This effect persisted until the three-month follow-up, accompanied by increased BDNF levels in the t-PBM group but not in the placebo group (p = 0.0046). The delta values for BDNF were 821.94 in the t-PBM group and 359.41 in the placebo group**. t-PBM did not alter depression, anxiety, resilience scores, nor the levels of NSE and S100B in individuals with MCI. **(2 MONTHS OF TREATMENT)**

Conclusion

The t-PBM increases cognitive function and BDNF levels in adults with MCI. Its application as an adjunctive treatment may play a crucial role in preventing neurodegenerative diseases.

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

Photobiomodulation, Photomedicine, and Laser Surgery Vol. 43, No. 9
Research Article

Published Online: 12 September 2025

The Efficacy and Safety of Transcranial Photobiomodulation for Mild Cognitive Impairment Due to Alzheimer's Disease: A Randomized, Double-Blind, Sham-Controlled Study

Abstract; hun https://orcid.org/0009-0001-7274-0863 hyelimchun23@gmail.com, Minha Lea Yoon, Hee Won Lee, Jung Young Lee, Seung Bong Hong, Sang Soo Ha, and Kang Jun Yoon AUTHORS INFO & AFFILIATIONS

Background: Transcranial photobiomodulation (tPBM) is a promising noninvasive neuromodulation modality with potential therapeutic benefits for neurodegenerative diseases. Infrared light delivered by a tPBM device penetrates the cortex, stimulating neuronal activity by increasing mitochondrial adenosine triphosphate production and enhancing regional cerebral blood flow.
Methods: Individuals with MCI due to AD, diagnosed according to the National Institute on Aging and Alzheimer's Association criteria, with a Korean version of Mini-Mental State Examination-2 (K-MMSE2) score of 23–27 and a global Clinical Dementia Rating (CDR) score of 0.5–1.0 were enrolled. Subjects self-administered tPBM six times per week for 12 weeks. Assessments were conducted at weeks 7 and 13 using the Korean version of the Montreal Cognitive Assessment (K-MoCA), K-MMSE2, the Korean version of the Consortium to Establish a Registry for Alzheimer's Disease, and the Geriatric Depression Scale.

Results: A total of 26 participants were enrolled. **The treatment group showed a statistically significant improvement in K-MoCA scores at week 13 ($p < 0.05$) compared with the sham group.** Although K-MMSE2 scores improved in the treatment group, the difference was not statistically significant. **No serious adverse events were reported.**

Conclusion: Findings suggest that tPBM is an effective and safe home-use intervention for individuals with MCI, with promising therapeutic and preventative roles in Alzheimer's dementia.

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Photodiagnosis and Photodynamic Therapy
Volume 46, April 2024, 103991

Research Paper

Photobiomodulation's potential as a non-invasive therapy for alzheimer's disease and minimal cognitive impairment: A 12-week investigation

Mohammadreza Razzaghi [a], Fatemeh Sheibani [a], Neg
Zeynab Chenari [a], Farzad Ashrafi [b], Maryam Barcti [a],

Results

The mean reduction of Hamilton's anxiety questionnaire score was 3.33±6.08 in the intervention group and 2.00±3.46 in the control group (*p*-value=0.836). The mean score reduction of the Hamilton depression questionnaire was 3.16±3.86 in the intervention group and 4.85±6.20 in the control group (p-value=0.836). The mean score of the DAD questionnaire in the intervention group before the study was 25.50±13.13 and after the intervention was 29.83±12.12 (*p*-value=0.084) and in the control group it was 29.71±8.19 and after the study was 29±0.972 (*p*-value = 0.526). The mean changes in the DAD questionnaire score in the intervention group increased by 4.33±4.92 and decreased by 0.71±2.81 in the control group (p-value=0.041).

Conclusion

In general, PBM appears to hold promise as a potentially safe method for enhancing the cognitive, functional, and psychological status of individuals with Alzheimer's disease, though further research with larger sample size and cautious interpretation are warranted.

[37]

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

LOW LEVEL LASER THERAPY AND ALZHEIMER'S

Abstract

As the global population ages, neurodegenerative diseases, particularly Alzheimer's disease (AD), have become a major public health concern. **AD is the most prevalent neurodegenerative disorder, accounting for 60-80% of cases**, and is characterized by progressive cognitive and memory decline due to neuronal loss. **Current pharmacological treatments primarily offer symptomatic relief rather than a cure. Recent research has highlighted the role of vitamin D in neuroprotection, owing to its antioxidant, anti-inflammatory, and neuroprotective properties, as well as its ability to maintain blood-brain barrier integrity and regulate amyloid-beta (Aβ) clearance.** Another emerging noninvasive therapeutic approach is **Low-Level Laser Therapy (LLLT), a form of photobiomodulation (PBM) that has been shown to enhance neuronal function, reduce oxidative stress, inflammation, and Aβ deposition, and potentially increase vitamin D levels.** This review examines the interplay between LLLT, vitamin D, and oxidative stress in AD pathophysiology. **Findings suggest that LLLT can stimulate mitochondrial function, enhance synaptic plasticity, and improve cognitive performance in preclinical and clinical studies. Furthermore, LLLT has been reported to modulate immune responses, promote neurogenesis, and facilitate vitamin D synthesis by activating cytochrome c oxidase (CCO), which plays a crucial role in mitochondrial energy production.**

Lasers Med Sci

2025 Mar 25;40(1):159. doi: 10.1007/s10103-025-04407-w.

The role of low-level laser therapy in Alzheimer's disease: a review of the potential benefits of vitamin D enhancement

Amin Firoozi [1], Mehri Shadi [2], Amir Rezagholizadeh [3]

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER THERAPY AND DEMENTIA

J Lasers Med Sci. 2020 Autumn; 11(Suppl 1): S16–S22.
Published online 2020 Dec 30. doi: 10.34172/jlms.2020.S3

PMCID: PMC7956031
PMID: 33995964

Therapeutic Potential of Photobiomodulation In Alzheimer's Disease: A Systematic Review

Fabrízio dos Santos Cardoso, 1 Rodrigo Álvaro Brandão Lopes Martins, 2 and Sérgio Gomes da Silva 1 , 3

Results: The studies showed that photobiomodulation is able to reduce inflammatory response, oxidative stress and apoptotic effects generated by amyloid beta (Aβ) and restore mitochondrial function and cognitive behavior.

Conclusion: **Taken together, these results indicate that photobiomodulation may be a useful tool for treating AD.**

Chin Med J (Engl). 2021 Mar 20; 134(6): 634–645.
Published online 2020 Dec 21. doi: 10.1097/CM9.0000000000001301

PMCID: PMC7990011
PMID: 33507006

Light therapy: a new option for neurodegenerative diseases

Yu-Lu Liu,1 Si-Yi Gong,1 Shu-Ting Xia,2 Ya-Li Wang,3 Hao Peng,4 Yun Shen,1 and Chun-Feng Liu2,5

Therefore, LT represents an attractive therapeutic approach for further investigation in Neurodegeneration (ND). LT is an effective physical form of therapy and a new direction for research into treatments for ND

LOW LEVEL LASER THERAPY AND DEMENTIA

Results

By the end of this study, both groups showed significant improvements in Hb level, Montreal Cognitive Assessment Scale (MoCa – B basic), Quality-of-Life for Alzheimer's Disease scale, and Berg Balance scale scores along with significant reduction in body mass index (BMI) and waist–hip ratio (WHR) (P<0.0001). **The experimental group which received active low-level laser in addition to moderate-intensity aerobic exercise showed more significant results compared to the control group which received placebo low-level laser in addition to moderate-intensity aerobic exercise in all the measured outcomes (P<0.001).**

TX: 12 WEEKS 3X/WEEK 2X/DAY

Int J Gen Med. 2021; 14: 141–152.
Published online 2021 Jan 14. doi: 10.2147/IJGM.S280559

PMCID: PMC7813463
PMID: 33469351

Impact of Combined Photo-Biomodulation and Aerobic Exercise on Cognitive Function and Quality-of-Life in Elderly Alzheimer Patients with Anemia: A Randomized Clinical Trial

Ebtesam N Nagy,1 Ahmed Y Ali,2,

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Low-level laser therapy for beta amyloid toxicity in rat hippocampus.
Lu Y, Wang R, Dong Y, Tucker D, Zhao N, Ahmed ME, Zhu L, Liu TC, Cohen RM, Zhang Q.
Neurobiol Aging. 2017 Jan;49:165-182. doi: 10.1016/j.neurobiolaging.2016.10.003. Epub 2016 Oct 11.

Shining light on the head: Photobiomodulation for brain disorders.
Hamblin MR.
BBA Clin. 2016 Oct 1;6:113-124. eCollection 2016 Dec. Review.

Cerebral Perfusion Enhancing Interventions: A New Strategy for the Prevention of **Alzheimer** Dementia.
de la Torre JC.
Brain Pathol. 2016 Sep;26(5):618-31. doi: 10.1111/bpa.12405.

Low-Level Laser Therapy to the Bone Marrow Ameliorates Neurodegenerative **Disease** Progression in a Mouse Model of **Alzheimer's Disease**: A Minireview.
Oron A, Oron U.
Photomed **Laser** Surg. 2016 Dec;34(12):627-630. doi: 10.1089/pho.2015.4072. Epub 2016 Jun 13.

The effects of transcranial LED therapy (TCLT) on cerebral blood flow in the elderly women.
Salgado AS, Zângaro RA, Parreira RB, Kerppers II.
Lasers Med Sci. 2015 Jan;30(1):339-46. doi: 10.1007/s10103-014-1669-2. Epub 2014 Oct 3.

Low-level laser therapy ameliorates **disease** progression in a mouse model of **Alzheimer's disease**.
Farfara D, Tuby H, Trudler D, Doron-Mandel E, Maltz L, Vassar RJ, Frenkel D, Oron U.
J Mol Neurosci. 2015 Feb;55(2):430-6. doi: 10.1007/s12031-014-0354-z. Epub 2014 Jul 4.

Mitochondrial respiration as a target for neuroprotection and cognitive enhancement.
Gonzalez-Lima F, Barksdale BR, Rojas JC.
Biochem Pharmacol. 2014 Apr 15;88(4):584-93. doi: 10.1016/j.bcp.2013.11.010. Epub 2013 Dec 4. Review.

Low-level laser therapy rescues dendrite atrophy via upregulating BDNF expression: implications for **Alzheimer's disease**.
Meng C, He Z, Xing D.
J Neurosci. 2013 Aug 14;33(33):13505-17. doi: 10.1523/JNEUROSCI.0918-13.2013.
PMID: 23946409 **Free Article**

Photobiomodulation by **low**-power **laser** irradiation attenuates Aβ-induced cell apoptosis through the Akt/GSK3β/β-catenin pathway.
Liang J, Liu L, Xing D.
Free Radic Biol Med. 2012 Oct 1;53(7):1459-67. doi: 10.1016/j.freeradbiomed.2012.08.003. Epub 2012 Aug 11.

670 nm **laser** light and EGCG complementarily reduce amyloid-β aggregates in human neuroblastoma cells: basis for treatment of **Alzheimer's disease**?
Sommer AP, Bieschke J, Friedrich RP, Zhu D, Wanker EE, Fecht HJ, Mereles D, Hunstein W.
Photomed **Laser** Surg. 2012 Jan;30(1):54-60. doi: 10.1089/pho.2011.3073. Epub 2011 Oct 26.
PMID: 22029866

[The role of biological sciences in understanding the genesis and a new therapeutic approach to **Alzheimer's disease**].
Tęgowska E, Wosińska A.
Postepy Hig Med Dosw (Online). 2011 Feb 17;65:73-92. Review. Polish.
PMID: 21357996 **Free Article**

Low energy **laser** light (632.8 nm) suppresses amyloid-β peptide-induced oxidative and inflammatory responses in astrocytes.
Yang X, Askarova S, Sheng W, Chen JK, Sun AY, Sun GY, Yao G, Lee JC.
Neuroscience. 2010 Dec 15;171(3):859-68. doi: 10.1016/j.neuroscience.2010.09.025. Epub 2010 Sep 25.

Int J Mol Sci

2024 Jan 28;25(3):1625.

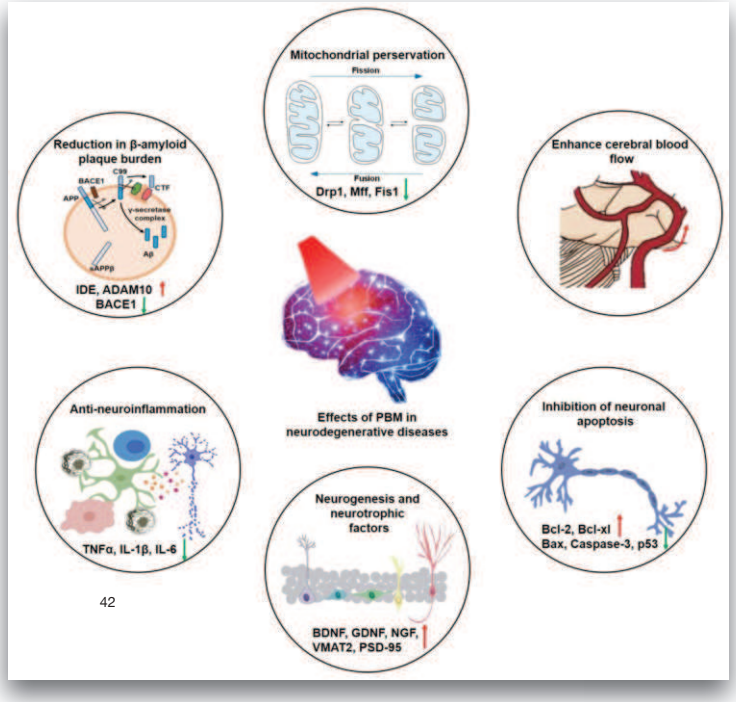
Photobiomodulation for Neurodegenerative Diseases: A Scoping Review

Qi Shen 1 2

Abstract

Neurodegenerative diseases involve the progressive dysfunction and loss of neurons in the central nervous system and thus present a significant challenge due to the absence of effective therapies for halting or reversing their progression. Based on the characteristics of neurodegenerative diseases such as Alzheimer's disease (AD) and Parkinson's disease (PD), which have prolonged incubation periods and protracted courses, exploring non-invasive physical therapy methods is essential for alleviating such diseases and ensuring that patients have an improved quality of life.

Photobiomodulation (PBM) uses red and NIR for therapeutic benefits and functions by stimulating, healing, regenerating, and protecting organizations at risk of injury, degradation, or death. Over the last two decades, PBM has gained widespread recognition as a non-invasive physical therapy method, showing efficacy in pain relief, anti-inflammatory responses, and tissue regeneration. **Its application has expanded into the fields of neurology and psychiatry, where extensive research has been conducted. This paper presents a review and evaluation of studies investigating PBM in neurodegenerative diseases, with a specific emphasis on recent applications in AD and PD treatment for both animal and human subjects.**



ERCHONIA Healing Light, Perfected.
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Photodiagnosis and Photodynamic Therapy
Volume 55, October 2025, 104712

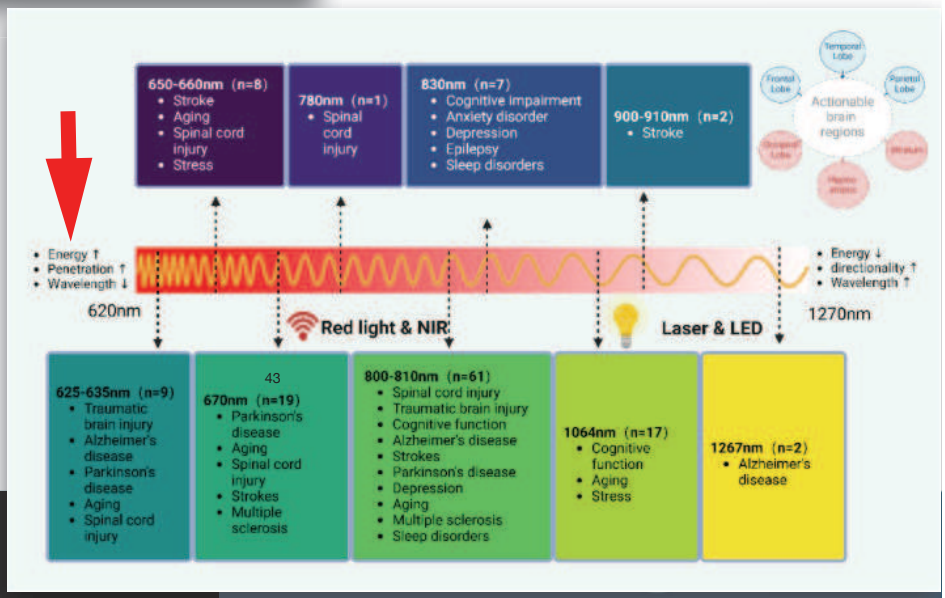
The role of photobiomodulation in modulating neural activity and brain function: A systematic bibliometric analysis

Zhixin Zhang [a b c d e f], Xinmiao Xue [a b c d e f], Peng Liu [a b c d e g], Chi Zhang [a b c d e], Jianan Li [a b c d e], Ya Liu [a b c d e], Yvke Jiang [a b c d e f], Shuhan Lv [a b c d e f], Li Wang [a b c d e f],

Abstract

This systematic bibliometric analysis evaluates the scientific evidence, impact, and trends of photobiomodulation (PBM) for central nervous system (CNS) disorders. **Analyzing 150 relevant PubMed-indexed studies (including 46 clinical trials) up to September 2024, we demonstrate PBM's positive effects on the CNS. Key mechanisms include enhanced ATP synthesis, modulated nitric oxide signaling, improved neuronal excitability, suppressed oxidative stress, anti-inflammatory effects, and ion channel modulation.**

we summarize in Fig. 5 the relationship between wavelength ranges and corresponding medical conditions. Our findings indicate that red light with wavelengths less than 760 nm exhibits strong penetration capabilities, making it particularly beneficial for conditions affecting deeper regions of the central nervous system, such as brain injuries, cerebrovascular diseases, and Parkinson's disease.



ERCHONIA
Healing Light, Perfected.

NEUROGENESIS AND ALZHEIMER'S

Neural Regen Res. 2020 May; 15(5): 824–827.
Published online 2019 Nov 8. doi: 10.4103/1673-5374.268896

PMCID: PMC6990771
PMID: 31719242

Alzheimer's disease, neural stem cells and neurogenesis: cellular phase at single-cell level

Mehmet Ilyas Cosacak,[1] Prabesh Bhattarai,[1] and Caghan Kizil, PhD[1,2*]

Abstract

Alzheimer's disease cannot be cured as of yet. Our current understanding on the causes of Alzheimer's disease is limited. To develop treatments, experimental models that represent a particular cellular phase of the disease and more rigorous scrutiny of the cellular pathological mechanisms are crucial. In recent years, Alzheimer's disease research underwent a paradigm shift. According to this tendency, Alzheimer's disease is increasingly being conceived of a disease where not only neurons but also multiple cell types synchronously partake to manifest the pathology. Knowledge on every cell type adds an alternative approach and hope for the efforts towards the treatment. Neural stem cells and their neurogenic ability are making an appearance as a new aspect of the disease manifestation based on the **recent findings that neurogenesis reduces dramatically in Alzheimer's disease patients compared to healthy individuals. Therefore, understanding how neural stem cells can form new neurons in Alzheimer's disease brains holds an immense potential for clinics**.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

NEUROGENESIS AND ALZHEIMER'S

• Published: 25 March 2019

Adult hippocampal neurogenesis is abundant in neurologically healthy subjects and drops sharply in patients with Alzheimer's disease

• Elena P. Moreno-Jiménez, Miguel Flor-García, Julia Terreros-Roncal, Alberto Rábano, Fabio Cafini, Noemí Pallas-Bazarra, Jesús Ávila & María Llorens-Martín

Nature Medicine

Abstract

The hippocampus is one of the most affected areas in Alzheimer's disease (AD)1. Moreover, this structure hosts one of the most unique phenomena of the adult mammalian brain, namely, the addition of new neurons throughout life2. This process, called adult hippocampal neurogenesis (AHN), confers an unparalleled degree of plasticity to the entire hippocampal circuitry3,4. Nonetheless, direct evidence of AHN in humans has remained elusive. Thus, determining whether new neurons are continuously incorporated into the human dentate gyrus (DG) during physiological and pathological aging is a crucial question with outstanding therapeutic potential. By combining human brain samples obtained under tightly controlled conditions and state-of-the-art tissue processing methods, we identified thousands of immature neurons in the DG of neurologically healthy human subjects up to the ninth decade of life. These neurons exhibited variable degrees of maturation along differentiation stages of AHN. In sharp contrast, the number and maturation of these neurons progressively declined as AD advanced. These results demonstrate the persistence of AHN during both physiological and pathological aging in humans and provide **evidence for impaired neurogenesis as a potentially relevant mechanism underlying memory deficits in AD that might be amenable to novel therapeutic strategies.**

RESTORING BRAIN —HEALTH—

NEUROGENESIS AND ALZHEIMER'S

~0.8 J/cm2 on the hippocampus

PBM treatment performed postischemia exerted a long-lasting protective effect on astrocytes and promoted endogenous neurogenesis in the hippocampal CA1 region

Front. Cell. Neurosci., 20 **August 2021** | https://doi.org/10.3389/fncel.2021.731855

Photobiomodulation Promotes Hippocampal CA1 NSC Differentiation Toward Neurons and Facilitates Cognitive Function Recovery Involving NLRP3 Inflammasome Mitigation Following Global Cerebral Ischemia

Sihan Guo1, Ruimin Wang2*

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER THERAPY AND DEMENTIA

Introduction

Several recent studies in animal models of Alzheimer's and Parkinson's disease have reported that **low-level near infrared light (NIr) therapy not only mitigates the behavioral deficits associated with these conditions but also has neuroprotective effects, slowing the underlying death of neurons.** Current clinical therapies for both diseases do not achieve a comparable slowing of degeneration and neuroprotection, though they do relieve motor signs in Parkinson's disease and, to a lesser extent, the cognitive, and memory deficits in Alzheimer's disease. **In this review, we consider the evidence for neuroprotection by NIr in animal models of these diseases, the putative mechanisms by which NIr may work to protect cells against insult, the safety of NIr therapy and finally, the potential effective use of NIr therapy in patients.** First, we provide an overview of Alzheimer's and Parkinson's disease and current treatment options for these conditions.

RESTORING BRAIN —HEALTH—

NEUROGENESIS AND ALZHEIMER'S

Results: It has been observed that both of these modalities were successful at neural cell differentiation. **PBM at 1 J/cm2** and low-dose PDT at 3 J/cm2 energy densities provided the best differentiation profiles which were proved by the over-expressions of SYN-1 and GAP43 genes. It was also observed that intracellular ROS production and NO release had pivotal roles in these mechanisms with more cell differentiation obtained especially in low-dose PDT application.

Conclusion: **It can be concluded that light-induced mechanisms with properly optimized light parameters have the capacity for neural cell regeneration and thus, can be a successful treatment for incurable neurodegenerative diseases.**

Photodiagnosis Photodyn Ther

2022 Mar;37:102702. doi: 10.1016/j.pdpdt.2021.102702. Epub 2021 Dec 23.

Mechanistic approaches to the light-induced neural cell differentiation: Photobiomodulation vs Low-Dose Photodynamic Therapy

YOU MUST ACTIVATE BRAIN REGIONS TO PROMOTE NEUROPLASTICITY AND FUNCTIONALITY. A HEALTHY BRAIN REQUIRES ALL SYSTEMS (THYROID, DIGESTIVE, ADRENALS ETC) TO BE ADDRESSED AND SUPPORTED. A HEALTHY BRAIN REQUIRES AN INTEGRATIVE APPROACH!

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

FUNCTIONAL BRAIN ACTIVITY USING ONLY ERCHONIA LLLT

CASE 2.

Figure 1.
In Figure 1, QEEG absolute power shows an EEG activation in the right hemisphere, mainly in the temporal lobe

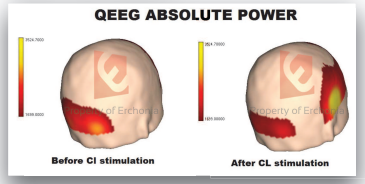
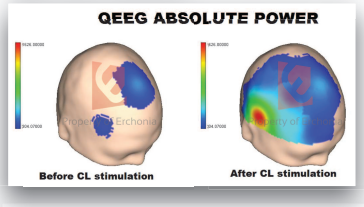
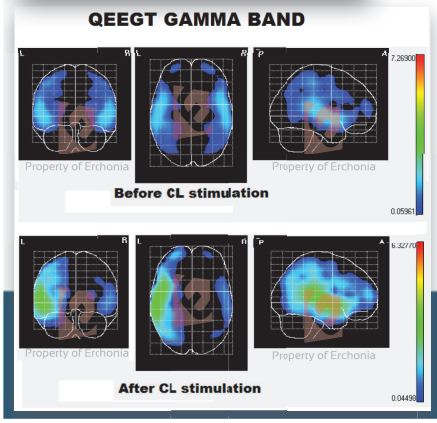


Figure 2.
In Figure 2, QEEG within the Gamma Band shows a clear EEG activation in the right hemisphere, mainly in the temporal region.

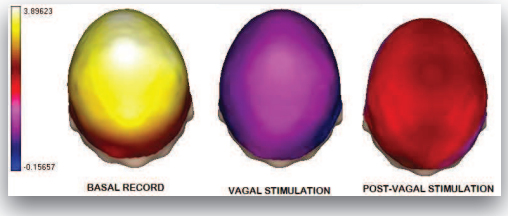




THE FIRST REAL TIME TRANSCRANIAL STUDY USING QEEG WITH THE ERCHONIA XLR8

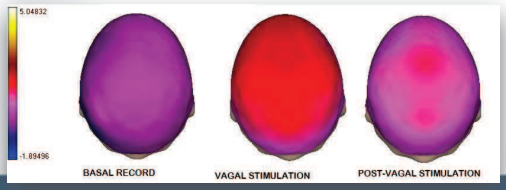
VAGAL NERVE STIMULATION ONLY OVER THE SCM REGION (VIOLET TOP, RED BOTTOM) REAL TIME QEEG

SUBJECT 10



QEEG RESULTS USING LLLT WITH RED LASER

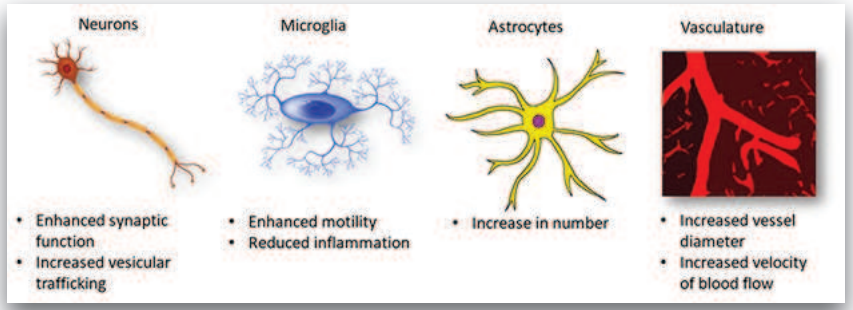
SUBJECT O1



RESTORING BRAIN —HEALTH—

MASTER BRAIN PROTOCOL FREQUENCIES: 40 HZ

SUMMARY OF FINDINGS TO DATE WITH LI-HUEI TSAI FROM M.I.T. AND COLLEAGUES HAVE FOUND IN AD ANIMAL MODELS, PILOT, PHASE 1 AND PHASE 2 CLINICAL TRIALS USING 40 HZ FREQUENCY APPLICATIONS ON THE BRAIN



REDUCTION OF AMYLOID PLAQUES
REDUCTION OF TAU PROTEIN MISFOLDS
IMPROVED MEMORY
VASODILATION/INCREASED BLOOD FLOW
INCREASED GLYMPHATIC WASTE CLEARANCE
DECREASED CORTICAL ATROPHY
PRESERVATION OF THE HIPPOCAMPUS
IMPROVED SLEEP
DECREASED BRAIN INFLAMMATION MARKERS

https://www.alzforum.org/news/conference-coverage/does-synchronizing-brain-waves-bring-harmony

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

SUMMARY OF BENEFITS OF 10 HZ FREQUENCY ON THE HUMAN BODY

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

A 10 Hz frequency, often associated with the "alpha wave" in the brain, is considered beneficial for promoting relaxation, reducing stress, improving focus, enhancing creativity, and facilitating a state of calm alertness, as it falls within the range of brainwave activity typically present during a relaxed, wakeful state; essentially acting as a "bridge" between conscious and subconscious mind.

Key benefits of a 10 Hz frequency:

- **Relaxation and stress reduction:**
  This frequency is often used in relaxation techniques like meditation and deep breathing due to its calming effect on the brain.

  Improved focus and concentration:
  A balanced alpha wave activity (around 10 Hz) can enhance mental clarity and focus, making it useful for tasks requiring concentration.

  Creativity enhancement:
  Some research suggests that alpha brainwaves can facilitate creative thinking and problem-solving.

  Improved sleep quality:
  Exposure to 10 Hz frequencies before sleep may help transition into a relaxed state, potentially aiding sleep onset.

  Pain management:
  Some studies suggest that low-frequency stimulation around 10 Hz might be beneficial for pain relief in certain conditions.

RESTORING BRAIN —HEALTH—

ALPHA FREQUENCIES

PEAK ALPHA FREQUENCY

10 HZ

- THE RESONANCE BETWEEN THE THALAMUS AND CORTEX
- STATE OF RELAXATION, PEACEFULNESS AND ALERTNESS
- THE OPTIMAL MENTAL RESOURCEFULNESS STATE READY TO MOVE QUICKLY AND ACCOMPLISH TASKS WHEN NEEDED
- A SENSE OF EASE AND CALM. IDEAL STATE FOR DAILY BASELINE
- IS THE BRIDGE BETWEEN CONSCIOUSNESS AND SUBCONSCIOUS ENDEAVORS
- IT IS THE ALERT STATE WHEN AWAKE BUT NOT BOGGED DOWN BY BEING OVERACTIVE (IE. HIGH BETA)
- ARE STRONGEST IN THE BACK 1/3 OF THE HEAD AND FRONTAL CORTEX AND SHOULD BE SLIGHTLY HIGHER IN THE RIGHT HEMISPHERE THAN LEFT
- WHEN EYES ARE CLOSED BUT AWAKE OR “DAYDREAMING”, SHOULD PUT YOU IN AN ALPHA STATE
- IT IS THE IDEAL WAKING STATE FOR MENTAL WORK AND CREATIVITY
- BEING IN OPTIMAL ALPHA WILL TYPICALLY CREATE A GOOD MOOD, SEE THE WORLD TRUTHFULLY AND SENSE OF CALMNESS
- IT IS A CRITICAL STATE FOR LEARNING AND USING INFORMATION TAUGHT
- INTROSPECTIVE MEDITATION AND BREATHING EXERCISES TYPICALLY PROMOTE ALPHA STATES
- BEING IN ALPHA ALLOWS US TO SHIFT EASILY FROM ONE TASK TO ANOTHER
- IT’S BETTER TO LEARN AND PROCESS INFORMATION WHEN YOUR “FOOT ISN’T STUCK ON THE GAS PEDAL” OR “THE ENGINE WON’T TURN ON OR IS SPUTTERING”. (TOO HIGH OR LOW BRAIN WAVE ACTIVITY)
- OTHER KEY TERMS WITH ALPHA OPTIMIZATION: TRANQUIL, RELAXED, HEALING, MEDITATION
- PEAK ALPHA IS 10 HZ. THIS IS THE OPTIMAL “IDLING ENGINE” FREQUENCY OF THE BRAIN FOR A FINELY TUNED VEHICLE
- THIS STATE ALLOWS US TO EFFECTIVELY KICK INTO GEAR AND COMMUNICATE, LEARN ETC WHILE TUNING OUT IRRELEVANT INFORMATION. IT LOWERS TO GO INTO SLEEP STATES
- 10 HZ HAS BEEN SHOWN TO PROMOTE “RESTING DIGESTING” PARASYMPATHETIC STATES IN BRAIN, VAGUS NERVE - SCM AND AURICULAR REGIONS

ERCHONIA® Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

LASER PULSED FREQUENCIES AND EEG BRAINWAVES

NOTE: PULSED FREQUENCIES OF LIGHT THERAPIES CAN INFLUENCE BRAIN EEG WAVES AT THE SAME FREQUENCY BUT THIS IS NOT A LINEAR RESPONSE AND DEPENDS ON SEVERAL FACTORS INCLUDING INDIVIDUAL BRAIN CHARACTERISTICS, LOCATION OF THE LASER APPLICATION AND MORE. WITH THAT SAID, THERE IS EVIDENCE IN THE LITERATURE THAT PULSING LASERS CAN SYNCHRONIZE WITH NEURONAL OSCILLATIONS (BRAINWAVES THAT ARE MEASURED IN EEG) IN INFLUENCING BRAIN ACTIVITY, PAIN REDUCTION AND CELLULAR RECOVERY.

Research in transcranial photobiomodulation (tPBM) has shown that applying specific pulse rates of near-infrared or low-level lasers to the scalp can alter EEG patterns, promoting desired brain states like relaxation or improved attention. However, individual responses vary, and clinical applications require precision to avoid overstimulation or undesired effects.

This phenomenon is called frequency following response or entrainment, where the brain's neural activity aligns with external stimuli of a specific frequency

Photobiomodulation and EEG Changes

- **Study**: Zomorrodi, R., Loheswaran, G., Pushparaj, A., & Lim, L. (2019). "The effects of transcranial photobiomodulation on resting-state functional connectivity in healthy adults." *Brain and Behavior*, 9(4 e01217.
  - **Relevance**: Demonstrates the effects of transcranial photobiomodulation (tPBM) on EEG activity and functional brain connectivity. Pulse rates were shown to modulate neural responses.

Frequency-Specific Effects of Laser Therapy

- **Study**: Naeser, M. A., Saltmarche, A., Krengel, M. H., Hamblin, M. R., & Knight, J. A. (2011). "Improved cognitive function after transcranial, light-emitting diode treatments in chronic, traumatic brain inj Two case reports." *Photomedicine and Laser Surgery*, 29(5), 351-358.
  - **Relevance**: Explores how specific pulse rates of transcranial light therapy influence cognitive recovery and EEG activity in brain injury patients.

Brainwave Entrainment Using Light Pulses

- **Study**: Barghout, J., Kumar, N., & Mann, W. (2009). "Brainwave entrainment to periodic light stimulation: An exploratory study." *Neuroscience Letters*, 454(2), 84-88.
  - **Relevance**: Investigates how periodic light pulses at specific frequencies entrain EEG activity, aligning brainwaves with the applied external stimuli.

"PROTOCOLS"

THE HEALTHCARE PROVIDER SHOULD MAKE EVERY ATTEMPT TO SUPPORT COMPLEX NEUROLOGICAL CASES WITH INDIVIDUALIZED CARE. FOR EXAMPLE, ALZHEIMER'S LIKE DEMENTIA MAY HAVE 6 DIFFERENT GROUPINGS WITH HUNDREDS OF VARIATIONS OF UNDERLYING CAUSES. WITH THAT SAID, I HAVE ATTEMPTED TO GIVE FOUNDATIONAL PROTOCOLS IN MOST OF THE SECTIONS OF THESE SEMINAR NOTES FOR YOU TO EASILY REFERENCE. LOOK FOR THIS SYMBOL:

NOTE: A SUMMARY SECTION OF THE PRIMARY "PROTOCOL" PAGES HAS BEEN ADDED IN AT THE END OF THE PRIMARY SEMINAR SLIDES, BEFORE THE MISCELLANEOUS SUPPORT SLIDE SECTION FOR EASY REFERENCE

DO NOT BE CONCERNED ABOUT CONTENT THAT IS COVERED BY DROP DOWN BOXES OF TEXT COVERING UNDERLYING TEXT. I HAVE MADE SURE TO NOT COVER ANY PERTINENT OR NECESSARY INFORMATION WHEN YOU GO BACK TO REVIEW YOUR NOTES

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trever Berry, DC, DACNB is prohibited.

NEURODEGENERATION SUPPORT PARAMETERS

HOW LONG SHOULD THE PATIENT IMPLEMENT LIFESTYLE AND TREATMENT PARAMETERS IN NEURODEGENERATIVE CONDITIONS TO SEE CHANGES?

A: 6 MONTHS
DIET, LIFESTYLE, NUTRITIONAL SUPPORT AND OTHER RECOMMENDATIONS OF THE BREDESEN PROTOCOL SHOULD BE IMPLEMENTED FOR 6 MONTHS BEFORE DECIDING IF THEY WILL BE A VIABLE SOLUTION FOR THE PATIENT.
THIS TIMEFRAME SHOULD ALSO BE CONSIDERED FOR FOOD ELIMINATION PROTOCOLS.

HOW LONG SHOULD SUPPLEMENTS/MEDICATIONS BE TAKEN FOR SHIFTS IN MICRONUTRIENT AND CHEMISTRY PROFILE CHANGES?

A: 3 MONTHS MINIMUM

HOW MANY IN-OFFICE LASER AND BRAIN BASED TREATMENTS SHOULD BE UTILIZED TO SEE IF ANY OBJECTIVE AND SUBJECTIVE MARKERS IMPROVE (TO ANY DEGREE)?

A: 12 VISITS MINIMUM (OVER A 4-6 WEEK PERIOD). TARGET 3 MONTHS TOTAL!

RESTORING BRAIN —HEALTH—

2026 FOUNDATIONAL PROTOCOLS FOR YOUR ERCHONIA DEVICES

MOST COMMONLY USED BRAIN FREQUENCIES:

BRAIN RESTORE **10-40 FOR ALL DIODES (HAND HELDS OR FX'S)**

MASTER BRAIN **1-10-40-60 (ADD 10s AND 40s ON FX DEVICES FOR THE REMAINING DIODES)**

BASE IT ON QEEG MAPPING SUGGESTIONS AND LOCATION (EX. SMR 12-15 UP OVER MIDLINE)

FIRST PRINCIPLES…ARE YOU DEALING WITH HIGH ANXIETY, OCD ETC = SLOW IT DOWN WITH A REST-DIGEST TYPE OF 1-10-1-10
IF THEY ARE BOGGED DOWN LIKE STUCK IN DELTA/THETA POST CONCUSSION USE MORE 40s

DEFAULT TIME PER REGION FOR DIFFERENT ERCHONIA HAND HELD DEVICES:
2 MINUTES PER REGION GVL
3 MINUTES PRE REGION EVRL
4 MINUTES PER REGION XLR8
MINIMUM 6 MINUTES FX 405 (SPLIT 3 AND 3 SAGITTAL/CORONAL)
AND 10 MINUTES WITH FX 635 (SPLIT 5 AND 5)

DO NOT GET ANALYSIS PARALYSIS! YOU CAN LEAVE THE FX ON SOMEONE'S HEAD FOR THE FULL 20 MINUTES WITHOUT GOING NEAR EXCESS DOSE PARAMETERS! AND THEY AREN'T GOING TO GROW A LIVER IN THEIR BRAIN IF YOU ACCIDENTALLY LEAVE IT ON THE LIVER SETTING AND PUT IT ON SOMEONE'S HEAD!

Duplication, citation or distribution of this material in whole or in part

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

SOME OF DR BERRYS FAVORITE FREQUENCY SETTINGS

MASTER BRAIN 1-10-40-60 MASTER GAMMA 40-40-40-40

VAGUS 10-10-10-10 BRAIN RESTORE 10-40-10-40

REST DIGEST 1-10-1-10 DELTA 1-1-1-1

MASTER LOVE 1-10-40-528

IMMUNE BOOST 20-73-465-728

DEFAULT VIRUS (ACUTE) 20-73-625-787

DEFAULT BACTERIA (ACUTE) 20-664-787-1560

DEFAULT MUSCULOSKELETAL 9-16-42-53

PAIN 4-60-5000-10000

ACUTE INJURY (GAIR) 8-25-42-125

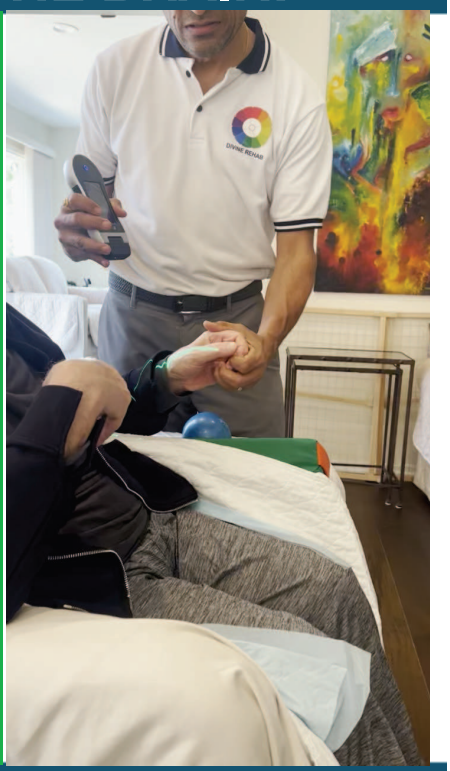
CHRONIC INJURY (GAIR) 8-25-42-279

CLINICAL GEM: ADD 10 HZ AND 40 HZ TO JEROME'S BASE 4 FREQUENCY PLATFORM FOR FX LASERS

REFER TO JEROME RERUCHA'S LASER A-Z PROTOCOL BOOK!

SOMETIMES IT'S BEST JUST TO TRY GETTING THE LASER ON THE BRAIN!

MANISH PATEL (A P.T.): HIS FATHERS PATIENT (ON THE LEFT) HAD A STROKE AND HAD NOT OPENED HIS HAND IN THE 2 YEARS SINCE. HE GOT HIS GVL, HEARD ME SAY "JUST GET THE LASER ON THE HEAD EVEN IF YOU DON'T FULLY UNDERSTAND NEUROLOGY. BIG THINGS CAN HAPPEN WHEN THE BRAIN HAS MORE ENERGY!"
FOLLOWED BY THIS MOST RECENT VIDEO SENT ON 9/17/25. MSA PATIENT (MULTIPLE SYSTEM ATROPHY)



Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MEDLEGAL VERBIAGE REGARDING SCOPE OF PRACTICE:

(IF YOUR SPECIALTY DOES NOT ALLOW FOR DIAGNOSIS AND TREATMENT OF MEDICAL CONDITIONS)

"YOU ARE SUPPORTING NORMAL ADAPTIVE PHYSIOLOGY OF THE NERVOUS SYSTEM"

HAVE THIS IN THE VERBIAGE OF YOUR DOCUMENTATION FORMS

CONSENSUS AI REVIEW 12/24/25

WHAT ARE THE WORST 5 DRUG CLASSES THAT MAY CAUSE OR CONTRIBUTE TO DEMENTIA AND COGNITIVE DECLINE?

Pro · 2 steps · 20 sources >

Several drug classes are consistently linked to higher dementia and cognitive-decline risk, especially with long-term, high-dose, or multi-drug use.

Do certain chronic medication classes increase dementia risk? N = 13

3. Antipsychotics

- Systematic data-driven review links antipsychotic use with increased dementia risk and worse cognition 3 7.
- Widely flagged as "potentially inappropriate medications" in dementia units 10.

4. Opioids

- Associated with delirium and chronic cognitive impairment; opioids with anticholinergic components are especially problematic 7 10.
- Polypharmacy with other CNS depressants further worsens cognition 7 18.

60

5. Glucocorticoids (systemic steroids)

- Can cause impaired attention, executive function, memory, delirium, and dementia, especially with chronic exposure and high doses via hippocampal damage 7.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS AI REVIEW 12/24/25

WHAT ARE THE WORST 5 DRUG CLASSES THAT MAY CAUSE OR CONTRIBUTE TO DEMENTIA AND COGNITIVE DECLINE?

Pro · 2 steps · 20 sources >

Several drug classes are consistently linked to higher dementia and cognitive-decline risk, especially with long-term, high-dose, or multi-drug use.

Do certain chronic medication classes increase dementia risk? N = 13

Highest-risk drug classes (evidence strongest in older adults)

1. Anticholinergic drugs (multiple indications)

Includes bladder antimuscarinics, many antidepressants (TCAs, paroxetine), antiparkinson, some antipsychotics, antihistamines, GI/vertigo agents.

- Long-term use (≥3–12 months) increases dementia risk by ~20–50%, with dose–response and stronger risk at higher cumulative burden 14 11 15 19.
- Particularly risky in Alzheimer's and Lewy body/Parkinson dementia due to existing cholinergic deficit 10 5 7.

2. Benzodiazepines and Z-drugs

61

- Frequently used in older adults; strongly associated with acute delirium and drug-induced cognitive impairment 7 10.
- Many observational studies show increased dementia risk, though some confounding remains 9 7.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

IF ANTICHOLINERGICS ARE THE WORST CLASS OF DRUGS CONTRIBUTING TO IATROGENIC CAUSES OF COGNITIVE DECLINE AND DEMENTIA, WOULD IT BE REASONABLE TO SUGGEST THAT SUPPORTING ACETYLCHOLINE PRODUCTION MIGHT BE A WISE DECISION FOR BRAIN HEALTH???

62

ERCHONIA
Healing Light, Perfected.

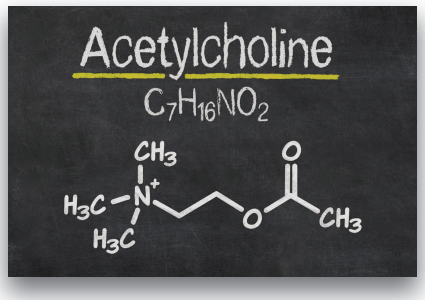
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ACETYLCHOLINE (ACh): THE BRAIN–BODY INTEGRATOR

Brain & Cognition
- Attention & Focus
- Learning & Memory
- Neuroplasticity
- REM Sleep
- Executive Function



Parasympathetic / Autonomic
- ↓ Heart Rate
- ↑ Digestion & GI Motility
- Bronchoconstriction
- Salivation & Lacrimation
- Bladder Contraction
- Sexual Arousal

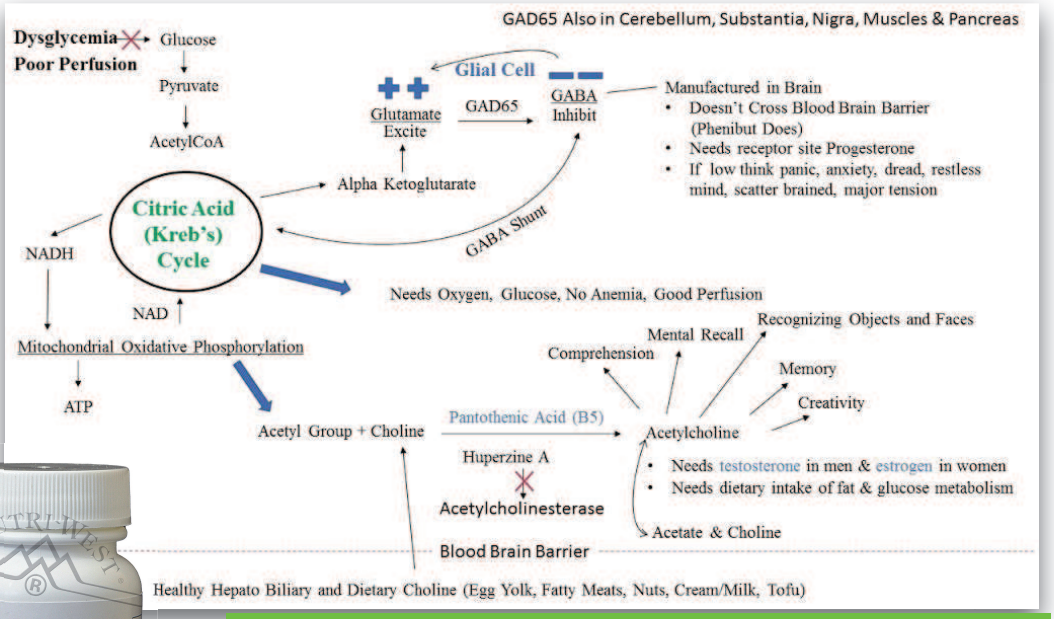
Immune & Inflammation
- Cholinergic Anti-Inflammatory Pathway
- ↓ TNF-α, IL-1β, IL-6
- Microglial Regulation
- Vagus Nerve → Immune Control

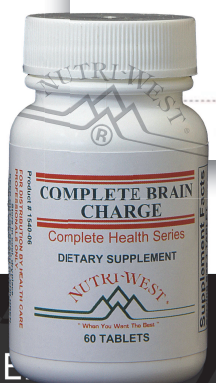
Movement
- Primary NMJ Neurotransmitter
- Skeletal Muscle Contraction
- Posture, Gait & Fine Motor Control

Acetylcholine = attention + memory + movement + parasympathetic tone + inflammation control

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

GABA, GLUTAMATE AND ACETYLCHOLINE





Ingredients :

Each Tablet Contains: Choline (as bitartrate) 24 mg. Proprietary blend 143 mg* of: Ginkgo Biloba Extract (24% ginkgosides, 6% terpene lactones), Phosphatidylcholine (from sunflower lecithin), Phosphatidylserine (from soybean), Alpha GPC (alpha glycerylphosphorylcholine), Turmeric (curcumin)(rhizome) Extract, Cytidine 5'-diphosphocholine.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EXOGENOUS CHOLINE SOURCES

DESIGNER CHOLINES: Alpha GPC and CDP-Choline

Alpha glycerylphosphocholine (Alpha GPC demonstrated a support benefit for patients in a number of clinical studies conducted on the brain and maintenance of brain function. In fact, the researchers, "being impressed not only with results but in lack of side effects, recommended that it is desirable to reconsider **alpha glycerylphosphocholine** in larger carefully controlled studies … for the maintenance of brain function" (Scapicchio PL. Revisiting choline alphoscerate profile: a new, perspective, role…Int J Neurosci. 2013 Jul;123(7):444-9.)

"Supplementation with alpha-GPC and CDP choline (cytidine 5'-diphosphocholine) has proven to be much superior to supplementation with just basic choline for brain support and normal neurotransmitter production. It is theorized that these designer cholines are able to be better incorporated and stored into the phospholipids in the brain, and therefore make better precursors for acetylcholine"

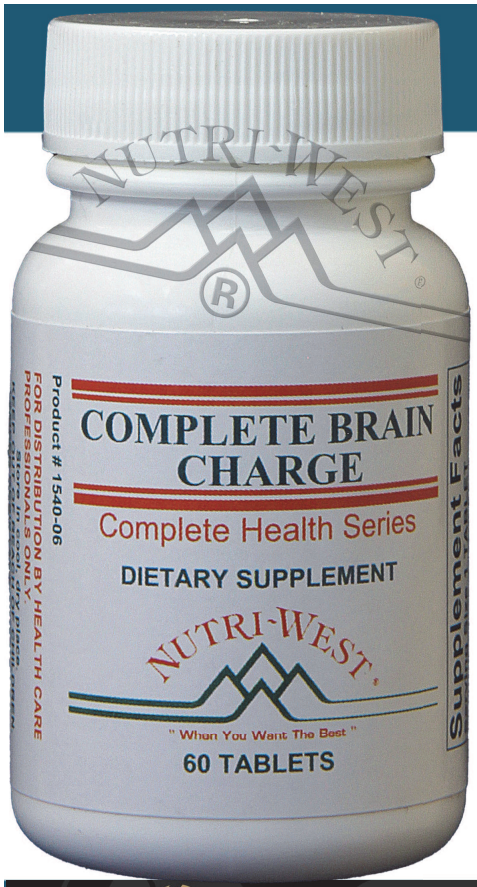
- Amenta F, Tayebati SK. Pathways of acetylcholine synthesis, transport and release. Curr Med

"Alpha-glyceryl-phosphorylcholine, (alpha-GPC) is a derivative of phosphatidylcholine known to support the parasympathetic nervous system. Alpha-GPC demonstrated benefit for patients in a number of clinical studies conducted on the brain and maintenance of brain function. The researchers, being impressed not only with results but in lack of side effects, recommended that "it is desirable to reconsider alpha-GPC in larger carefully controlled studies", … for the maintenance of brain function. *Scapicchio PL. Revisiting choline alphoscerate profile: a new, perspective, role…? Int J Neurosci. 2013 Jul;123(7):444-9.*

65

CDP choline undergoes quick hydrolysis and dephosphorylation after ingestion to yield cytidine and choline, which then enter the brain separately and are used to resynthesize CDP-choline inside brain cells. A research paper reviews the roles of choline and of choline-containing phospholipids on brain metabolism, stating that (CCPLs) are safe, effective compounds developed a long time ago that have a place in this support category and should be further investigated by new appropriate clinical studies. They also report that CDP-choline has displayed interesting effects documented in preclinical studies and limited clinical trials for its neurotransmission support (Clin Chem Lab Med. 2013 Mar 1;51(3):513-21. Choline-containing phospholipids: relevance to brain functional pathways. Tayebati SK1, Amenta F.) In an open, multicenter trial, CDP choline supported biosynthesis of phospholipids in neuronal membranes, brain metabolism, neurotransmitters, and the central nervous system (Clin Interv Aging. 2013;8:131-7. Effectiveness and safety of citicoline…: the IDEALE study. Cotroneo AM1, et al.)

ERCHONIA®
Healing Light, Perfected.



Ingredients :

Each Tablet Contains: Choline (as bitartrate) 24 mg. Proprietary blend 143 mg* of: Ginkgo Biloba Extract (24% ginkgosides, 6% terpene lactones), Phosphatidylcholine (from sunflower lecithin), Phosphatidylserine (from soybean), Alpha GPC (alpha glycerylphosphorylcholine), Turmeric (curcumin)(rhizome) Extract, Cytidine 5-diphosphocholine.

66

COMPLETE BRAIN CHARGE
"Where Membranes Meet Acetylcholine Messaging."

ERCHONIA®
Healing Light, Perfected.

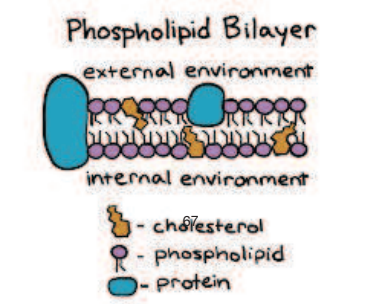
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Core Concept:

You cannot optimize brain signaling, neuroplasticity, or neuromodulation without healthy membranes.



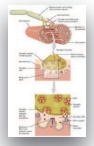
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

MEMBRANE SUPPORT AS WELL!

Complete Brain Charge® is designed to **build, stabilize, and optimize neuronal phospholipid membranes**, which are essential for brain signaling, plasticity, and energy efficiency.

- **Provides direct phospholipid building blocks (GREAT TO STACK WITH OMEGA 3'S)**
  - **Phosphatidylcholine (PC)** and **Phosphatidylserine (PS)** are core structural components of neuronal and mitochondrial membranes
  - Supports membrane integrity, fluidity, and receptor function
- **Supplies "designer cholines" for membrane synthesis**
  - **Alpha-GPC** and **CDP-Choline (Citicoline)** are superior choline sources that are efficiently incorporated into brain phospholipids
  - Promotes ongoing renewal of damaged or aging neuronal membranes



- **Enhances membrane-based neurotransmission**
  - Healthy phospholipid membranes are required for acetylcholine synthesis, vesicle release, receptor docking, and signals
- **Improves nutrient delivery & bioavailability**
  - PC and PS act as lipid carriers, enhancing absorption and effectiveness of co-nutrients such as curcumin and ginkgo
- **Creates the structural foundation for neuromodulation**
  - Optimized membranes improve responsiveness to photobiomodulation, neurofeedback, HBOT, and other brain-based therapies

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Brain-Training App 'Reverses 10 Years' of Decline in a Key Brain System

In a rigorous, double-blinded controlled trial, 82 healthy adults over the age of 65 were randomly assigned to play either Posit's 'BrainHQ' app or a recreational computer game for thirty minutes a day.

After 10 weeks, brain scans revealed that those who played BrainHQ, which focused on speed-based challenges, showed measurable increases in cholinergic activity, a type of brain signalling.

Published on 13.Oct.2025 in Vol 13 (2025)

Effects of Computerized Cognitive Training on Vesicular Acetylcholine Transporter Levels using [18F]Fluoroethoxybenzovesamicol Positron Emission Tomography in Healthy Older Adults: Results from the Improving Neurological Health in Aging via Neuroplasticity-based Computerized Exercise (INHANCE) Randomized Clinical Trial

Mouna Attarha[1] ; Ana de Figueiredo Pelegrino[2] ; Lydia Ouellet[2] ; Paule-Joanne Toussaint[2] ; Sarah-Jane Grant[1] ; Thomas Van Vleet[1] ; Etienne de Villers-Sidani[2]

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

"STACKING" MODALITIES

A longevity expert says combining brain games with exercise could be the secret to healthy aging—here's how to try it yourself

Twenty-four healthy sedentary women (65–78 years) were randomly allocated to one of three training groups: BET, exercise training, and control (no training). **The BET and exercise training groups completed the same physical training protocol comprising three 45-min exercise sessions (20-min resistance exercise plus 25-min endurance exercise) per week for eight weeks. The BET group completed a 20-min cognitive task prior to exercise tasks.** Cognitive (tasks: psychomotor vigilance, Stroop) and physical (tests: walk, chair-stand, arm curl) performance was tested when fresh and fatigued (before and after a 30-min cognitive task) at weeks 0 (pre-test), 4 (mid-test), 8 (post-test), and 12 (follow-up test).

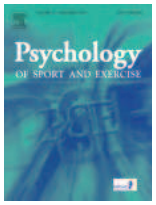
Highlights

Cognitive and physical performance were improved by 6-weeks of training compared to control.

BET improved performance on some cognitive and exercise tasks more than standard exercise training, especially when fatigued.

BET can improve cognitive and performance performance in older adults.

Combined training interventions can tackle aging-related declines in performance.



Psychology of Sport and Exercise
Volume 76, January 2025, 102757

Brain endurance training improves sedentary older adults' cognitive and physical performance when fresh and fatigued

Jesús Díaz-García

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN HEALTH APPS

APPS FOR BRAIN TRAINING AND OPTIMIZATION:

- FOCUS BUILDER (NEEDS DOCTOR RX INSTRUCTIONS)
- LUMOSITY
- NEURONATION
- **BRAIN HQ**
- ELEVATE (WIDE RANGE OF MENTAL SKILLS)
- COGNIFIT
- PERSONAL ZEN (ANTI-ANXIETY TRAINING)
- HAPPIFY
- RELIEFLINK (SUICIDE PREVENTION AND MOOD TRACKER)
- BRAIN TAP

CONSIDER A SECOND LANGUAGE APP:

- BABBEL
- DUOLINGO
- ROSETTA STONE

CONSIDER AI TRAINING. NOT IN A CO-DEPENDENT WAY BUT RATHER BY PROMPTING DAILY EXERCISES

THE BRAIN-MEMORY STACK

AFTER DINNER CONSIDER THE FOLLOWING:

1. TARGET SLEEP START BETWEEN 10-11 PM (7.5 HRS)
2. TAKE SUPPLEMENT SUPPORT (MG, L-THEANINE, GABA, KAVA, VALERIAN, CORTISOL ADAPTOGENS)
3. ACETYLCHOLINE (ALPHA GPC) AND MG L-THREONATE SUPPORT
4. WHEN AND WHY USE CBD AND MELATONIN?
5. VAGAL STACK - ERCHONIA LASER THE VAGUS NERVE (10 HZ) AND GUT MIN 2 MINUTES EACH, MASSAGE GUT, BOX BREATHING, GARGLE WITH NIGHT TOOTH BRUSHING
6. AROMATHERAPY (PLEASANT SMELL UNDER NOSTRIL OR DIFFUSER)
7. SLEEP BINAURALS, SOLFEGGIO, OR BRAIN TAP (BRAIN HQ DURING ACTIVE REHAB) WHILE LASERING THE TEMPORAL LOBE ABOVE THE EAR 5 MINUTES EACH SIDE USING 1-10-40-10 SETTING (REVIEW WHY LASER IS SO IMPORTANT. REVIEW MUSIC SUMMATION)
8. IF TRYING TO LEARN SOMETHING, MAKE THAT YOUR LAST READ BEFORE BED (NO BLUE LIGHT AND USE NIGHT SETTING IF ELECTRONIC)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

2025 SLIDES

Eli Lilly's Alzheimer's drug rejected in Europe

The EU medicines regulator on Friday (MARCH 28, 2025) rejected Eli Lilly's Alzheimer's drug, saying the treatment's ability to slow cognitive decline was not large enough to outweigh the risk of serious brain swelling in patients.

Like Leqembi, Kisunla is designed to clear sticky clumps of a protein called amyloid beta, believed to be a hallmark of Alzheimer's, from the brain.

Lilly has expanded its direct-to-consumer online platform LillyDirect to include telehealth providers to diagnose and care for patients with Alzheimer's disease, in its efforts to boost Kisunla sales.

Kisunla, which is given as a monthly infusion and has an annual U.S. list price of around $32,000, brought in sales of $9.3 million last year. (LEQEMBI IS $26,500/ YEAR)

PHARMACEUTICALS AND DEMENTIA

These 5 Popular Medications Have Been Linked With Alzheimer's, Research Shows

Sezeryadigar/iStock

1. **Benzodiazepines (BZD)** have been associated with an increased dementia risk, the Alzheimer's Association reports. However, the interpretation of this data is "uniquely challenging because the conditions for which BZDs are prescribed (anxiety, agitations, insomnia) are also symptoms of ADRD," warns the site.

The National Library of Medicine reported that a meta-analysis of ten different studies found "that BZD significantly increases the risk of dementia in the elderly population" noting the significance of long-term use: "The risk is higher in patients taking BZD with a longer half-life (greater than 20 hours) and for a longer duration (greater than three years)."

Bet_Noire/iStock

2. **ANTICHOLINERGICS**. Long-term use was also found to be significant in research regarding anticholinergics. "Anticholinergics are drugs that block the action of acetylcholine," explains Healthline. "Anticholinergics can treat a variety of conditions, including urinary incontinence, overactive bladder (OAB); [and] chronic obstructive pulmonary disorder (COPD)" among other conditions. *Tricyclic antidepressants (for example: amitriptyline) first generation antihistamines (for example: chlorpheniramine, diphenhydramine) overactive bladder antimuscarinics (for example: oxybutynin)*

When the researchers examined medication use, they found that people who used anticholinergic drugs were more likely to have developed dementia than those who didn't use them," says Harvard Health, emphasizing that this increased with "the cumulative dose."

"Taking an anticholinergic for the equivalent of three years or more was associated with a 54 percent higher dementia risk than taking the same dose for three months or less," warned the site.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

These 5 Popular Medications Have Been Linked With Alzheimer's, Research Shows

3. Statins

A class of statins called lipophilic statins—which include drugs such as Lipitor, Lescol, and Livalo—can be very effective at lowering high levels of cholesterol. But **studies have shown a possible link between use of these drugs and cognitive decline.**

"While you would expect that statin use would reduce the risk of cognitive decline and dementia because statins lower cardiovascular risks and the risk of stroke, it hasn't been clearly shown to be the case," **JoAnn Manson, MD, told Harvard Health. "It's surprising that there's not a clearer reduction seen. If anything, some of these studies have raised concerns about cognitive risks."**

4. Anti-seizure drugs

One type of anti-seizure drug, Levetiracetam, was found in 2021 to actually improve cognitive functions in Alzheimer's patients. But other anti-seizure meds are linked to an increased risk of dementia, revealed a study which focused on patients in Germany and Finland and was published in the ***Journal of American Geriatrics Society*. These include drugs like Depakote and Topamax.**

"Continuous use of anti-epileptic drugs for a period exceeding one year was associated with a 15 percent increased risk of Alzheimer's disease in the Finnish dataset, and with a 30 percent increased risk of dementia in the German dataset," says Science Daily.

PHARMACEUTICALS AND DEMENTIA

These 5 Popular Medications Have Been Linked With Alzheimer's, Research Shows

5. Parkinson's disease medications

"Parkinson's disease is a progressive disorder that affects the nervous system and the parts of the body controlled by the nerves," the Mayo Clinic explains. "Although Parkinson's disease can't be cured, medications might significantly improve your symptoms."

Unfortunately, some of the medications used to treat the disease "may lead to major side effects such as memory loss, confusion, delusions, and compulsive behaviors" as a result of their effects on the pathways for the neurotransmitter dopamine, reports *Pharmacy Times*.

ERCHONIA Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

MEDICATION INDUCED DEMENTIA

Popular medication could increase dementia risk by staggering 79 per cent

A new study says you may want to think twice before reaching for prescription sleeping tablets. Research published in the Journal of Alzheimer's Disease discovered that white people had a 79 per cent higher risk of developing dementia when on sleeping meds.

- **Benzodiazepines such as Halcion, Dalmane or Restoril, which are prescribed for chronic insomnia — almost twice as likely**
- **Trazodone (trade name Desyrel or Oleptro), an antidepressant that is sometimes prescribed to help people sleep — 10 times as likely**
- **"Z-drugs" such as Ambien, which are sedative-hypnotics — more than seven times as likely**

Journal: Journal of Alzheimer's Disease vol. 91, no. 3, pp. 1133-1139, 2023
Accepted 24 November 2022 | **Published:** 31 January 2023

PHARMACEUTICALS AND DEMENTIA

Study finds link between proton pump inhibitors and dementia

Avoidance of PPI medication may prevent development of dementia, researchers suggest

Older people treated with **proton pump inhibitors (PPIs), which are typically used for gastroesophageal reflux disease and peptic ulcers, may be at increased risk of developing dementia, according to the results of a German study published in the *Journal of American Medical Association Neurology***
[1] on 15 February 2016.

Researchers discovered that the patients who used PPIs regularly — at least one prescription per quarter in an 18-month period – had a 44% higher risk of developing dementia (hazard ratio [HR], 1.44 [95% confidence interval [CI], 1.36–1.52]; P<0.001), than patients not prescribed PPIs.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

PPI'S AND DEMENTIA

HIGHLIGHTS

- After following 1,983,785 individuals for a median of 10 years, 99,384 developed dementia
- PPIs were used by 21.2% of cases and 18.9% of controls
- PPI use was associated with increased dementia rate regardless of time of treatment onset
- Magnitude of associations increased with younger age at diagnosis
- PPI use was not associated with dementia occurring after age 90 years

In conclusion, exposure to PPIs was found to be associated with an increased rate of all-cause dementia occurring before 90 years of age regardless of time of treatment initiation according to the diagnosis. Longer cumulative duration of PPI use yielded higher risk estimates

Proton pump inhibitors and dementia: A nationwide population-based study

Nelsan Pourhadi, Janet Janbek, Christina Jensen-Dahm, Christiane Gasse, Thomas Munk Laursen, Gunhild Waldemar

First published: 05 October 2023

RESTORING BRAIN —HEALTH—

Computational BiologyResearch Article5 September 2025

A hidden confounder for microbiome studies: medications used years before sample collection

Authors: Oliver Aasmets

"The effects of antibiotics, psycholeptics, antidepressants, proton pump inhibitors, and beta-blockers are detectable several years after use."

ABSTRACT

We show that past usage of medications is associated with the gut microbiome. For example, the effects of antibiotics, psycholeptics, antidepressants, proton pump inhibitors, and beta-blockers are detectable several years after use. Furthermore, by analyzing a subcohort ($n = 328$) with a second microbiome characterization, we show that similar changes in the gut microbiome occur after treatment initiation or discontinuation, possibly indicating causal effects.

Surprisingly, our results showed that benzodiazepine derivatives have an even broader effect on the whole microbiome composition as well as on the presence and abundance of individual microbial species compared to several antibiotic classes. Moreover, benzodiazepine derivatives show remarkable carryover effects several years after their use, which is comparable to effects observed with the broad-spectrum antibiotic classes.

ERCHONIA Healing Light, Perfected.

Common Drugs Can Rewire Your Gut For Several Years, Study Finds

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

IF PILLS ARE TOO TOXIC TO FLUSH DOWN THE TOILET, EVER ASK, WHY IS IT SAFE TO SWALLOW THEM???

BRAIN BALANCE = BODY BALANCE

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN BALANCING AND RESTORATION

START WITH OBJECTIVE MEASURES:

MOCA (MONTREAL COGNITIVE ASSESSMENT)

BALANCE TESTING (CDP)

FOUNDATIONAL LAB TESTS

BEDSIDE NEURO EXAM

QEEG BRAIN MAP

EASY TO ADMINISTER TESTS. EVEN YOUR STAFF COULD DO IT!

HANDS ON DEMO FOR CEREBELLAR BRAIN BALANCING AND CENTER OF PRESSURE

CLINICAL GEM:
ALWAYS CONSIDER BALANCING OUT THE CEREBELLUM AND COP BEFORE EMBARKING ON NEUROMUSCULOSKELETAL TREATMENT STRATEGIES!

- FINGER TO NOSE/FINGER TO FINGER (EYES OPEN AND CLOSED)
- RAPID ALTERNATING MOVEMENT (DYSDIADOCHOKINESIA)
- REBOUND PHENOMENA
- STATIONARY STANCE/RHOMBERG'S (TECHNICALLY PROPRIO BUT DOES INVOLVE CB FEEDBACK) ALSO WATCH FOR TITUBITION (NUCHAL OR TRUNK)
- GAIT (ESPECIALLY WIDE BASED "ATAXIC" GAIT)
- HEEL-TOE STANCE/WALK (BACK FOOT BIAS)
- HEEL TO SHIN
- PENDULAR DTR'S
- FLOPPY TONE OF MUSCLES
- END GAZE NYSTAGMUS/HORIZONTAL SQUARE WAVE JERKS/LEAKY NEURAL INTEGRATORS
- DYSMETRIA OF FAST EYE MOVEMENTS
- SCANNING/STACCATO CEREBELLAR SPEECH

IT'S MUCH MORE DIFFICULT TO OBTAIN INJURY AND PAIN RECOVERY, SPORTS OPTIMIZATION AND IMPROVED BRAIN FUNCTION WHEN THE REGION OF THE CNS THAT QUARTERBACKS ALL OF THIS IS NOT WORKING PROPERLY!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

2026 MINI MASTER PROTOCOL

ALL HAND HELDS SET FOR 10-40-10-40
2 MINUTES EACH REGION FOR GVL
3 MINUTES FOR EVRL
4 MINUTES FOR XLR8

DLPF/TEMPORAL/ PARIETAL

STACK WITH CEREBELLAR COMPLEX

BRAINSTEM/ CEREBELLUM/ VAGUS

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

5 MINUTE BEDSIDE NEURO EXAM TIME PERMITTING

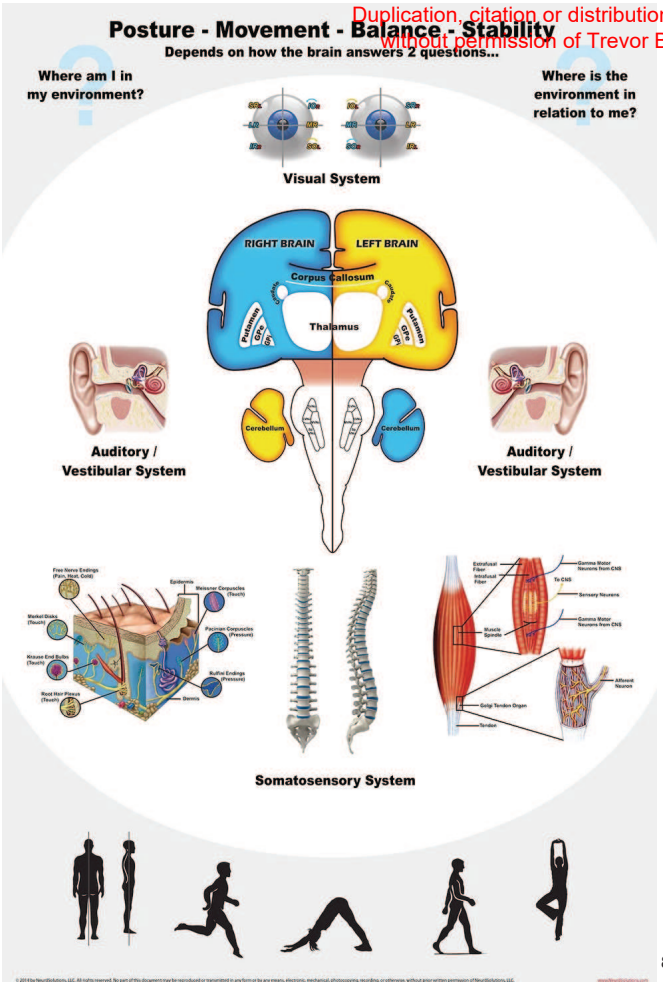
GET CAMERA OUT TO RECORD LAST 5 MINUTES!

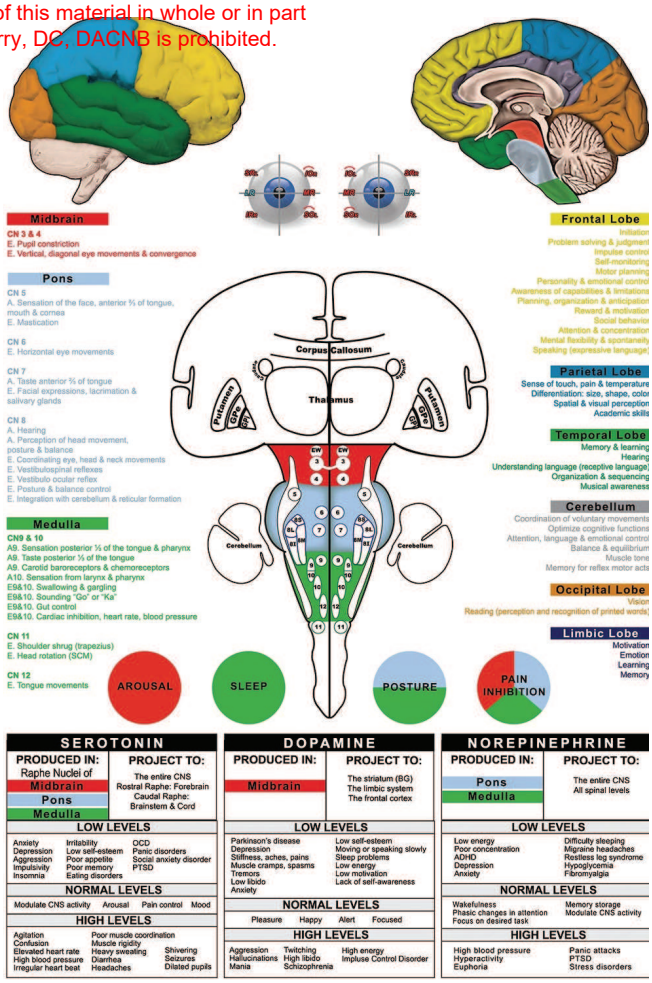
RESTORING BRAIN —HEALTH—

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

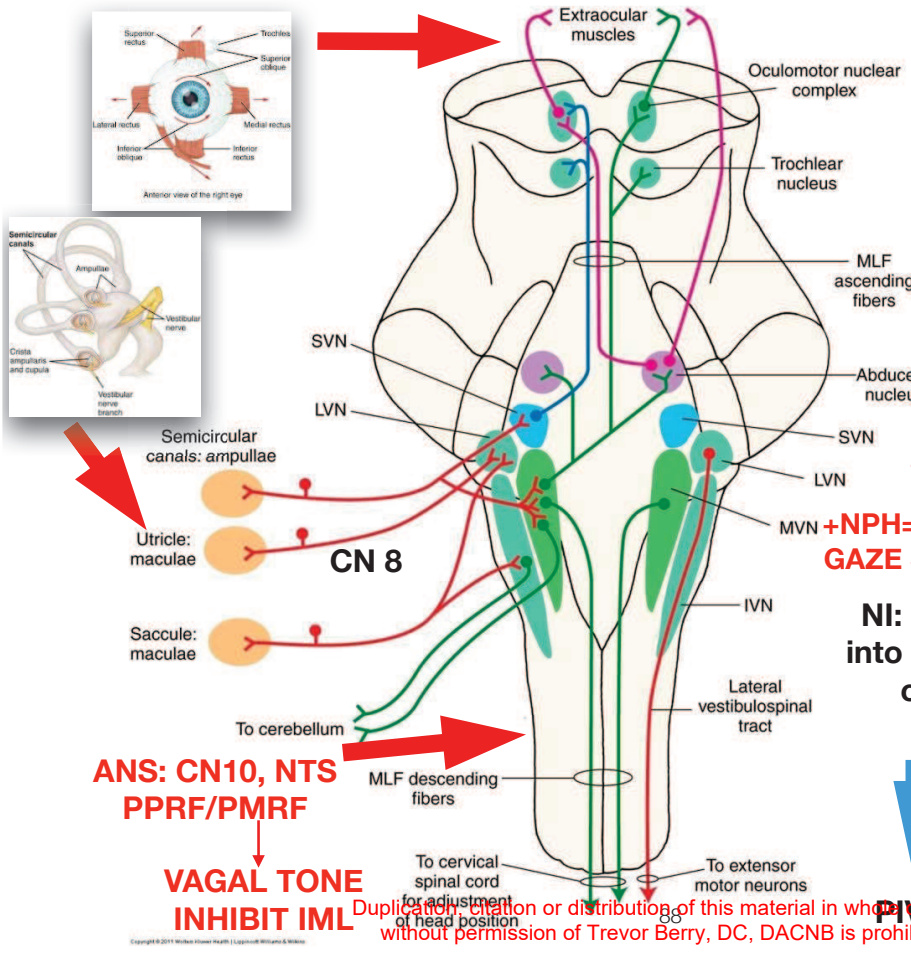
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.





"THE EYES HAVE IT"



ANS: CN10, NTS PPRF/PMRF

VAGAL TONE INHIBIT IML

Vestibular/Sensory Mismatch?

Are our visual, auditory, vestibular and proprioceptive/LDA systems matching

Filtered through a common neural integrator

"Y"

Cerebellum

+NPH=horizontal GAZE STABILITY

"Leaky NI"

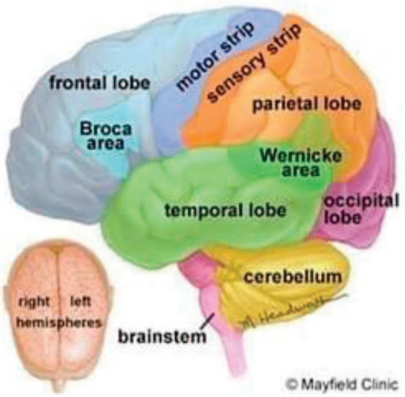
NI: Takes the pulse command into step command or a velocity command into a position command

BALANCE TRACKING SYSTEMS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

REGIONAL BRAIN OVERVIEW

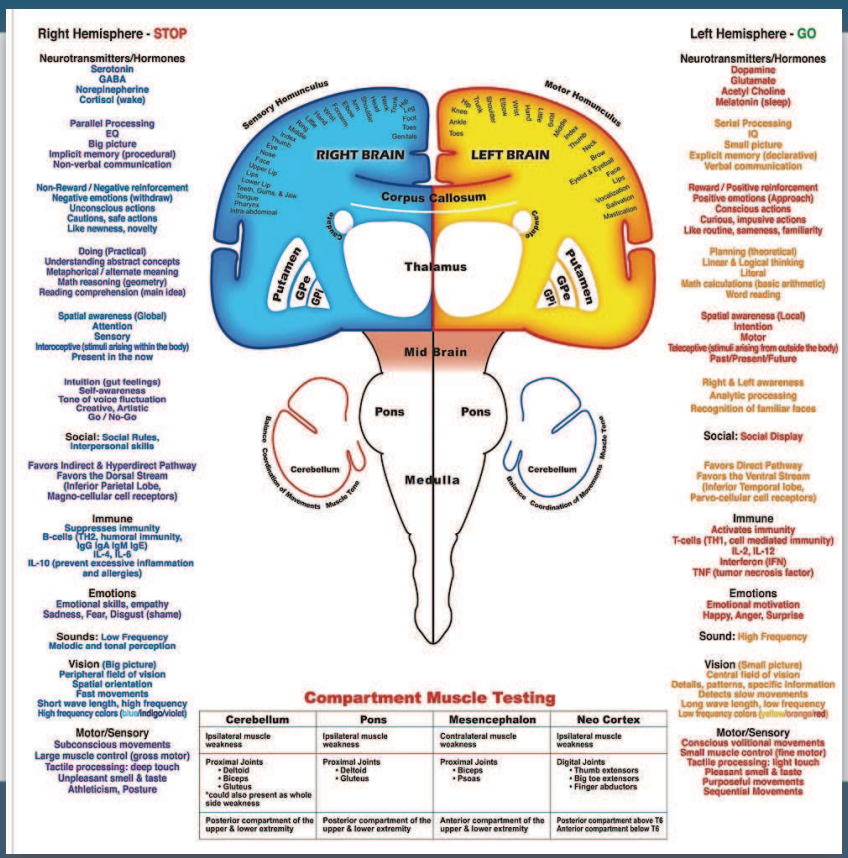


HOW MIGHT YOU STACK/CO-ACTIVATE EACH REGION OF THE CNS WITH LASER AND OTHER RECEPTOR EXERCISES?

	Healthy Brain	Injured Brain
Frontal lobe	Personality / emotions Intelligence Attention / concentration Judgment Body movement Problem solving Speech (speak & write)	Loss of movement (paralysis) Repetition of a single thought Unable to focus on a task Mood swings, irritability, impulsiveness Changes in social behavior and personality Difficulty with problem solving Difficulty with language; can't get the words out (aphasia)
Parietal lobe	Sense of touch, pain and temperature Distinguishing size, shape and color Spatial perception Visual perception	Difficulty distinguishing left from right Lack of awareness or neglect of certain body parts Difficulties with eye-hand coordination Problems with reading, writing, naming Difficulty with mathematics
Occipital lobe	Vision	Defects in vision or blind spots Blurred vision Visual illusions / hallucinations Difficulty reading and writing
Temporal lobe	Speech (understanding language) Memory Hearing Sequencing Organization	Difficulty understanding language and speaking (aphasia) Difficulty recognizing faces Difficulty identifying / naming objects Problems with short- and long-term memory Changes in sexual behavior Increased aggressive behavior
Cerebellum	Balance Coordination	Difficulty coordinating fine movements Difficulty walking Tremors Dizziness (vertigo) Slurred speech
Brainstem	Breathing Heart rate Alertness / consciousness	Changes in breathing Difficulty swallowing food and water Problems with balance and movement Dizziness and nausea (vertigo)

RESTORING BRAIN —HEALTH—

BRAIN INTEGRATION



FIRST PRINCIPLES:

POTENTIAL SPEECH PROTOCOLS (EX EXPRESSIVE APHASIA, STUTTER ETC)

ADHD/OCD/PTSD CO MANAGMENT

CRANIAL NERVE BRAIN BALANCE

LEFT VS. RIGHT HEMISPHERICITY

POSTERS
COURTESY OF DR. CEDRICK NOEL
NEURD SOLUTIONS

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

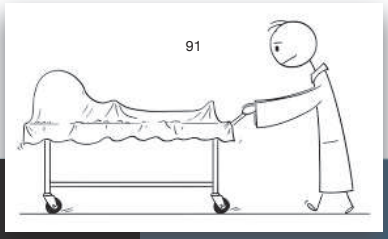
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LONGEVITY METRICS

WHEN IT COMES TO "ALL CAUSE MORTALITY" IT IS COMMON KNOWLEDGE CERTAIN RISK FACTORS HAVE A HIGH ASSOCIATION WITH DYING. FOR EXAMPLE, THE HAZARD RATIO OF HYPERTENSION IS ABOUT 20% INCREASED RISK OF MORTALITY. TYPE 2 DIABETES CARRIES ABOUT A 30% INCREASED RISK. SMOKING ABOUT A 50% INCREASED RISK.

HOWEVER, BEING WEAK VS BEING STRONG FOR YOUR DEMOGRAPHIC CARRIES ABOUT A 250% INCREASED RISK OF ALL CAUSE MORTALITY!!!

HAVING POOR CARDIORESPIRATORY INTEGRITY (BOTTOM 25% VS TOP 2.5%) AS MEASURED BY VO2 MAX, HAS ABOUT A 400% CORRELATION TO INCREASED MORTALITY!!!



ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

LONGEVITY METRICS

Direct Cardiopulmonary Exercise Testing (CPET) is considered the gold standard for VO2 max testing in research

VO_2 Max (CPET) Reference Chart

Direct measurement via metabolic cart

MEN

VO_2 Max (mL/kg/min)	Category	Clinical Interpretation
< 20	Very Low	**High mortality risk**, severe cardiorespiratory limitation
20–29	Low	Elevated CV, metabolic, and neurologic risk
30–39	Moderate	Average functional reserve
40–49	Good	Protective; strong longevity signal
≥ 50	Excellent	Elite cardiopulmonary reserve

High-Risk Thresholds (commonly cited)

- Men: VO_2 max < 30 mL/kg/min
- Women: VO_2 max < 27 mL/kg/min

WOMEN

VO_2 Max (mL/kg/min)	Category	Clinical Interpretation
< 18	Very Low	**High mortality risk**, frailty trajectory
18–27	Low	Increased cardiometabolic and neuro risk
28–37	Moderate	Average functional reserve
38–47	Good	Protective; longevity-associated
≥ 48	Excellent	Elite cardiopulmonary reserve

92

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LONGEVITY METRICS

ALTHOUGH A VARIETY OF TESTS ARE USED AS METRICS FOR STRENGTH SUCH AS CHAIR STAND TEST (5 TIMES SIT TO STAND OUT OF A CHAIR WITH ARMS ACROSS CHEST), AND GAIT SPEED, THE MOST WIDELY USED AND ACCEPTED TEST IS STANDARD GRIP STRENGTH USING A DYNAMOMETER!

Handgrip Strength Reference Chart (Dynamometer)

Units: pounds (lb)

Best of 2–3 maximal efforts; dominant hand

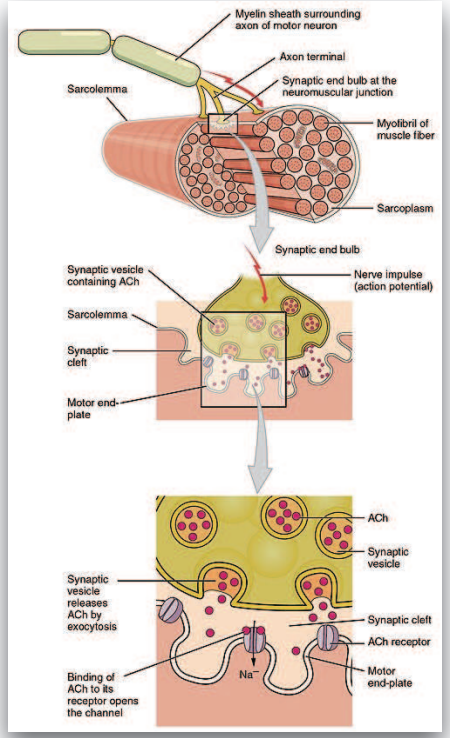
Sex	Low / High Risk	Moderate	Good / Protective
Men	< 57 lb	57–77 lb	> 77 lb
Women	< 35 lb	35–55 lb	> 55 lb

(Conversion used: 1 kg = 2.205 lb)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

LONGEVITY METRICS



REMEMBER, ACETYLCHOLINE IS NOT JUST FOR BRAIN HEALTH, MEMORY AND COGNITIVE FUNCTION. EVERY VOLUNTARY MUSCLE CONTRACTION STARTS WITH ACH AT THE MYONEURAL JUNCTION!!! AND WITH REPETITIVE FIRING, RESISTANCE, COMPLEX MOVEMENTS ETC YOU GET MORE PROTEIN REPLICATION (EAT YOUR PROTEIN!), MORE LEAN MUSCLE MASS AND STRENGTH, BETTER LARGE DIAMETER FEEDBACK TO KEEP THE BRAIN ALIVE (GRAVITY!) AND MORE GROWTH FACTORS PRODUCED WITH THE MUSCLE ACTIVATION (BDNF!). ALSO, PROMOTES OTHER MECHANISMS LIKE BETTER PROPRIOCEPTIVE FEEDBACK (IE. SPINOCEREBELLAR SYSTEMS) FOR BALANCE AND FALL PREVENTION (#1 PREVENTABLE DEATH IN THE ELDERLY), POSTURAL DYNAMICS WHICH HELP MANY THINGS (PAIN AND ORTHOPEDIC INJURY REDUCTION, BREATHING CAPACITY (POSTURE/RIB MECHANICS/DIAPHRAGM) AND MUCH MORE!!!

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Science News *from research organizations*

A 47-year study reveals when fitness and strength start to fade

Date: January 18, 2026

Source: Karolinska Insitutet

Summary: A long-running Swedish study has followed adults for nearly five decades, uncovering when physical decline truly begins. Fitness and strength start slipping around age 35, then worsen gradually with age. The encouraging twist: adults who began exercising later still improved their physical capacity by up to 10 percent. It's a powerful reminder that staying active matters, even if you start late.

Most earlier research in this area relied on cross-sectional data, comparing different age groups rather than following the same individuals. **In contrast, the SPAF study repeatedly measured fitness and strength in the same participants across Sweden for nearly half a century, making it one of the most comprehensive efforts of its kind.**

The results show that both fitness and strength start to decrease as early as age 35, regardless of how much people trained earlier in life. From that point forward, physical decline continues gradually and tends to speed up with advancing age. Despite this pattern, the researchers found encouraging evidence that exercise remains valuable at any stage. **Participants who became physically active during adulthood increased their physical capacity by 5-10 percent.**

The research is ongoing. **Next year, the participants will be examined again when they reach age 68. The team hopes to better understand how changes in physical performance are connected to lifestyle choices, overall health, and underlying biological processes.**

1. Maria Westerståhl, Gustav Jörnåker, Eva Jansson, Ulrika Aasa, Michael Ingre, Kaveh Pourhamidi, Brun Ulfhake, Thomas Gustafsson. **Rise and Fall of Physical Capacity in a General Population: A 47-Year Longitudinal Study**. *Journal of Cachexia, Sarcopenia and Muscle*, **2025**; 16 (6) DOI: 10.1002/jcsm.70134

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PROTEIN RDA = 0.8 g/kg/day

- **This is a minimum to avoid deficiency, not optimal brain health.**

🔬 **Core brain functions dependent on protein**

- **Neurotransmitters**
  - *Tryptophan → serotonin*
  - *Tyrosine → dopamine, norepinephrine*
  - *Glutamine → glutamate & GABA*
- **Structural integrity**
  - Neuronal membranes
  - Myelin repair
  - Cytoskeletal proteins
- **Neuroplasticity**
  - Synaptogenesis
  - BDNF signaling
- **Detox & resilience**
  - Glutathione synthesis
  - Methylation support
- **Recovery & repair**
  - Post-TBI, stroke, neuroinflammation, aging

- **Many adults:**
  - Eat **most protein at dinner**
  - Skip protein at breakfast
  - Under-consume total daily protein
- **Older adults (50+)** are the *most deficient*, despite the *highest need*.

Who is most under-proteinized?

- Older adults
- Women
- Chronic dieters
- Vegetarians/vegans without planning
- Patients with GI dysfunction, inflammation, or poor digestion

Paradox:
Americans often eat *too many calories* but **too little usable protein**.

ERCHONIA® Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS PROTEIN INTAKE SUMMARY

Practical brain-health takeaways (adults):

- Ensure at least **RDA or above**; many experts recommend **~1.0–1.2 g/kg/day minimum in older adults** to preserve muscle, which is strongly tied to cognitive outcomes [3] [11] [16].
- Within the **1.0–1.6 g/kg/day** range, focus on **protein quality and sources**: more **plant protein, fish, legumes, lean poultry**, and fewer processed meats and high-sugar foods, as these patterns associate with better cognitive outcomes [2] [4] [14] [8] [18].
- Distribute protein across the day (especially **adequate dinner protein**, which is associated with better cognition in older adults) rather than concentrating everything at one meal [10] [15] [19].

Summary

- For **lean muscle**: strongest evidence supports **~1.2–1.6 g/kg/day**, with resistance training, and ~0.25–0.4 g/kg per meal [1] [5] [7].
- For **brain health**: no precise gram/kg "optimum," but **adequate-to-higher protein (≥1.0–1.2 g/kg/day)**, maintaining muscle, and favoring **high-quality, largely unprocessed protein sources** is associated with better specific cognitive domains and lower subjective cognitive decline [2] [4] [6] [11] [14] [18].

97

MINIMUM 1.2 g/kg/day FOR ADULTS BUT TARGET AS MUCH AS 1.6 g/kg/day IN CERTAIN SITUATIONS LIKE HEAVY TRAINING, NEURODEGENERATION AND HEAD INJURY RECOVERY

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

CONSENSUS PROTEIN INTAKE SUMMARY

Daily Protein Intake at 1.2 g/kg

Adult Females

Body Weight	Weight (kg)	Protein / Day
120 lb	54.5 kg	65 g
130 lb	59 kg	71 g
140 lb	63.6 kg	76 g
150 lb	68 kg	82 g
160 lb	72.7 kg	87 g
170 lb	77 kg	92 g

Adult Males

Body Weight	Weight (kg)	Protein / Day
150 lb	68 kg	82 g
160 lb	72.7 kg	87 g
170 lb	77 kg	92 g
180 lb	81.8 kg	98 g
190 lb	86.4 kg	104 g
200 lb	90.9 kg	109 g

98

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PROTEIN CACULATION APPS

Top Recommendations:

- **MyFitnessPal (MFP): A very popular choice with a massive food database and barcode scanner, making it simple to log meals and get instant protein, calorie, and macro breakdowns.**
- **Cronometer: Known for its accuracy, it tracks not just macros but also micronutrients, giving a detailed view of your diet with a robust food database.**
- **Protein Pal / Protein Tracker: Specifically designed for protein tracking, these apps focus on simplicity, allowing for quick logging and monitoring of your daily protein goals with a high-protein food list.**
- **Foodnoms / MacrosFirst: Good options for tracking overall nutrition (macros, calories) and protein, often with user-friendly interfaces.**

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

WATCH THE JOE ROGAN PODCAST #2461
ROBERT F. KENNEDY, JR.

~60% of the brain is fat (dry weight).

DHA, phospholipids, cholesterol are structural, not optional.

Muscle is the brain's metabolic battery. This pyramid supports lean muscle mass and development.

What inflames the gut, inflames the brain. By replacing pesticides, toxins, bad carbs and ultra processed foods with good organic fats and protein, the gut and brain will be less inflamed.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Common Sweetener May Damage Critical Brain Barrier, Risking Stroke

In the recent study, researchers exposed blood-brain barrier cells to levels of erythritol typically found **after drinking a soft drink sweetened with the compound.** They saw a chain reaction of cell damage that could make the brain more vulnerable to blood clots – a leading cause of stroke.

Erythritol triggered what scientists call oxidative stress, **flooding cells with harmful, highly reactive molecules known as free radicals, while simultaneously reducing the body's natural antioxidant defences.** This double assault **damaged the cells' ability to function properly, and in some cases killed them outright.**

The study found that erythritol disrupted this critical system, **reducing nitric oxide production while ramping up endothelin-1. The result would be blood vessels that remain dangerously constricted**, potentially starving the brain of oxygen and nutrients.

This imbalance is a known warning sign of ischaemic stroke – the type caused by blood clots blocking vessels in the brain.

The non-nutritive sweetener erythritol adversely affects brain microvascular endothelial cell function

Authors: Auburn R. Berry https://orcid.org/0009-0002-6326-193X, Samuel T. Ruzzene, Emily I. Ostrander, Kendra N. Wegerson, Nathalie C. Orozco-Fersiva, Madeleine F. Stone, Whitney B. Valenti, Joao E. Izaias, Joshua P. Holzer, Jared J. Greiner, Vinicius P. Garcia https://orcid.org/0000-0003-3022-1616, and Christopher A. DeSouza https://orcid.org/0000-0001-9372-5083 AUTHORS INFO & AFFILIATIONS
Publication: Journal of Applied Physiology
Volume 138, Issue 6
https://doi.org/10.1152/japplphysiol.00276.2025

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

"THE DIRTY DOZEN" 2024

THE DIRTY DOZEN
1. STRAWBERRIES
2. SPINACH
3. KALE, COLLARD, MUSTARD GREENS
4. GRAPES
5. PEACHES
6. PEARS
7. NECTARINES
8. APPLES
9. BELL AND HOT PEPPERS
10. CHERRIES
11. BLUEBERRIES
12. GREEN BEANS

Farmworkers who use or are exposed to pesticides are at highest risk, according to studies. A 2022 meta-analysis found workers exposed to pesticides were nearly five times as likely to have DNA damage while a February study found children exposed at an early age showed poorer neurodevelopment from infancy to adolescence.

Approximately 95% of nonorganic strawberries, leafy greens such as spinach and kale, collard and mustard greens, grapes, peaches and pears tested by the United States government contained detectable levels of pesticides, according to the 2024 Shopper's Guide to Pesticides in Produce.

THE CLEAN 15
1. AVOCADOS
2. SWEET CORN
3. PINEAPPLE
4. ONIONS
5. PAPAYA
6. SWEET PEAS (FROZEN)
7. ASPARAGUS
8. HONEYDEW MELON
9. KIWI
10. CABBAGE
11. WATERMELON
12. MUSHROOMS
13. MANGOES
14. SWEET POTATOES
15. CARROTS

Pesticides have been linked in studies to preterm births, congenital malformations such as neural tube defects, spontaneous abortions and an increase in genetic damage in humans. Exposure to pesticides has also been associated with lower sperm concentrations, heart disease, cancer and other disorders.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RECEPTOR ACTIVATION AND NEUROPLASTICITY

Exercise Alters Brain Chemistry to Protect Aging Synapses

FeaturedNeuroscience January 7, 2022

Summary: *Older adults who remain active have more of a class of proteins that enhance synapses to help maintain healthy cognitive function.*

Source: *UCSF*

When elderly people stay active, their brains have more of a class of proteins that enhances the connections between neurons to maintain healthy cognition, a UC San Francisco study has found.

This protective impact was found even in people whose brains at autopsy were riddled with toxic proteins associated with Alzheimer's and other neurodegenerative diseases.

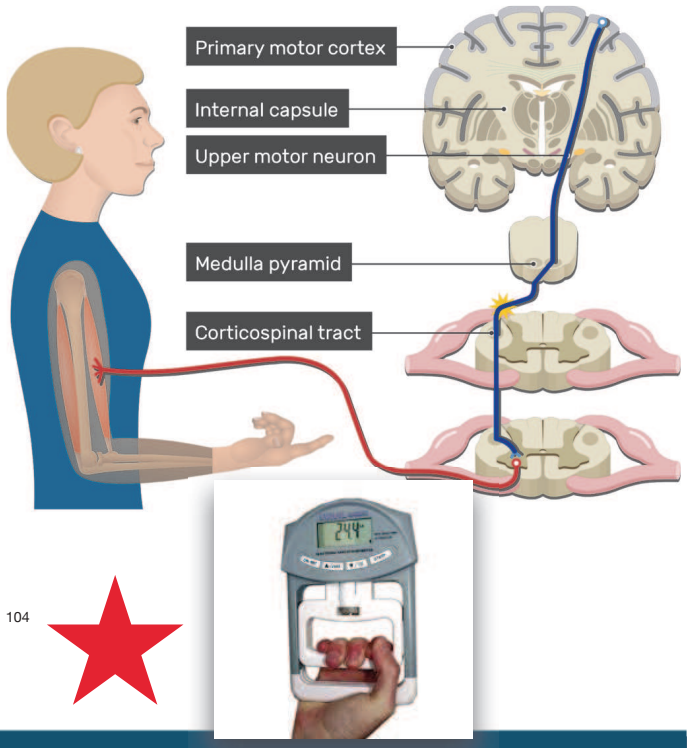
LET'S WALK THROUGH THE NEUROLOGY OF RECEPTOR ACTIVATION SUCH AS HOW A CHIROPRACTIC ADJUSTMENT OR EXERCISE MAY PROMOTE NEUROPLASTICITY AND PROTECT YOUR BRAIN FROM NEURODEGENERATIVE CONDITIONS. (HINT RECEPTOR SYSTEMS AND GROWTH FACTORS)

RESTORING BRAIN —HEALTH—

DOWNSTREAM DYNAMOMETER PROTOCOL

HOW TO MOTOR CALIBRATE SYSTEMS FROM CENTRAL TO END TARGET

WITH CROSSOVER INTO PAIN MANAGEMENT



ERCHONIA Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

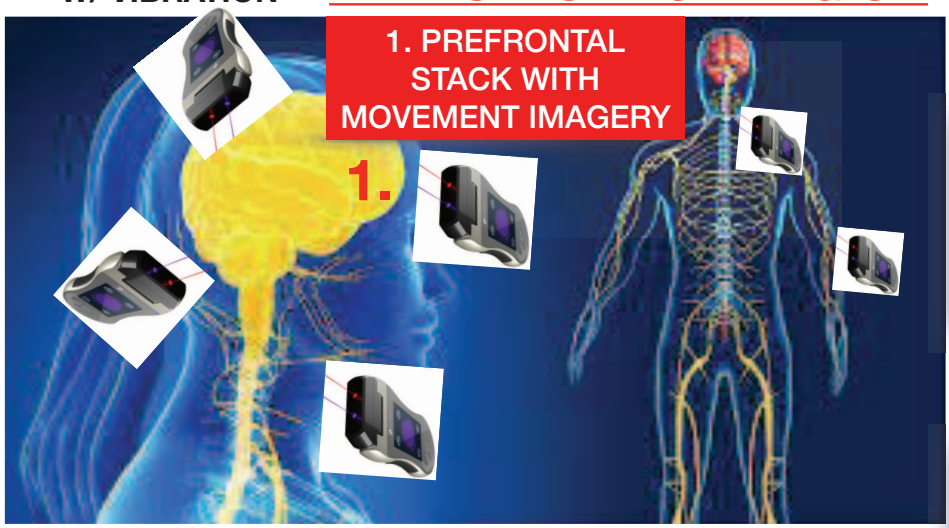
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THE "DOWNSTREAM" MOTOR PROTOCOL

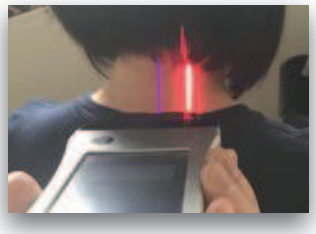
(FOR EVEN BETTER RESULTS, PERFORM AFTER BRAIN BALANCE)

SUMMARY OVERVIEW
2 MINUTES EACH REGION

2. PARIETAL LOBE W/ VIBRATION



5. SPINAL CORD/ NERVE ROOT



6. TARGET MUSCLE/ DISTAL TISSUE

3. IPSI CEREBELLUM STACK WITH MOVEMENT/ISOMETRICS

4. VAGAL NERVE STIMULATION IPSILATERAL TO INVOLVED SIDE

USE 10-40-10-40

MOTOR AND SENSORY MIRROR THERAPIES

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

COVID VACCINE INJURY STUDY

Vaccine
Volume 42, Issue 9, 2 April 2024, Pages 2200-2211

COVID-19 vaccines and adverse events of special interest: A multinational Global Vaccine Data Network (GVDN) cohort study of 99 million vaccinated individuals

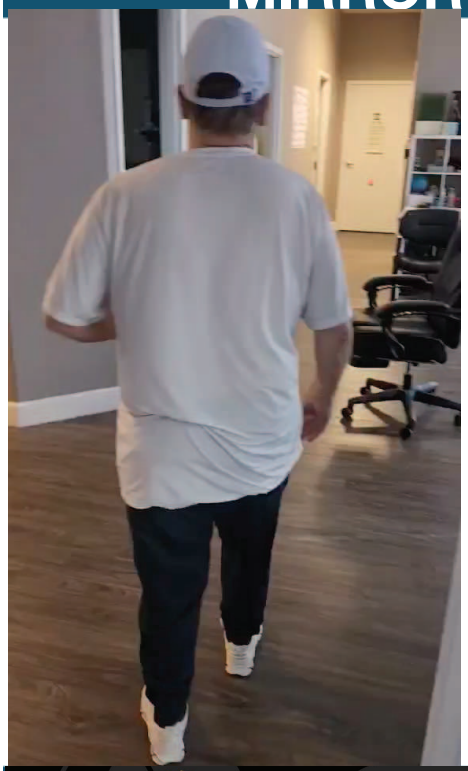
K. Faksova a

, **The identified increased risk of CVST (CEREBRAL VENOUS SINUS THROMBOSIS) following ChAdOx1 vaccination in this study is corroborated by multiple studies**. An increased OE ratio was observed in a nationwide cohort study from Denmark and Norway, with increased rates of venous thromboembolic events, including CVST with an excess rate of 2.5 events per 100,000 vaccinations following ChAdOx1 [7]. **Based on a variety of methodologies, other studies have also reported increased incidence of CVST after vaccination [31], [32]**

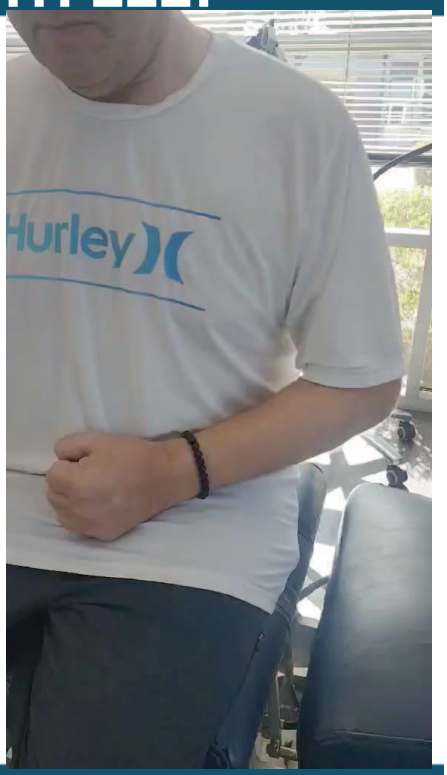
Risk periods following homologous vaccination schedules contributed 23,168,335 person-years of follow-up. OE ratios with LBCI > 1.5 were observed for **Guillain-Barré syndrome** (2.49, 95 % CI: 2.15, 2.87) **and cerebral venous sinus thrombosis** (3.23, 95 % CI: 2.51, 4.09) following the first dose of ChAdOx1 vaccine. **Acute disseminated encephalomyelitis** showed an OE ratio of 3.78 (95 % CI: 1.52, 7.78) following the first dose of mRNA-1273 vaccine. The OE ratios for **myocarditis and pericarditis following BNT162b2, mRNA-1273, and ChAdOx1 were significantly increased with LBCIs > 1.5**.

ERCHONIA Healing Light, Perfected.
RESTORING BRAIN HEALTH

COVID VACCINE INJURIES, CVA MIRROR THERAPIES WITH LLLT



SHAWN K. INITIAL EXAM AND TREATMENT DAY FOR INTENSIVE (FATHER BROUGHT HIM IN FROM IDAHO JANUARY 13, 2025. WAS A HEALTHY SURFING, SCUBA DIVING, HELICOPTER PILOT IN HAWAII. RIGHT HEMI CVA THE DAY AFTER HE RECEIVED A COVID AND FLU SHOT.



ERCHONIA Healing Light, Perfected.
RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

COVID VACCINE INJURY

CONTINUATION OF DAY 1 "DIAGNOSTIC TREATMENT". STACK YOUR LASER WITH YOUR TREATMENT MODALITY TO SEE IF CAN HELP THE NERVOUS SYSTEM "TURN ON"

USING THE GVL ON HIS LEFT CEREBELLUM AND EVRL ON HIS RIGHT PARIETAL FRONTAL REGION USING THE MASTER BRAIN SETTING

CVA POST COVID VACCINE

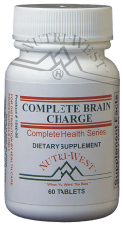
SHAWN K. DAY 3 OF TREATMENT 1/21/2025

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LONG COVID CONSIDERATIONS

1. CLEAR OUT THE S2 SPIKE PROTEIN (NATTOKINASE)
2. IMMUNE SUPPORT (NAC, HIGH DOSE VIT D/A, TURMERIC, OMEGA 3/PRM) - ALL CIS CONDITIONS
3. GUT MICROBIOME
4. ACETYLCHOLINE SUPPORT (nACH RECEPTORS)
5. ADIPOSE TISSUE REDUCTION AND SYSTEMIC INFLAMMATION
6. HISTAMINES/MCAS (QUERCITIN, DAO, VIT C, GREEN TEA/EGCG)
7. DYSAUTONOMIA! (FUNCTIONAL NEUROLOGY SUPPORT - TILT TABLE, OVAR, POTS)



ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

S2 SPIKE PROTEIN

Published online 2022 Aug 24. doi: 10.3390/molecules27175405

PMCID: PMC9458005
PMID: 36080170

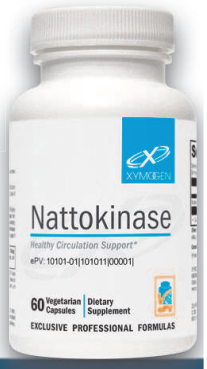
Degradative Effect of Nattokinase on Spike Protein of SARS-CoV-2

Takashi Tanikawa,

Abstract

The coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged as a pandemic and has inflicted enormous damage on the lives of the people and economy of many countries worldwide. However, therapeutic agents against SARS-CoV-2 remain unclear. SARS-CoV-2 has a spike protein (S protein), and cleavage of the S protein is essential for viral entry. Nattokinase is produced by *Bacillus subtilis* var. *natto* and is beneficial to human health. In this study, we examined the effect of nattokinase on the S protein of SARS-CoV-2. When cell lysates transfected with S protein were incubated with nattokinase, the S protein was degraded in a dose- and time-dependent manner. Immunofluorescence analysis showed that S protein on the cell surface was degraded when nattokinase was added to the culture medium.

Thus, our findings suggest that nattokinase exhibits potential for the inhibition of SARS-CoV-2 infection via S protein degradation.



ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
Without permission of Trevor Berry, DC, DACNB is prohibited.

CARDIOVASCULAR DISEASE AND LLLT

Research Article ISSN 2771-9057

Recent Advances in Clinical Trials

Exploring A Novel Approach Using Red and Violet Laser Therapy For Carotid Artery Stenosis - A Case Series

Andrzej Eberhardt[1] and Travis Sammons[2*]

[1]General surgery and Vascular, Vascular Surgery and Phlebology in Femmed Clinic, Warsaw, Poland.

[2]Erchonia Corporation, Melbourne, FL, USA.

*Correspondence:
Travis M. Sammons, Erchonia Corporation, 112 Southchase Bvld., Fountain Inn, SC 29644, Tel: 888-242-0571, Fax: 321.473.1608.

Received: 20 Mar 2024; Accepted: 26 Apr 2024; Published: 03 May 2024

Citation: Eberhardt A, Sammons T. Exploring A Novel Approach Using Red and Violet Laser Therapy For Carotid Artery Stenosis - A Case Series. Recent Adv Clin Trials. 2024; 4(2); 1-6.

Keywords
Carotid artery stenosis, Violet laser therapy, Ischemic stroke, Novel therapeutic approaches.

Introduction

Carotid artery stenosis (CS), characterized by atherosclerotic plaque accumulation within the carotid arteries, is a prominent risk factor for ischemic stroke [1]. The World Health Organization recognizes ischemic stroke as the second leading cause of mortality worldwide [2]. Complications arising from unstable atherosclerotic plaque, primarily through embolization (blood clot formation and travel), are responsible for approximately 75-80% of ischemic strokes [3]. However, reduced blood flow (hypoperfusion) due to severe stenosis also plays a role, potentially contributing to an estimated 20-25% of cases. This established link between CS and stroke necessitates the exploration of novel therapeutic approaches.

Current management strategies for CS primarily focus on lifestyle modifications, such as a healthy diet, exercise, and smoking cessation, which aim to improve overall cardiovascular health and potentially slow atherosclerotic plaque progression [4]. Antiplatelet medications are a mainstay of medical therapy [5], while these drugs cannot reverse arterial narrowing, they play a crucial role in reducing platelet activity and consequently the risk of stroke. However, long-term use of antiplatelet drugs comes with its own set of side effects, including stomach ulcers, kidney problems, high blood pressure, and tinnitus. For symptomatic patients with severe stenosis (70% to 99%), carotid endarterectomy, a surgical procedure to remove plaque, is often recommended [6].

Recent studies investigating the effects of low-level laser therapy (LLLT) on vascular health have shown promising results, and may provide a non-invasive, and non- pharmacological therapeutic option for CS. LLLT appears to influence factors like vascular endothelial growth factor (VEGF) and may potentially reduce inflammation within the vessel wall and plaque structure [7,8]. This study aims to contribute to the expanding body of research by evaluating the potential therapeutic effects of LLLT on both plaque size and morphology in patients diagnosed with CS.

Material and Methods

This study enrolled a total of 30 patients (17 male, 13 female) receiving treatment at a vascular surgery Femmed clinic in Warsaw, Poland. All participants were on established pharmacological therapy regimens, adhering to the 2023 Clinical Practice Guidelines on the Management of Atherosclerotic Carotid and Vertebral Artery Disease set forth by the European Society for Vascular Surgery. Throughout the 12-week study period, each patient underwent bi-weekly ultrasound examinations to monitor their condition. Both the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria and the Gray-Scale Median (GSM) scale were employed to assess the severity of carotid artery stenosis and atherosclerotic plaque instability, respectively. The GSM value is one of the most widespread methods of studying atherosclerotic plaques and it is important to comprehensively standardize image post-processing. Higher GSM values indicate denser, more calcified plaques, which are generally considered more stable. A lower GSM value suggest softer plaques with a higher lipid or fluid content, potentially indicating a more unstable plaque and a worse prognosis for CS.

All participants received laser therapy using the Erchonia EVRL device, which emits a combination of red (635 nm) and blue-violet (405 nm) lasers, each delivering a power output of 7.5

Conclusions

Our investigation explored the application of **low-level laser therapy with red and violet/blue wavelengths in patients with carotid artery stenosis. The results demonstrated a significant reduction in stenotic changes, along with enhanced stability of atherosclerotic plaque and reduced inflammation. Notably, these positive outcomes were independent of factors such as gender, smoking history, or diabetic status. These findings suggest that low-level laser therapy holds promise as a therapeutic strategy to not only impede atherosclerotic progression but also potentially mitigate its complications, including ischemic stroke.** Further exploration through multicenter trials is crucial to elucidate the underlying mechanisms of action and firmly establish this modality as a novel and efficacious treatment for vascular diseases.

(12 WEEKS OF TREATMENT. BI-WEEKLY 15 MINUTES EVRL)

CARDIOVASCULAR SUPPORT

OPTIMAL XYMOGEN MEDPAX FOR CARDIOVASCULAR SUPPORT:

DIOVASC = DIOSMIN THINK STRONG BLOOD VESSELS, IMPROVED MICROCIRCULATION AND LYMPHATICS, GLUCOSE METABOLISM AND ANTIOXIDANT SUPPORT. MAY ALSO HELP WITH S2 SPIKE AND CAPILLARY STABILITY WHICH CAN LEAD TO END ORGAN FAILURE IN COVID-19

BERGACOR PLUS = TARGETS POOR CARDIOMETABOLIC PROFILE ALONG WITH PERIPHERAL INSULIN RESISTANCE FOR HEALTHY LIPID AND GLUCOSE LEVELS. CONTAINS THE HIGHEST DOSE OF BERGAMOT ON THE MARKET ALONG WITH THE SUPER FRUIT AMLA. WILL ALSO SUPPORT HIGH HS-CRP

NIAVASC = NIACIN FOR HEALTHY LIPIDS AND TUNICA INTIMA-MEDIA

ENDOCALYX SUPPORTS AND MAINTAINS A HEALTHY GLYCOCALYX WHICH IS A LIKE A MICRO-THIN SHIELD PROTECTING THE BLOOD VESSELS INCLUDING CORONARY ARTERIES AND MICROCIRCULATION LIKE CAPILLARY BEDS. IT KEEPS BLOOD VESSELS SLICK, SMOOTH AND PROTECTED WHILE OPTIMIZING BLOOD FLOW, GAS AND NUTRIENT EXCHANGE

* ALWAYS ADDRESS UNDERLYING CO-MORBIDITIES, ELEVATED HOMOCYSTEINE, SAD DIET AND OTHER SOURCES OF INFLAMMATION, SEDENTARY LIFESTYLE ETC.

Duplication, citation or distribution of this material in whole or in part
Without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MALE WITH CONCUSSION INJURY AND LOSS OF MOTION IN LEFT SHOULDER

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
HEALTH

PAIN MANAGEMENT

PERIPHERAL SENSITIZATION DOWN REGULATION

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THE NEED FOR SAFE AND RESEARCHED ALTERNATIVES TO PAIN

Abstract

Introduction: Gabapentin is widely used to treat chronic pain, but its association with cognitive decline and dementia remains unclear. **This study examined whether gabapentin prescription is associated with dementia in adults with chronic low back pain.**

Methods: We conducted a retrospective cohort study using the TriNetX national database of de-identified patient records from 2004 to 2024. Adults diagnosed with chronic low back pain were included; those with prior gabapentin use, dementia, epilepsy, stroke, or cancer were excluded. **Propensity score matching controlled for demographics, comorbidities, and pain medications. Patients were stratified by age and gabapentin prescription frequency. Primary outcomes were dementia and mild cognitive impairment.**

Results: 26,416 adults we analyzed following propensity-score matching. Patients with six or more gabapentin prescriptions had an increased incidence of dementia (RR: 1.29; 95% CI: 1.18-1.40) and mild cognitive impairment (RR: 1.85; 95% CI: 1.63-2.10). When stratified by age, ***non-elderly adults (18-64) prescribed gabapentin had over twice the risk of dementia*** **(RR: 2.10; 95% CI: 1.75-2.51) and mild cognitive impairment (RR: 2.50; 95% CI: 2.04-3.05) compared to those not prescribed gabapentin.** ***Risk increased further with prescription frequency*****: patients with 12 or more prescriptions had a higher incidence of dementia (RR: 1.40; 95% CI: 1.25-1.57) and mild cognitive impairment (RR: 1.65; 95% CI: 1.42-1.91) than those prescribed gabapentin 3-11 times.**

Conclusions: ***Gabapentin prescription in adults with chronic low back pain is associated with increased risk of dementia and cognitive impairment, particularly in non-elderly adults.*** Physicians should monitor cognitive outcomes in patients prescribed gabapentin.

Reg Anesth Pain Med

2025 Jul 10:rapm-2025-106577. doi: 10.1136/rapm-2025-106577. Online ahead of print.

Risk of dementia following gabapentin prescription in chronic low back pain patients

Nafis B Eghrari 1

ERCHONIA Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

WHAT TYPES OF NEUROMUSCULOSKELETAL CONDITIONS HAVE LASERS BEEN RESEARCHED FOR HELPING…

MOJ Orthop Rheumatol. 2015; 2(5): 00068.
Published online 2015 Jun 9. doi: 10.15406/mojor.2015.02.00068

The Use of Low Level Laser Therapy (LLLT) For Musculoskeletal Pain

Howard B Cotler,1,2,* Roberta T Chow,3 Michael R Hamblin,4,5,6 and James Carroll7

Abstract

LLLT in the treatment of pain

Acute orthopedic conditions such as sprains [52,53], strains, post-surgical pain, a whiplash injury [54], muscular back pain, cervical or lumbar radiculopathy [55,56], tendinitis [57,58] and chronic conditions such as osteoarthritis [59–64], rheumatoid arthritis, frozen shoulder [65], neck and back pain [56], epicondylitis [66], carpal tunnel syndrome [67,68], tendinopathy [69], fibromyalgia [70], plantar fasciitis [70], post tibial fracture surgery [9] and chronic regional pain syndrome are amenable to LLLT. Dental conditions producing pain such as orthodontic procedures [71], dentine hypersensitivity [72], and third molar surgery [73] respond well to treatment with LLLT. Neuropathic pain conditions can also be treated such as post herpetic neuralgia [74], trigeminal neuralgia (10), and diabetic neuropathy [75]. Due to the wide spectrum of conditions one would surmise that multiple mechanisms can operate to achieve pain relief.

The peripheral nerve endings of nociceptors, consisting of the thinly myelinated Aϑ and unmyelinated, slow-conducting C fibers, lie within the epidermis. This complex network transduces noxious stimuli into action potentials. Moreover these nerve endings are very superficial in nature and thus are easily within the penetration depths of the wavelengths used in LLLT

ERCHONIA Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CHRONIC LOW BACK STUDY KEY POINTS

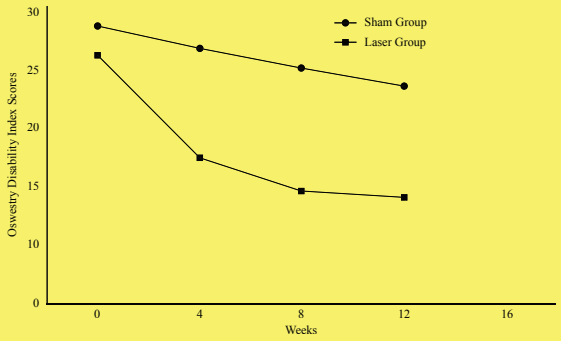
- ON AVERAGE PARTICIPANTS HAD LOW BACK PAIN FOR 97.8 MONTHS (OVER 8 YEARS)
- ONLY THE FX 635 COULD BE USED DURING THE STUDY. THEY COULD NOT RECEIVE NARCOTICS, CHIROPRACTIC, MASSAGE ETC
- PATIENTS CONTINUED TO IMPROVE WELL AFTER THE END OF THE ACTIVE TREATMENTS FOR THE TREATMENT GROUP (VS LED PLACEBO BEGAN TO RETURN TO INITIAL PAIN STATUS)
- NO ADVERSE EVENTS WERE REPORTED DURING THE STUDY FOR THE TREATMENT GROUP

Chronic Low Back Pain Study

	Test subjects	Placebo subjects
n	29	29
n meeting success criteria	21	8
% meeting success criteria	**72.4%**	**27.6%**

Table 16: Individual Success Criteria met by procedure group

There is a difference of 44.8% in the proportion of subjects who met the individual success criteria between procedure groups, such that 44.8% more test group than placebo group subjects evidenced a decrease in low back pain rating on the VAS of 30% or greater from baseline to study endpoint, exceeding the pre-established target of a 35% difference between procedure groups by +9.8%, indicative of **superior treatment effect of the Erchonia® FX-635™ Laser over placebo**.



	Test Group (n=29)		Placebo Group (n=29)	
	Mean	SD	Mean	SD
Baseline	59.00	11.77	59.21	13.23
Endpoint	24.76	24.27	48.24	26.90
# Change	***-34.24***	*25.94*	***-10.97***	*24.69*

Table 17: Baseline and endpoint VAS by procedure group

The 34.24-point mean decrease in VAS ratings from study Baseline to Endpoint for test group subjects is more than 3 times greater than the relative 10.97-point mean decrease in VAS ratings attained for placebo group subjects.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

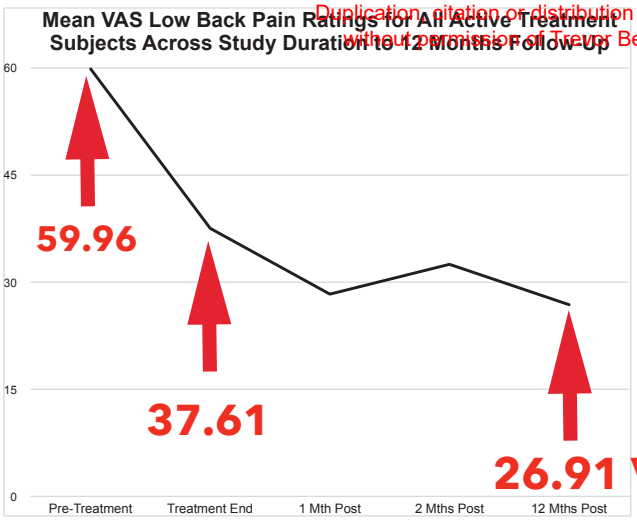


Chart 1: Mean VAS Low Back Rating for Active Treatment Subjects from Baseline to 12-Months Follow-up

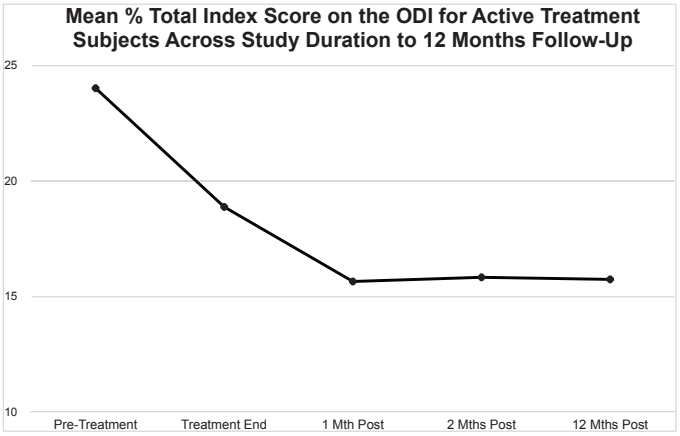


Chart 3: Mean % Total Index Score on the ODI for Active Treatment Subjects from Baseline to 12-Months Follow-up

DEPARTMENT OF HEALTH AND HUMAN SERVICES
Food and Drug Administration

Indications for Use

Form Approved: OMB No. 0910-0120 122
Expiration Date: 06/30/2020
See PRA Statement below.

510(k) Number *(if known)*
K180197

Device Name
Erchonia® FX-635

Indications for Use *(Describe)*
The Erchonia® FX-635 laser is indicated for the following two indications:
a. as an adjunct to provide relief of minor chronic low back pain of musculoskeletal origin.
b. as an adjunct to reducing chronic heel pain arising from plantar fasciitis.

IT'S OFFICIAL. ERCHONIA RECEIVED FORMAL FDA CLEARANCE FOR THE FX-635 TO TREAT CHRONIC LOW BACK PAIN!!!!!

ERCHONIA
World Leaders in Low Level Laser Technology

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FDA U.S. FOOD & DRUG ADMINISTRATION

Erchonia Corporation c/o Steven Shanks President
650 Atlantis Road Melbourne, FL 32904

June 1, 2019

Re: K190572
Trade/Device Name:
Regulation Number: 21 CFR 890.5500

Regulatory Class: Class II
Product Code: NHN
Dated: March 5, 2019
Received: March 6, 2019

THE FX 635 (17.5 MW X 3 ROTATING LINE BEAM GENERATION DIODES) WAS GRANTED FDA APPROVAL (JUNE 1, 2019) FOR TREATMENT OF *ALL* NOCICEPTIVE MUSCULOSKELETAL PAIN CONDITIONS!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LOW BACK PAIN PROTOCOLS

PAIN AND WAVELENGTHS

	Red Laser Only	Red & Violet Combination	Green & Violet Combination
Subjects (n)	N=43	N=44	N=43
Duration of pain (months)	61.7	76.58	89.19
Subjects meeting study success criteria, ≥ 30% pain reduction	65%	75%	81.4%
(%) Improvement in VAS from baseline to immediately after treatment	48.17%	45.84%	52.61%
(%) Improvement in VAS from study endpoint to 48 hrs. post-treatment	43.19%	50.17%	65.20%

COMPARISON

1 TREATMENT NECK AND SHOULDER PAIN: XLR8 VS EVRL VS GVL

© 2022 Erchonia Corporation
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

SAFE AND EFFICACIOUS

THROUGHOUT THE ENTIRE HISTORY OF THE COMPANY, OF ALL OF ERCHONIA'S LEVEL 1 FDA CLEARANCE STUDIES THERE HAS NEVER BEEN A REPORTED ADVERSE EVENT OR REACTION TO THE LASER TREATMENTS!

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

127

ERCHONIA 510K FDA CLEARANCES

ERCHONIA
World Leaders in Low Level Laser Technology

Erchonia Corporation

Changing Medicine Through Technology

- First company in the World to Garner FDA clearance for LLLT in 2002
- Erchonia holds 22 FDA 510(k) Clearances in LLLT with specific "IFU" (Indication for Use) which is more than all other low-level lasers combined
  - Substantial Equivalent- *"Heating Lamp"*

Erchonia 510(k)s

- 2002. CHRONIC NECK AND SHOULDER PAIN
- 2004. POST SURGICAL PAIN FOLLOWING LIPOSUCTION
- 2005. ACNE VULGARIS
- 2008. POST SURGICAL PAIN FOLLOWING BREAST AUGMENTATION
- 2010. CIRCUMFERENCE REDUCTION OF THE WAIST, HIPS AND THIGHS
- 2012. CIRCUMFERENCE REDUCTION OF THE ARMS
- 2013. CIRCUMFERENCE REDUCTION WAIST, HIPS, THIGH (GREEN LASER)
- 2013. CELLULITE
- 2014. CHRONIC HEEL PAIN AND PLANTAR FASCIITIS
- 2014. OBESITY CIR_CUMFERENTIAL REDUCTION
- 2015. ZERONA OTC (OVER THE COUNTER)
- 2015. ZERONA EZ6 PROTOCOL
- 2016. ONYCHOMYCOSIS
- 2016. OVERALL BODY INDICATION FOR BODY CONTOURING
- 2018. CHRONIC LOWER BACK PAIN
- 2019. OVERALL MUSCULOSKELETAL PAIN
- 2019. CHRONIC NECK AND SHOULDER PAIN USING EVRL (VIOLET & RED LASER)
- 2020. NON-EVASIVE FAT LOSS FOR PATIENTS WITH UP TO 40 BMI (GREEN LASER)
- 2021. POST OPERATIVE PAIN
- 2021. OVERALL MUSCULOSKELETAL PAIN USING FX 405 (VIOLET & RED LASER SCANNER)
- 2022. CHRONIC NECK & SHOULDER PAIN USING GVL (GREEN & VIOLET LASER)
- 2023. NON-INVASIVE DERMATOLOGICAL AESTHETIC TREATMENT FOR THE REDUCTION OF BODY CIRCUMFERENCE. (VIOLET ZERONA® VZ8 OTC)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA FDA INDICATIONS FOR USE

Erchonia FDA 510(k) Indications for Use *Updated-9/27/24*

1. Indication – ***Chronic neck and shoulder pain***
   - January 17th, 2002 • FDA Market Clearance K012580
   - Device – Red single diode • Results Published - Funct Neurol Rehabil Ergon 2016;6(2):97-104

2. Indication -- ***Low Level Laser Assisted Liposuction and reduction of pain associated with surgery***
   - September 30th, 2004 • FDA Market Clearance K041139
   - Device – Red multi-diode • Results Published - The American Journal of Cosmetic Surgery

3. Indication – ***Erchonia EVRL –***
   a. while using the red diode, for adjunctive use in providing temporary relief of minor chronic neck and shoulder pain of musculoskeletal origin,
   b. and while using the violet diode, to treat dermatological conditions, and specifically indicated to treat moderate inflammatory Acne Vulgaris
   - May 2nd, 2005 • FDA Market Clearance K050672
   - Device – Red/Violet multi-diode • Results Will Not be Published

4. Indication – ***Breast Augmentation and Pain Associated with Surgery***
   - April 24th, 2008 • FDA Market Clearance K072206
   - Device – Multi-diode red • Results Published - Breast Augmentation American Journal of Cosmetic Surgery

5. Indication – ***Non-Invasive Body Contouring and Fat Reduction***
   - August 28th, 2010 • FDA Market Clearance K082609
   - Device – Erchonia MLS Scanner (Zerona) • Results Published - Lasers in Surgery and Medicine 41:799–809 (2009)

6. Indication – ***Arm Circumference Reduction of the Upper Arms***
   - May 14th, 2012 • FDA Market Clearance K121690
   - Device – Erchonia MLS Scanner (Zerona) • Results Published - Seminars in Cutaneous Medicine and Surgery

7. Indication – ***Reduction in the Appearance of Cellulite***
   - May 17th, 2013 • FDA Market Clearance K130922
   - Device – Erchonia Verju Laser System with Massager • Results Published - Lasers in Surgery and Medicine

8. Indication – ***Non-invasive Body Contouring of the Waist, Hips and Thighs***
   - May 17th, 2013 • FDA Market Clearance K130922
   - Device – Erchonia Verju Laser System with Massager • Results Published - American Journal of Cosmetic Surgery

9. Indication – ***Adjunct to Chronic Heel Pain Arising from Plantar Fasciitis***
   - April 14th, 2014 • FDA Market Clearance - K132940
   - Device – Erchonia ALLAY (FX 635 Laser) • Results Published - American Orthopaedic Foot & Ankle Society

10. Indication – ***Non-Invasive Body Contouring of the Waist, Hips and Upper Abdomen for BMI 30-40***
    - October 21st, 2014 • FDA Market Clearance K142042
    - Device – Erchonia SHL (10 Head) • Results Published - Photomedicine and Laser Surgery

11. Indication – ***Zerona-Z6 OTC - Non-Invasive Dermatological Aesthetic Treatment for the reduction of the circumference of the hips, waist and thighs***
    - January 15th, 2015 • FDA Market Clearance K143007
    - Device – Zerona-Z6 • Results Will Not be Published

12. Indication – ***Zerona-Z6 (6) Week Protocol - Non-Invasive Dermatological Aesthetic Treatment for the Reduction of Circumference of Hips, Waist, Thighs and Upper Abdomen*** (1 Tx per Week for 6 Weeks)
    - May 21st, 2015 • FDA Market Clearance K150446
    - Device – Zerona-Z6 Dermatology • Results Published – The Journal of Clinical & Aesthetic

13. Indication - ***The LunulaLaser device is indicated for use for the temporary increase of clear nail in patients with onychomycosis (e.g., dermatophytes Trichophyton rubrum and T. mentagrophytes, and/or yeasts Candida albicans, etc.)***
    - June 3rd, 2016 • FDA Market Clearance K153164

Erchonia FDA 510(k) Indications for Use *Updated-9/27/24*

15. Indication – ***The FX 635 laser is indicated for the following two indications:***
    a. as an adjunct to provide relief of minor chronic low back pain of musculoskeletal origin.
    b. as an adjunct to reducing chronic heel pain arising from plantar fasciitis.
    - May 21st, 2018 • FDA Market Clearance K180197
    - Device-Erchonia FX 635 • Results Published eMedical Research & Journal of Pain

16. Indication – ***The FX-635 laser is indicated for the adjunctive use in providing temporary relief of nociceptive musculoskeleta***
    - June 1st, 2019 • FDA Market Clearance K190572
    - Device-Erchonia FX 635 • Results Published Orthopedics and Rheumatology Journ

17. Indication – ***Erchonia EVRL –***
    a. while using the red and violet diode simultaneously, for adjunctive use in providing temporary relief of minor chro and shoulder pain of musculoskeletal origin,
    b. and while using the violet diode, to treat dermatological conditions, and specifically indicated to treat moderate inf Acne Vulgaris
    - August 8th, 2019 • FDA Market Clearance K191257
    - Device – Red/Violet multi-diode • Results Published Medical Devices: Evidence and Rese

18. Indication – ***Emerald Laser is indicated for use as a non-invasive dermatological aesthetic treatment for the reduction of boc circumference in individuals with a Body Mass Index (BMI) up to 40 kg/m2***
    - September 13th, 2019 • FDA Market Clearance K192544
    - Device-Erchonia Emerald Laser (SHL) • Results Will Not be Published

19. Indication – ***Erchonia Red Laser is indicated as an adjunctive treatment of postoperative pain***
    - October 22, 2021 • FDA Market Clearance K211186
    - Device – Red multi-diode • Results Submitted to be Published

20. Indication – ***Erchonia FX-405 Laser is indicated for relief of nociceptive musculoskeletal pain.***
    - November 12th, 2021 • FDA Market Clearance K212595
    - Device – Red/Violet multi-diode • Results Will Not be Published

21. Indication - The Erchonia GVL Laser -
    a. while using the green and violet diode simultaneously, for adjunctive use in providing temporary relief of minor ch neck and shoulder pain of musculoskeletal origin,
    b. and while using the violet diode, to treat dermatological conditions, and specifically indicated to treat moderate inflammatory Acne Vulgaris.
    - September 1st, 2022 • FDA Market Clearance K221987
    - Device – Green/Violet multi-diode • Results Will Be Published

22. Indication - Erchonia Violet ZERONA® Z6 OTC is indicated for use as a non-invasive dermatological aesthetic treatment for t of body circumference.
    - July 21st, 2023 • FDA Market Clearance K231474
    - Device – Violet multi-diode • Results Will Be Published

ERCHONIA® *Healing Light, Perfected.*

RESTORING BRAIN —HEALTH—

LASERS A - Z MANUAL
JEROME RERUCHA

BASE MUSCULOSKELETAL/PAIN DEFAULT UPREGULATION

9-16-42-53

LIGAMENT
5-9-125-2720

CARTILAGE
20-690-787-2720

BONE
20-45-30594-8687

BURSITIS
9-16-1442-6568

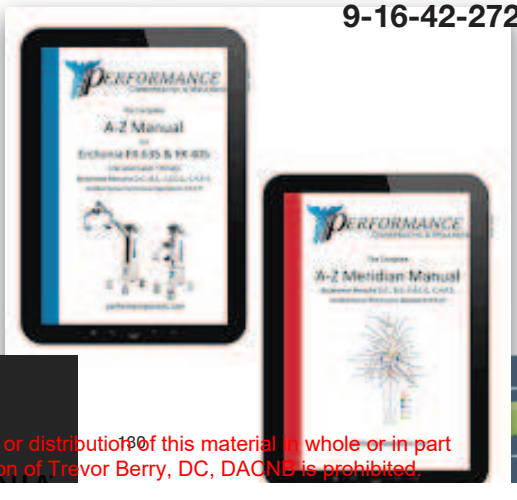
EDEMA
21-33-43-5000

TENDONITIS
1-21-250-2720

NEUROGENIC INFLAMMATION/FASCIITIS
9-16-33-36

WOUNDS
3-16-24-111

INFLAMMATION
9-16-42-2720



ERCHONIA® *Healing Light, Perfected.*

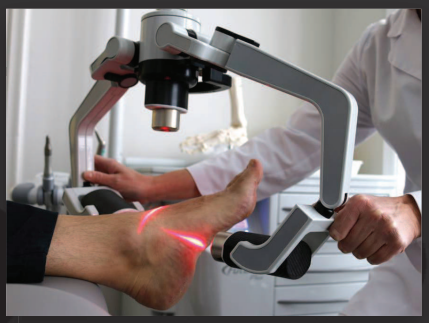
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FDA STUDY 6 VISITS. SET IT AND FORGET IT
SWIM UP STREAM (USE FX OR LINE BEAM TO TARGET
FOOT POPLITEAL/BIFURCATION AND
LOW BACK NERVE ROOT/SPINAL CORD ALL
AT ONCE. 9-16-42-53 (ADD 2HZ TO FX MODELS)

ERCHONIA®
Healing Light, Perfected.



FX LE PROTOCOL BASIC

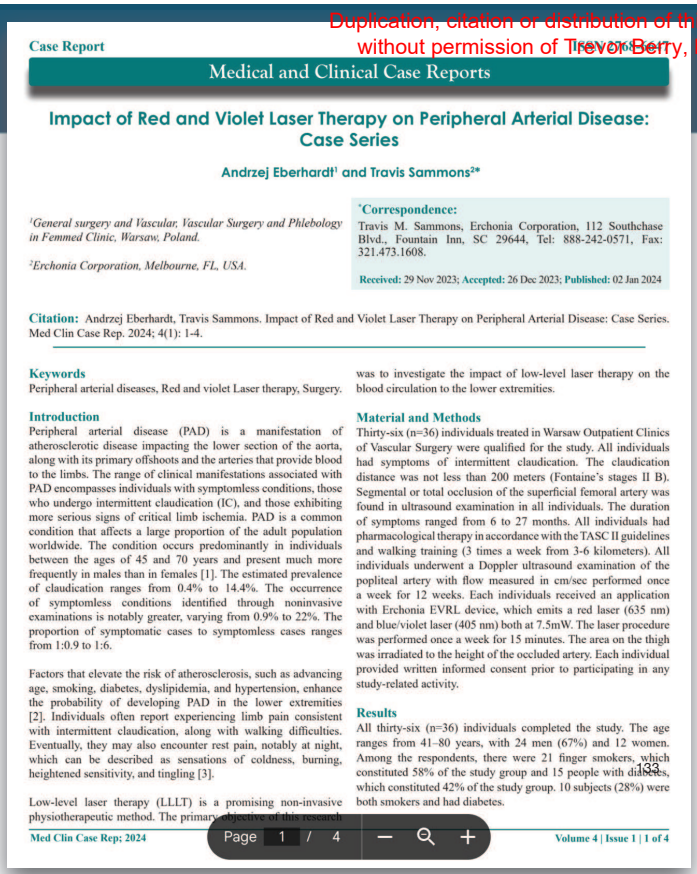
CONSIDER 10 MINUTES EACH LEG/NERVE DISTRIBUTION
L/S PLEXUS, POPLITEAL BIFURCATION AND COMMON PERONEAL
TARSAL TUNNEL/PLANTAR REGION

ERCHONIA®
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



Case Report

Medical and Clinical Case Reports

Impact of Red and Violet Laser Therapy on Peripheral Arterial Disease: Case Series

Andrzej Eberhardt[1] and Travis Sammons[2]*

[1]General surgery and Vascular, Vascular Surgery and Phlebology in Femmed Clinic, Warsaw, Poland.

[2]Erchonia Corporation, Melbourne, FL, USA.

*Correspondence:
Travis M. Sammons, Erchonia Corporation, 112 Southchase Blvd., Fountain Inn, SC 29644, Tel: 888-242-0571, Fax: 321.473.1608.

Received: 29 Nov 2023; Accepted: 26 Dec 2023; Published: 02 Jan 2024

Citation: Andrzej Eberhardt, Travis Sammons. Impact of Red and Violet Laser Therapy on Peripheral Arterial Disease: Case Series. Med Clin Case Rep. 2024; 4(1): 1-4.

Keywords
Peripheral arterial diseases, Red and violet Laser therapy, Surgery.

Introduction

Peripheral arterial disease (PAD) is a manifestation of atherosclerotic disease impacting the lower section of the aorta, along with its primary offshoots and the arteries that provide blood to the limbs. The range of clinical manifestations associated with PAD encompasses individuals with symptomless conditions, those who undergo intermittent claudication (IC), and those exhibiting more serious signs of critical limb ischemia. PAD is a common condition that affects a large proportion of the adult population worldwide. The condition occurs predominantly in individuals between the ages of 45 and 70 years and present much more frequently in males than in females [1]. The estimated prevalence of claudication ranges from 0.4% to 14.4%. The occurrence of symptomless conditions identified through noninvasive examinations is notably greater, varying from 0.9% to 22%. The proportion of symptomatic cases to symptomless cases ranges from 1:0.9 to 1:6.

Factors that elevate the risk of atherosclerosis, such as advancing age, smoking, diabetes, dyslipidemia, and hypertension, enhance the probability of developing PAD in the lower extremities [2]. Individuals often report experiencing limb pain consistent with intermittent claudication, along with walking difficulties. Eventually, they may also encounter rest pain, notably at night, which can be described as sensations of coldness, burning, heightened sensitivity, and tingling [3].

Low-level laser therapy (LLLT) is a promising non-invasive physiotherapeutic method. The primary objective of this research was to investigate the impact of low-level laser therapy on the blood circulation to the lower extremities.

Material and Methods

Thirty-six (n=36) individuals treated in Warsaw Outpatient Clinics of Vascular Surgery were qualified for the study. All individuals had symptoms of intermittent claudication. The claudication distance was not less than 200 meters (Fontaine's stages II B). Segmental or total occlusion of the superficial femoral artery was found in ultrasound examination in all individuals. The duration of symptoms ranged from 6 to 27 months. All individuals had pharmacological therapy in accordance with the TASC II guidelines and walking training (3 times a week from 3-6 kilometers). All individuals underwent a Doppler ultrasound examination of the popliteal artery with flow measured in cm/sec performed once a week for 12 weeks. Each individuals received an application with Erchonia EVRL device, which emits a red laser (635 nm) and blue/violet laser (405 nm) both at 7.5mW. The laser procedure was performed once a week for 15 minutes. The area on the thigh was irradiated to the height of the occluded artery. Each individual provided written informed consent prior to participating in any study-related activity.

Results

All thirty-six (n=36) individuals completed the study. The age ranges from 41–80 years, with 24 men (67%) and 12 women. Among the respondents, there were 21 finger smokers, which constituted 58% of the study group and 15 people with diabetes, which constituted 42% of the study group. 10 subjects (28%) were both smokers and had diabetes.

ERCHONIA RESEARCH 2025

36 PATIENTS WITH PERIPHERAL ARTERY DISEASE FROM ATHEROSCLEROSIS. PRIMARY OBJECTIVE MEASUREMENT WAS DOPPLER FLOW RATE TO THE POPLITEAL ARTERY (MEASURED IN CM/SEC). THIS STUDY INCLUDED DIABETICS, SMOKERS AND MORE WITH NO CHANGE IN THEIR MEDICATION OR EXERCISE SCHEDULES. ALL PARTICIPANTS HAD FULL OR PARTIAL OCCLUSION OF THE SUPERFICIAL FEMORAL ARTERY BASED ON ULTRASOUND AND ALL HAD CLAUDICATION TO THE LOWER EXTREMITIES AT <200 METERS.

DEVICE AND TREATMENT:
ERCHONIA RED/VIOLET
15 MINUTES OVER THE THIGH TO THE HEIGHT OF THE OCCLUDED ARTERY
1X/WEEK FOR 12 WEEKS

MEAN DOPPLER FLOW RATE IMPROVED FROM:
32.86 CM/SEC
TO
79.31 CM/SEC (WHICH IS CONSIDERED WNL!)
OVER DOUBLE FLOW IMPROVEMENT (46.44 CM/SEC)
USING ONLY ERCHONIA RED/VIOLET LASER.
NO OTHER LIFESTYLE OR MEDICATION CHANGES!

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PAIN MANAGEMENT

MOST OF YOUR CASES WILL RESPOND TO A "SET IT AND FORGET IT" APPROACH
PUT YOUR LASER OVER THE TARGET TISSUE (KNEE, LOW BACK ETC)
WITH A BASE SETTING LIKE 9-16-42-53
5 MINUTE MINIMUM UP TO 20 MINUTE MAX

HOWEVER, BECAUSE PAIN CENTRALIZES TO THE BRAIN AND WITH ALL OF THE KNOWN BENEFITS OF LASERING THE BRAIN, WE ALWAYS UTILIZE A "DOWNSTREAM" APPROACH FOR PAIN MANAGEMENT…

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN AND PAIN

Understanding that chronic back pain originates from within the brain could lead to quicker recovery, a new study finds

Published: November 3, 2023 8.43am EDT

When a person has been in pain for months, years or even decades, pain processing pathways are more likely to fire, and brain regions that typically are not involved in pain start to be involved. Chronic pain also leads to increased levels of activity in glial cells, which are part of the brain's immune system. All these changes in the brain then serve to "entrench" the pain, making it persist.

One of the main goals of pain reprocessing therapy is to help people think differently about the causes of their pain. After we treated participants with **pain reprocessing therapy,** about half the causes of pain that people described were related to the mind or brain. They said things like "anxiety," "fear" or "neural pathways" were the causes of their pain.

The more that people shifted to this kind of understanding, the more their back pain went down. **We think this shift in understanding reduces fear and avoidance of pain, which can tamp down pain pathways in the brain and promote healthy, pain-reducing behaviors like exercise and socializing.**

Reattribution to Mind-Brain Processes and Recovery From Chronic Back Pain

Yoni K. Ashar, PhD[1]; Mark A. Lumley, PhD[2]; Roy H. Perlis, MD, MSc[3]; et al
Conor Liston, MD, PhD[4]; Faith M. Gunning, PhD[4]; Tor D. Wager, PhD[5]
Author Affiliations Article Information

JAMA Netw Open. 2023;6(9):e2333846. doi:10.1001/jamanetworkopen.2023.33846

RESTORING BRAIN —HEALTH—

CURRENT BACK PAIN TREATMENT MODEL

84% More Successful – Scientists Reveal the Most Effective Treatment for Back Pain

"The higher effort required for individual treatment is worthwhile because patients benefit to an extent that is clinically important," says lead author Dr. Johannes Fleckenstein from the Institute of Sport Sciences at Goethe University Frankfurt.

They stop being afraid to move or are taught tactics for coping with pain. This makes them realize that they are by no means helpless. But what does psychotherapeutic support through CBT actually contribute to the success of the treatment? Analysis of the data revealed the following: **When an individualized approach and CBT were combined, the success rate in terms of pain relief was an impressive 84 percent higher than with standard treatment. The combined therapy, also called multimodal therapy, thus led to the best result by far.**

Reference: "Individualized Exercise in Chronic Non-Specific Low Back Pain: A Systematic Review with Meta-Analysis on the Effects of Exercise Alone or in Combination with Psychological Interventions on Pain and Disability" by Johannes Fleckenstein, Philipp Floessel, Tilman Engel, Laura Krempel, Josefine Stoll, Martin Behrens and Daniel Niederer, **29 July 2022, *Journal of Pain*.**
DOI: 10.1016/j.jpain.2022.07.005

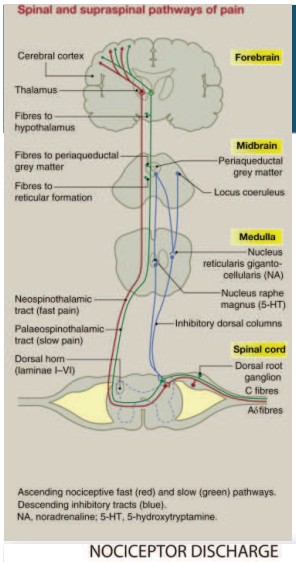
ERCHONIA Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

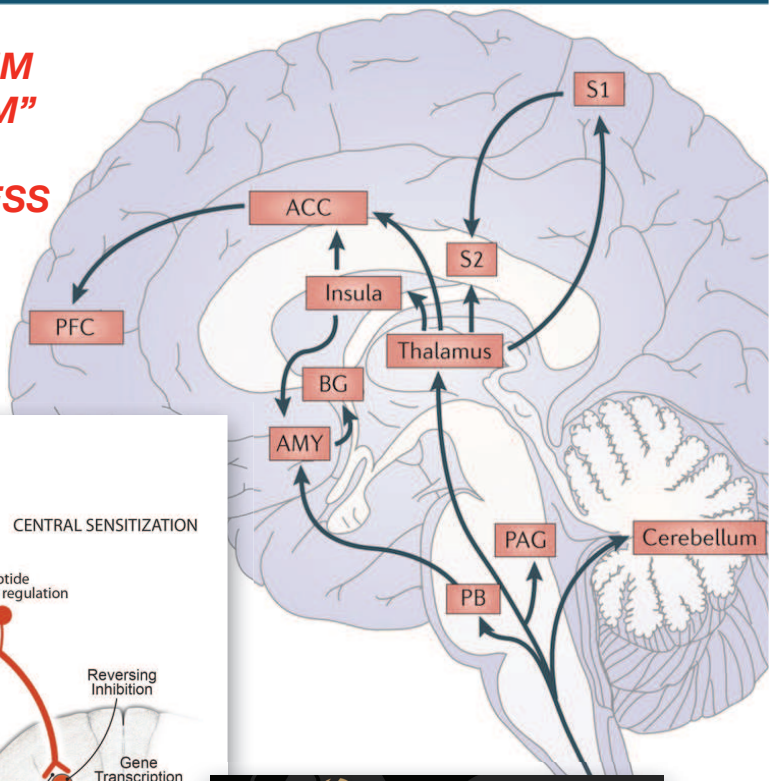
RESTORING BRAIN —HEALTH—

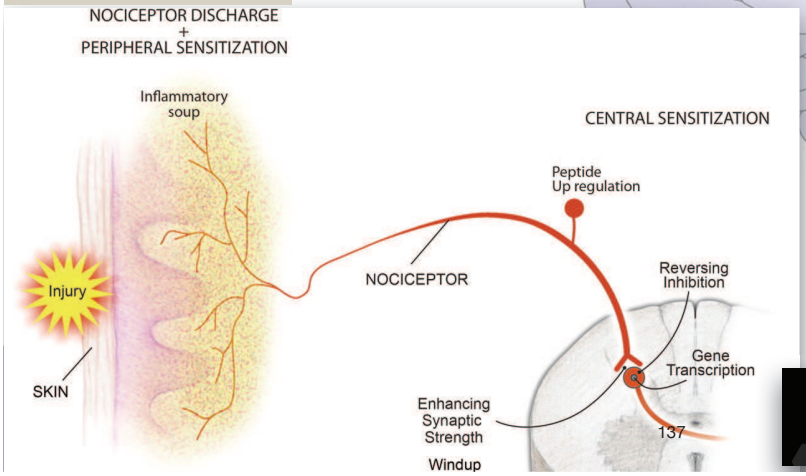
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THE NEUROLOGY OF PAIN



WHY WE "SWIM DOWNSTREAM" WITH OUR LASER. ADDRESS THE CENTRAL NERVOUS SYSTEM!





ERCHONIA
Healing Light, Perfected.

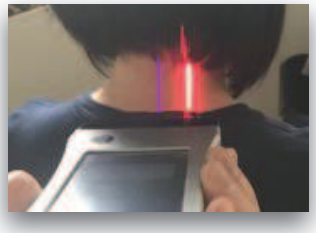
THE "DOWNSTREAM" PROTOCOL

(FOR EVEN BETTER RESULTS, PERFORM AFTER BRAIN BALANCE)

SUMMARY OVERVIEW
2 MINUTES EACH REGION

2. PARIETAL LOBE W/ VIBRATION

5. SPINAL CORD/ NERVE ROOT



6. INVOLVED AREA/ DISTAL TISSUE

3. IPSI CEREBELLUM STACK WITH MOVEMENT

4. VAGAL NERVE STIMULATION IPSILATERAL TO INVOLVED SIDE

USE 10-40-10-40

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA'S FDA CLEARANCE FOR POST-SURGICAL PAIN

FDA Grants 510(k) Market Clearance for Whole Body Postoperative Pain to World Leader in Low Level Laser Technology Randomized and controlled peer reviewed published clinical trials on 635 nm red laser prove effectiveness for treatment As the World Leader in Low Level Laser Technology, we have always believed we need to continue expanding the science of this amazing technology. This latest U.S. FDA 510(k) statement adds to our commitment. MELBOURNE, Fla. (PRWEB) November 01, 2021 Erchonia, the World Leader in Low Level laser Technology announces that on October 22, 2021, they have received their latest 510(k) statement from the U.S. FDA based on level 1 blinded and controlled clinical trials. 510(k) #211186 The new FDA 510(k) statement for whole body treatment of postoperative pain was based on the company's previously granted 510(k) statements from 2004 and 2008 along with data from seven randomized clinical trials on coronary artery bypass graft surgery, coronary bypass surgery with internal mammary artery grafts, tibial fracture surgery, cesarean section, endodontic surgery and tonsillectomy. This groundbreaking FDA market clearance provides a drug free & safe treatment option for postoperative pain. Based on clinical data, subjects who received Erchonia laser treatments

AVERAGE PAIN REDUCTION POST-OP >50%
REDUCTION IN NARCOTICS USE
RED LASER 635 NM 7.5 MW DIODES
5 MINUTES PRE-SURGICAL
5 MINUTES POST-SURGICAL

ERCHONIA
Healing Light, Perfected.

139

RESTORING BRAIN —HEALTH—

LASER NEUROLOGY FOR HEADACHES

ERCHONIA
Healing Light, Perfected.

140

THE GVL ADVANCED PROTOCOL

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.
© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Most common neurologic symptom domains:

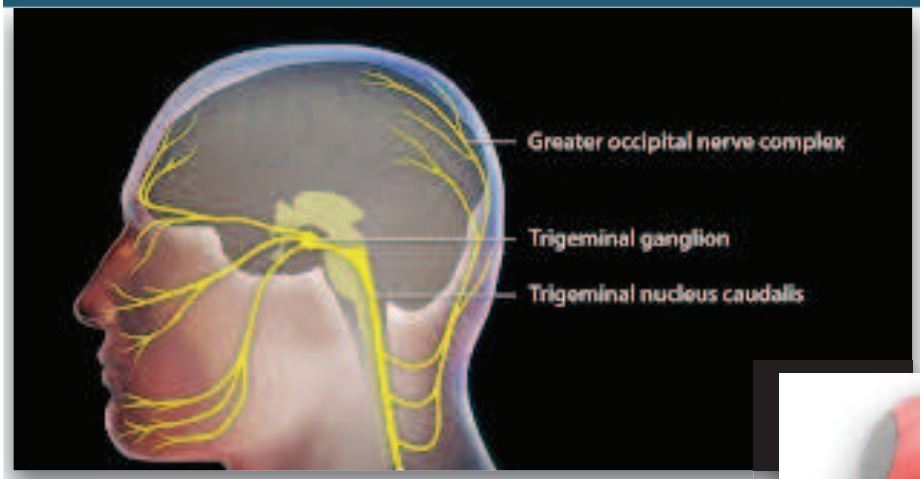
1. **Headache**
2. Dizziness/vertigo/lightheadedness
3. Numbness/tingling/burning pain
4. Weakness (focal or generalized)
5. Spells (seizures, blackouts, transient events)
6. Tremor/abnormal movements
7. Visual disturbance
8. Cognitive/behavioral change
9. Gait imbalance/falls
10. Sleep disturbance/fatigue with other neuro symptoms

ERCHONIA
Healing Light, Perfected.

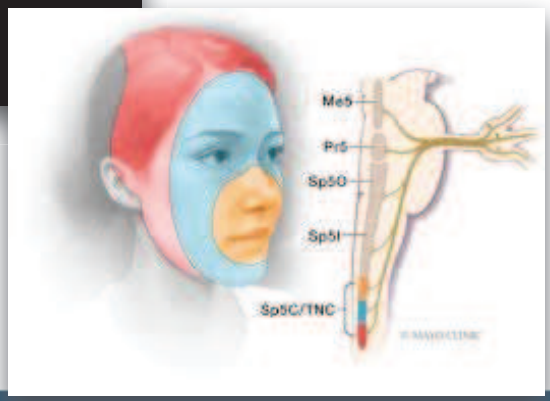
Semin Neurol 2021; 41(06): 632
DOI: 10.1055/s-0041-1735894
Preface
Outpatient Issues in Neurology

RESTORING BRAIN
—HEALTH—

THE TRIGEMINAL COMPLEX: OVERVIEW



OF THE KEY INFLAMMATORY MEDIATORS, CGRP (CALCITONIN GENE RELATED PEPTIDE) IS ONE OF THE MOST IMPORTANT TO MITIGATE ITS EFFECTS ON CN 5



ALL MIGRAINES AND HEADACHES IN GENERAL, HAVE A TRIGEMINAL NERVE INVOLVEMENT

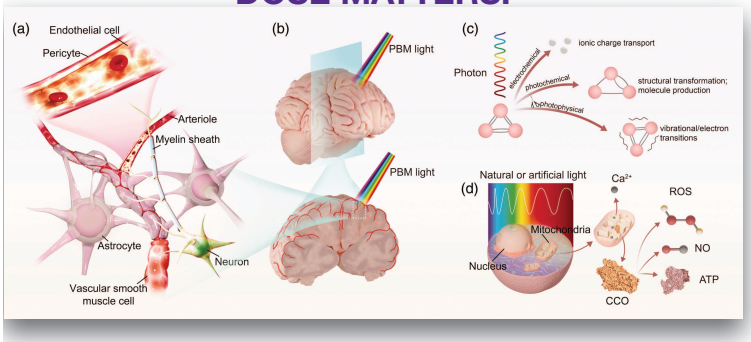
ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
—HEALTH—
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

First published online January 7, 2025
Request permissions
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Unlocking the potential of photobiomodulation therapy for brain neurovascular coupling: The biological effects and medical applications

Bingzi Yan, Jie Zhou, [...],

AN EXCELLENT REVIEW PAPER (FREE) WITH MULTIPLE EXAMPLES OF WHY DOSE MATTERS!



A mechanistic review of PBM indicates that CGRP release from perivascular nerve fibres (via TRPV1 activation) is part of pain/inflammatory pathways, and that PBM may modify neurovascular / inflammatory signalling.

ROS have a dual-edged nature, with their effects on cellular physiology depending on concentration, rendering the selection of irradiation parameters critically important. **Research has indeed established a link between ROS generation and laser fluence** Figure 4(G) [46] **In a separate study, where the optimal radiant exposure for PBM was found to be 3 J/cm2, the alterations in various redox effect indicators—including ROS, NF-κB, tumor necrosis factor (TNF)-α, IL-1β, intracellular SOD, and TAC—were found to be dose-dependent.[47] Higher doses, such as 120 J/cm2, can lead to excessive ROS generation and activate apoptotic pathways,[48] indicating the biphasic-dose responses of different PBM parameters, which require meticulous management to avoid adverse effects.**

ERCHONIA Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

Arq Neuropsiquiatr
2018 Oct;76(10):663-667. doi: 10.1590/0004-282X20180109.

Botulinum toxin A (BT-A) versus low-level laser therapy (LLLT) in chronic migraine treatment: a comparison

Luana Mazzacoratti Loeb

Abstract

Objective: The aim of this work was to evaluate patients with chronic migraine treated with botulinum toxin A (BT-A) and compare this with low level laser therapy (LLLT), referencing: pain days, pain intensity, intake of drugs/self-medication, anxiety and sleep disorders.

Methods: Patients were randomized into two groups: BT-A group (n = 18) and LLLT group (n = 18). Each patient kept three pain diaries: one before (baseline) (30 days), one during treatment (30 days) and one after the post-treatment phase (30 days). Repeated ANOVA plus the Bonferroni post-test, Student's t test, and factorial analysis were applied, and $p < 0.05$ was accepted as significant.

Results: **Our data showed that both treatments were able to reduce headache days, acute medication intake and decrease the intensity of pain. Anxiety was reduced in the BT-A group, while sleep disturbance was reduced in the LLLT group.**

Conclusion: Our data showed that both treatments can be used to treat chronic migraine, without notable differences between them.

ERCHONIA Healing Light, Perfected.
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS AI SEARCH

Effects of low level laser therapy on the trigeminal nerve

20 sources

Low-level laser therapy (LLLT) significantly reduces pain and improves nerve function in trigeminal neuralgia, but optimal protocols remain unstandardized.

Mechanisms and Biological Effects

LLLT, also known as photobiomodulation, uses low-intensity light to stimulate biological processes without heating tissue. In the trigeminal nerve, LLLT increases mitochondrial ATP production, promotes neurogenesis, enhances myelin synthesis, and provides neuroprotection. These effects help repair or prevent further nerve damage, reduce the release of pain mediators, and increase pain thresholds by stimulating endorphin synthesis

Clinical Efficacy and Outcomes

Multiple randomized controlled trials and systematic reviews show that LLLT leads to significant reductions in pain intensity and frequency in patients with trigeminal neuralgia, often outperforming placebo and sometimes matching or exceeding conventional therapies.

Comparative Effectiveness Table

Outcome	LLLT vs. Placebo	LLLT vs. Medication	LLLT + Medication	Citatio
Pain reduction	Superior 145	Comparable or better	Additive benefit	1 19
Nerve function improvement	Yes	Not directly compared	Yes	2
Side effects	Minimal/none	Fewer	Fewer	1

FIGURE 1 LLLT consistently reduces pain and improves nerve function in trigeminal neuralgia.

ERCHONIA Healing Light, Perfected.

CONSENSUS AI SEARCH

Effects of low level laser therapy on headaches

16 sources

Low-level laser therapy (LLLT) shows potential for reducing headache frequency and intensity

Does low-level laser therapy reduce headache frequency or severity? N = 9

Key Clinical Outcomes from Recent Studies

Headache Type	Main Outcome	LLLT Effectiveness	Citations
Chronic migraine	↓ Migraine days, ↓ meds	Significant improvement	6 8 14
Tension-type	↓ Pain, ↑ QoL	Significant improvement	4 10
Sinus-related	↓ Headache, ↓ fatigue	Significant improvement	1
Neuropathic/orofacial	↓ Pain intensity	Effective, but more studies needed	16

FIGURE 2 Table summarizing LLLT effects by headache type.

ERCHONIA Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GREEN LIGHT AND IMMUNITY

Abstract

The present study examined the effects of different light spectra (red and green light-emitting diodes [LEDs]) and light intensities (0.3 and 0.5 W m_{-2}) on antioxidant systems (superoxide dismutase, H_2O_2 and lipid peroxidation) and immunity (lysozyme, melatonin and endorphin) in gold-striped amberjack, *Seriola lalandi.* **In groups exposed to green LED, the levels of antioxidant enzyme were significantly lower than in the control, whereas the levels of immune-related parameters were significantly higher compared to other groups.** The levels of antioxidant enzyme in individuals exposed to red light increased with increasing light intensity, whereas these levels were not correlated to the intensity of green light and were increased even at low light intensity. **These results indicate that exposure to low-intensity green light accelerates the antioxidant system and immune system.**

The effect of green and red light spectra and their intensity on the oxidative stress and non-specific immune responses in gold-striped amberjack, *Seriola lalandi*

Young Jae Choi,Ji Yong Choi,Sang-Geun Yang,Bong-Seok Kim &**Cheol Young Choi**

Pages 223-234 | Received 06 Jan 2016, Accepted 10 Mar 2016, Published online: 07 Apr 2016

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

GREEN LIGHT AND FIBROMYALGIA

In another study by Dr. Ibrahim, published in *Pain Medicine* in 2020, 21 adults with fibromyalgia were exposed to white light for one to two hours daily for 10 weeks, followed by green light therapy. During the green light exposure, patients reported a reduction in pain intensity.

Green Light Exposure Improves Pain and Quality of Life in Fibromyalgia Patients: A Preliminary One-Way Crossover Clinical Trial

Laurent Martin, PhD, Frank Porreca, PhD, Elizabeth I Mata, BS, Michelle Salloum, MD, Vasudha Goel, MD, Pooja Gunnala, BS, Wiliam D S Killgore, PhD, Sejal Jain, MD, Felesia N Jones-MacFarland, MD, Rajesh Khanna, PhD ... Show more

Pain Medicine, Volume 22, Issue 1, January 2021, Pages 118–130,

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Long-lasting antinociceptive effects of green light in acute and chronic pain in rats

Ibrahim, Mohab M.[a,b,*]; Patwardhan, Amol[a,b]; Gilbraith, Kerry B.[a]; Moutal, Aubin[b]; Yang, Xiaofang[b]; Chew, Lindsey A.[a]; Largent-Milnes, Tally[b]; Malan, T. Philip[a,b]; Vanderah, Todd W.[a,b]; Porreca, Frank[a,b]; Khanna, Rajesh[b]

Author Information

PAIN: February 2017 - Volume 158 - Issue 2 - p 347-360

Abstract

Treatments for chronic pain are inadequate, and new options are needed. Nonpharmaceutical approaches are especially attractive with many potential advantages including safety. Light therapy has been suggested to be beneficial in certain medical conditions such as depression, but this approach remains to be explored for modulation of pain. We investigated the effects of light-emitting diodes (LEDs), in the visible spectrum, on acute sensory thresholds in naive rats as well as in experimental neuropathic pain. Rats receiving green LED light (wavelength 525 nm, 8 h/d) showed significantly increased paw withdrawal latency to a noxious thermal stimulus; this antinociceptive effect persisted for 4 days after termination of last exposure without development of tolerance. No apparent side effects were noted and motor performance was not impaired. Despite LED exposure, opaque contact lenses prevented antinociception. Rats fitted with green contact lenses exposed to room light exhibited antinociception arguing for a role of the visual system. Antinociception was not due to stress/anxiety but likely due to increased enkephalins expression in the spinal cord. Naloxone reversed the antinociception, suggesting involvement of central opioid circuits. Rostral ventromedial medulla inactivation prevented expression of light-induced antinociception suggesting engagement of descending inhibition. Green LED exposure also reversed thermal and mechanical hyperalgesia in rats with spinal nerve ligation. Pharmacological a nd proteomic profiling of dorsal root ganglion neurons from green LED-exposed rats identified changes in calcium channel activity, including a decrease in the N-type (CaV2.2) channel, a primary analgesic target.

Thus, green therapy may represent a novel, nonpharmacological approach for managing pain.

RESTORING BRAIN —HEALTH—

Research by Mary Heinricher suggests that light has the potential to engage pain-modulating systems such that normally unharmful inputs are perceived as painful. Her experiments documented substantial light intolerance in patients with fibromyalgia, and raised the possibility that this abnormal photosensitivity could be explained by abnormal engagement of pain-facilitating systems by light.

and suggest that light has the potential to engage pain-modulating systems such that normally innocuous inputs are perceived as aversive or even painful.

Pain. Author manuscript; available in PMC 2017 Apr 1.

Published in final edited form as:
Pain. 2016 Apr; 157(4): 868–878.
doi: 10.1097/j.pain.0000000000000450

PMCID: PMC4794405
NIHMSID: NIHMS743647
PMID: 26785323

A possible neural mechanism for photosensitivity in chronic pain

Melissa E. Martenson,a

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GREEN LIGHT AND PAIN

Green light analgesia in mice is mediated by visual activation of enkephalinergic neurons in the ventrolateral geniculate nucleus

YU-LONG TANG HTTPS://ORCID.ORG/0000-0001-8047-6751, AI-LIN LIU HTTPS://ORCID.ORG/0000-0002-0774-0701, SU-SU LV, ZI-RUI ZHOU, HONG CAO HTTPS://ORCID.ORG/0000-0001-5048-3379 , SHI-JUN WENG HTTPS://ORCID.ORG/0000-0001-6550-892X , AND YU-QIU ZHANG HTTPS://ORCID.ORG/0000-0001-6623-9629 Authors Info & Affiliations

SCIENCE TRANSLATIONAL MEDICINE

7 Dec 2022

KEY NEURONAL POOLS ARE REGIONS SUCH AS THE ROSTRAL VENTRAL MEDULLA (LIKE AN ON-OFF SWITCH), THE LOCUS CERULEUS (HELPS FACILITATE NOREPINEPHRINE IN REGIONS LIKE THE HYPOTHALAMUS) AND THE NUCLEUS RAPHE MAGNUS (SEROTONIN OUTPUT CENTERS OF THE BRAINSTEM).

RETINAL EXPOSURE TO GREEN LIGHT AND LOW LEVEL LASER TO THESE REGIONS IN GENERAL CAN FACILITATE THEIR OUTPUT.

CONSIDER OCULAR WISPING!

HIGHTLIGHTS:

-EFFECTS LASTED FOR 4 DAYS

-BY ACTIVATING THE RODS AND CONES WITH GREEN LIGHT THEY ACTIVATED VENTROLATERAL GENICULATE WHICH ACTIVATES THE DORSAL RAPHE NUCLEUS

ERCHONIA® Healing Light, Perfected.

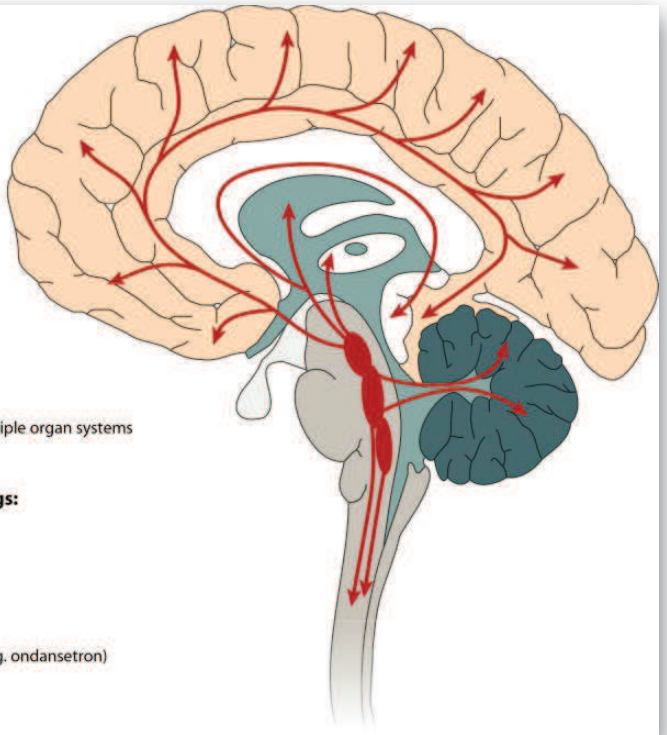
RESTORING BRAIN —HEALTH—

SEROTONIN PRODUCTION IN THE BRAIN

Behavioral effects:
Mood
Perception
Memory
Anger
Aggression
Fear
Stress responses
Appetite
Addiction
Sexuality

Other CNS effects:
Motor control
Cerebellar regulation
Sleep/circadian rhythms
CNS vascular tone
Emesis
Respiratory drive
Body temperature
Descending regulation of multiple organ systems

Central serotonergic drugs:
SSRIs
Tricyclic antidepressants
MAOIs
Other antidepressants
Buspirone
Atypical antipsychotics
Triptans
5-HT_3 receptor antagonists (e.g. ondansetron)
Fenfluramine
Ergotamine/methysergide
Hallucinogens



MANUFACTURED IN THE RAPHE NUCLEI LOCATED IN THE BRAINSTEM

MADE FROM TRYPTOPHAN, AN ESSENTIAL AMINO ACID

YOU ALSO NEED CO-FACTORS AND TRANSPORT SYSTEMS

A HEALTHY GUT AND MICROBIOME ARE ALSO CRITICAL FACTORS (UP TO 90% OF THE BODY'S SEROTONIN IS MADE IN THE GUT

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MOST COMMONLY USED HEADACHE PROTOCOL FREQUENCIES:

DR. HARCOURT
ALL DIODES AT 1-1-1-1

DR. BERRY
ALL DIODES AT 1-10-1-10

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

EMERGING RESEARCH OF FREQUENCY SPECIFIC APPLICATIONS:

Headache Frequency Cheat-Sheet (by modality)

rTMS – Migraine (prevention)

- **Frequency & target:** **10 Hz over left DLPFC**
- **Dose:** 10–20 sessions, ~1,000–2,000 pulses/session (varies by trial).
- **Effect:** ↓ monthly migraine days vs sham in several RCTs/meta-analyses.
- **Key sources:** reviews/meta syntheses of rTMS for migraine prevention; sites effective include LDLPFC and M1. Frontiers+1

rTMS – Chronic Tension-Type Headache (CTTH)

- **Frequency & target:** **1 Hz over right DLPFC.**
- **Dose:** ~10 sessions over 2 weeks in RCTs.
- **Effect:** ↓ pain and EMG activity vs sham (emerging but promising).
- **Key source:** Rajain et al., 2023 RCT; plus prior CTTH pilot. Cureus+1

External Trigeminal Nerve Stimulation (e-TNS, Cefaly) – Migraine

- **Prevention program:** **60 Hz**, 20 min daily; **Acute** program often **100 Hz**.
- **Effect:** PREMICE trial showed ↓ migraine days/drug intake; FDA De Novo and 510(k) docs specify frequencies.
- **Key sources:** PREMICE/replications; FDA summaries K122566, K171446, K160237; 2024 update. FDA Access Data+4Nature+4FDA Access Data+4

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EMERGING RESEARCH OF FREQUENCY SPECIFIC APPLICATIONS:

Headache Frequency Cheat-Sheet (by modality)

tACS – Acute Migraine

- **Frequency & target:** **140 Hz** over visual cortex (occipital montage).
- **Dose:** ~15 min at low current (e.g., 0.4 mA) during attack.
- **Effect:** Pain abatement in a higher proportion of attacks vs sham in a double-blind home-use RCT.
- **Key sources:** Antal et al., 2020 RCT + open summaries/reviews. PMC+2PubMed+2

tACS – Mechanistic (pain networks; not migraine-specific)

- **Frequencies:** **~10 Hz (alpha) can modulate pain perception/attention networks in humans; frequency-specific somatosensory effects reported.**
- **Use:** Consider alpha-tACS (~10 Hz) for experimental modulation; migraine-specific RCTs at 10 Hz are limited.
- **Key sources:** human pain/tACS studies and frequency-specific SI work. MDPI+2ScienceDirect+2

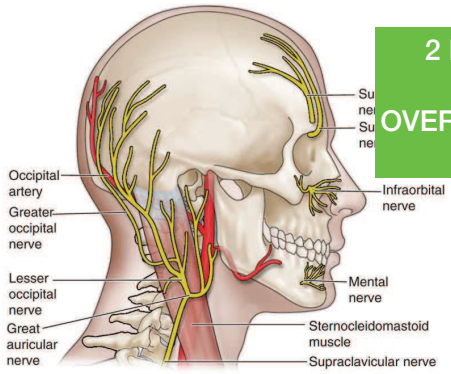
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

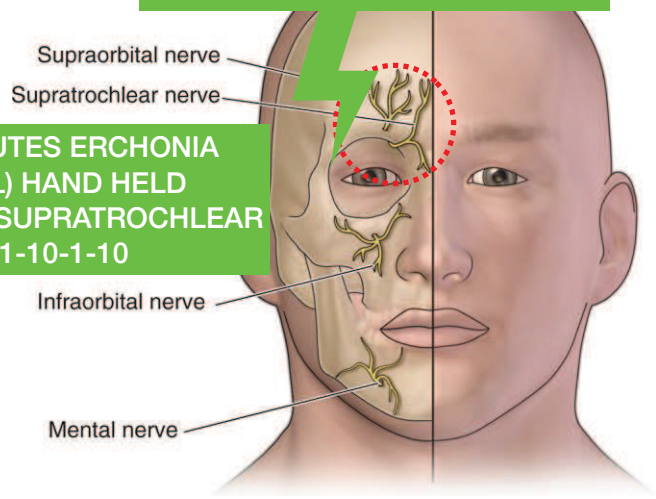
HEADACHES

30 SECONDS OCULAR WISPING
(GREEN DIODE)
1-10-1-10
EYES OPEN VS CLOSED

SUPRAORBITAL NERVE



2 MINUTES ERCHONIA
(GVL) HAND HELD
OVER V1/SUPRATROCHLEAR
1-10-1-10



ERCHONIA
Healing Light, Perfected.

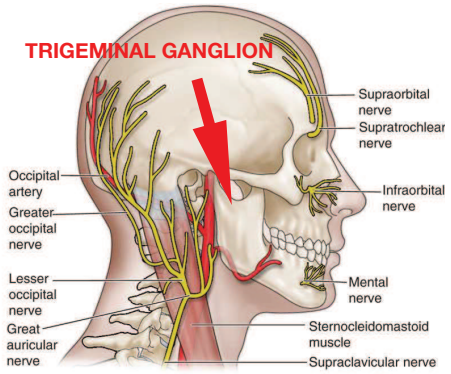
RESTORING BRAIN
—HEALTH—

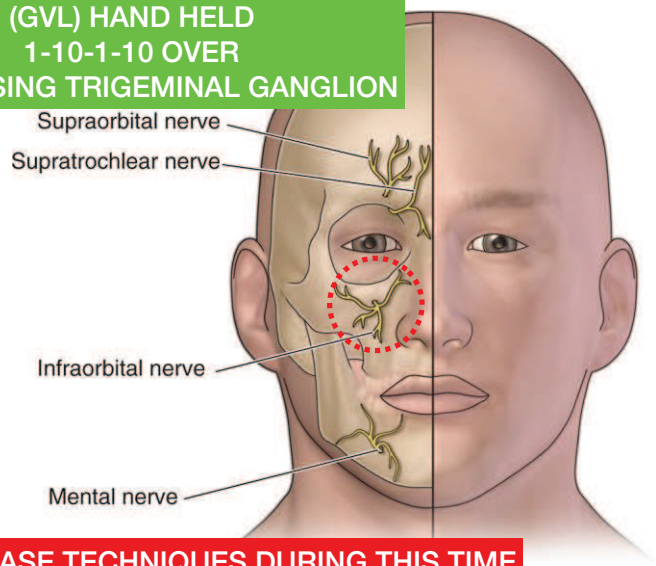
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

INFRATROCHLEAR NERVE AND TRIGEMINAL GANGLIA

2 MINUTES ERCHONIA
(GVL) HAND HELD
1-10-1-10 OVER
V2 CROSSING TRIGEMINAL GANGLION





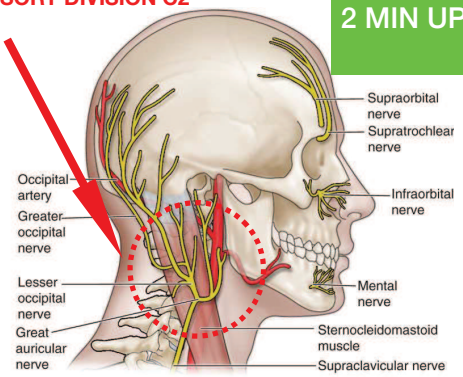
CONSIDER MASTICATION RELEASE TECHNIQUES DURING THIS TIME
(INTERNAL AND EXTERNAL)

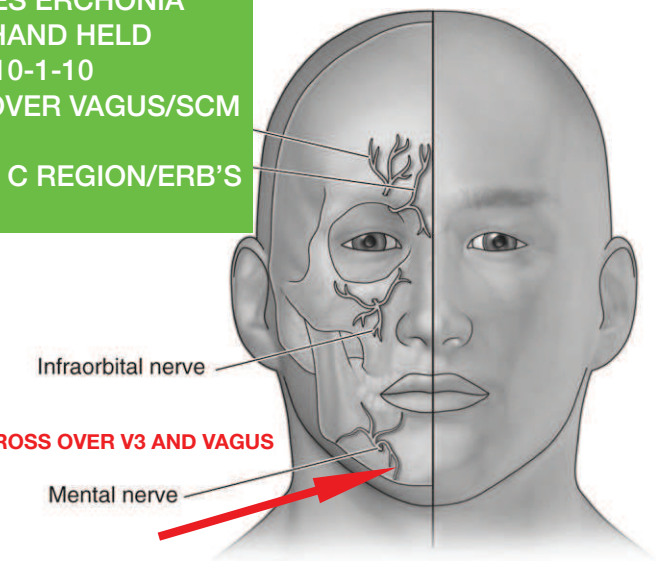
ERB'S POINT

2 MINUTES ERCHONIA
(GVL) HAND HELD
1-10-1-10
V3/MENTAL OVER VAGUS/SCM

2 MIN UPPER C REGION/ERB'S

UPPER CERVICAL COMPLEX
SENSORY DIVISION C2





CLINICAL GEM: ONCE BASE HEADACHE PROTOCOLS AND FOUNDATIONAL FUNC MED DIFFERENTIALS (IE. ANEMIA PATTERNS, MICRONUTRIENT DEPLETION ETC) HAVE BEEN RULED OUT, CONSIDER GUT MICROBIOME, HISTAMINES AND DYSAUTONOMIA FOR MIGRAINES

INSUFFLATION TECHNIQUE

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HEADACHE STACKING

STACKING THE BOX!

1. MSK CORRECTION ONCE BUCKET IS BIG ENOUGH TO TOLERATE (COUPLED CMT, OCULOMOTOR CORRECTION VERGENCE, MASTICATION/CRANIAL FASCIA - TIME PERMITTING DEMONS)
2. VAGAL QUAD WITH INSUFFLATION (DURING MIGRAINES LASER THE UPPER C/SCM, TO PREVENT MIGRAINES LASER THE AURICULAR BRANCH)
3. HEADACHE LLLT PROTOCOL (GVL IF AVAILABLE)
4. FX 405 SCAN TO CN5 DISTRIBUTION
5. TOPICAL APPLICATION (OFFICE FINISH AND HOME) PROLOGEL, CBD WITH MG, GLUTATHIONE
6. SUPPLEMENT SUPPORT (MQ-7 PREVENTION,)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

INSUFFLATION TECHNIQUE

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

INSUFFLATION TECHNIQUE

Prepare

- Ensure the ear(s) to be treated are free of known perforation, infection, middle ear pathology, active otitis media/externa, etc.
- Have the INSUFFLATION device clean (sterilised ear-tip or personal use) and ready.
- Sit in a comfortable, stable chair in a quiet room.

Method

- **Step 1: Squeeze (deflate) the rubber bulb of the device so that it is "deflated".**
- **Step 2: While the bulb is deflated, gently insert the INSUFFLATION ear-tip into the outer ear to create an air-tight seal at or just past the entrance to the ear canal (but not further into the canal).**
  - **Warning: do *not* insert deeply into the ear canal.**
- **Step 3: Maintain the bulb in the deflated state for 20-30 seconds. During this time the user may feel a "mild sensation of increased pressure".**
- **Step 4: After 20-30 s, pinch the bulb (or release whatever stop the seal) and gently remove the device from the ear.**
- **Step 5: Repeat steps 1-4 on the other ear.**
- **Step 6: Repeat the cycle 3-5 times per ear or until the sensation of "pressure or tension in head" has eased. MAX 5 OR IT'S NOT GOING TO WORK. ALSO IF ON TRIPTANS FOR 5HT IT MAY NOT WORK**
- **Note: Device is typically applied to side of headache symptoms but can be used bilaterally or contralateral.**
- **ICE FRONT OF NECK (DR. HARCOURT)**

Frequency / Duration of Use

- Can be used as frequently as necessary.
- For best results perform **3-5 times per day** for **3-4 weeks**.

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

CONSENSUS AI SEARCH

Top 5 Medication Classes Known to Cause Headaches

Several medication classes are well-documented to cause headaches, especially when overused or as a side effect. The most consistently implicated are:

- **Analgesics (including acetaminophen, NSAIDs, and combination painkillers)**
- **Opioids**
- **Triptans**
- **Ergotamine derivatives**
- **Caffeine-containing medications**

These drugs are most commonly associated with medication overuse headache (MOH) and can also cause headaches as a direct adverse effect or during withdrawal.

Key references:
1 Holland et al., Cephalalgia, 2020
2 Da Silva & Lake, Headache, 2014
3 Saper & Da Silva, CNS Drugs, 2013
4 Monteiro et al., Int J Environ Res Public Health, 2021
5 Ferrari, Curr Drug Saf, 2006
6 Saengjaroentham et al., Brain, 2020
9 Thorlund et al., J Headache Pain, 2016
12 Vandenbussche et al., J Headache Pain, 2018
13 Telang et al., J Affect Disord, 2018
15 Pan et al., Psychol Med, 2021
16 Aleksenko et al., 2020
20 Kebede et al., Front Pain Res, 2023

ERCHONIA
Healing Light, Perfected.
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.
RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

MIGRAINE PREVENTION

Supplement Facts
Serving Size: 4 Capsules
Servings Per Container: 60

Amount Per Serving		%DV†
Vitamin C (buffered, from Magnesium Ascorbate)	150 mg	166
Vitamin D3 (Cholecalciferol)	10 mcg	50
Riboflavin	100 mg	7,692
Vitamin B6 (Pyridoxal-5-Phosphate)	12.5 mg	735
Folic Acid (1 mg reported in new regulation format)	1,700 mcg DFE	425
Vitamin B12 (Hydroxycobalamin)	200 mcg	8,333
Pantothenic Acid (from d-Calcium Pantothenate)	35 mg	700
Magnesium (from Dimagnesium Malate and Magnesium Ascorbate)	150 mg	35
Selenium (from Selenium Amino Acid Chelate)	55 mcg	100
Proprietary Equilibrate™ Blend	2,075 mg	*

Contains: L-Tyrosine, L-Methionine, L-Cysteine, 5-HTP (Hydroxytryptophan), Coenzyme Q10, Butterbur Extract, and Feverfew Extract.

† - % Daily Value * - Daily Value Not Established

Other Ingredients: Vegetable Cellulose (from Capsule Shell), Silica, and Coconut Stearate.

https://biogenicnutrition.com/new-doctor-account-application-neurodocb/

FINANCIAL DISCLOSURE: THIS IS AN AFFILIATE ACCOUNT OF DR. BERRY

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

ACUTE MIGRAINE SUPPORT

Proprietary extraction method for our sublingual oils to isolate the active, fat-soluble compounds in ginger and provide rapid absorption and quicker relief of migraines. Can also be swallowed for nausea symptoms. Clinical gem: Ginger is good for aborting migraines but not for preventing them!

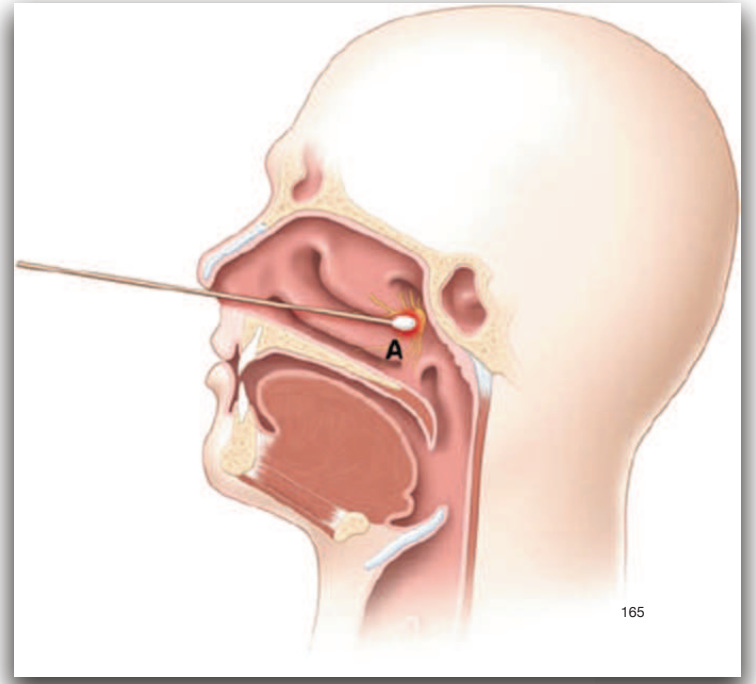
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
with out permission of Trevor Berry, DC. DACNB is prohibited

SPG (SPHENOPALATINE) TOPICAL



COTTON TIP APPLICATION OF DRAGONS BLOOD TO THE SPG. SATURATE THE COTTON TIP AND GENTLY APPLY TO THE BACK OF THE SPHENOID SINUS (THINK COVID SWAB). 4 APPLICATIONS (1 EVERY 3 DAYS INITIALLY) THEN PRN.

CONSIDER FULL MEDICAL SPG BLOCK

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

HEADACHES

MISCELLANEOUS CLINICAL GEMS (MANY COURTESY OF DR. HARCOURT):

- MIGRAINES ARE THE 2ND MOST COMMON CAUSE OF TEMPORARY DISABILITY IN THE USA (LOW BACK PAIN #1)
-USE YOUR GREEN PROTECTIVE ERCHONIA LASER GLASSES IN OFFICE TO HELP WITH MIGRAINES. ALSO CONSIDER 520 NM GREEN LAMPS FOR HOME OR OFFICE
-TO PREVENT MIGRAINES, USE VAGAL STIMULATION ON THE AURICULAR BRANCH, DURING MIGRAINES DO IT ON THE UPPER CERVICAL PORTION OF THE VAGUS (DR. HARCOURT)
-IN COMPLEX MIGRAINE PATIENTS, START WITH LASER/VAGAL PROTOCOLS, THEN PNS STIMULATION, DIET/ SUPPLEMENTS THEN GO INTO FUNCTIONAL NEURO REHAB (ESPECIALLY VERGENCE INSUFFICIENCY - TEST) AND MUSCULOSKELETAL. MANY CHRONIC MIGRAINE PATIENTS CANNOT TOLERATE UPPER CERVICAL CMT UNTIL "THEIR BUCKET GETS BIGGER". EVEN WITH CMT, CONSIDER STARTING LOWER IN SPINE AND RIB ADJUSTING/02 AND THEN LOWER AMPLITUDE TECHNIQUES FOR C/UPPER C LIKE ACTIVATOR
-WHEN USING LASER FOR ALTERATION OF THE DEFAULT MODE NETWORK, SHINE THE BEAMS UP THE NOSE
-IN COMPLEX MIGRAINE PATIENTS (20+/MONTH) AND THERAPIES START DECREASING THE FREQUENCY/SEVERITY, YOU CAN ALMOST COUNT ON THE REMAINING HEADACHES THAT MONTH WILL BE RIGHT BEFORE THE FEMALE CYCLE WHEN ESTROGEN DROPS (FEMQUIL?) AND DURING BAROMETRIC PRESSURE SHIFTS
-ADDRESS HORMONE IMBALANCES USING A FULL FUNCTIONAL HORMONE PANEL SUCH AS THE DUTCH TEST OR VIBRANT HORMONE PANEL!
-ALSO CONSIDER OTHER SYSTEMIC INFLAMMATORY MEDIATORS SUCH AS CHRONIC PATHOGENS (MOLD, VIRUSES ETC) AND FOOD SENSITIVITIES OR ALLERGIES. USE A MINIMUM 4 WEEK STRICT ELIMINATION TRIAL (WHILE DOING VAGAL/GUT REPAIR)
-CONSIDER LOW GLYCEMIC, KETOGENIC DIET
-ALL MIGRAINE PATIENTS MUST ELIMINATE COFFEE/CAFFEINE (THIS IS NON-NEGOTIABLE ACCORDING TO DR. HARCOURT). HAVE THEM START ON THE FRIDAY SO THEY HAVE WEEKEND TO ENDURE INITIAL WITHDRAWAL)
-TO TAKE A DEEPER DIVE, READ PETER GOSBY. ATTEND THE DR. HARCOURT HEADACHE SEMINAR SERIES!

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC. DACNB is prohibited.

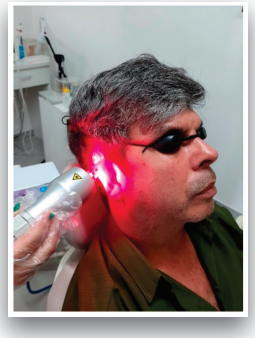
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

TINNITUS

Abstract

Background: **tinnitus is a symptom with no specific cause known to date, and there are no associated pharmacogenomics of hearing disorders and no FDA-approved drugs for tinnitus treatment. The effectiveness of drug treatments is not reproducible on idiopathic patients and inexistent in refractory patients.** Personalized treatments for these patients are a great clinical need. Our study investigated the outcome of potential alternative and complementary treatment modalities for idiopathic and refractory tinnitus patients. Methods: we were the first to evaluate the tinnitus handicap inventory (THI) score changes over the course of treatment up to 15 days after complete cessation of treatment for novel transmeatal low-level laser therapy (LLLT) modalities using light alone, as well as LLLT combined with vacuum therapy (VT), ultrasound (US), Ginkgo biloba (GB) and flunarizine dihydrochloride (FD), while also comparing all treatment outcomes with laser puncture (LP), FD alone and GB alone. Results: a positive treatment outcome (superior to a placebo effect) was achieved by using either LP or transmeatal LLLT, whereas short-term antagonistic effects of VT, US, GB and FD when combined with LLLT. For transmeatal LLLT, an improvement in the treatment outcome was observed by increasing the irradiation time from 6 min to 15 min (with 100-mW of applied laser power at 660 nm). Finally, a lasting therapeutic effect higher than the placebo was observed at 15 days after treatment upon combining LLLT with VT, GB or by using FD alone, by using the transmeatal LLLT alone or by using LP. **Conclusions: LP and Transmeatal LLLT can be promising alternative treatments for idiopathic and refractory tinnitus patients**. Future studies should investigate the long-term effects of LLLT in tinnitus patients, as well as the dosimetry and wavelength of transmeatal LLLT.



The laser power was set at 100 mW for each laser spot. Laser was applied at 660 nm (3 spots; red light) and 808 nm (3 spots; infrared light) wavelengths. Vacuum was applied for 3 min around the ear in the MP7 mode (continuous) and at a pressure of −120 mbar. 8 SESSIONS OVER 4 WEEKS

Effects of Red and Infrared Laser Therapy in Patients with Tinnitus: A Double-Blind, Clinical, Randomized Controlled Study Combining Light with Ultrasound, Drugs and Vacuum Therapy

by **Vitor Hugo Panhóca**

J. Pers. Med. **2023**, *13*(4), 581; https://doi.org/10.3390/jpm13040581

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

TINNITUS
J Speech Lang Hear Res
2021 Jan 14;64(1):279-298. doi: 10.1044/2020_JSLHR-20-00066. Epub 2020 Dec 29.

Effects of Low-Level Laser Therapy as a Therapeutic Strategy for Patients With Tinnitus: A Systematic Review

Maria Carolina Ferreira 1, Izabella Lima de Matos 1, Isabela Porto de Toledo 2, Heitor Marques Honório 3, Maria Fernanda Capoani Garcia Mondelli 4

All the seven selected studies found different degrees of significant results regarding tinnitus severity; however, there was no consensus among the results. Conclusion Even though the LLLT showed positive effects in the tinnitus severity in some studies, it is not possible yet to make any recommendation over its uses for the treatment of tinnitus severity.

CLINICAL CONSIDERATIONS:
LLLT - VAGAL PROTOCOLS (SCM AND AURICULAR/EAM)
TEMPORAL LOBE APPLICATIONS WITH STACKING 2+ MIN EACH MASTER 1-10-40-60
VESTIBULAR CO-ACTIVATION/TONE GENERATOR/TONGUE STIM (SEE NEXT SLIDE)
HEARING AIDS? UPPER C ADJUSTING
TMD THERAPIES/MASTICATION/EAR ADJUSTING/NASAL SPECIFIC (MURPHY)

OTHER CLINICAL CONSIDERATIONS:
OTC PAIN MEDS, NOISE POLLUTION, ANEMIA PATTERNS, METALS, Mg DEFICIENCY, LOW SODIUM DIET, (MICRONUTRIENTS PANEL), SOURCES OF CYOTOKINES TO TECTAL SITES (RUN FOOD SENSITIVITIES. CHRONIC PATHOGENS/MOLD, PAIN), THYROID DYSFUNCTION

TINNITUS

- Open access
- **Published: 19 August 2024**

Combining sound with tongue stimulation for the treatment of tinnitus: a multi-site single-arm controlled pivotal trial

- Michael Boedts,

Abstract

Bimodal neuromodulation is emerging as a nonsurgical treatment for tinnitus. **Bimodal treatment combining sound therapy with electrical tongue stimulation using the Lenire device is evaluated in a controlled pivotal trial** (TENT-A3, NCT05227365) consisting of 6-weeks of sound-only stimulation (Stage 1) followed by 6-weeks of bimodal treatment (Stage 2) with 112 participants serving as their own control. The primary endpoint compares the responder rate observed in Stage 2 versus Stage 1, where a responder exceeds 7 points in the Tinnitus Handicap Inventory. In participants with moderate or more severe tinnitus, there is a clinically superior performance of bimodal treatment (58.6%; 95% CI: 43.5%, 73.6%; $p = 0.022$) compared to sound therapy alone (43.2%; 95% CI: 29.7%, 57.8%), which is not observed in the full cohort across all severity groups. Consistent results are observed for the secondary endpoint based on the Tinnitus Functional Index (bimodal treatment: 45.5%; 95% CI: 31.7%, 59.9%; sound-only stimulation: 29.6%; 95% CI: 18.2%, 44.2%; $p = 0.010$), where a responder exceeds 13 points. There are no device related serious adverse events. These positive outcomes led to FDA De Novo approval of the Lenire device for tinnitus treatment.

ADVANCED TREATMENT STRATEGY: LENIRE DEVICE. MINISTIM OR STIMPOD TO TONGUE WITH AUDITORY THERAPIES LIKE TONE GENERATOR STACKED WITH LOW LEVEL LASER PROTOCOLS DESCRIBED IN PRIOR SLIDE

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE BRAIN-GUT AXIS

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

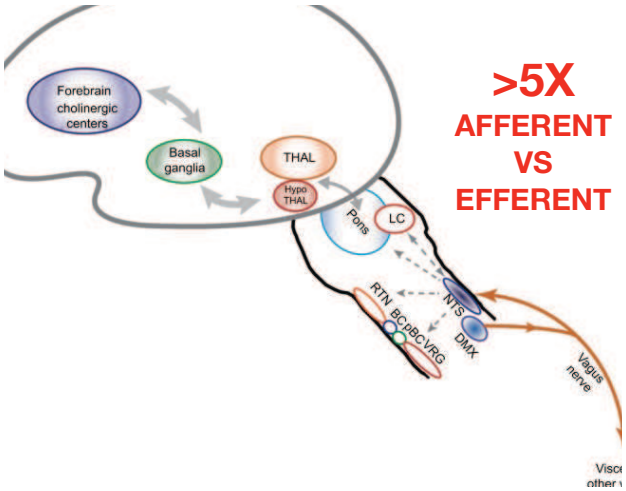
VAGAL NERVE STIMULATION

J Inflamm Res. 2018; 11: 203–213.
Published online 2018 May 16. doi: 10.2147/JIR.S163248 PMCID: PMC5961632PMID: _____

A review of vagus nerve stimulation as a therapeutic intervention

_____1 and _____1,2
Author information Copyright and License information Disclaimer

- EPILEPSY
- DIABETES
- PARKINSON'S
- TRAUMATIC BRAIN INJURY
- DEPRESSION
- SEPSIS/SYSTEMIC INFECTION
- POTS
- PATHOGENS AND DYSAUTONOMIA
- PAIN MANAGEMENT/FIBROMYALGIA
- LUNG INJURY
- STROKE
- OBESITY
- LEAKY GUT AND BRAIN
- CARDIOVASCULAR DISEASE AND MYOCARDIAL INFARCTIONS
- RHEUMATOID ARTHRITIS AND OTHER AUTOIMMUNE CONDITIONS
- POTENTIALLY CANCER!



>5X AFFERENT VS EFFERENT

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

VAGAL NERVE STIMULATION

Study Provides Better Insight Into the Vagus Nerve's Link to the Brain

FeaturedNeuroscience · July 28, 2022

Summary: Using stimulation, researchers found a direct connection between the vagus nerve and learning centers of the brain. Vagus nerve stimulation, they discovered, increases learning in a healthy nervous system.

"We concluded that there is a direct connection between the vagus nerve, the cholinergic system that regulates certain aspects of brain function, and motor cortex neurons that are essential in learning new skills," said Cristin Welle, PhD, senior author of the paper and the vice chair of research for the Department of Neurosurgery at the University of Colorado School of Medicine.

"Vagus nerve stimulation drives selective circuit modulation through cholinergic reinforcement" by Cristin Welle et al. *Neuron*

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PARASYMPATHETIC REGULATION

Modulation of the autonomic nervous system by one session of spinal low-level laser therapy in patients with chronic colonic motility dysfunction

M. Khawar Ali[1,2]

Patients with a defecation disorder may not evoke a normal defecation reflex, or the reflex may be excessive, as a dysfunction of the spinal autonomic nervous system. Treatment with various forms of lumbar and sacral neuromodulation have shown symptom improvement, but potential changes in autonomic functioning are rarely studied. Here we evaluate the effects on autonomic function of a single session of low-level laser therapy (LLLT) on the lumbar and sacral spine in 41 patients with chronic gastrointestinal motor dysfunction. The LLLT protocol used red LED light at a wavelength of 660 nm for 10 min and infrared LED light at a wavelength of 840 nm for 10 min, followed by infrared laser light at a wavelength of 825 nm for 10 min. Effects on the autonomic nervous system were assessed by measuring heart rate variability (HRV) changes. Respiratory Sinus Arrhythmia (RSA) and Root Mean Square of Successive Differences (RMSSD) were used to quantify parasympathetic reactivity; the Baevsky's Stress Index (SI) reflected sympathetic activity while the ratios SI/RSA and SI/RMSSD were used to show shifts in autonomic dominance. The results indicate that lumbar and sacral neuromodulation using light arrays reduced, whereas stimulation by the laser probes significantly increased parasympathetic activity. The light arrays increased whereas the laser probes significantly decreased sympathetic activity (SI). The entire protocol shifted the autonomic balance toward parasympathetic activity. **The comparison of actual vs. sham neuromodulation proved that the change in HRV parameters was due to actual light stimulation and not due to the arrays and probe touching the skin. In conclusion, a single session of LLLT markedly affects autonomic nervous system activity reflected in changes in HRV which is only possible by generating activity in the spinal autonomic nerves. These results warrant a study into the effects of LLLT on restoring autonomic dysfunction in chronic refractory colonic motility disorders.**

Front. Neurosci., 31 August 2022
Sec. Autonomic Neuroscience
Volume 16 - 2022 | https://doi.org/10.3389/fnins.2022.882602

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

VAGAL NERVE STIMULATION PROTOCOL

CONSIDER THE SACRAL PLEXUS!

RESTORING BRAIN —HEALTH—

RIGHT (rVNS) VS. LEFT (lVNS) VAGAL NERVE STIMULATION (Invasive iVNS vs Non-Invasive nVNS)

Table 1.

Outcomes of L-VNS and R-VNS for epilepsy treatment in human patients.

Type of VNS	Model	Outcome(s)	Reference
L-VNS			
iVNS	Pediatric patients with medical refractory epilepsy	68% clinical response rate in children	Patwardhan et al. (2000)
iVNS	Adult patients with epilepsy	46% decrease in seizure frequency (3 months)	Englot et al. (2011)
iVNS	Adult patients with epilepsy	50% decrease in seizure frequency	García-Navarrete et al. (2013)
iVNS	Adult patients with epilepsy	63% long-term response rate (2 years)	Englot et al. (2015)
iVNS	Pediatric patients with primary generalized epilepsy	64% improvement in seizure frequency (1 year)	Welch et al. (2018)
iVNS	Adult patients with epilepsy	90% decrease in seizure frequency	Batson et al. (2022)
R-VNS			
iVNS	Pediatric patient with epilepsy	Seizure-free for over 2 years with minor respiratory events	McGregor et al. (2005)
iVNS	Medically refractory epilepsy	Seizure suppression, minor cardiac symptoms	Spuck et al. (2008)
iVNS	Adult patients with epilepsy	50 and 90% seizure reduction	Navas et al. (2010)
iVNS	Adult patients with epilepsy	Marked seizure reduction without cardiac side effects	Galbarriatu et al. (2015)

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RIGHT (rVNS) VS. LEFT (lVNS) VAGAL NERVE STIMULATION (Invasive iVNS vs Non-Invasive nVNS)

Impacts of L-VNS and R-VNS on cognitive function in human subjects.

Type of VNS	Model	Outcome(s)	Reference
L-VNS			
iVNS	Patients with epilepsy	Improved word recognition memory	Clark et al. (1999)
iVNS	Patients with Alzheimer's disease	Positive effect on cognition	Sjögren et al. (2002)
iVNS	Patients with epilepsy	Enhancements in figural recognition tasks	Helmstaedter et al. (2001)
iVNS	Patients with epilepsy	Improved verbal memory retention	Ghacibeh et al. (2006)
tcVNS	Older adults	Increased associative memory performance	Jacobs et al. (2015)
taVNS	Patients with epilepsy	Reduced errors in working memory tasks	Sun et al. (2017)
tcVNS	Healthy adults	Increase performance in matrix reasoning tasks and fewer false negative errors in recognition tasks	Klaming et al. (2022)
R-VNS			
R-tVNS +L- tVNS	Sleep-deprived healthy adults	Enhancement in multitasking, arousal, and reduced fatigue-induced cognitive decline	McIntire et al. (2021)

RESTORING BRAIN —HEALTH—

RIGHT (rVNS) VS. LEFT (lVNS) VAGAL NERVE STIMULATION (Invasive iVNS vs Non-Invasive nVNS)

Effects of L-VNS and R-VNS on heart failure management in human patients.

Type of VNS	Model	Outcome(s)	Reference
L-VNS			
iVNS	Patients with heart failure	Improve absolute LVEF by 4.5% and six-minute walk distance by 56 meters after 6 months	Premchand et al. (2016)
R-VNS			
iVNS	Patients with heart failure	Improvements in NYHA class and left ventricular function	Schwartz et al. (2008)
iVNS	Patients with heart failure	No significant impact on remodeling but improved quality of life	Zannad et al. (2015)
iVNS	Patients with heart failure	Improved quality of life	Gold et al. (2016)
iVNS	Patients with heart failure	Well-tolerated with maintained improvements (no significant differences when compared with L-VNS)	Premchand et al. (2016)
taVNS	Patients with heart failure	Reduced inflammatory cytokines and improved quality of life	Stavrakis et al. (2022)

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RIGHT (rVNS) VS. LEFT (lVNS) VAGAL NERVE STIMULATION (Invasive iVNS vs Non-Invasive nVNS)

Results of L-VNS in treating depression and PTSD in human patients.

Type of VNS	Model	Outcome(s)	Reference
L-VNS			
iVNS	Patients with treatment-resistant depression	30% positive response	Rush et al. (2000)
iVNS	Patients with chronic or recurrent major depression	Progressive response and remission rates	Nahas et al. (2005); Schlaepfer et al.
iVNS	Patients with depression	Significant reductions in BDI scores	Cristancho et al. (2011)
iVNS	Patients with depression	20% response during the acute phase	Aaronson et al. (2013)
tcVNS	Patients with PTSD	Reversing neurobiological changes	Wittbrodt et al. (2021)
R-VNS			
No instances of R-VNS for applications in depression found at time of literature search			

RIGHT (rVNS) VS. LEFT (lVNS) VAGAL NERVE STIMULATION (Invasive iVNS vs Non-Invasive nVNS)

Front Neurosci. 2020 Apr 28;14:284. doi: 10.3389/fnins.2020.00284

Critical Review of Transcutaneous Vagus Nerve Stimulation: Challenges for Translation to Clinical Practice

Jonathan Y Y Yap

There's no robust clinical evidence that right-side VNS outperforms left for dystonia or other movement disorders; the safety convention still favors left.

AS A GENERAL RULE, FAVOR THE LEFT SIDE FOR NEUROLOGICAL CONDITIONS AND OVERALL SAFETY OF AUTONOMICS

HOWEVER, THE RIGHT SIDE MAY BE INDICATED IN AUTONOMIC REGULATION ESPECIALLY IF SA NODE MODULATION IS NEEDED

MONITOR AUTONOMICS (PULSE-OX, HRV ETC), AND BASE IT ON BEDSIDE NEUROLOGICAL EXAM

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

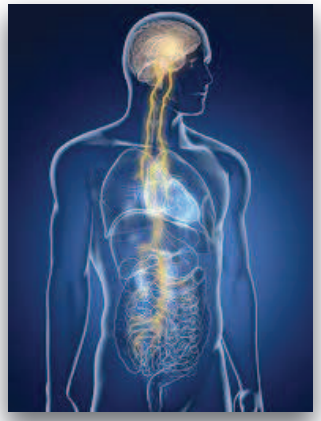
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

"DOWNSTREAM PROTOCOL"

COMBINE WITH VAGUS PROTOCOL AND THE SACRAL PLEXUS!

THE BRAIN-GUT AXIS

CLINICAL GEM: YOU CAN'T HAVE A HEALTHY GUT WITHOUT A HEALTHY BRAIN. CONSIDER VAGAL NERVE STIMULATION AND OTHER FUNCTIONAL NEUROLOGY PRINCIPLES FOR A HEALTHY BRAINSTEM AND OTHER AUTONOMIC REGULATION CENTERS



A HEALTHY BRAIN ALSO NEEDS A HEALTHY GUT. USE AN INTEGRATIVE APPROACH COMBINING FUNCTIONAL NEUROLOGY WITH FUNCTIONAL MEDICINE TO OPTIMIZE THIS CONNECTION

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

74 Year Old Female with Brain Fog, Fatigue, Loss of Voice and Severe Digestive Problems

BRAIN RESTORE AND VAGAL PROTOCOLS USING ERCHONIA LLLT
10-40-10-40
TRANSCRANIAL FRONTAL (ESPECIALLY BROCA'S AREA)
CEREBELLUM (R>L)
AND VAGUS LASER ONLY
(5 MIN SCM, 5 MIN GUT)
1.3 ATA HBOT 40+ MIN
NEUROFEEDBACK
(T3-T4)
NO NUTRITION DUE TO HER IMMUNOSUPPRESSION MEDICATIONS DUE TO A HEART TRANSPLANT
***GUT HEALTH NEEDS A HEALTHY BRAIN-GUT NEUROLOGICAL PATHWAY! IT'S NOT JUST ABOUT DIET AND SUPPLEMENTS.**

CASE STUDY: 5 DAY INTENSIVE BID (10 VISITS)

RESTORING BRAIN —HEALTH—

Nutrients. 2020 Mar; 12(3): 665.
Published online 2020 Feb 29. doi: 10.3390/nu12030665

GERD

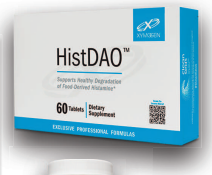
A Review of Zinc-L-Carnosine and Its Positive Effects on Oral Mucositis, Taste Disorders, and Gastrointestinal Disorders

Susan Hewlings[1],

Evidence supports the safety and efficacy of ZnC for the maintenance, prevention, and treatment of the mucosal lining and other epithelial tissues. The research supports its use for gastric ulcers (approved in Japan) and conditions of the upper GI and suggests other applications, particularly for oral mucositis.

ERCHONIA LLLT: VAGAL (10HZ) + LINE BEAM LINEAR DOWN ESOPHOGEAL/ EPIGASTRIC REGION 2 MINUTES/ VISIT

FOR AT LEAST 1 WEEK PRIOR TO PPI/H2 BLOCKER WITHDRAWAL CONSUME ZINC L-CARNITINE (XYMOGEN PEPCIX). IF CHEWABLE TABLET, CHEW AND SWALLOW TO TARGET ENTIRE GI LINING. DURING ACTIVE WITHDRAWAL PHASE USE 1 PEPCIX AND 1 HISTDAO PRE-MEAL(S) AND 1 AM XYMOZYME HCL



VIBRANT LAB CONSIDERATIONS:
FOOD ALLERGY
FOOD SENSITIVITY
LECTIN ZOOMER
FOOD ZOOMERS

OTHER CLINICAL AND LIFESTYLE CONSIDERATIONS:
SLOW AND RELAXED EATING/CHEW FOOD
HOME VAGAL STIM
AM APPLE CIDER VINEGAR 2 TBSP (DO CHALLENGE FIRST)
BEFORE BED ALOE JUICE 2 TBSP AND ANY TIME OF PAIN POST MEAL
CHIROPRACTIC/MSK REGIONAL T SPINE/RIB/PEC AND GI RELEASE

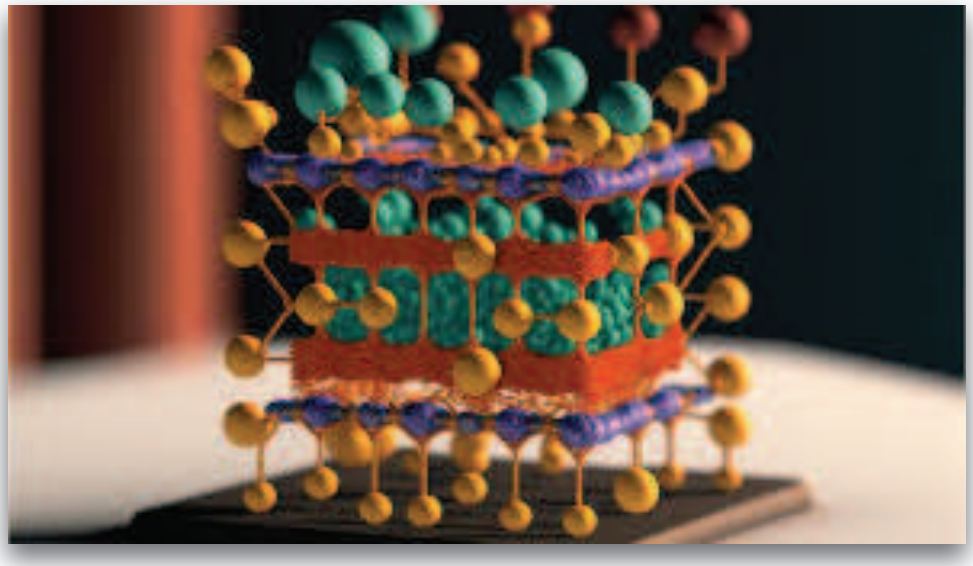
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND LPS



© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

INFLAMMATION AND CAUSES OF DEATH

The 15 leading causes of death in 2019 were:

1. Diseases of heart (heart disease)
2. Malignant neoplasms (cancer)
3. Accidents (unintentional injuries)
4. Chronic lower respiratory diseases
5. Cerebrovascular diseases (stroke)
6. Alzheimer disease
7. Diabetes mellitus (diabetes)
8. Nephritis, nephrotic syndrome and nephrosis (kidney disease)
9. Influenza and pneumonia
10. Intentional self-harm (suicide)
11. Chronic liver disease and cirrhosis
12. Septicemia
13. Essential hypertension and hypertensive renal disease (hypertension)
14. Parkinson disease
15. Pneumonitis due to solids and liquids

National Vital Statistics Reports

Volume 70, Number 8 — July 26, 2021

Deaths: Final Data for 2019

by Jiaquan Xu, M.D., Sherry L. Murphy, B.S., Kenneth D. Kochanek, M.A., and Elizabeth Arias, Ph.D., Division of Vital Statistics

Abstract

Objectives—This report presents final 2019 data on U.S. deaths, death rates, life expectancy, infant and maternal mortality, and trends by selected characteristics such as age, sex, Hispanic origin and race, state of residence, and cause of death. The race categories are consistent with 1997 Office of Management and Budget (OMB) standards, different from reports prior to 2018, which followed 1977 OMB standards.

Methods—Information reported on death certificates is presented in descriptive tabulations. The original records are filed in state registration offices. Statistical information is compiled in a national database through the Vital Statistics Cooperative Program of the National Center for Health Statistics. Causes of death are processed according to the *International Classification of Diseases, 10th Revision*. Beginning in 2018, all states and the District of Columbia were using the 2003 revised certificate of death for the entire year, which includes the 1997 OMB revised standards for race. The 2019 and 2018 data based on the revised standards are not completely comparable to previous years. Selected estimates are presented in this report for both the revised and previous race standards to provide some reference for interpretation of trends.

Results—In 2019, a total of 2,854,838 deaths were reported in the United States. The age-adjusted death rate was 715.2 deaths per 100,000 U.S. standard population, a decrease of 1.2% from the 2018 rate. Life expectancy at birth was 78.8 years, an increase of 0.1 year from 2018. Age-specific death rates decreased in 2019 from 2018 for age groups 45–54, 65–74, 75–84, and 85 and over and increased for age group 35–44. The 15 leading causes of death in 2019 remained the same as in 2018, although two causes exchanged ranks. Influenza and pneumonia, the eighth leading cause of death in 2018, became the ninth leading cause of death in 2019, while kidney disease, the ninth leading cause of death in 2018, became the eighth leading cause of death in 2019. The infant mortality rate, 5.58 infant deaths per 1,000 live births in 2019, remained a historic low but was not statistically different from the rate in 2018.

Conclusions—In 2019, the age-adjusted death rate decreased and life expectancy at birth increased for the total, male, and female populations for the second consecutive year.

Keywords: mortality • cause of death • life expectancy • National Vital Statistics System

Highlights

Mortality experience in 2019

- In 2019, a total of 2,854,838 resident deaths were registered in the United States, yielding a crude death rate of 869.7 per 100,000 population.
- The age-adjusted death rate, which accounts for the aging of the population, was 715.2 deaths per 100,000 U.S. standard population.
- Life expectancy at birth was 78.8 years.
- The 15 leading causes of death in 2019 were:

1. Diseases of heart (heart disease)
2. Malignant neoplasms (cancer)
3. Accidents (unintentional injuries)
4. Chronic lower respiratory diseases
5. Cerebrovascular diseases (stroke)
6. Alzheimer disease
7. Diabetes mellitus (diabetes)
8. Nephritis, nephrotic syndrome and nephrosis (kidney disease)
9. Influenza and pneumonia
10. Intentional self-harm (suicide)
11. Chronic liver disease and cirrhosis
12. Septicemia
13. Essential hypertension and hypertensive renal disease (hypertension)
14. Parkinson disease
15. Pneumonitis due to solids and liquids

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Centers for Disease Control and Prevention
National Center for Health Statistics
National Vital Statistics System
CDC

SPECIAL THANKS TO DR. TOM O'BRYAN

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS AI RESEARCH ANALYSIS: INFLAMMATION

WHAT WOULD BE AN EASY TO RUN LAB MARKER TO ASSESS OVERALL SYSTEMIC INFLAMMATION LEVELS?

WHAT DIET FORM WOULD BE THE BEST WAY TO ADDRESS THIS INFLAMMATION?

WHAT SUPPLEMENT IS THE BEST OVERALL TO PUT OUT SYSTEMIC INFLAMMATION?

WHAT SINGLE MODALITY WOULD BE BEST TO TAMP OUT THAT INFLAMMATION?

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

CONSENSUS AI RESEARCH ANALYSIS: INFLAMMATION

Best single lab for inflammation

- **hs-CRP**

Best diet

- **Mediterranean / Keto-flex anti-inflammatory diet**

Best supplement

- **Omega-3 fatty acids (EPA/DHA)**

Best single modality

- **Low-Level Laser Therapy** [188] **(Photobiomodulation)**

KEY REFERENCES

Inflammation markers

- Ridker PM. *Circulation*. 2003.
- Pearson TA et al. *Circulation*. 2003.

Diet & inflammation

- Estruch R et al. *NEJM*. 2013.
- Schwingshackl L et al. *BMJ*. 2018.

Omega-3 inflammation resolution

- Calder PC. *Nutrients*. 2017.
- Serhan CN. *Nature*. 2014.

Photobiomodulation & inflammation

- Hamblin MR. *BBA – Bioenergetics*. 2016.
- de Freitas LF, Hamblin MR. *Photomed Laser*
- Chung H et al. *Ann Biomed Eng*. 2012.
- Huang YY et al. *J Biophotonics*. 2011.

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

PRIMARY MECHANISMS OF INFLAMMATION AND NEURODEGENERATION

Wheat Zoomer

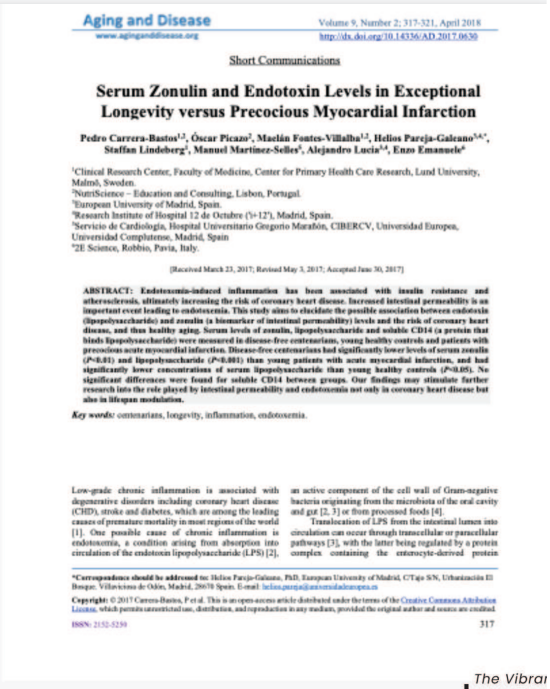
Intestinal Permeability Panel		Current	Previous	Result	Reference
Zonulin (ng/mL)		16.6		0 — 45.3 — 55.3	≤45.3
Intestinal Permeability Panel		**Current**	**Previous**	**Result**	**Reference**
Anti-Zonulin	IgA	1.34		0 — 0.89 — 1.1	≤0.89
	IgG	2.97		0 — 0.89 — 1.1	≤0.89
Anti-Actin	IgA	0.34		0 — 0.89 — 1.1	≤0.89
	IgG	1.08		0 — 0.89 — 1.1	≤0.89
Human Anti-Lipopolysaccharide	IgA	23.7		0 — 30	≤30.0 (U/ml)
	IgG	411.9		0 — 281	≤281.0 (U/ml)

IS IT POSSIBLE THAT THESE TEST MARKERS ARE THE ROOT MECHANISM OF DISEASE INCLUDING NEURODEGENERATIVE CONDITIONS SUCH AS ALZHEIMER'S AND CARDIOVASCULAR DISEASE??

INFLAMMATION AND CAUSES OF DEATH

Disease-free centenarians had significantly lower levels of serum zonulin (P<0.01) and LPS (P<0.001) than young patients with acute myocardial infarction, and had significantly lower concentrations of serum LPS than young healthy controls

Aging and Disease Volume 9, Number 2; 317-321, April 2018



Aging and Disease
www.agingandisease.org
Volume 9, Number 2; 317-321, April 2018
http://dx.doi.org/10.14336/AD.2017.0630

Short Communications

Serum Zonulin and Endotoxin Levels in Exceptional Longevity versus Precocious Myocardial Infarction

Pedro Carrera-Bastos[1,2], Óscar Picazo[3], Maelán Fontes-Villalba[1,2], Helios Pareja-Galeano[3,4,*], Staffan Lindeberg[5], Manuel Martínez-Selles[6], Alejandro Lucia[3,4], Enzo Emanuele[6]

[1]Clinical Research Center, Faculty of Medicine, Center for Primary Health Care Research, Lund University, Malmö, Sweden.
[2]NutriScience – Education and Consulting, Lisbon, Portugal.
[3]European University of Madrid, Spain.
[4]Research Institute of Hospital 12 de Octubre (i+12), Madrid, Spain.
[5]Servicio de Cardiología, Hospital Universitario Gregorio Marañón, CIBERCV, Universidad Europea, Universidad Complutense, Madrid, Spain
[6]2E Science, Robbio, Pavia, Italy.

[Received March 23, 2017; Revised May 3, 2017; Accepted June 30, 2017]

ABSTRACT: Endotoxemia-induced inflammation has been associated with insulin resistance and atherosclerosis, ultimately increasing the risk of coronary heart disease. Increased intestinal permeability is an important event leading to endotoxemia. This study aims to elucidate the possible association between endotoxin (lipopolysaccharide) and zonulin (a biomarker of intestinal permeability) levels and the risk of coronary heart disease, and thus healthy aging. Serum levels of zonulin, lipopolysaccharide and soluble CD14 (a protein that binds lipopolysaccharide) were measured in disease-free centenarians, young healthy controls and patients with precocious acute myocardial infarction. Disease-free centenarians had significantly lower levels of serum zonulin ($P<0.01$) and lipopolysaccharide ($P<0.001$) than young patients with acute myocardial infarction, and had significantly lower concentrations of serum lipopolysaccharide than young healthy controls ($P<0.05$). No significant differences were found for soluble CD14 between groups. Our findings may stimulate further research into the role played by intestinal permeability and endotoxemia not only in coronary heart disease but also in lifespan modulation.

Key words: centenarians, longevity, inflammation, endotoxemia.

Low-grade chronic inflammation is associated with degenerative disorders including coronary heart disease (CHD), stroke and diabetes, which are among the leading causes of premature mortality in most regions of the world [1]. One possible cause of chronic inflammation is endotoxemia, a condition arising from absorption into circulation of the endotoxin lipopolysaccharide (LPS) [2], an active component of the cell wall of Gram-negative bacteria originating from the microbiota of the oral cavity and gut [2, 3] or from processed foods [4].

Translocation of LPS from the intestinal lumen into circulation can occur through transcellular or paracellular pathways [3], with the latter being regulated by a protein complex containing the enterocyte-derived protein

*Correspondence should be addressed to: Helios Pareja-Galeano, PhD. European University of Madrid, C/Tajo S/N, Urbanización El Bosque. Villaviciosa de Odón, Madrid, 28670 Spain. E-mail: helios.pareja@universidadeuropea.es

Copyright: © 2017 Carrera-Bastos, P et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

ISSN: 2152-5250

The Vibra

SPECIAL THANKS TO DR. TOM O'BRYAN

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND CAUSES OF DEATH

Aging and Disease
www.agingandisease.org
Volume 9, Number 2; 317-321, April 2018
http://dx.doi.org/10.14336/AD.2017.0630

Short Communications

Serum Zonulin and Endotoxin Levels in Exceptional Longevity versus Precocious Myocardial Infarction

Pedro Carrera-Bastos[1,2], Óscar Picazo[3], Maelán Fontes-Villalba[1,2], Helios Pareja-Galeano[3,4,*], Staffan Lindeberg[5], Manuel Martínez-Selles[6], Alejandro Lucia[3,4], Enzo Emanuele[7]

[1]Clinical Research Center, Faculty of Medicine, Center for Primary Health Care Research, Lund University, Malmö, Sweden.
[2]NutriScience – Education and Consulting, Lisbon, Portugal.
[3]European University of Madrid, Spain.
[4]Research Institute of Hospital 12 de Octubre (i+12), Madrid, Spain.
[5]Servicio de Cardiología, Hospital Universitario Gregorio Marañón, CIBERCV, Universidad Europea, Universidad Complutense, Madrid, Spain
[7]2E Science, Robbio, Pavia, Italy.

[Received March 23, 2017; Revised May 3, 2017; Accepted June 30, 2017]

ABSTRACT: Endotoxemia-induced inflammation has been associated with insulin resistance and atherosclerosis, ultimately increasing the risk of coronary heart disease. Increased intestinal permeability is an important event leading to endotoxemia. This study aims to elucidate the possible association between endotoxin (lipopolysaccharide) and zonulin (a biomarker of intestinal permeability) levels and the risk of coronary heart disease, and thus healthy aging. Serum levels of zonulin, lipopolysaccharide and soluble CD14 (a protein that binds lipopolysaccharide) were measured in disease-free centenarians, young healthy controls and patients with precocious acute myocardial infarction. Disease-free centenarians had significantly lower levels of serum zonulin ($P<0.01$) and lipopolysaccharide ($P<0.001$) than young patients with acute myocardial infarction, and had significantly lower concentrations of serum lipopolysaccharide than young healthy controls ($P<0.05$). No significant differences were found for soluble CD14 between groups. Our findings may stimulate further research into the role played by intestinal permeability and endotoxemia not only in coronary heart disease but also in lifespan modulation.

Key words: centenarians, longevity, inflammation, endotoxemia.

Low-grade chronic inflammation is associated with degenerative disorders including coronary heart disease (CHD), stroke and diabetes, which are among the leading causes of premature mortality in most regions of the world [1]. One possible cause of chronic inflammation is endotoxemia, a condition arising from absorption into circulation of the endotoxin lipopolysaccharide (LPS) [2], an active component of the cell wall of Gram-negative bacteria originating from the microbiota of the oral cavity and gut [2, 3] or from processed foods [4].

Translocation of LPS from the intestinal lumen into circulation can occur through transcellular or paracellular pathways [3], with the latter being regulated by a protein complex containing the enterocyte-derived protein

*Correspondence should be addressed to: Helios Pareja-Galeano, PhD, European University of Madrid, C/Tajo S/N, Urbanización El Bosque. Villaviciosa de Odón, Madrid, 28670 Spain. E-mail: helios.pareja@universidadeuropea.es

Copyright: © 2017 Carrera-Bastos, P et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

ISSN: 2152-5250

Intestinal hyperpermeability may cause endotoxemia, which in turn could lead to inflammation and hence insulin resistance, atherosclerosis and hypercoagulation

Aging and Disease Volume 9, Number 2; 317-321, April 2018

SPECIAL THANKS TO DR. TOM O'BRYAN

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

INFLAMMATION AND CAUSES OF DEATH

REVIEW
All disease begins in the (leaky) gut: role of zonulin-mediated gut permeability in the pathogenesis of some chronic inflammatory diseases [version 1; peer review: 3 approved]

Alessio Fasano [1,2]

[1]Mucosal Immunology and Biology Research Center, Center for Celiac Research and Treatment and Division of Pediatric Gastroenterology and Nutrition, Massachusetts General Hospital for Children, Boston, Massachusetts, USA
[2]European Biomedical Research Institute of Salerno, Salerno, Italy

V1 First published: 31 Jan 2020, 9(F1000 Faculty Rev):69 (https://doi.org/10.12688/f1000research.20510.1)
Latest published: 31 Jan 2020, 9(F1000 Faculty Rev):69 (https://doi.org/10.12688/f1000research.20510.1)

Abstract
Improved hygiene leading to reduced exposure to microorganisms has been implicated as one possible cause for the recent "epidemic" of chronic inflammatory diseases (CIDs) in industrialized countries. That is the essence of the hygiene hypothesis that argues that rising incidence of CIDs may be, at least in part, the result of lifestyle and environmental changes that have made us too "clean" for our own good, so causing changes in our microbiota. Apart from genetic makeup and exposure to environmental triggers, inappropriate increase in intestinal permeability (which may be influenced by the composition of the gut microbiota), a "hyper-belligerent" immune system responsible for the tolerance–immune response balance, and the composition of gut microbiome and its epigenetic influence on the host genomic expression have been identified as three additional elements in causing CIDs. During the past decade, a growing number of publications have focused on human genetics, the gut microbiome, and proteomics, suggesting that loss of mucosal barrier function, particularly in the gastrointestinal tract, may substantially affect antigen trafficking, ultimately influencing the close bidirectional interaction between gut microbiome and our immune system. This cross-talk is highly influential in shaping the host gut immune system function and ultimately shifting genetic predisposition to clinical outcome. This observation led to a re-visitation of the possible causes of CIDs epidemics, suggesting a key pathogenic role of gut permeability. Pre-clinical and clinical studies have shown that the zonulin family, a group of proteins modulating gut permeability, is implicated in a variety of CIDs, including autoimmune, infective, metabolic, and tumoral diseases. These data offer novel therapeutic targets for a variety of CIDs in which the zonulin pathway is implicated in their pathogenesis.

Keywords
Chronic inflammatory diseases, Gut permeability, microbiome, zonulin

Open Peer Review
Reviewer Status
Invited Reviewers 1 2 3
version 1

F1000 Faculty Reviews are written by members of the prestigious F1000 Faculty. They are commissioned and peer reviewed before publication to ensure that the final, published version is comprehensive and accessible. The reviewers who approved the final version are listed with their names and affiliations.

1 Xin M. Luo, Virginia Tech, Blacksburg, USA
2 Michael Maes, Chulalongkorn University, Bangkok, Thailand
3 Arul Jayaraman, Texas A&M Health Science Center, Bryan, USA

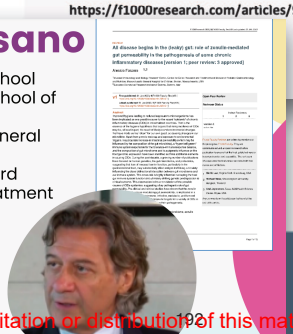
Any comments on the article can be found at the end of the article.

Among the several potential intestinal luminal stimuli that can stimulate zonulin release (thus Intestinal Permeability), small exposure to large amounts of bacteria (and its exhaust LPS) and gluten, have been identified as the two most powerful triggers.

https://f1000research.com/articles/9-69/v1

Professor Alessio Fasano

- Professor of Pediatrics at Harvard Medical School
- Professor of Nutrition at Harvard T.H. Chan School of Public Health
- Chief of Pediatric Gastroenterology, Mass General Hospital
- Director, Mucosal Immunology Center, Harvard
- Director, Center for Celiac Research and Treatment
- Director, Mucosal Immunology and Biology Research Center;
- Associate Chief for Basic, Clinical and Translational Research
- 327 publications on pubmed.gov
- Identified zonulin as the protein activated in intestinal permeability



SPECIAL THANKS TO DR. TOM O'BRYAN

RESTORING BRAIN —HEALTH—

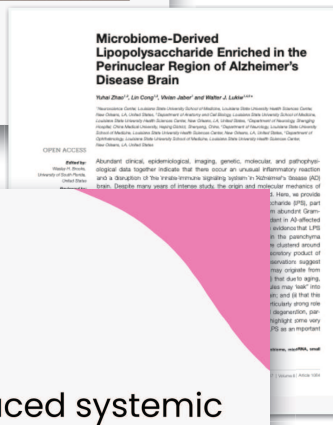
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND CAUSES OF DEATH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

One of the main toxins responsible for inflammation induction are lipopolysaccharides (LPS, endotoxin) from Gram-negative bacteria, which rank among the most potent immunostimulants found in nature.

Int. J. Mol. Sci. 2023, 24, 15169.



LPS is recognized by the immune system (TLR4) as a marker for the detection of bacterial pathogen invasion and responsible for the development of inflammatory response.

Front. Immunol. 8:1064. doi: 10.3389/fimmu.2017

LPS-induced systemic inflammatory toxicity is termed 'endotoxemia'

Cells 2022, 11,4038

SPECIAL THANKS TO DR. TOM O'BRYAN

RESTORING BRAIN —HEALTH—

INFLAMMATION AND CAUSES OF DEATH

LPS causes a substantial increase in the production of cytokines, chemokines, and the synthesis of a broad group of lipid inflammatory mediators

International Journ

SPECIAL THANKS TO DR. TOM O'BRYAN

For instance, complex formation between all lipoprotein subclasses (HDL, LDL, VLDL, oxidized LDL), and both bacteria and viruses has been demonstrated by electron microscopy, enzyme-linked immunoabsorbence assay, and column chromatography

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

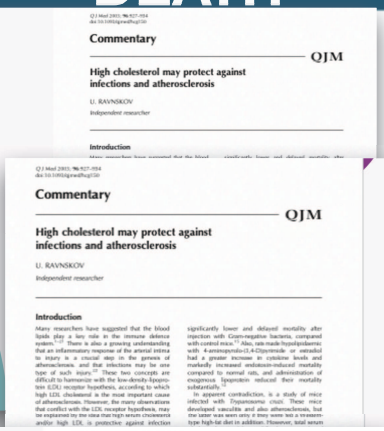
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND CAUSES OF DEATH

High serum cholesterol and/or high LDL is protective against infection and atherosclerosis

QJM 2013, Vol. 96, no. 12, 927-934



Commentary

QJM

High cholesterol may protect against infections and atherosclerosis

U. RAVNSKOV

Independent researcher

Introduction

Many researchers have suggested that the blood lipids play a key role in the immune defence system. [1–3] There is also a growing understanding that an inflammatory response of the arterial intima to injury is a crucial step in the genesis of atherosclerosis, and that infections may be one type of such injury. [4–6] These two concepts are difficult to harmonize with the low-density-lipoprotein (LDL) receptor hypothesis, according to which high LDL cholesterol is the most important cause of atherosclerosis. However, the many observations that conflict with the LDL receptor hypothesis, may be explained by the idea that high serum cholesterol and/or high LDL is protective against infection

significantly lower and delayed mortality after injection with Gram-negative bacteria, compared with control mice. [7] Also, rats made hypolipidaemic with 4-aminopyrido-[3,4-d]pyrimidin- or estradiol had a greater increase in cytokine levels and markedly increased endotoxin-induced mortality compared to normal rats, and administration of exogenous lipoprotein reduced their mortality substantially. [8]

In apparent contradiction, is a study of mice infected with *Trypanosoma cruzi*. These mice developed vasculitis and also atherosclerosis, but the latter was seen only if they were fed a treatment-type high-fat diet in addition. However, fatal serum

Commentary

QJM

High cholesterol may protect against infections and atherosclerosis

U. RAVNSKOV

Independent researcher

Introduction

Many researchers have suggested that the blood lipids play a key role in the immune defence system. [1–3] There is also a growing understanding that an inflammatory response of the arterial intima to injury is a crucial step in the genesis of atherosclerosis, and that infections may be one type of such injury. [4–6] These two concepts are difficult to harmonize with the low-density-lipoprotein (LDL) receptor hypothesis, according to which high LDL cholesterol is the most important cause of atherosclerosis. However, the many observations that conflict with the LDL receptor hypothesis, may be explained by the idea that high serum cholesterol and/or high LDL is protective against infection

significantly lower and delayed mortality after injection with Gram-negative bacteria, compared with control mice. [7] Also, rats made hypolipidaemic with 4-aminopyrido-[3,4-d]pyrimidin- or estradiol had a greater increase in cytokine levels and markedly increased endotoxin-induced mortality compared to normal rats, and administration of exogenous lipoprotein reduced their mortality substantially. [8]

In apparent contradiction, is a study of mice infected with *Trypanosoma cruzi*. These mice developed vasculitis and also atherosclerosis, but the latter was seen only if they were fed a treatment-type high-fat diet in addition. However, fatal serum

LPS, or endotoxin, the main pathogenic factor of Gram-negative bacteria, binds rapidly to lipoproteins, mainly LDL, and lipoprotein-bound LPS is <u>unable to activate the secretion of various cytokines by monocytes</u>.

Lipoprotein(a) [Lp(a)] was as potent as low-density lipoproteins (LDL) in inhibiting LPS-stimulated TNF synthesis by human mononuclear cells.

INFECTION AND IMMUNITY, May 1998, p. 2365–2367

INFECTION AND IMMUNITY, May 1998, p. 2365–2367 Vol. 66, No. 5

Copyright © 1998, American Society for Microbiology

Lipoprotein(a) Inhibits Lipopolysaccharide-Induced Tumor Necrosis Factor Alpha Production by Human Mononuclear Cells

MIHAI G. NETEA, NATASJA nn BONT, PIERRE N. M. DEMACKER, BART JAN KULLBERG, LIESBETH E. H. JACOBS, THEES J. G. VERVER-JANSEN, ANTON F. H. STALENHOEF, AND JOS W. M. VAN DER MEER*

Division of General Internal Medicine, Department of Medicine, University Hospital Nijmegen, 6500 HB Nijmegen, The Netherlands

Received 15 November 1997/Returned for modification 15 December 1997/Accepted 5 February 1998

Lipoproteins can bind lipopolysaccharide (LPS) and decrease LPS-stimulated cytokine production. Lipoprotein(a) [Lp(a)] was as potent as low-density lipoproteins (LDL) in inhibiting LPS-stimulated tumor necrosis factor synthesis by human mononuclear cells. The kinetics of LPS inhibition by Lp(a) was similar to that of LDL. This suggests that circulating Lp(a) may be an important factor determining the amplitude of the response to LPS in humans.

The systemic toxicity of gram-negative sepsis is largely due to endotoxin, a lipopolysaccharide (LPS) component of the outer membrane of gram-negative bacteria. LPS stimulates the production of proinflammatory cytokines such as tumor necrosis factor alpha (TNF) and interleukin-1, which in turn may induce disseminated intravascular coagulation, hypotension, and multiple organ failure (reviews in refs. 1 and 2). In the circulation, initial interactions of LPS with LPS-binding protein and lipoproteins are essential for stimulation of cytokine production. While LPS-binding protein mediates binding of LPS to CD14, the main LPS receptor, leading to stimulation of cytokine synthesis (23), hydrophobic interaction between the lipid A component of LPS and lipoproteins neutralizes LPS and inhibits LPS-induced cytokine release (14). Preincubation of LPS with low-density lipoproteins (LDL) (26), high-density lipoproteins (HDL) (14), or very-low-density lipoproteins (VLDL) reduces LPS-induced cytokine production in vitro, whereas hypolipoproteinemia protects animals against lethal endotoxemia (11, 13) and gram-negative sepsis (17, 21) in vivo. In humans, reconstituted HDL inhibits LPS-induced cytokine release in whole blood (20), and infusion of reconstituted HDL has potent anti-inflammatory effects during human endotoxemia (12). Thus, the ability of lipoproteins to neutralize LPS is mediated by lipoprotein-in the neutralization of LPS during endotoxemia and gram-negative infections. All the studies to date have focused on the interaction of LPS with the major lipoprotein subfractions LDL, HDL, and very-low-density lipoproteins, while studies investigated the LPS-neutralizing properties of the relatively recently described lipoprotein(a) [Lp(a)]. Lp(a) is a lipoprotein particle having apolipoprotein B-100 (apoB-100) as a protein moiety, linked by disulfide bridges to apolipoprotein(a) [apo(a)] (reviews in refs. 3 and 4). It is accepted that Lp(a) is an important and independent risk factor for the development of atherosclerosis (for a review see references 22). Lp(a) levels in plasma are genetically determined and vary widely, with differences as high as 100-fold being found among individuals, ranging from 20 to 2,000 mg/

* Corresponding author. Mailing address: Department of Medicine (543), University Hospital Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands. Phone: 31-24-3619876. Fax: 31-24-3540734. E-mail: j.vandermeer@int.azn.nl.

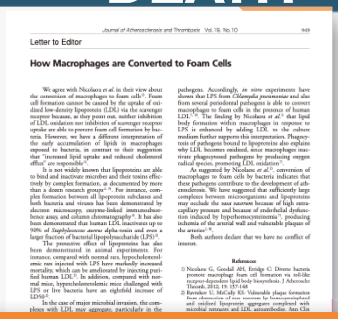
SPECIAL THANKS TO DR. TOM O'BRYAN

RESTORING BRAIN —HEALTH—

INFLAMMATION AND CAUSES OF DEATH

It has also been demonstrated that human LDL inactivates up to 90% of *Staphylococcus aureus* alpha-toxin and even a larger fraction of bacterial lipopolysaccharide (LPS)

Journal of Atherosclerosis and Thrombosis Vol.19, No.10



Journal of Atherosclerosis and Thrombosis Vol. 19, No. 10 949

Letter to Editor

How Macrophages are Converted to Foam Cells

We agree with Nicolaes *et al.* in their view about the conversion of macrophages to foam cells[1]. Foam cell formation cannot be caused by the uptake of oxidized low-density lipoprotein (LDL) via the scavenger receptor because, as they point out, neither inhibition of LDL oxidation nor inhibition of scavenger receptor uptake are able to prevent foam cell formation by bacteria. However, we have a different interpretation of the early accumulation of lipids in macrophages exposed to bacteria, in contrast to their suggestion that "increased lipid uptake and reduced cholesterol efflux" are responsible[1].

It is not widely known that lipoproteins are able to bind and inactivate microbes and their toxins effectively by complex formation, as documented by more than a dozen research groups[2–4]. For instance, complex formation between all lipoprotein subclasses and both bacteria and viruses has been demonstrated by electron microscopy, enzyme-linked immunosorbent assay, and column chromatography[5]. It has also been demonstrated that human LDL inactivates up to 90% of *Staphylococcus aureus* alpha-toxin and even a larger fraction of bacterial lipopolysaccharide (LPS)[6].

The protective effect of lipoproteins has also been demonstrated in animal experiments. For instance, compared with normal rats, hypercholesterolemic rats injected with LPS have markedly increased mortality, which can be ameliorated by injecting purified human LDL[7]. In addition, compared with normal mice, hypercholesterolemic mice challenged with LPS or live bacteria have an eightfold increase of LD50[8].

In the case of major microbial invasion, the complexes with LDL may aggregate, particularly in the

pathogens. Accordingly, *in vitro* experiments have shown that LPS from *Chlamydia pneumoniae* and also from several periodontal pathogens is able to convert macrophages to foam cells in the presence of human LDL[9–11]. The finding by Nicolaes *et al.*[1] that lipid body formation within macrophages in response to LPS is enhanced by adding LDL to the culture medium further supports this interpretation. Phagocytosis of pathogens bound to lipoproteins also explains why LDL becomes oxidized, since macrophages inactivate phagocytosed pathogens by producing oxygen radical species, promoting LDL oxidation[12].

As suggested by Nicolaes *et al.*[1], conversion of macrophages to foam cells by bacteria indicates that these pathogens contribute to the development of atherosclerosis. We have suggested that sufficiently large complexes between microorganisms and lipoproteins may occlude the vasa vasorum because of high extracapillary pressure and because of endothelial dysfunction induced by hypercholesterolemia[13], producing ischemia of the arterial wall and vulnerable plaques of the arteries[14].

Both authors declare that we have no conflict of interest.

References

1) Nicolaes G, Goedkoop AH, Erridge C: Diverse bacteria promote macrophage foam cell formation via toll-like receptor-dependent lipid body biosynthesis. J Atheroscler Thromb. 2012; 19: 157-168

2) Ravnskov U, McCully KS: Vulnerable plaque formation from obstruction of vasa vasorum by homocysteinylated and oxidized lipoprotein aggregates complexed with microbial remnants and LDL autoantibodies. Ann Clin Lab Sci. 2009; 39: 3-16

3) Hurt B: Plasma lipoproteins are important components of the immune system. Microbiol Immunol. 2010; 54: 246-253

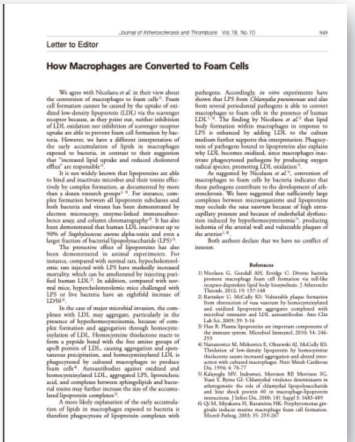
4) Hoeneveld M, Mokotoff E, Oliveroli AJ, McCully KS: Lipoprotein(a) and homocysteine in relation to atherosclerosis: stress-induced aggregation and altered interaction with cultured macrophages. Nutr Metab Cardiovasc Dis. 1996; 6: 70-77

5) Kalayoglu MV, Indrawati, Morrison RP, Morrison SG, Yoast T, Byrne GI: Cellular and vascular responses to intragastric infection of C3H/HeJ mice with *Chlamydia pneumoniae*. Am Heart Assoc Circ Arterioscler Thromb Vasc Biol. 2000; 20: 1884-1888

6) Qi M, Miyakawa H, Kuramitsu HK: Porphyromonas gingivalis induces murine macrophage foam cell formation. Microb Pathog. 2003; 35: 259-267

Phagocytosis of LPS bound to lipoproteins also explains why LDL becomes oxidized, since macrophages inactivate phagocytosed pathogens by producing oxygen radical species, promoting LDL oxidation

Journal of Atherosclerosis and Thrombosis Vol.19, No.10



Journal of Atherosclerosis and Thrombosis Vol. 19, No. 10 949

Letter to Editor

How Macrophages are Converted to Foam Cells

We agree with Nicolaes *et al.* in their view about the conversion of macrophages to foam cells[1]. Foam cell formation cannot be caused by the uptake of oxidized low-density lipoprotein (LDL) via the scavenger receptor because, as they point out, neither inhibition of LDL oxidation nor inhibition of scavenger receptor uptake are able to prevent foam cell formation by bacteria. However, we have a different interpretation of the early accumulation of lipids in macrophages exposed to bacteria, in contrast to their suggestion that "increased lipid uptake and reduced cholesterol efflux" are responsible[1].

It is not widely known that lipoproteins are able to bind and inactivate microbes and their toxins effectively by complex formation, as documented by more than a dozen research groups[2–4]. For instance, complex formation between all lipoprotein subclasses and both bacteria and viruses has been demonstrated by electron microscopy, enzyme-linked immunosorbent assay, and column chromatography[5]. It has also been demonstrated that human LDL inactivates up to 90% of *Staphylococcus aureus* alpha-toxin and even a larger fraction of bacterial lipopolysaccharide (LPS)[6].

The protective effect of lipoproteins has also been demonstrated in animal experiments. For instance, compared with normal rats, hypercholesterolemic rats injected with LPS have markedly increased mortality, which can be ameliorated by injecting purified human LDL[7]. In addition, compared with normal mice, hypercholesterolemic mice challenged with LPS or live bacteria have an eightfold increase of LD50[8].

In the case of major microbial invasion, the complexes with LDL may aggregate, particularly in the presence of hypercholesterolemia, because of complex formation and aggregation through homocysteinylation of LDL. Homocysteine thiolactone reacts to form a peptide bond with the free amino groups of apoB protein of LDL, causing aggregation and spontaneous precipitation, and homocysteinylated LDL is phagocytosed by cultured macrophages to produce foam cells[9]. Autoantibodies against oxidized and homocysteinylated LDL, aggregated LPS, lipoteichoic acid, and complexes between sphingolipids and bacterial toxins may further increase the size of the accumulated lipoprotein complexes[10].

A more likely explanation of the early accumulation of lipids in macrophages exposed to bacteria is therefore phagocytosis of lipoprotein complexes with

pathogens. Accordingly, *in vitro* experiments have shown that LPS from *Chlamydia pneumoniae* and also from several periodontal pathogens is able to convert macrophages to foam cells in the presence of human LDL[9–11]. The finding by Nicolaes *et al.*[1] that lipid body formation within macrophages in response to LPS is enhanced by adding LDL to the culture medium further supports this interpretation. Phagocytosis of pathogens bound to lipoproteins also explains why LDL becomes oxidized, since macrophages inactivate phagocytosed pathogens by producing oxygen radical species, promoting LDL oxidation[12].

As suggested by Nicolaes *et al.*[1], conversion of macrophages to foam cells by bacteria indicates that these pathogens contribute to the development of atherosclerosis. We have suggested that sufficiently large complexes between microorganisms and lipoproteins may occlude the vasa vasorum because of high extracapillary pressure and because of endothelial dysfunction induced by hypercholesterolemia[13], producing ischemia of the arterial wall and vulnerable plaques of the arteries[14].

Both authors declare that we have no conflict of interest.

References

1) Nicolaes G, Goedkoop AH, Erridge C: Diverse bacteria promote macrophage foam cell formation via toll-like receptor-dependent lipid body biosynthesis. J Atheroscler Thromb. 2012; 19: 157-168

2) Ravnskov U, McCully KS: Vulnerable plaque formation from obstruction of vasa vasorum by homocysteinylated and oxidized lipoprotein aggregates complexed with microbial remnants and LDL autoantibodies. Ann Clin Lab Sci. 2009; 39: 3-16

3) Hurt B: Plasma lipoproteins are important components of the immune system. Microbiol Immunol. 2010; 54: 246-253

4) Hoeneveld M, Mokotoff E, Oliveroli AJ, McCully KS: Lipoprotein(a) and homocysteine in relation to atherosclerosis: stress-induced aggregation and altered interaction with cultured macrophages. Nutr Metab Cardiovasc Dis. 1996; 6: 70-77

5) Kalayoglu MV, Indrawati, Morrison RP, Morrison SG, Yoast T, Byrne GI: Cellular and vascular responses to intragastric infection of C3H/HeJ mice with *Chlamydia pneumoniae*. Am Heart Assoc Circ Arterioscler Thromb Vasc Biol. 2000; 20: 1884-1888

6) Qi M, Miyakawa H, Kuramitsu HK: Porphyromonas gingivalis induces murine macrophage foam cell formation. Microb Pathog. 2003; 35: 259-267

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND CAUSES OF DEATH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Letter to Editor

How Macrophages are Converted to Foam Cells

Conversion of macrophages to foam cells by bacteria indicates that these pathogens contribute to the development of atherosclerosis.

Journal of Atherosclerosis and Thrombosis Vol.19, No.10

SPECIAL THANKS TO DR. TOM O'BRYAN

Compared with normal rats, hypocholesterolemic rats injected with LPS have markedly increased mortality (8-fold), which can be ameliorated by injecting purified human LDL

Letter to Editor

How Macrophages are Converted to Foam Cells

INFLAMMATION AND CAUSES OF DEATH

Could this mean that sometimes, elevated cholesterol (total, HDL, LDL, oxidized LDL, and/or Lp(a)) may be life-saving attempts by the body?

SPECIAL THANKS TO DR. TOM O'BRYAN

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LPS INDUCED ENDOTOXEMIA

LDL, VLDL, and HDL act as LPS scavengers

- Lipoproteins bind LPS in circulation due to Leaky Gut
- This reduces free endotoxin interaction with immune cells
- The liver then clears the LPS–lipoprotein complexes

Oxidation happens downstream

- Chronic LPS exposure → chronic inflammation
- Inflammation → oxidative stress
- Oxidative stress → **oxidized LDL**

Oxidized lipids:

- Disrupt BBB integrity
- Prime microglia
- Increase neuroinflammatory tone
- (Not just a cardiovascular disruptor)

Lipid oxidation products alter:

- Dopamine signaling
- Mitochondrial membranes
- Synaptic fluidity

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

INFLAMMATION AND CAUSES OF DEATH

Brain Behav Immun Health. 2022 Apr 22;22:100462. doi: 10.1016/j.bbih.2022.100462

Infection and inflammation: New perspectives on Alzheimer's disease

Heather E Whitson [a,]

Once a fringe theory, the possibility of microbial involvement in AD has garnered increasing research interest in recent decades. The number of publications on this topic rose from 601 papers between 1991 and 2000 to 3138 papers between 2011 and 2020, according to a PubMed search for "Alzheimer AND infection."

Rudolph E. Tanzi, PhD
Joseph P. and Rose F. Kennedy Professor of Neurology, Harvard Medical School
Vice-Chair, Neurology Department, Massachusetts General Hospital
Director, Genetics and Aging Research Unit, Massachusetts General Hospital

THE PRIMARY EPIGENETIC TRIPWIRE OF ALZHEIMER'S GENE SNP's IS SYSTEMIC INFLAMMATION.
A PRIMARY MECHANISM OF SYSTEMIC INFLAMMATION IS LPS ENDOTOXEMIA DUE TO INTESTINAL PERMEABILITY. AI AND STANDARD AMERICAN DIET (HIGH SUGAR/CARBS, ULTRA PROCESSED FOODS, SEED OILS ETC)

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

INFLAMMATION AND CAUSES OF DEATH

Photochem Photobiol. 2013 Jan-Feb;89(1):199-207. doi: 10.1111/j.1751-1097.2012.01225.x. Epub 2012 Sep 18.

Modulation of lipopolysaccharide-induced NF-κB signaling pathway by 635 nm irradiation via heat shock protein 27 in human gingival fibroblast cells.

Lim W1, Kim J, Kim S, Karna S, Won J, Jeon SM, Kim SY, Choi Y, Choi H, Kim O.

Heat shock protein-27 (HSP27) is a member of the small HSP family which has been linked to the nuclear factor-kappa B (NF-κB) signaling pathway regulating inflammatory responses. Clinical reports have suggested that low-level light therapy/laser irradiation (LLLT) could be an effective alternative treatment to relieve inflammation during bacterial infection associated with periodontal disease. However, it remains unclear how light irradiation can modulate the NF-κB signaling pathway. We examined whether or not 635 nm irradiation could lead to a modulation of the NF-kB signaling pathway in HSP27-silenced cells and analyzed the functional cross-talk between these factors in NF-κB activation. **The results showed that *635 nm* irradiation led to a decrease in the HSP27 phosphorylation, reactive oxygen species (ROS) generation, I-κB kinase (IKK)/inhibitor of κB (IκB)/NF-κB phosphorylation, NF-κB p65 translocation and a subsequent decrease in the COX-1/2 expression and prostaglandin (PGE(2))** release in lipopolysaccharide(LPS)-induced human gingival fibroblast cells (hGFs). However, in HSP27-silenced hGFs, no obvious changes were observed in ROS generation, IKK/IκB/NF-κB phosphorylation, NF-κB p65 translocation, nor in COX-1/2 expression, or PGE(2) release. This could be a mechanism by which ***635 nm irradiation modulates LPS-induced NF-κB signaling pathway via HSP27 in inflammation***. Thus, HSP27 may play a role in regulating the anti-inflammatory response of LLLT.

INFLAMMATION AND LPS

Reduction in LPS Bioactivity: **The 405 nm violet phototreatment was shown to inhibit LPS bioactivity by up to 42%, as measured by the reduction in nitrite formation, which is an indicator of inflammatory response in macrophages**

Effects of photodynamic laser and violet-blue led irradiation on Staphylococcus aureus biofilm and Escherichia coli lipopolysaccharide attached to moderately rough titanium surface: in vitro study

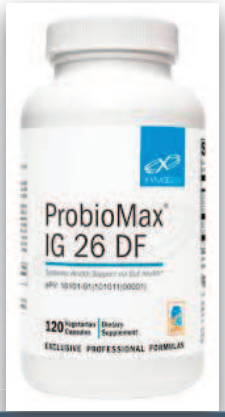
Published Mar 9, 2017 · M. Giannelli, G. Landini, Fabrizio Materassi+7 more
Lasers in Medical Science

ERCHONIA Healing Light, Perfected.
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.
RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

INFLAMMATION AND CAUSES OF DEATH

CLINICAL GUIDELINES FOR LPS INDUCED INFLAMMATION REDUCTION AND ENDOTOXEMIA:
1. FIX THE LEAKY GUT. (GI BALANCE XYMOGEN, REPAIR AND CLEAR APEX, TOTAL LEAKY GUT NUTRIWEST). RUN TESTS LIKE VIBRANT WHEAT ZOOMER AND FOOD SENSITIVITIES TESTS
2. BRAIN GUT AXIS. VAGAL NERVE STIMULATION PROTOCOL, PREFRONTAL INHIBITION/GABA
3. IMMUNOGLOBULIN THERAPIES (IGG/IGY, IVIG, COLOSTRUM)
4. LOW LEVEL LASER PROTOCOLS (FOCUS ON MASTER BRAIN, VAGAL/BRAIN-GUT AXIS, AND GLOBAL BODY PROTOCOLS
5. TARGET 3 MONTHS OF GUT REPAIR, FOOD ELIMINATION, SUPPLEMENT SUPPORT AND LLLT
6. AVOID GLUTEN/WESTERN WHEAT



Front. Nutr., 21 June 2018
Sec. Nutritional Immunology
Volume 5 - 2018 | https://doi.org/10.3389/fnut.2018.00052

Effects of Bovine Immunoglobulins on Immune Function, Allergy, and Infection

.2018 Apr;49(4):474-482. doi: 10.1097/SHK.0000000000000937.

Protection Against Lipopolysaccharide-Induced Immunosuppression by IgG and IgM

Christiana Kyvelidou 1, Dimitris Sotiriou, Ioanna Zerva, Irene Athanassakis

RESTORING BRAIN —HEALTH—

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

Supplement Facts

Serving Size: 2 Scoops (about 13.7 g)
Servings Per Container: 14

	Amount Per Serving	%DV
Calories	40	
Total Carbohydrate	7 g	3%†
Dietary Fiber	2 g	7%
Total Sugars	1 g	**
Zinc (as zinc L-carnosine)	32.25 mg	293%
L-Glutamine	4 g	**
Slippery Elm (*Ulmus fulva*)(bark)	2 g	**
N-Acetyl-D-Glucosamine	2 g	**
Aloe Vera 200:1 Extract (*Aloe barbadensis*)(leaf gel)	600 mg	**
Porcine Gastric Mucin	400 mg	**
Green Kiwifruit (*Actinidia deliciosa*)(fruit)(62,500 AU actinidin)[S1]	400 mg	**
Marshmallow (*Althaea officinalis*)(root)	400 mg	**
Licorice Extract (*Glycyrrhiza glabra*)(root)(3.5% glabridin)[S2]	300 mg	**
Quercetin (as quercetin dihydrate)	200 mg	**
Prune (*Prunus domestica*)(fruit)	200 mg	**
Chamomile Extract (*Matricaria recutita*)(flowering head)(1% apigenin-7-glucoside)	200 mg	**
Cat's Claw (*Uncaria tomentosa*)(bark)	200 mg	**
Okra (*Hibiscus esculentus*)(pod)	200 mg	**

† Percent Daily Values are based on a 2,000 calorie diet.
** Daily Value (DV) not established.

Other Ingredients: Guar gum, natural flavors, and stevia leaf extract.

EXAMPLE OF A BROAD SPECTRUM GI AND BARRIER SYSTEM REPAIR PRODUCT

NOTE: There is No Evidence of Glutamate Toxicity with Oral Doses
Clinical studies using **5–30 g/day of glutamine** in gut healing, cancer, and trauma patients **do not show increased neurotoxicity.**

In fact, glutamine often reduces inflammation and enhances gut–brain axis stability.

Reference:
Cury-Boaventura, M. F., et al. (2006). Effect of oral glutamine on gut permeability and plasma cytokines in severely ill patients. **JPEN Journal of Parenteral and Enteral Nutrition**, 30(5), 480–486. https://07106030005480

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

TURMERIC
BCM-95 VS PIPERINE
DOSE: BASELINE 500 MG DAILY
CHRONIC INFLAMMATORY CONDITIONS AND
NEURODEGENERATIVE CONDITIONS
MIN 1000 MG DAILY (500 MG BID OR TID)
BEST TAKEN WITH FOOD FOR ABSORPTION
INHIBITS NF-KB, IL-6, TNF-ALPHA

OMEGA 3 FATTY ACIDS
MIN 2G, TARGET 3G/DAY
(2G EPA/ 1G DHA)
WITH MEALS
INHIBITS PGE2
SPM/PRMS?

RESVERATROL (ACTIVATED TRANS-RESVERO)
MINIMUM 200 MG BUT TARGET UP TO 1,000 MG (500 MG BID)
SYNERGISTIC EFFECT WHEN COMBINED WITH TURMERIC
ALSO GOOD FOR BARRIERS

1. Choi, J. Y., Kim, Y. H., & Lee, J. (2010).
"Synergistic neuroprotective effects of resveratrol and curcumin against oxidative stress in rat primary cortical neurons."
Journal of Medicinal Food, 13(3), 608–614.
https://doi.org/10.1089/jmf.2009.1271
• Conclusion: Co-treatment more effectively reduced markers of oxidative stress and neuronal damage compared to single treatment.

2. Wang, H., Liu, Y., Briesacher, J. A., et al. (2018).
"Synergistic anti-inflammatory effects of curcumin and resveratrol in astrocyte cultures."
Neurochemical Research, *43*(5), 1025–1033.
• Demonstrated that the combination significantly reduced proinflammatory cytokine release (TNF-α, IL-1β) from activated glial cells more effectively than either agent alone.

Sample of low level laser under different wavelengths and inflammation reduction

- **Albertini et al., 2007 (Inflammation Research)** — Carrageenan rat-paw model → LLLT ↓ **COX-2 mRNA** in inflamed tissue (early evidence that PBM downshifts the COX-2→PGE_2 axis). SpringerLink
- **Prianti Jr. et al., 2014 (J Biophotonics/PMID 24532118)** — Carrageenan inflammation (paw) with CNS readouts → single exposure PBM ↓ **COX-2 mRNA** in subplantar tissue **and brain**; COX-1 unchanged. PubMed
- **Torres-Silva et al., 2015 (Lasers Med Sci)** — Rat collagenase tendinitis, 660 nm → ↓ **COX-2, TNF-α, IL-6** (with compensatory ↑ **IL-10**); gene-level modulation consistent with analgesia/repair. PubMed+1
- **Chen et al., 2021 (Photonics)** — Periodontal models; **630–635 nm** PBM → ↓ **PGE_2** release in human gingival fibroblasts; animal periodontitis shows reduced inflammation and faster healing. MDPI
- **Tomazoni et al., 2021 (Lasers Surg Med)** — Chronic pain patients; multi-site PBMT (clinical RCT) → acute systemic shifts in inflammatory mediators including ↓ **PGE_2** with pain improvement. Wiley Online Library
- **Sammons et al., 2023 (J Clin Med)** — Human intervertebral disc cell model; **405 nm** violet vs 532/650 → all wavelengths ↓ **IL-8**, but **only 405 nm** significantly ↓ **IL-6** (wavelength-specific cytokine control). PMC
- **Tamimi et al., 2022 (Sci Rep, open-access PMC)** — Human adipose-derived MSCs, **532 nm** green → RNA-seq/RT-PCR show broad **down-regulation of inflammatory genes**; supports green-light anti-inflammation in regenerative contexts. PMC
- **Shamloo et al., 2023 (Biomedicines)** — Mouse systemic inflammation → PBM induces **anti-inflammatory serum profile** (e.g., ↑ **IL-10**) and mitigates tissue inflammatory markers. PMC
- **Hamblin, 2017 (AIMS Biophysics review)** — Mechanistic synthesis: PBM modulates **NF-κB/MAPK**, brief ROS/NO/ATP signal, and downstream cytokines (↓ **IL-6/IL-1β/TNF-α**; context- and dose-dependent; 405 nm uniquely noted for **IL-6** suppression in some models). AIMS Press
- **Tian et al., 2023 (Lasers Med Sci)** — Macrophages in vitro → PBM promotes **M1→M2 polarization** via **PI3K/AKT/mTOR**, aligning with reduced pro-inflammatory mediator expression. PubMed
- **Woo et al., 2023/2024 (Oral-facial cell lines & review)** — **630 nm LED**, ~30 J: shifts macrophages toward **M2**, reducing CCR2/**IL-1β** and favoring **IL-1RA**; broader review affirms PBM-driven macrophage reprogramming. ScienceDirect+1
- **Ma et al., 2022 (Frontiers Immunol.)** — Spinal cord injury mice → PBM inhibits neurotoxic macrophage/microglia polarization via **Notch1–HIF-1α/NF-κB**, lowering inflammatory mediator release and improving function. Frontiers
- **Cardoso et al., 2022 (Frontiers Neurosci.)** — Narrative review across TBI/brain models: PBM ↓ **pro-inflammatory cytokines** and microglial activation; supports CNS applicability. Frontiers
- **Zhang et al., 2023 (Cells review)** — Broad PBM overview: confirms **COX-2 inhibition** and cytokine modulation across tissues; collates parameters and mechanistic pathways relevant to dosing. PMC
- **Fernandes et al., 2015 (J Formos Med Assoc)** — M1 J774 macrophages, **660 nm** → ↓ **TNF-α, iNOS, COX-2**; (note: IL-6 rose under these conditions—dose/model nuance matters). ScienceDirect
- **Pasternak-Mnich et al., 2024 (Sci Rep)** — Human PBMCs, dual NIR (808/905 nm): framework paper highlighting **parameter-specific** cytokine responses and need for standardized protocols (supports biphasic/dose-sensitive effects). Nature+1

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Red (≈630–660 nm): **Strongest and most replicated evidence for ↓ COX-2 → ↓ PGE_2, plus broad cytokine down-modulation (IL-1β/ IL-6/TNF-α) and macrophage M2 shift; CNS and peripheral data.** PubMed+4SpringerLink+4PubMed+4

Violet (≈405 nm): **Distinct IL-6 suppression (with shared IL-8 reduction) in disc/periodontal cellular models—useful where IL-6 is a dominant driver.** PMC

Green (≈520-532 nm): **RNA-seq/RT-PCR evidence for inflammatory gene down-regulation in hADMSCs; promising for regenerative/soft-tissue settings.** PMC

INFLAMMATION AND CAUSES OF DEATH

SUMMARY:

- INTESTINAL PERMEABILITY (LEAKY GUT) ALLOWS FOR BACTERIA TO INFILTRATE THE BLOOD STREAM TRIGGERING IMMUNE INFLAMMATORY RESPONSES TO LPS ON THE CELL WALLS OF THE GRAM NEGATIVE BACTERIA
- SYSTEMIC INFLAMMATION IS THE MAJOR MECHANISM ALMOST EVERY MAJOR CAUSE OF DEATH, HEART/CARDIOVASCULAR DISEASE AND CAN ALSO TRIGGER GENETIC RISK FACTORS IN NEURODEGENERATION LIKE ALZHEIMER'S
- LEAKY GUT CAN BE CAUSED BY MANY FACTORS BUT MAJOR ONES TO CONSIDER ARE WESTERN WHEAT/GLUTEN, TBI/POST-CONCUSSION SYNDROME, CHRONIC STRESS, AND POOR AUTONOMIC REGULATION OF PARASYMPATHETIC TONE
- CARDIOVASCULAR MARKERS MAY BE A PROTECTIVE REACTION TO THIS PROCESS
- FIX THE BARRIER SYSTEMS USING GUT REPAIR PROTOCOLS, FOOD ELIMINATION, HEALTHY IMMUNE NEUTRAL DIETS, FUNCTIONAL NEUROLOGY/BRAINSTEM/AUTONOMIC REGULATION
- DECREASE LOCAL AND SYSTEMIC INFLAMMATION AND PROMOTE IMMUNE REGULATION

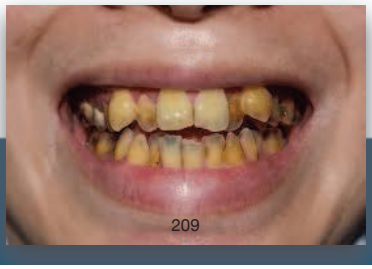
LOW LEVEL LASER THERAPY HAS THE POTENTIAL TO HELP EVERY STAGE OF THIS PROCESS!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE ORAL TROJAN HORSE

Oral diseases may be a direct cause of infectious disease based neurodegeneration. Oral bacteria like porphyromonas gingivalis and strep mutans can lead to gut barrier destruction and systemic inflammation that can damage the BBB and trigger amyloid beta, inflammatory immune responses and more.

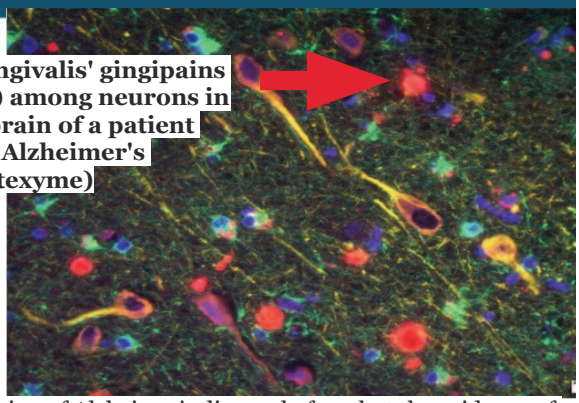


RESTORING BRAIN —HEALTH—

ORAL PATHOGENS AND ALZHEIMERS

Porphyromonas gingivalis in Alzheimer's disease brains: Evidence for disease causation and treatment with small-molecule inhibitors

In a new paper led by senior author Jan Potempa, a microbiologist from the University of Louisville, researchers report the discovery of *Porphyromonas gingivalis* – the pathogen behind chronic periodontitis (aka gum disease) – in the brains of deceased Alzheimer's patients.



P. gingivalis' gingipains (red) among neurons in the brain of a patient with Alzheimer's (Cortexyme)

"Infectious agents have been implicated in the development and progression of Alzheimer's disease before, but the evidence of causation hasn't been convincing," Dominy says.

"Now, for the first time, we have solid evidence connecting the intracellular, Gram-negative pathogen, *P. gingivalis*, and Alzheimer's pathogenesis."

In addition, ***the team identified toxic enzymes called gingipains secreted by the bacteria in the brains of Alzheimer's patients, which correlated with two separate markers of the disease: the tau protein, and a protein tag called ubiquitin.***

But even more compellingly, the team identified these toxic gingipains in the brains of deceased people who were never diagnosed with Alzheimer's.

That's important, because while *P. gingivalis* and the disease have been linked before, it's never been known – to put it simply – whether gum disease causes Alzheimers, or whether dementia leads to poor oral care.

The fact that low levels of gingipains were evident even in people who were never diagnosed with Alzheimer's could be a smoking gun – suggesting they might have developed the condition if they had lived longer.

"Our identification of gingipain antigens in the brains of individuals with AD and also with AD pathology but no diagnosis of dementia argues that brain infection with *P. gingivalis* is not a result of poor dental care following the onset of dementia or a consequence of late-stage disease, but is an early event that can explain the pathology found in middle-aged individuals before cognitive decline," the authors write in their paper.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ORAL PATHOGENS AND ALZHEIMERS

J Oral Microbiol. 2016 Nov 22;8:33029. doi: 10.3402/jom.v8.33029. eCollection 2016.

Porphyromonas gingivalis suppresses adaptive immunity in periodontitis, atherosclerosis, and Alzheimer's disease.

Olsen I1, Taubman MA2,3, Singhrao SK4.

Author information Abstract

Porphyromonas gingivalis, a keystone pathogen in chronic periodontitis, has been found to associate with remote body organ inflammatory pathologies, including atherosclerosis and Alzheimer's disease (AD). Although *P. gingivalis* has a plethora of virulence factors, much of its pathogenicity is surprisingly related to the overall immunosuppression of the host. This review focuses on ***P. gingivalis* aiding suppression of the host's adaptive immune system involving manipulation of cellular immunological responses, specifically T cells and B cells in periodontitis and related conditions. In periodontitis, this bacterium inhibits the synthesis of IL-2 and increases humoral responses**. This reduces the inflammatory responses related to T- and B-cell activation, and subsequent IFN-γ secretion by a subset of T cells. The T cells further suppress upregulation of programmed cell death-1 (PD-1)-receptor on CD+cells and its ligand PD-L1 on CD11b+-subset of T cells. IL-2 downregulates genes regulated by immune response and induces a cytokine pattern in which the Th17 lineage is favored, thereby modulating the Th17/T-regulatory cell (Treg) imbalance. The suppression of IFN-γ-stimulated release of interferon-inducible protein-10 (IP-10) chemokine ligands [ITAC (CXCL11) and Mig (CXCL9)] by *P. gingivalis* capsular serotypes triggers distinct T cell responses and contributes to local immune evasion by release of its outer membrane vesicles. In atherosclerosis, ***P. gingivalis* reduces Tregs, transforms growth factor beta-1 (TGFβ-1), and causes imbalance in the Th17 lineage of the Treg population**. ***In AD, P. gingivalis may affect the blood-brain barrier permeability and inhibit local IFN-γ response by preventing entry of immune cells into the brain. The scarcity of adaptive immune cells in AD neuropathology implies P. gingivalis infection of the brain likely causing impaired clearance of insoluble amyloid and inducing immunosuppression***. By the effective manipulation of the armory of adaptive immune suppression through a plethora of virulence factors, *P. gingivalis* may act as a keystone organism in periodontitis and in related systemic diseases and other remote body inflammatory pathologies.

ORAL PATHOGENS AND ALZHEIMERS

alzheimer's association | Diagnosis, Assessment & Disease Monitoring

Periodontal dysbiosis associates with reduced CSF Aβ42 in cognitively normal elderly 12 April 2021

Angela R. Kamer Smruti Pushalkar

Discussion

We show a selective relationship between periodontal disease bacterial dysbiosis and CSF biomarkers of amyloidosis, but not for tau. Further modeling is needed to establish the direct link between oral bacteria and Aβ.

Harmful oral bacteria were identified as *Prevotella, Porphyromonas,* and *Fretibacterium.*

"To our knowledge, this is the first report of an association between subgingival periodontal bacteria and CSF biomarkers of AD pathology in cognitively normal elderly people. We found that subgingival periodontal dysbiosis characterized by increases in periodontal-associated bacteria and decreases in health-related bacteria associated with reduced CSF Aβ42 but not with CSF P-tau," the authors wrote in the paper.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ORAL PATHOGENS AND ALZHEIMERS

Curr Med Chem. 2018 Mar 8. doi: 10.2174/0929867325666180309103645. [Epub ahead of print]

Linking Antimicrobial Potential of Natural Products Derived from Aquatic Organisms and Microbes Involved in Alzheimer's Disease - A Review.

Stojkovic D1, Kostic M1, Smiljkovic M1, Aleksic M2, Vasiljevic P2, Nikolic M1, Sokovic M1.

Author information Abstract

The following review is oriented towards microbes linked to Alzheimer's disease (AD) and

Major group of microbes linked to AD include bacteria: Chlamydia pneumoniae, Helicobacter pylori, Porphyromonas gingivalis, Fusobacterium nucleatum, Prevotella intermedia, Actinomyces naeslundii, spirochete group; fungi: Candida sp., Cryptococcus sp., Saccharomyces sp., Malassezia sp., Botrytis sp., and viruses: herpes simplex virus type 1 (HSV-1), Human cytomegalovirus (CMV), hepatitis C virus (HCV).

ORAL PATHOGENS AND ALZHEIMERS

Arch Oral Biol. 1992 Nov;37(11):883-7.

Sensitization of oral bacteria in biofilms to killing by light from a low-power laser.

Dobson J1, Wilson M.

Author information

Abstract

Biofilms of Streptococcus sanguis, Porphyromonas gingivalis, Fusobacterium nucleatum and Actinobacillus actinomycetemcomitans were prepared on the surfaces of agar plates and a number of compounds were screened for their ability to sensitize bacteria in these biofilms to **killing by light from a 7.3 mW Helium/Neon (He/Ne) laser.** Toluidine blue O and methylene blue enabled detectable killing of all four target organisms after exposure to He/Ne light for 30 s.

These findings suggest that lethal photosensitization may be an effective means of eliminating periodontopathogenic bacteria from dental plaque.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ORAL PATHOGENS AND ALZHEIMERS

Photodiagnosis Photodyn Ther. 2016 Mar;13:344-6. doi: 10.1016/j.pdpdt.2015.07.173. Epub 2015 Jul 26.

Lethal photosensitisation of Prevotellaceae under anaerobic conditions by their endogenous porphyrins.

Hope CK1, Strother M2, Creber HK3, Higham SM4.
Author information

The oral anaerobes, Prevotella intermedia and P. nigrescens are known to be photosensitive…

The Prevotellaceae were exposed to two 405 nm light sources for different exposure times…

Lethal photosensitivity of Prevotella Intermedia and Nigrescens was demonstrated in two species of Prevotella under anaerobic conditions.

RESTORING BRAIN —HEALTH—

Violet-Blue Light Arrays at 405 Nanometers Exert Enhanced Antimicrobial Activity for Photodisinfection of Monomicrobial Nosocomial Biofilms

Appl Environ Microbiol. 2019 Nov 1; 85(21): e01346-19.

Conclusion (Highlights):

-Optimal antimicrobial effects of the blue-violet spectrum are obtained with 405 nm

-Wavelengths outside of this range (375nm, 420nm, and 450nm) are most likely not absorbed by endogenous porphyrins and are therefore unlikely to generate ROS through Soret band absorption.

-It was interesting to observe that the majority of the bacteria irradiated with 375-nm and 450-nm LEDs exhibited increases in biofilm seeding. This seems logical, since these wavelengths are unlikely to be absorbed by the Soret band contained within the porphyrins, and hence, the antimicrobial effects seen with 395 and 405nm are likely absent or reduced. The growth and increased seeding of these biofilms may be explained through (non-Soret band) absorption of the light, resulting in the generation of heat and the stimulation of bacterial growth owing to heat. Indeed, heating may also produce small amounts of ROS, which may also be beneficial for bacterial growth (30).

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REFERENCES FOR ORAL PATHOGENS, NEURODEGENERATION AND LOW LEVEL LASER

Dominy SS et al. *Sci Adv*. 2019;5:eau3333 — *P. gingivalis* and gingipains in Alzheimer's brains.

Wu H et al. *Front Aging Neurosci*. 2022;12:912709 — *F. nucleatum* worsens AD pathology in mice.

Miklossy J. *Front Aging Neurosci*. 2015;7:46 — Spirochetes and dementia/AD hallmarks.

Li X et al. *J Clin Med*. 2022;11:6518 — Periodontitis–neuroinflammation link review.

Fukui M et al. *Lasers Surg Med*. 2008;40:494–500 — 405 nm violet light suppresses *P. gingivalis*.

Nakada T et al. *BPB Reports*. 2022;6:81–87 — 405 nm violet reduces *A. actinomycetemcomitans* without dye.

Perumal MKK et al. *Lasers Med Sci*. 2024 — 660 nm methylene blue aPDT reduces periodontal pathogens.

Teerakapong A et al. *Photodiagn Photodyn Ther*. 2017;20: — 532 nm (green) + erythrosine/C3G disrupt *P. gingivalis* biofilms.

Martu MA et al. *Int J Mol Sci*. 2023;24:1898 — Periodontal laser/aPDT overview, biofilm efficacy.

Kikuchi T et al. *Int J Mol Sci*. 2015;16:24111 — Low-power lasers + dyes (MB/TBO/ICG/RB) effective vs spirochetes.

Goulart RC et al. *Photomed Laser Surg*. 2010;28:S85–S90 — aPDT inactivates *A. actinomycetemcomitans*.

ERCHONIA
Healing Light, Perfected.

217

EVRL FOR ORAL PATHOGENS

ERCHONIA eCommunity Education

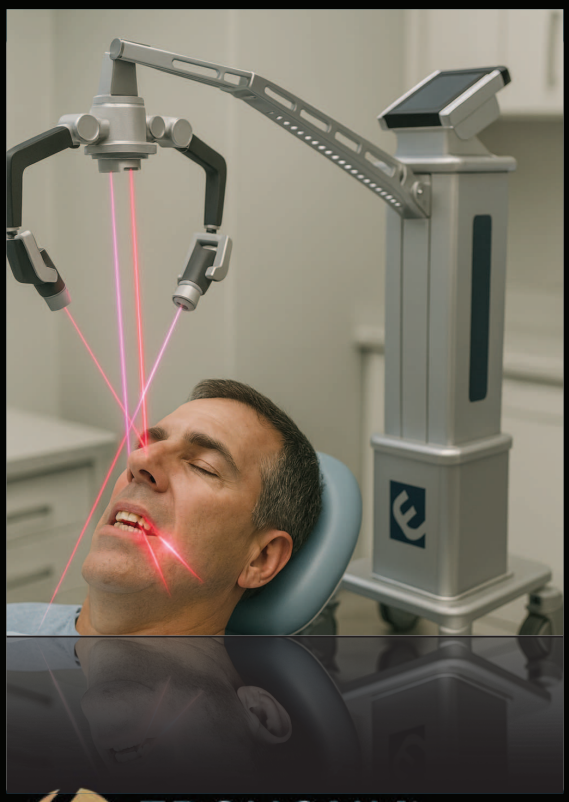
EVRL 405 NM LASER

"BACTERIA SETTING" 20-664-787-1560
4 MINUTES
1 MINUTE NASAL, 1 MINUTE THROAT
1 MINUTE MAXILLARY GUM LINE
1 MINUTE MANDIBULAR GUM LINE

OTHER CONSIDERATIONS:
REGULAR FLOSSING, BRUSHING AND DENTAL HYGIENE
LOW SUGAR/CARB DIET
"OIL PULLING" WITH COCONUT OIL
ARGENTYN SILVER

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



ERCHONIA
Healing Light, Perfected.

LASERS AND LONG LASTING SMILES:
THE ULTIMATE DENTAL STACK

PROGRAM YOUR “MASTER DENTAL” SETTING ON AN FX 405 LASER TO COMBINE BRAIN FREQUENCIES WITH ANTI-MICROBIAL

10-20
40-664
10-787
40-1560

10 MINUTES OVER THE HEAD AND FACE AREA DURING YOUR PROCEDURES

CONSIDER UTILIZING YOUR ERCHONIA LASER FOR ALL PRE- , DURING AND POST- DENTAL PROCEDURES:

- -PRE-CONDITIONING THE TISSUES. IE. LESS OXIDATIVE DAMAGE
- -IMPROVE STERILE FIELD
- -PROMOTE HEALING AND FASTER RECOVERY TIMES
- -REDUCE INFLAMMATION AND EDEMA
- -ENHANCE BONE REGENERATION
- -IMPROVES MICROCIRCULATION AND OXYGEN DELIVERY
- -MINIMIZE PAIN AND POST-PROCEDURAL RECOVERY MEDS
- -PROMOTE NERVE REPAIR AND REDUCE PARESTHESIA RISK
- -IMPROVE PATIENT EXPERIENCE DUE TO THE AFOREMENTIONED FACTORS
- -REDUCE INFECTION RISK. ALTERNATIVE TO ANTIBIOTICS???

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GLP-1s: THE NEXT GREAT MEDICAL MYTH?!

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

1. Most Patients Regain the Weight When They Stop

GLP-1 drugs work primarily by suppressing appetite. When the medication is discontinued, appetite and metabolic signaling often return to baseline.

This is why many experts now consider GLP-1 drugs **chronic medications similar to statins or blood-pressure drugs**. YOU ARE STUCK ON THEM INDEFINITELY!

Weight Maintenance after GLP-1 RA Withdrawal Exposes Critical Research Gaps

Can GLP-1-influenced weight loss be maintained?

Dec 31, 2025

Diabetes Obes Metab. 2022 May 19;24(8):1553–1564. doi: 10.1111/dom.14725
Weight regain and cardiometabolic effects after withdrawal of semaglutide: The STEP 1 trial extension
John P H Wilding [1]

Articles
Trajectory of weight regain after cessation of GLP-1 receptor agonists: a systematic review and nonlinear meta-regression

Brajan Budini a e

ELSEVIER

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

2. They Are Very Expensive and Often Required Long-Term

GLP-1 drugs can cost **$900–$1,400 per month in the U.S. without insurance**.

Because weight regain occurs when the medication stops, many patients must stay on them long-term.

Patients may face
$10,000–$15,000 per year in medication costs

ERCHONIA
Healing Light, Perfected.
AVERAGE ZERONA PROGRAM
$1500-$3000
RESTORING BRAIN
—HEALTH—

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

3. A Large Portion of Weight Loss Can Be Lean Mass (Muscle)

Weight loss from GLP-1 therapy is not purely fat.

UPWARDS OF 40% OF WEIGHT LOSS IS LEAN MUSCLE MASS!

J Endocr Soc. 2021 May 3;5(Suppl 1):A16–A17. doi: 10.1210/jendso/bvab048.030
Impact of Semaglutide on Body Composition in Adults With Overweight or Obesity: Exploratory Analysis of the STEP 1 Study
John P H Wilding [1]

Beyond fat: Does semaglutide affect lean mass?
Tannaz Jamialahmadi a b,
Clinical Nutrition
Volume 44, January 2025, Pages 104-108

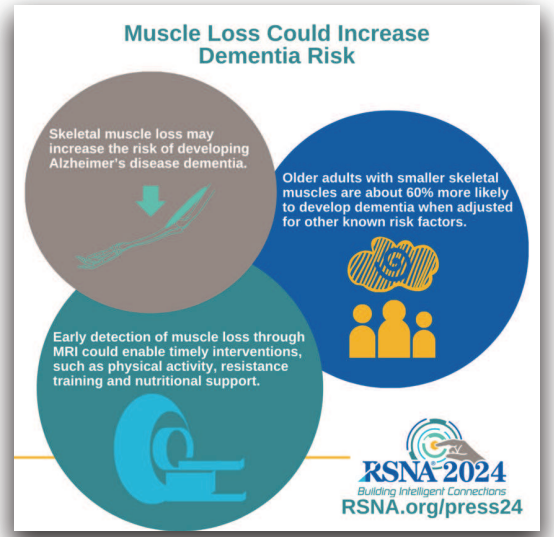
Changes in lean body mass with glucagon-like peptide-1-based therapies and mitigation strategies
Ian J. Neeland MD, Jennifer Linge PhD, Andreas L. Birkenfeld MD
First published: 27 June 2024

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS



- OLDER ADULTS WITH SMALLER MUSCLES ARE ABOUT 60% MORE LIKELY TO DEVELOP DEMENTIA WHEN ADJUSTED FOR OTHER KNOW RISK FACTORS
- PEOPLE WITH SARCOPENIA (LOSS OF SKELETAL MUSCLE MASS AND STRENGTH) MORE THAN DOUBLES THE RISK OF COGNITIVE IMPAIRMENT
- LOW MUSCLE MASS IS A DIRECT PREDICTOR OF COGNITIVE DECLINE
- SKELETAL MUSCLE IS NOW RECOGNIZED AS AN ENDOCRINE ORGAN THAT PROTECTS THE BRAIN! (REGULATES INSULIN SIGNALING, MITOCHONDRIAL FUNCTION, REDUCES INFLAMMATION AND RELEASES GROWTH FACTORS FOR NEUROPLASTICITY!)

"Loss of muscle mass is one of the strongest predictors of frailty, disability, and cognitive decline with aging."

Skeletal Muscle Health and Cognitive Function: A Narrative Review

by Sophia X. Sui 1,*,Lana J. Williams 1,Kara L. Holloway-Kew 1,Natalie K. Hyde 1 andJulie A. Pasco 1,2,3,4

Int. J. Mol. Sci. **2021**, *22*(1),

Archives of Gerontology and Geriatrics
Volume 117, February 2024, 105160

Sarcopenia using muscle mass prediction model and cognitive impairment: A longitudinal analysis from the English longitudinal study on ageing

Laura Maniscalco a

ELSEVIER

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

4. They Significantly Slow Digestion and Can Cause GI Disorders

GLP-1 drugs are associated with **gastroparesis, bowel obstruction, pancreatitis, and biliary disease**.

Patients using GLP-1 drugs were reported to be **9.1× more likely to develop pancreatitis and 3.7× more likely to develop gastroparesis compared with some other weight-loss medications**.

These drugs may worsen conditions such as:

- **SIBO/SIFO**
- **IBS**
- **existing GI motility disorders**

≈24,499 emergency-department visits in the U.S. between 2022–2023

U.S. Emergency Department Visits Attributed by Clinicians to Semaglutide Adverse Events, 2022–2023 FREE

Authors: Maribeth C. Lovegrove, MPH,

- JAMA
- Vol. 330, No. 18

Risk of Gastrointestinal Adverse Events Associated With Glucagon-Like Peptide-1 Receptor Agonists for Weight Loss

- Mohit Sodhi, MSc[1];

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

5. Nutrient Deficiencies Can Develop Due to Appetite Suppression and Malabsorption

Because appetite drops significantly, some patients consume much less protein and micronutrients. Also, with slowed GI function and increased risk of dysbiosis and other GI related matters, abnormal breakdown and absorption of foods, vitamins and minerals is common.

The systematic review included data from 480,825 adults with obesity who were taking GLP-1 medications. One study found that 12.7% of participants had a nutritional deficiency after six months on GLP-1. **Vitamin D deficiency was the most common, followed by low iron and B-complex vitamins.2**

Scott Butsch W, Sulo S, Chang AT, et al. Nutritional deficiencies and muscle loss in adults with type 2 diabetes using GLP-1 receptor agonists: A retrospective observational study. Obes Pillars. 2025;15:100186. Published 2025 Jun 10. doi:10.1016/j.obpill.2025.100186

1

ERCHONIA
Healing Light, Perfected.

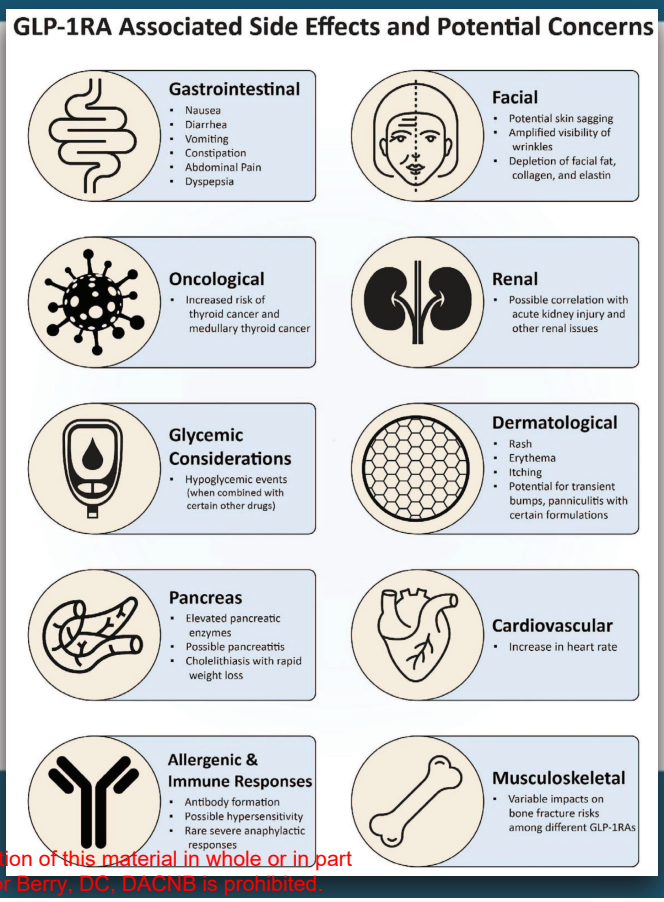
RESTORING BRAIN HEALTH

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

6. Long-Term Metabolic and Skeletal Effects Are Still Being Studied

Although the drugs are effective, long-term physiologic effects are still under investigation.

A large study of ~150,000 patients found 30% higher osteoporosis risk and over 150% higher osteomalacia risk among GLP-1 users.



ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

7:

- ~162 deaths in the U.S. have been reported in association with GLP-1 drugs (Ozempic, Wegovy, Mounjaro, etc.) since ~2018
- Of those, ~94 involved semaglutide (Wegovy/Ozempic)

SOURCE:

FDA Adverse Event Monitoring System (AEMS) Public Dashboard

[Formerly FDA Adverse Event Reporting System (FAERS)]

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

HMB

New miracle supplement found to boost memory and muscle strength in old age

The study found HMB significantly enhanced the effect of resistance training on muscle strength, physical performance, and muscle quality. It also reduced inflammatory markers.

Studies in mice with Alzheimer's disease have shown that **HMB successfully reduces plaques and increases factors for neuronal growth to protect learning and memory.**

In a mice study, published in the journal *Cell Reports*, researchers examined whether after oral administration, HMB could enter into the brain. **Three days after oral treatment, HMB was detected in the hippocampus of HMB-fed mice compared with control untreated mice, indicating that HMB is capable of crossing the blood-brain barrier.** The hippocampus region of the brain is responsible for learning and memory, and leakage in the blood-brain barrier is associated with forgetfulness in ageing.

"Our study found that **after oral consumption, HMB enters into the brain to increase these beneficial proteins, restore neuronal connections and improve memory and learning in mice with Alzheimer's-like pathology, such as plaques and tangles,"** said Kalipada Pahan, Professor of Neurology at RUSH Medical College.

The journal of nutrition, health & aging Article

Effects of Beta-Hydroxy-Beta-Methylbutyrate Supplementation on Older Adults with Sarcopenia: A Randomized, Double-Blind, Placebo-Controlled Study

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GLP-1 ALTERNATIVE?

OZEMPIC (SEMAGLUTIDE) VS BERBERINE

BOTH ARE GLP-1 AGONISTS (GLUCAGON-LIKE PEPTIDE-1)
GLP-1 IS RELEASED BY THE SMALL INTESTINE IN RESPONSE TO FOOD
GLP-1 HAS 3 PRIMARY MECHANISMS OF ACTION:
1. DECREASES APPETITE VIA HYPOTHALAMIC STIMULATION OF SATIETY
2. SLOWS GASTRIC EMPTYING OF THE STOMACH
3. STIMULATES THE PANCREAS TO PROMOTE INSULIN SECRETION AND SUPPRESS GLUCAGON
(INSULIN BINDS TO GLUT 4 RECEPTORS FOR GLUCOSE ENTRANCE INTO CELLS AND PREVENTS GLUCOSE FROM GETTING TOO HIGH WHERE AS GLUCAGON PREVENTS GLUCOSE LEVELS FROM GETTING TOO LOW)
BERBERINE WILL NOT YIELD THE SAME WEIGHT LOSS EFFECTS AS OZEMPIC BUT HAS FAR FEWER SIDE EFFECTS
AND IS MUCH LESS EXPENSIVE (~$50/MONTH VS $900-1200/MONTH)

BERBERINE ALSO PROMOTES:

- **GIP INCREASE (GASTRIC INHIBITORY POLYPEPTIDE) PROMOTING FURTHER INSULIN AND SOME INHIBITION OF GASTRIC ACID (Similar to Tirzepatides)**
- **BHB (B-HYDROXYBUTYRATE) ONE OF THREE MAIN KETONES THAT KEEP THE BODY IN FAT BURNING STATE AND MUSCLE PRESERVATION**
- **AMPK (AMP-ACTIVATED PROTEIN KINASE) RELEASE FROM MOSTLY MUSCLES AND IS A MAJOR FAT BURNING ENZYME**
- **PCSK-9 INHIBITION. BREAKS DOWN LDL RECEPTORS TO REMOVE LDL FROM BLOODSTREAM**

BERBERINE ES-5
5X BIOAVAILABILITY
AS REGULAR
BERBERINE

ACTIVE FOR 8 HOURS
VS
4 HOURS REGULAR
BERBERINE

TAKE BID
1 WITH EACH
OF 2 BIGGEST
MEALS

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

GLP-1 Drugs and Brain Health

Myth vs Reality

Common Claim (Myth)	What the Research Actually Shows (Reality)
"GLP-1 drugs improve brain health."	Evidence for neuroprotection is **preliminary and mostly from animal studies or diabetic populations**, not healthy individuals. Large human trials are still ongoing.
"They may help prevent Alzheimer's."	Some observational studies suggest reduced dementia risk in diabetics taking GLP-1 drugs, but **causation has not been proven**. More controlled trials are needed.
"Weight loss from GLP-1 drugs helps the brain."	Rapid pharmacologic weight loss often includes **25–40% lean muscle loss**, which may undermine brain resilience because skeletal muscle releases brain-protective myokines.
"These drugs improve metabolic health."	They suppress appetite and slow gastric emptying, but **do not correct underlying drivers of metabolic dysfunction** such as mitochondrial dysfunction, circadian disruption, or insulin resistance.
"They are safe for long-term neurological health."	Emerging concerns include **nutrient deficiencies, optic nerve injury signals, and potential neurovascular effects**, though research is still evolving.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

7 UNCOMFORTABLE TRUTHS ABOUT GLP-1 DRUGS

Supporting Citations

Neuroprotection claims still under investigation

- Hölscher C. GLP-1 receptor agonists as treatments for neurodegenerative disease. J Neural Transmission.
- Journal of Neurology, Neurosurgery & Psychiatry review highlighting need for larger trials.

Weight regain and long-term dependence

- Wilding JPH et al. Semaglutide treatment effects. New England Journal of Medicine (STEP trials).

Lean mass loss during GLP-1 weight loss

- Body composition analyses from semaglutide trials show **significant lean mass reduction**.
- STEP trial body composition substudy.

Muscle–brain connection

- Pedersen BK & Febbraio MA. Myokines and brain health. Nature Reviews Endocrinology.

Nutrient deficiency concerns

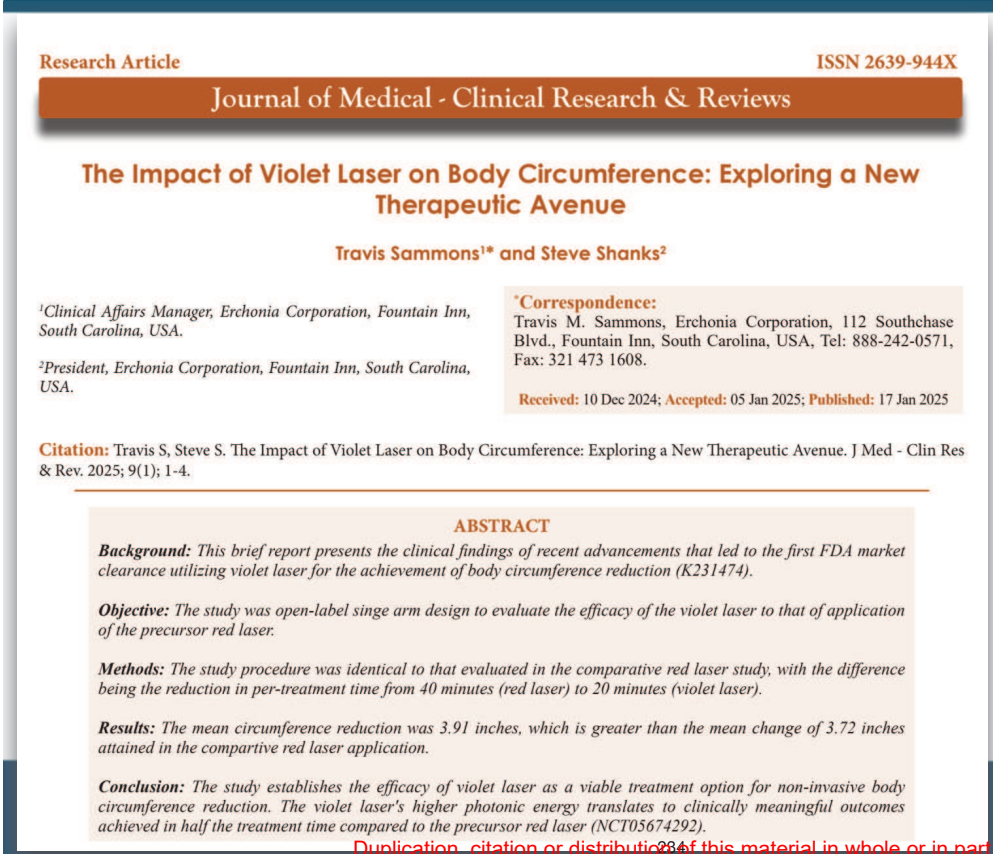
- Large population analyses showing vitamin and mineral deficiencies in GLP-1 users.

SAFER ALTERNATIVES?

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

THE NEW VZ8



Research Article ISSN 2639-944X

Journal of Medical - Clinical Research & Reviews

The Impact of Violet Laser on Body Circumference: Exploring a New Therapeutic Avenue

Travis Sammons[1]* and Steve Shanks[2]

[1]Clinical Affairs Manager, Erchonia Corporation, Fountain Inn, South Carolina, USA.

[2]President, Erchonia Corporation, Fountain Inn, South Carolina, USA.

*Correspondence:
Travis M. Sammons, Erchonia Corporation, 112 Southchase Blvd., Fountain Inn, South Carolina, USA, Tel: 888-242-0571, Fax: 321 473 1608.

Received: 10 Dec 2024; Accepted: 05 Jan 2025; Published: 17 Jan 2025

Citation: Travis S, Steve S. The Impact of Violet Laser on Body Circumference: Exploring a New Therapeutic Avenue. J Med - Clin Res & Rev. 2025; 9(1); 1-4.

ABSTRACT

Background: *This brief report presents the clinical findings of recent advancements that led to the first FDA market clearance utilizing violet laser for the achievement of body circumference reduction (K231474).*

Objective: *The study was open-label singe arm design to evaluate the efficacy of the violet laser to that of application of the precursor red laser.*

Methods: *The study procedure was identical to that evaluated in the comparative red laser study, with the difference being the reduction in per-treatment time from 40 minutes (red laser) to 20 minutes (violet laser).*

Results: *The mean circumference reduction was 3.91 inches, which is greater than the mean change of 3.72 inches attained in the compartive red laser application.*

Conclusion: *The study establishes the efficacy of violet laser as a viable treatment option for non-invasive body circumference reduction. The violet laser's higher photonic energy translates to clinically meaningful outcomes achieved in half the treatment time compared to the precursor red laser (NCT05674292).*

THE NEW ERCHONIA VZ8, MORE THAN JUST A BODY CIRCUMFERENCE LASER!

- 1. Sammons & Shanks (2025). Impact of violet laser on body circumference. J Med Clin Res & Rev, 9(1), 1-4.
- 2. Huang et al. (2023). Photobiomodulation effects of 405 nm laser on adipose tissue. Lasers Med Sci, 38(4), 795-810.
- 3. Kim & Park (2021). Mechanisms of 405 nm laser on inflammation. Int J Laser Therapy, 29(3), 233-248.
- 4. Suzuki & Nakamura (2020). 405 nm laser on neuroinflammation. Neurobiol Phototherapy, 16(4), 321-335.
- 5. Patel & Johnson (2022). Anti-inflammatory effects of violet laser. J Pain & Laser Med, 35(2), 203-221.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ADIPOSE REDUCTION

"Obesity is now the top modifiable dementia risk factor in the U.S.," reports the Journal of the American Medical Association in its latest bulletin. **Obesity in midlife is now leading to more cases of dementia than any of the other usual suspects, including depression, smoking, diabetes, high blood pressure, physical inactivity, or low levels of education. About a third of dementia cases in America are now associated with some combination of these factors, with midlife obesity, physical inactivity and low education levels leading the way. According to the medical study, midlife obesity was a contributing factor to some 18% or just under 1 in 5 cases of dementia in America.**

PUBLISHED JULY 11, 2022

RESTORING BRAIN —HEALTH—

ADIPOSE REDUCTION

Harmful Body Fat Not Only Increases Your Waistline, but Also Your Risk of Dementia

Featured Neurology Neuroscience **July 26, 2021**

Examining grey brain matter of about 28,000 people, the world first research showed that increased body fat incrementally leads to increased atrophy of grey matter in the brain and consequently higher risk of declining brain health.

"Unlocking the causal link of metabolically different adiposity subtypes with brain volumes and the risks of dementia and stroke: A Mendelian randomization study" by Anwar Mulugeta et al. *Neurobiology of Aging*

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

OBESITY AND BRAIN

This is the #1 Cause of Obesity, as Study Ties it to Poor Brain Health

Obesity is terrible for your brain—here's why.

By Ferozan Mast

Published on January 3, 2023 I 7:01 AM

According to the CDC, over 41% of US adults are obese and one in three are overweight, making obesity an epidemic. Now **new research shows obesity in childhood negatively impacts brain health, which is concerning considering 20% of American children are obese.** "We know being obese as an adult is associated with poor brain health," says researcher Simone Kaltenhauser, a post-graduate research fellow in radiology and biomedical imaging at the Yale School of Medicine in New Haven, Connecticut. "However, previous studies on children have often focused on small, specific study populations or single aspects of brain health."

Using MRI scans, the researchers studied the brain health of 5,169 children, of whom 21% were overweight, and 17.6% were obese. The results? The MRIs of the overweight and obese children showed structural brain changes, especially significant impairment to white matter.

"It is striking that these changes were visible early on during childhood," Kaltenhauser says. **Researchers also noticed thinning in the outermost layer of the brain (the cortex).**

One factor Dr. Ludwig discusses is t**he role carbohydrates play.**

"Processed, rapidly digestible carbohydrates – foods like sweetened breakfast cereals, potato chips and sugary beverages – raise our insulin level too high," he says. "This causes our fat cells to take in and store too many calories, leaving fewer available for the rest of the body. A few hours after eating a high-carb meal, the number of calories in the bloodstream plummets, so we get hungrier sooner after eating.

RESTORING BRAIN —HEALTH—

ZERONA RED AND VZ8: LOOKING GOOD VS FEELING GOOD

Brain Health via Gut–Brain Axis

- **Gut microbiome + visceral fat + inflammation** directly feed into neuroinflammatory loops → BBB permeability, microglial priming, amyloid/tau pathology.
- PBM reducing gut inflammation, pathogens, and adipokine signaling may **decrease risk of neurodegeneration and mood disorders**.
- **Evidence:**
  • Cryan JF et al., *Nat Rev Neurosci* 2019;20:703–718 — gut–brain axis in mood/ neurodegeneration.
  • Salehpour F et al., *Neurophotonics* 2021;8(4):045006 — systemic PBM effects relevant for brain function.

Deduction: Abdominal PBM is not just cosmetic — it may be a **gut–brain therapy** with implications for Alzheimer's, Parkinson's, and depression.

ERCHONIA Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ZERONA RED ADVICE: LOOKING GOOD VS FEELING GOOD

Adipose Tissue → Inflammation & Cytokine Modulation

- **Low-level red laser (Zerona 635 nm)** increases adipocyte membrane permeability → release of lipids without necrosis.
- Mobilization of stored fat reduces **visceral adiposity**, which is strongly linked to systemic inflammation and brain risk (IL-6, TNF-α, CRP).
- **Evidence**:
  • Jackson RF et al., *Obes Surg* 2010;20(6):722–729 — Zerona significantly reduced waist circumference.
  • Wajchenberg BL, *Endocr Rev* 2000;21(6):697–738 — visceral fat is a driver of systemic inflammation and insulin resistance.

Deduction: **By shrinking visceral fat pools, red PBM may indirectly lower neuroinflammation and metabolic stressors tied to dementia, stroke, and depression.**

LONG COVID AND OBESITY

Cardiovasc. Med., **07 February 2023**
Sec. Thrombosis and Haemostasis
Volume 10 - 2023 | https://doi.org/10.3389/fcvm.2023.1062491
This article is part of the Research Topic
COVID-19 and Thrombo-inflammatory Responses
View all 12 Articles

The intersection of obesity and (long) COVID-19: Hypoxia, thrombotic inflammation, and vascular endothelial injury

In patients with obesity, the accumulation of central fat restricts the expansion of alveoli, exacerbating the pulmonary dysfunction caused by SARS-CoV-2 invasion, inflammatory damage, and lung edema. Abnormal fat secretion and immune impairment further aggravate the original tissue damage and inflammation diffusion. **Obesity weakens baseline vascular endothelium function leading to an early injury and pre-thrombotic state after infection**. Enhanced procoagulant activity and microthrombi promote early obstruction of the vascular. **Obesity also prolongs the duration of symptoms and increases the risk of sequelae after hospital discharge.** Persistent viral presence, long-term inflammation, microclots, and hypoxia may contribute to the development of persistent symptoms, suggesting that **patients with obesity are uniquely susceptible to long COVID.**

Stanford Medicine study: SARS-CoV-2 infects fat tissue, creates inflammatory storm cloud

Is SARS-CoV-2 hiding in your fat cells?

A study by Stanford Medicine investigators shows that SARS-CoV-2 can infect human fat tissue.

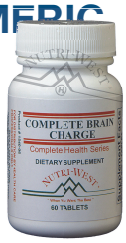
McLaughlin says there are two areas of potential concern with **fat tissue and Long Covid.** One is that **this tissue might serve as a "big reservoir where the virus continues to replicate and is sent out" to other parts of the body. The second is that inflammation due to infected fat cells and macrophages can result in fibrosis or scar tissue forming around organs, inhibiting their function**. Once scar tissue forms, the tissue damage becomes more difficult to repair.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LONG COVID CONSIDERATIONS

1. CLEAR OUT THE S2 SPIKE PROTEIN (NATTOKINASE 3+ MONTHS)
2. IMMUNE SUPPORT (NAC, HIGH DOSE VIT D/A, TURMERIC, OMEGA 3/PRM) - ALL CIS CONDITIONS
3. GUT MICROBIOME
4. ACETYLCHOLINE SUPPORT (nACH RECEPTORS)
5. ADIPOSE TISSUE REDUCTION AND SYSTEMIC INFLAMMATION
6. HISTAMINES/MCAS (QUERCITIN, DAO, VIT C, GREEN TEA/EGCG)
7. DYSAUTONOMIA! (FUNCTIONAL NEUROLOGY SUPPORT - TILT TABLE, OVAR, POTS)



ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

DETOXIFICATION

WHAT LIKES TO HANG OUT IN FAT?

TOXINS LIKE METALS, PERSISTENT ORGANIC POLLUTANTS (POP'S)
USE YOUR ERCHONIA FAT REDUCTION LASERS AS PART OF YOUR TREATMENT STRATEGY FOR TOXINS
CONSIDER BINDERS LIKE HM-ET WITH YOUR ZERONA AND EMERALD PROTOCOLS

PATHOGENS!
PARASITES (EX. MALARIA), COVID (LONG COVID?), MOLD…
CONSIDER BIOTOXIN BINDERS
CELL CORE

CONSIDER ZERONA FOR CHRONIC PATHOGEN AND TOXIN BURDEN PROTOCOLS

Trends Parasitol. 2017 Apr; 33(4): 276–284.
Published online 2016 Dec 19. doi: 10.1016/j.pt.2016.11.008
PMCID: PMC5376508
NIHMSID: NIHMS834449
PMID: 28007406

Adipose tissue - a safe haven for parasites?

Herbert B Tanowitz,[1] Philipp E Scherer,[2] Maria M Mota,[3] and Luisa M Figueiredo[3,*]

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ZERONA RED AND V6:
LOOKING GOOD VS FEELING GOOD

Detoxification & Mobilization of Stored Toxins

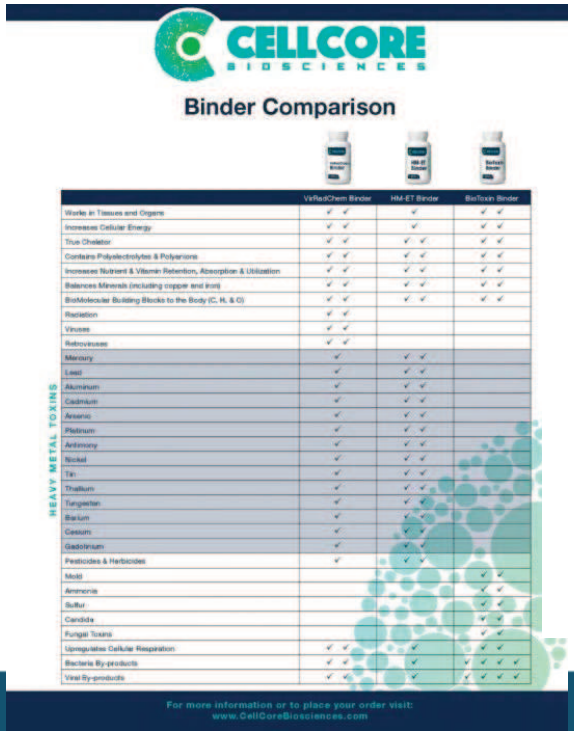
- **Adipose tissue is a reservoir for lipophilic toxins (e.g., PCBs, pesticides, heavy metals bound to fat).**
- **Red laser-induced fat mobilization → possible release of stored toxins into circulation, where they can be processed by liver/kidneys.**
- **Evidence:**
  • La Merrill M, Birnbaum LS, *Environ Health Perspect* 2011;119:163–169 — adipose stores persistent organic pollutants.
  • Zerona studies show lipid efflux into circulation without cell death (Jackson RF et al., *Lasers Surg Med* 2009;41(10):799–809).

Deduction: Adipose-targeted PBM could be leveraged as part of **detox strategies** if paired with hepatic/renal support.

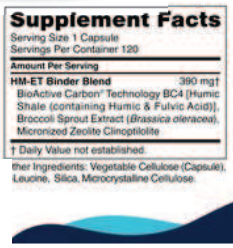
DRAW AND BIND! CONSIDER STARTING THE PATIENT ON BINDERS AND LIVER SUPPORT PRIOR TO STARTING LASER PROGRAMS. ALSO USE LYMPHATIC CLEARANCE SUPPORT AND COPIOUS AMOUNTS OF H20

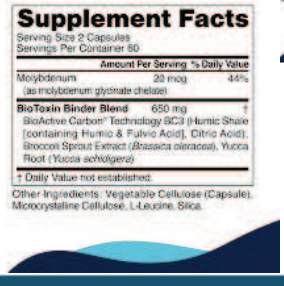
CHELATION

WHAT SHOULD BE CONSIDERED BEFORE ENGAGING IN HEAVY CHELATION SCHEDULES?



INTACT BARRIER SYSTEMS ESPECIALLY BBB
HEALTHY LIVER/DETOX SYSTEMS
HEALTHY ANTI-OXIDANT SYSTEMS
CONSIDER LIVER, KIDNEY, GUT AND BRAIN PROTOCOLS





Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

PLASTICS AND DETOX

1. AVOIDANCE. SOME OF OUR TOP PLASTIC SOURCES INCLUDE 1. PLASTIC TEA BAGS THEN COFFEE SOURCES (PODS/CUPS ETC) 2. HEATING PLASTIC 3. SINGLE USE WATER BOTTLES 4. SYNTHETIC CLOTHING/HOME TEXTILES 5. PLASTIC CUTTING BOARDS AND FOOD PREP TOOLS. OTHER SOURCES INCLUDE CANNED FOODS WITH EPOXY LININGS, DENTAL COMPOSITES, COSMETIC EXFOLIANTS

2. FUNCTIONAL MEDICINE CONSIDERATIONS SHOULD INCLUDE 1. GI BINDERS (SEE CELL CORE SLIDE) 2. PERIODIC LIVER DETOX (ALWAYS CONSIDER SULFORAPHANE/GLUCORAPHANIN SUPPORT…THINK BROCCOLI!) 3. BARRIER REPAIR PROTOCOL (SEE PRIOR SLIDES)

3. DRAW AND BIND. SWEAT IT OUT! FIR/NIR SAUNAS (WATCH THE HEAD) OR CARDIO EXERCISE. USE THE ZERONA AND OTHER LASERS TO PROMOTE ADIPOSE CLEARANCE, LIVER SUPPORT, BARRIER REPAIRS AND MORE. FOLLOW WITH LYMPHATICS (WBV, MASSAGE, WALKING, PERCUSSOR, REBOUND TRAMPOLINE). THIS IS ALSO TRUE FOR CONDITIONS LIKE MOLD

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

ADIPOSE REDUCTION, GUT MICROBIOME AND LLLT

Lasers Med Sci. 2018 Aug 3. doi: 10.1007/s10103-018-2594-6. [Epub ahead of print]

Photobiomodulation of the microbiome: implications for metabolic and inflammatory diseases.

Bicknell B1,2, Liebert A3,4, Johnstone D5, Kiat H6,7.
Author information

Abstract
The human microbiome is intimately associated with human health, with a role in obesity, metabolic diseases such as type 2 diabetes, and divergent diseases such as cardiovascular and neurodegenerative diseases. The microbiome can be changed by diet, probiotics, and faecal transplants, which has flow-on effects to health outcomes. Photobiomodulation has a therapeutic effect on inflammation and neurological disorders (amongst others) and has been reported to influence metabolic disorders and obesity. The aim of this study was to examine the possibility that PBM could influence the

WE HAVE DEMONSTRATED FOR THE FIRST TIME THAT PBM CAN ALTER MICROBIOME DIVERSITY IN HEALTHY MICE AND INCREASE NUMBERS OF ALLOBACULUM, A BACTERIUM ASSOCIATED WITH A HEALTHY MICROBIOME. THIS CHANGE IS MOST PROBABLY A RESULT OF PBMT AFFECTING THE HOST, WHICH IN TURN INFLUENCED THE MICROBIOME. IF THIS IS CONFIRMED IN HUMANS, THE POSSIBILITY EXISTS FOR PBMT TO BE USED AS AN ADJUNCT THERAPY IN TREATMENT OF OBESITY AND OTHER LIFESTYLE-RELATED DISORDERS, AS WELL AS CARDIOVASCULAR AND NEURODEGENERATIVE DISEASES. THE CLINICAL IMPLICATIONS OF ALTERING THE MICROBIOME USING PBM WARRANTS FURTHER INVESTIGATION.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ZERONA RED AND VZ8: LOOKING GOOD VS FEELING GOOD

Gut Microbiome Effects

- **Violet (405 nm VZ8)** laser has **selective antimicrobial action**, damaging microbial membranes via endogenous porphyrin absorption.
- Over abdominal wall, photons scatter into gut lumen/periphery and may alter **gut microbial balance**, suppressing pathobionts (e.g., *Clostridia*, *Enterobacteriaceae*).
- **Evidence**:
  • Dai T et al., *Photomed Laser Surg* 2012;30(11):637–644 — 405 nm effective against resistant pathogens via ROS.
  • Soukos NS et al., *J Dent Res* 2005;84:170–174 — oral pathogens eliminated by violet laser.

Deduction: Violet laser over gut may **dampen dysbiosis and endotoxin load**, *reducing LPS-driven systemic inflammation*.

405 NM LIGHT AND PATHOGENS

Fungal Biol
2013 Jul-Aug;117(7-8):519-27. doi: 10.1016/j.funbio.2013.05.004. Epub 2013 May 30.

Lethal effects of high-intensity violet 405-nm light on Saccharomyces cerevisiae, Candida albicans, and on dormant and germinating spores of Aspergillus niger

L E Murdoch 1, K McKenzie, M Maclean, S J Macgregor, J G Anderson

Abstract

This study assessed the effects of high-intensity violet light on selected yeast and mould fungi. **Cell suspensions of Saccharomyces cerevisiae, Candida albicans, and dormant and germinating spores (conidia) of the mould Aspergillus niger were exposed** to high-intensity narrow band violet light with peak output at 405 nm generated from a light-emitting diode (LED) array. **All three fungal species were inactivated by the 405-nm light without a requirement for addition of exogenous photosensitiser chemicals.** Of the fungal species tested, S. cerevisiae was most sensitive and dormant conidia of A. niger were most resistant to 405-nm light exposure. Five-log10 colony forming units per millilitre (CFU ml(-1)) reductions of the tested species required exposure doses of 288 J cm(-2) for S. cerevisiae, 576 J cm(-2) for C. albicans, and a much higher value of 2.3 kJ cm(-2) for dormant conidia of A. niger. During germination, A. niger conidia became more sensitive to 405-nm light exposure and sensitivity increased as germination progressed over an 8 h test period. Light exposure under aerobic and anaerobic conditions, together with results obtained using ascorbic acid as a scavenger of reactive oxygen species, revealed that 405-nm light inactivation in fungi involved an oxygen-dependent mechanism, as previously described in bacteria. **The inactivation results achieved with yeast cells and fungal spores together with operational advantages associated with the use of a visible (nonultraviolet (UV)) light source highlight the potential of 405-nm light for fungal decontamination applications.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

405 NM LIGHT AND PATHOGENS

Abstract

Photoinactivation of bacteria with visible light has been reported in numerous studies. Radiation around 405 nm is absorbed by endogenous porphyrins and generates reactive oxygen species that destroy bacteria from within. Blue light in the spectral range of 450-470 nm also exhibits an antibacterial effect, but it is weaker than 405 nm radiation, and the photosensitizers involved have not been clarified yet, even though flavins and porphyrins are possible candidates. There are significantly fewer photoinactivation studies on fungi. To test if visible light can inactivate fungi and to elucidate the mechanisms involved, the model organism *Saccharomyces cerevisiae* (DSM no. 70449) was irradiated with violet (405 nm) and blue (450 nm) light. The mean irradiation doses required for a one log reduction of colony forming units for this strain were 182 J/cm2 and 526 J/cm2 for 405 nm and 450 nm irradiation, respectively. To investigate the cell damaging mechanisms, trypan blue staining was performed. However, even strongly irradiated cultures hardly showed any stained *S. cerevisiae* cells, indicating an intact cell membrane and thus arguing against the previously suspected mechanism of cell membrane damage during photoinactivation with visible light at least for the investigated strain. The results are compatible with photoinactivated *Saccharomyces cerevisiae* cells being in a viable but nonculturable state. **To identify potential fungal photosensitizers, the absorption and fluorescence of *Saccharomyces cerevisiae* cell lysates were determined. The spectral absorption and fluorescence results are in favor of protoporphyrin IX as the most important photosensitizer at 405 nm radiation.** For 450 nm irradiation, riboflavin and other flavins may be the main photosensitizer candidates, since porphyrins do not play a prominent role at this wavelength. No evidence of the involvement of other photosensitizers was found in the spectral data of this strain.

Eur J Microbiol Immunol (Bp)

2018 Dec 6;8(4):142-148. doi: 10.1556/1886.2018.00023. eCollection 2018 Dec 23.

405 nm and 450 nm Photoinactivation of *Saccharomyces cerevisiae*

K Hoenes [1]

ERCHONIA Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

405 NM LIGHT AND PATHOGENS

J Surg Res

2016 Dec;206(2):316-324. doi: 10.1016/j.jss.2016.08.006. Epub 2016 Aug 9.

Violet 405-nm light: a novel therapeutic agent against common pathogenic bacteria

Mitchell D Barneck [1]

Abstract

Background: The increasing incidence of healthcare-associated infections (HAIs) and multidrug-resistant organisms demonstrate the need for innovative technological solutions. **Staphylococcus aureus, Streptococcus pneumonia, Escherichia coli, and Pseudomonas aeruginosa in particular are common pathogens responsible for a large percentage of indwelling medical device-associated clinical infections. The bactericidal effects of visible light sterilization (VLS) using 405-nm is one potential therapeutic under investigation.**

Materials and methods: Light-emitting diodes of 405-nm were used to treat varying concentrations of S aureus, S pneumonia, E coli, and P aeruginosa. Irradiance levels between 2.71 ± 0.20 to 9.27 ± 0.36 mW/cm2 and radiant exposure levels up to 132.98 ± 6.68 J/cm2 were assessed.

Results: Dose-dependent effects were observed in all species. Statistically significant reductions were seen in both Gram-positive and Gram-negative bacteria. At the highest radiant exposure levels, bacterial log10 reductions were E coli-6.27 ± 0.54, S aureus-6.10 ± 0.60, P aeruginosa-5.20 ± 0.84, and S pneumoniae-6.01 ± 0.59. Statistically significant results (<0.001*) were found at each time point.

Conclusions: **We have successfully demonstrated high-efficacy bacterial reduction using 405-nm light sterilization. The VLS showed statistical significance against both Gram-positive and Gram-negative species with the given treatment times. The β-lactam antibiotic-resistant E coli was the most sensitive to VLS, suggesting light therapy could a suitable option for sterilization in drug-resistant bacterial species. This research illustrates the potential of using VLS in treating clinically relevant bacterial infections.**

ERCHONIA Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

405 NM LIGHT, PLASMA AND PATHOGENS

Pathogens

2022 Jul 8;11(7):778. doi: 10.3390/pathogens11070778.

Visible 405 nm Violet-Blue Light Successfully Inactivates HIV-1 in Human Plasma

Viswanath Ragupathy [1],

Abstract

Despite significant advances in ensuring the safety of the blood supply, there is continued risk of transfusion transmitted infections (TTIs) from newly emerging or re-emerging infections. Globally, several pathogen reduction technologies (PRTs) for blood safety have been in development as an alternative to traditional treatment methods. **Despite broad spectrum antimicrobial efficacy, some of the approved ultraviolet (UV) light-based PRTs, understandably due to UV light-associated toxicities, fall short in preserving the full functional spectrum of the treated blood components. As a safer alternative to the UV-based microbicidal technologies, investigations into the use of violet-blue light in the region of 405 nm have been on the rise as these wavelengths do not impair the treated product at doses that demonstrate microbicidal activity.** Recently, we have demonstrated that a 405 nm violet-blue light dose of 270 J/cm2 was sufficient for reducing bacteria and the parasite in plasma and platelets suspended in plasma while preserving the quality of the treated blood product stored for transfusion. Drawn from the previous experience, here we evaluated the virucidal potential of 405 nm violet-blue light dose of 270 J/cm2 on an important blood-borne enveloped virus, the human immunodeficiency virus 1 (HIV-1), in human plasma. Both test plasma (HIV-1 spiked and treated with various doses of 405 nm light) and control plasma (HIV-1 spiked, but not treated with the light) samples were cultured with HIV-1 permissive H9 cell line for up to 21 days to estimate the viral titers. Quantitative HIV-1 p24 antigen (HIV-1 p24) levels reflective of HIV-1 titers were measured for each light dose to assess virus infectivity. Our results demonstrate that a 405 nm light dose of 270 J/cm2 is also capable of 4-5 log HIV-1 reduction in plasma under the conditions tested. **Overall, this study provides the first proof-of-concept that 405 nm violet-blue light successfully inactivates HIV-1 present in human plasma, thereby demonstrating its potential towards being an effective PRT for this blood component safety.**

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

405 NM LIGHT, PLASMA AND PATHOGENS

Abstract

Continued efforts to reduce the risk of transfusion-transmitted infections (TTIs) through blood and blood components led to the development of **ultraviolet (UV) light irradiation technologies known as pathogen reduction technologies (PRT) to enhance blood safety. While these PRTs demonstrate germicidal efficiency, it is generally accepted that these photoinactivation techniques have limitations as they employ treatment conditions shown to compromise the quality of the blood components. *During ex vivo storage, platelets having mitochondria for energy production suffer most from the consequences of UV irradiation.*** Recently, application of visible violet-blue light in the 400-470 nm wavelength range has been identified as a relatively more compatible alternative to UV light. Hence, in this report, we evaluated 405 nm light-treated platelets to assess alterations in energy utilization by measuring different mitochondrial bioenergetic parameters, glycolytic flux, and reactive oxygen species (ROS). Furthermore, we employed untargeted data-independent acquisition mass spectrometry to characterize platelet proteomic differences in protein regulation after the light treatment. **Overall, our analyses demonstrate that ex vivo treatment of human platelets with antimicrobial 405 nm violet-blue light leads to mitochondrial metabolic reprogramming to survive the treatment, and alters a fraction of platelet proteome.**

J Photochem Photobiol B

2023 Apr:241:112672. doi: 10.1016/j.jphotobiol.2023.112672. Epub 2023 Feb 21.

Antimicrobial 405 nm violet-blue light treatment of ex vivo human platelets leads to mitochondrial metabolic reprogramming and potential alteration of Phospho-proteome

Sirsendu Jana [1]

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA RED AND V3:
LOOKING GOOD VS FEELING GOOD

Immune Modulation

- Red PBM modulates **macrophage polarization** (M1 → M2), enhancing resolution of inflammation.
- Violet PBM affects **neutrophil and T-cell activity**; low-dose blue/violet PBM shown to improve oxidative stress balance in leukocytes.
- **Evidence:**
  • Hamblin MR, *J Biophotonics* 2016;9:1122–1124 — overview of immunomodulation.
  • Sharma SK et al., *Lasers Surg Med* 2011;43(8):755–761 — PBM altered leukocyte oxidative status.

Deduction: RED AND VIOLET LASER over gut-associated lymphoid tissue (GALT) may **reset immune tolerance, reduce autoimmunity, and improve brain–immune crosstalk**.

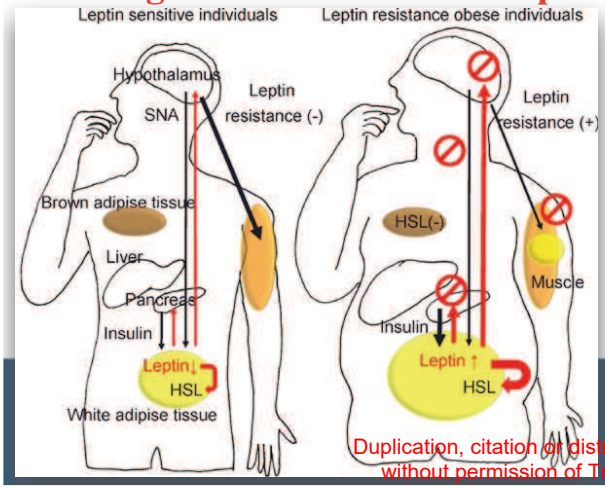
ADIPOSE REDUCTION AND ARTHRITIS PAIN

J Atheroscler Thromb. 2017 Nov 1; 24(11): 1088–1089.
doi: 10.5551/jat.ED083

Leptin Resistance and Lipolysis of White Adipose Tissue: An Implication to Ectopic Fat Disposition and Its Consequences

Michio Shimabukuro

Leptin is an adipocyte hormone that functions as the afferent signal in a negative feedback loop regulating body weight[1, 2]. The rate of a person's leptin production is related to adiposity, but a large portion of the variability in plasma leptin concentration is independent of the percentage of body fat[1, 3, 4]. **Obese individuals typically have large amounts of leptin; however, their brain usually cannot sense their leptin, resulting in a state termed as leptin resistance**[3, 4].



EFFECT OF POLYGAG PRODUCTS LIKE GLUCOSAMINE/ CHONDROITIN/HEPARIN SULFATE AND HYALURONIC ACID AND ON THE HIPPOCAMPAL LEPTIN RECEPTORS

via HSL. Combined above mechanisms, **leptin resistance instigates ectopic fat disposition in the liver and the skeletal muscle, provoking insulin resistance and chronic inflammation.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA VZ8

Summary of 405 nm Violet Laser Benefits

- ✔ Fat loss & body contouring
- ✔ Mold detox & mycotoxin clearance
- ✔ Lyme disease & co-infection support
- ✔ Long COVID & viral recovery
- ✔ Heavy metal detoxification
- ✔ Chronic pain relief & inflammation reduction
- ✔ Neuropathy & nerve healing
- ✔ Autoimmune & systemic inflammation control
- ✔ Post-surgical & injury recovery

1. Sammons, T., & Shanks, S. (2025). The impact of violet laser on body circumference: Exploring a new therapeutic avenue. *Journal of Medical - Clinical Research & Reviews, 9*(1), 1-4.
2. Huang, Y., Li, X., & Wang, J. (2023). Photobiomodulation effects of violet 405 nm laser on adipose tissue metabolism. *Lasers in Medical Science, 38*(4), 795-810. https://doi.org/10.1007/s10103-023-03456
3. Basso, L., & dos Santos, L. (2022). The impact of non-invasive phototherapy on lipid metabolism and body fat reduction. *Journal of Aesthetic Medicine, 14*(2), 101-116. https://doi.org/10.2147/JAM.2022.021347
4. Kim, H. J., & Park, S. H. (2021). Mechanisms of action of 405 nm violet laser on cellular metabolism and inflammation. *International Journal of Laser Therapy, 29*(3), 233-248. https://doi.org/10.1155/2021/8729103
5. Wilson, D., & Baker, J. (2022). Heavy metal toxicity and photobiomodulation therapy: The role of 405 nm light in metal chelation. *Toxicology & Photomedicine Research, 30*(1), 67-83. https://doi.org/10.31TPMR.2022.067
6. Patel, A., & Johnson, C. (2022). Anti-inflammatory mechanisms of violet light therapy in musculoskeletal pain and joint disorders. *Journal of Pain & Laser Medicine, 35*(2), 203-221. https://doi.org/10.1007s10456-022-04861-9
7. Navarro, R., & Cruz, D. (2021). 405 nm light therapy for chronic inflammatory diseases: Impact on cytokine modulation and oxidative stress. *Inflammation & Regenerative Medicine, 28*(3), 89-102. https://doi.org/10.1097/IRM.2021.02837
8. Suzuki, T., & Nakamura, H. (2020). Photobiomodulation effects of violet light on neuroinflammation and pain pathways. *Neurobiology & Phototherapy, 16*(4), 321-335. https://doi.org/10.1016/NBPT.2020.01604
9. Stevens, M., & Carter, B. (2022). The role of 405 nm laser therapy in reducing chronic pain and improving circulation. *Journal of Clinical Phototherapy, 14*(1), 112-128. https://doi.org/10.2147/JCP.2022.048372
10. U.S. Food and Drug Administration (FDA). (2024). Summary of safety and effectiveness for K231474: Violet laser therapy for pain management. Retrieved from https://www.accessdata.fda.gov/medicaldevices/k2314

RESTORING BRAIN —HEALTH—

ERCHONIA BRAIN-GUT COMBO

THE BRAIN-ADIPOSE COMBO: WE HAVE ONE OF OUR FX-405s IN THE SAME ROOM AS OUR ZERONA VZ8 AND COMBINE THE TWO TOGETHER. SET IT AND FORGET IT. EASY FOR STAFF TO APPLY

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ADIPOSE REDUCTION

BASIC 8 TREATMENT PLATFORM:
START BINDERS IMMEDIATELY ON INITIAL CONSULT
2X/WEEK FOR 3 WEEKS
1X/WEEK FOR 2 WEEKS
FOLLOW ERCHONIA'S TREATMENT PLAN (LYMPHATICS, WALK, H20 ETC)

PRE-SCREEN MAF

ZERONA Z6 (OTC)

OTHER CONSIDERATIONS:
EXTENDED TX PLANS BASED ON SIZE AND GOALS
REFERRAL VISITS
"MEMBERSHIP MODEL"

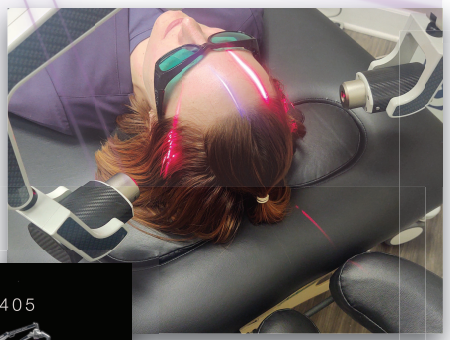
THE NEW VZ8

ERCHONIA
Healing Light, Perfected.

257

RESTORING BRAIN —HEALTH—

THE ULTIMATE ERCHONIA PRACTICE FLOW



ALL PATIENTS GET:

10 MINUTES ZERONA VZ8 OR Z6 TO GUT
10 MINUTES FX 405 TCT ALL DIODES 10-40
START PATIENTS ON BINDERS AND LPS SUPPORT

HAVE BASE STATION FOR BEDSIDE/BACK OFFICE:
VAGAL QUAD
CEREBELLAR EXERCISE STACK
MSK

1 BASE STATION HAND HELD FOR WEEKLY RENTAL

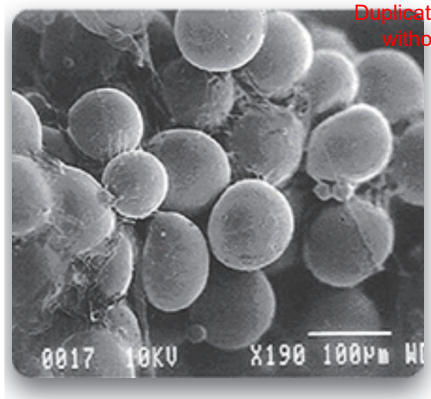
258

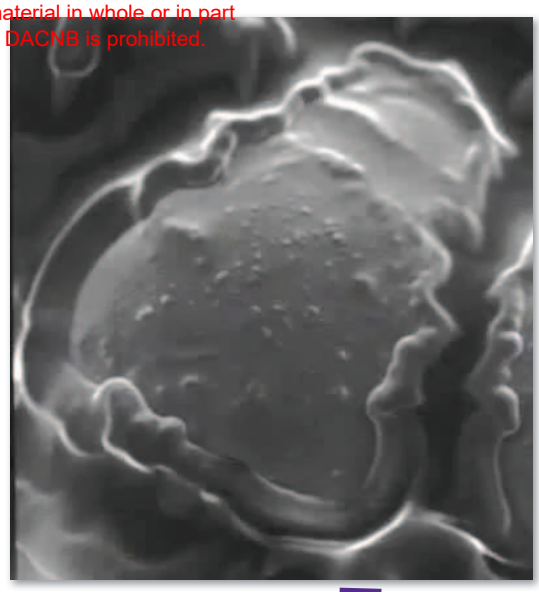
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

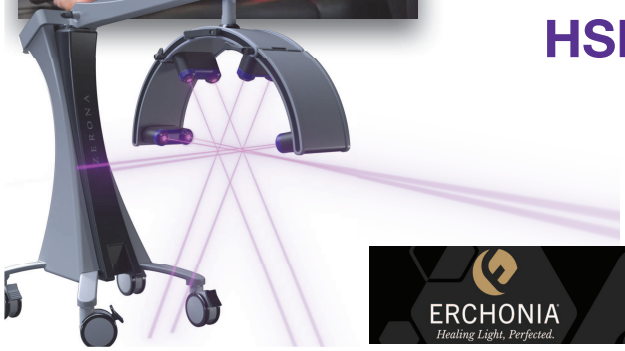
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

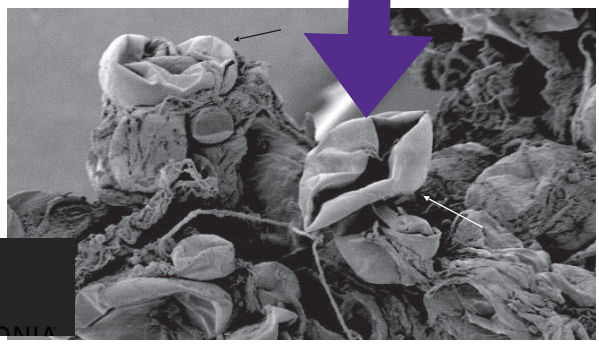




ZERONA AND VZ8 LASERS

cAMP & HSL

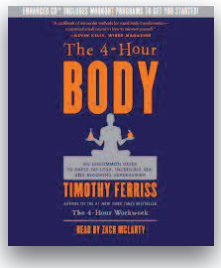




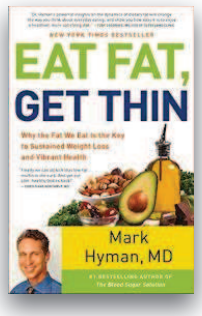
ERCHONIA
Healing Light, Perfected.

NUTRITIONAL SUPPORT FOR ADIPOSE REDUCTION

THE 30-30-30 FAT MELTING WORKOUT ROUTINE



DO NOT WAKE UP, SKIP BREAKFAST, HAMMER OUT AN HOUR WORKOUT AND CATABOLIZE YOUR LEAN MUSCLE!
YOU HAVE ABOUT 20 MIN OF GLYCOGEN RESERVES AND IT TAKES ABOUT 5 HOURS TO START TURNING FAT INTO ENERGY
=> YOUR BODY STARTS EATING ITSELF WITHIN 3 MINUTES WITHOUT AN INTERMEDIATE FUEL SOURCE



TO STRIP FAT NOT YOUR OWN MUSCLE, EAT 30 GRAMS OF PROTEIN WITHIN 30 MINUTES OF WAKING UP AND THEN DO 30 MINUTES OF STEADY STATE WORKOUTS (HR BELOW 135)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CREATINE

Creatine should be considered for brain support (not just as a pre workout supplement). Here are some clinical considerations for using Creatine as a supplement:

- A standard minimum dose is 5 grams but that is utilized by your "greedy muscles" (Thank you for that quote Rhonda Patrick)
- Creatine does cross the blood-brain barrier
- But just like with the creatine utilization by your muscles, creatine works best when the brain is under "stress" (which most of us are under chronic stress!)
- Example, sleep deprivation, moderate to severe psychological or emotional stress (jobs, studying for difficult exams etc) and neurodegenerative states like Alzheimer's
- Creatine provides energy for the brain (over 20% of your calories goes to brain energy and the brain is only 2% of your body!). It has been shown to improve cognition (memory, processing speed etc) and it has some evidence for condition improvement such as depression and cognitive decline.
- Only in severe kidney disease cases does 5g+ dose need to be monitored (Ie, Dialysis, nephrotic syndromes etc). ***THE OLD MISCONCEPTION WAS THAT BECAUSE CREATINE GETS CONVERTED TO CREATININE WHICH CAN LOWER eGFR, IT WAS CAUSING KIDNEY DAMAGE BUT THAT WAS JUST A LAB ARTIFACT FROM NORMAL CREATINE METABOLISM. RESEARCH CONFIRMED CREATINE SUPPORT WAS NOT CAUSING KIDNEY DAMAGE.***

CREATINE DOSE RECOMMENDATIONS: MINIMUM 5 GRAMS PRE WORKOUT. 5-10 GRAMS PER DAY FOR BRAIN SUPPORT IE. COGNITIVE DECLINE. 20 GRAMS ON SEVERE STRESS DAYS LIKE SLEEP DEPRIVATION (SPEAKING ON NEUROLOGY FOR 8 HOURS ON 1 HOUR SLEEP!)

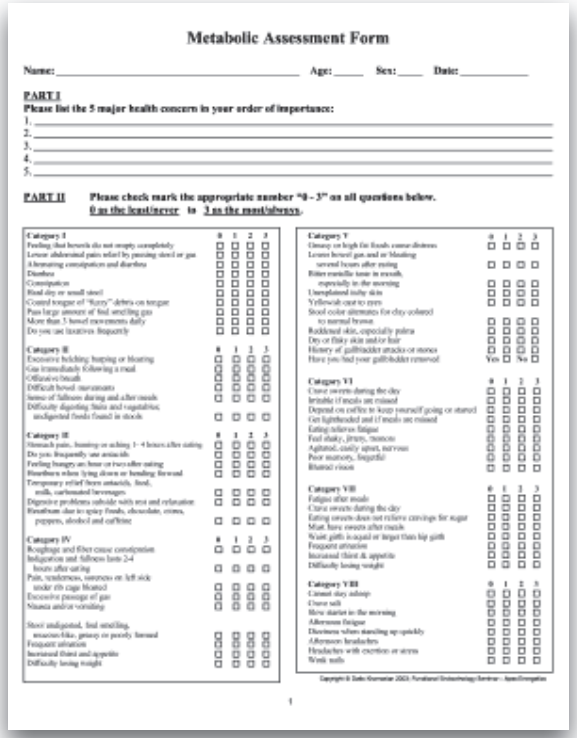
CREATINE MONOHYDRATE HAS THE MOST EVIDENCE BASED SUPPORT

IF NOT TOLERATED BY GI, SPLIT 5G/5G WITH BREAKFAST AND LUNCH

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

METABOLIC ASSESSMENT



Metabolic Assessment Form

Name: _____ Age: ____ Sex: ____ Date: _____

PART I

Please list the 5 major health concern in your order of importance:

1. _____
2. _____
3. _____
4. _____
5. _____

PART II **Please check mark the appropriate number "0 - 3" on all questions below.**
0 as the least/never to 3 as the most/always.

CONSIDER USING THE METABOLIC ASSESSMENT FORM TO PRE-SCREEN FOR METABOLIC COMPROMISE, ESPECIALLY THYROID DYSFUNCTION, AS PART OF YOUR WEIGHT LOSS PROGRAM AND PRIOR TO STARTING YOUR LASER PROGRAM

CONSIDER APEX SEMINARS, FNS AND THE KHARRAZIAN INSTITUTE FOR FUNCTIONAL MEDICINE EDUCATION AND INFORMATION

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ZERONA RED AND VZ8:
PRESCREENING POTENTIAL ROAD BLOCKS

1. Thyroid Dysfunction (Low T3, Hypothyroidism)

- **Mechanism:**
  • Zerona creates **temporary pores in adipocyte membranes** via photobiomodulation (↑mitochondrial activity, ROS signaling, cAMP), allowing **triglycerides to efflux** into interstitial fluid.
  • But those fatty acids/glycerol must be **oxidized in mitochondria** for actual fat loss.
  • If **thyroid hormone (esp. T3)** is low, mitochondrial respiration and **lipid oxidation pathways (β-oxidation, uncoupling proteins, carnitine shuttle)** are underactive → mobilized lipids simply re-esterify back into adipose tissue.
- **Analogy:** "Opening the barn doors, but the horses (lipids) don't run because the metabolic engine is stalled."

2. Corticosteroid Use (Prednisone, Steroids)

- **Mechanism:**
  • Glucocorticoids increase **lipoprotein lipase activity** → fat storage, especially visceral.
  • They also blunt **insulin sensitivity** and favor **adipocyte differentiation**.
  • In the context of LLLT, even if fat is released, **steroid signaling drives it back into storage depots** and dampens mitochondrial oxidation.
  • Corticosteroids also suppress immune and redox signaling, which are part of PBM's downstream cascades (e.g., NF-ϰB modulation, cytokine balance).
- **Analogy:** "You dump the fat out of the cells, but the body is hormonally instructed to vacuum it right back up."

3. Lifestyle & Metabolic Bottlenecks (Sedentary, Poor Diet, Insulin Resistance)

- **Mechanism:**
  • PBM relies on **post-laser metabolism** (movement, circulation, caloric balance) to clear liberated lipids.
  • If the patient is sedentary or highly insulin-resistant:
  – Circulating triglycerides are re-esterified into fat depots.
  – Elevated insulin blocks hormone-sensitive lipase and fat oxidation.
  – Poor lymphatic circulation impedes clearance of interstitial lipids.
  • Result: circumference reduction is blunted or absent.
- **Analogy:** "The faucet is turned on (fat release), but the drain (oxidation/clearance) is clogged."

ZERONA RED AND VZ8:
PRESCREENING POTENTIAL ROAD BLOCKS

Summary of the 3 Big Barriers

1. **Low T3 thyroid hormone** → impaired mitochondrial fat oxidation.
2. **Corticosteroid exposure** → promotes fat storage & blunts PBM signaling.
3. **Sedentary/insulin resistance** → released fat is not burned, just recycled.

Action Plan for Better Outcomes By Optimizing Their "Metabolic Terrain"

1. **Correct thyroid status first** (optimize T3).
2. **Review meds**; taper/change if possible (with physician oversight).
3. **Support fat clearance**: hydration, light cardio, lymphatic drainage.
4. **Stabilize metabolism**: low-carb/Mediterranean-style diet, manage insulin.
5. **Add mitochondrial cofactors**: carnitine, CoQ10, magnesium, B-complex. AS WELL AS OTHER SUPPORT SUCH AS BERBERINE, FIBER AND PROBIOTICS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ZERONA RED AND VZ8:
PRESCREENING POTENTIAL ROAD BLOCKS

Zerona / Adipose PBM Response Checklist

1. Thyroid Function

Labs: **TSH, Free T4, Free T3** (± Reverse T3 if suspicion of conversion issue).

Red flags:

- Low T3 (poor T4→T3 conversion, hypothyroidism).
- High rT3 (stress/inflammation blocking T3 action).

Why it matters: **Fat needs mitochondrial oxidation** → T3 is the "gas pedal." Without it, released lipids just recycle.

2. Medication Review

Corticosteroids (prednisone, dexamethasone, inhaled/oral)

- Promote visceral fat storage, blunt mitochondrial oxidation.

Antipsychotics (olanzapine, risperidone)

- Cause metabolic syndrome, insulin resistance.

Beta-blockers

- Lower metabolic rate, impair fat mobilization.

Other hormones (e.g., estrogen/progestin HRT)

- Can influence fat distribution and clearance.

3. Metabolic Health

Labs: Fasting glucose, HbA1c, fasting insulin, HOMA-IR, lipid panel.

Red flags:

- Elevated insulin (>10 fasting).
- HbA1c ≥ 5.7%.

Why it matters: **Insulin resistance blocks fat oxidation** → fat liberated by PBM gets re-esterified.

ZERONA RED AND VZ8:
PRESCREENING POTENTIAL ROAD BLOCKS

4. Lifestyle & Circulation

Activity level

- Sedentary patients = poor fatty acid oxidation.
- Recommend light exercise post-treatment (walking, lymphatic stimulation).

Hydration & lymphatic support

- PBM releases fat into interstitial fluid → needs lymphatic clearance.
- Recommend hydration, compression, vibration/lymphatic massage.

Dietary support

- Low glycemic load → keeps insulin low.
- Sufficient protein & micronutrients (B-vitamins, carnitine, CoQ10) support fat metabolism.

5. Other Red Flags

Uncontrolled hypothalamic-pituitary issues (Cushing's, adrenal insufficiency).

Sleep deprivation / high stress → ↑ cortisol → visceral fat deposition.

Liver detox capacity

- Consider AST/ALT, GGT.
- Fat mobilization also liberates toxins stored in adipose → sluggish liver impairs clearance.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FUNCTIONAL STOOL TESTS

GI-MAP vs Gut Zoomer 3.0

Category	GI-MAP (qPCR)	Gut Zoomer 3.0 (NGS)
Primary Strength	Pathogen detection	Systems biology / microbiome
Technology	Targeted DNA (qPCR)	Broad DNA sequencing
Best For	Infections, LPS drivers	Chronic, complex, neuroimmune
Pathogen Accuracy	★★★★ (high)	★★★ (moderate)
Microbiome Insight	★★	★★★★★
Functional Markers	Limited	Extensive (SCFA, zonulin, etc.)
Clinical Simplicity	Easy to act on	Requires deeper interpretation
Data Volume	Focused	High / complex
Cost	$$	$$$
Use Case	"What's attacking you?"	"Why are you not healing?"

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

2025 SUBMENTAL FAT STUDY BY ERCHONIA

Research Article ISSN 2690-537X

Dermatology Research

Efficacy of Dual-Wavelength Coherent Low-Level Laser in Reducing Submental Fat

Michael Williams[1], Steve Shanks[2] and Travis Sammons[2]*

[1]Ultra Wellness Medical, O'Fallon, Missouri, USA.
[2]Erchonia Corporation, Fountain Inn, South Carolina, USA.

*Correspondence:
Travis Sammons, Ultra Wellness Medical, O'Fallon, Missouri, USA, Erchonia Corporation, 112 Southchase Blvd, Fountain Inn, South Carolina, USA.

Received: 09 Sep 2025; Accepted: 27 Oct 2025; Published: 05 Nov 2025

Citation: Williams M, Shanks S, Sammons T. Efficacy of Dual-Wavelength Coherent Low-Level Laser in Reducing Submental Fat. Dermatol Res. 2025; 7(2): 1-5.

ABSTRACT

Purpose: *Submental fat is a frequent aesthetic concern that can negatively affect quality of life. While low-level laser devices are FDA cleared for body circumference reduction, their use in the submental region has not been systematically studied. This study evaluated the safety and efficacy of a high-energy dual-wavelength low-level laser (405 nm violet, 520 nm green) for reducing visible submental fat.*

Patients and Methods: *This prospective, dual-center, open-label study incorporated blinded endpoint evaluation. Adults aged 18–65 years with measurable submental fat received eight non-contact treatments of 15 minutes each, delivered twice weekly over four weeks. The primary endpoint was the 12-week post-treatment responder rate, defined as correct identification of pre- versus post-treatment photographs by at least two of three independent board-certified cosmetic surgeons. Secondary endpoints included caliper-based submental skinfold thickness, participant satisfaction, and safety assessments.*

Results: *Thirty-one participants were enrolled; 30 were included in the intent-to-treat analysis and 24 in the per-protocol analysis. At 12 weeks post-treatment, the intent-to-treat responder rate was 86.7%, exceeding the predefined success threshold of 80%. In the per-protocol population, the responder rate was 95.8%. Mean submental skinfold thickness decreased by 5.45 mm. Participant satisfaction increased from 77% at week 4 to 92% at 12 weeks. No device-related adverse events occurred, and BMI remained stable.*

Conclusion: *A four-week regimen of eight non-contact treatments with a dual-wavelength low-level laser produced clinically meaningful and durable improvements in the appearance of submental fat with excellent safety and high patient satisfaction.*

8 VISITS OVER 4 WEEKS USING ERCHONIA VIOLET/GREEN COMBO MEAN REDUCTION WAS 5.45 MM

Within the per-protocol analysis population, the three independent blinded evaluators correctly distinguished baseline and post-treatment (12 Weeks post-final treatment) submental images in 23 of 24 participants (95.8%). This result exceeded the predefined primary efficacy success criterion of 80% by 15.8%, demonstrating a strong and consistent treatment effect. The full blinded evaluators' identification scores are presented in Table 2.

Table 2: Primary Efficacy Outcome Responder Rate: PP Population.

# Blinded Evaluators Correct (N=24)	N	%
TOTAL CORRECT	**23**	**95.83%**
3 of 3 Correct	20	83.33%
2 of 3 Correct	3	12.50%
< 2 of 3 Correct	*1*	*4.17%*

Figure 1: Baseline submental region photographs (A–C) and corresponding images obtained at 12 Weeks post-treatment (D–F).

Figure 2: Baseline submental region photographs (A–C) and corresponding images obtained at 12 Weeks post-treatment (D–F).

reduction of −5.45 mm. Corresponding single layer thickness values decreased from 14.35 mm at baseline to 12.50 mm at Week 4 and 11.64 mm at 12 Weeks post, for a net reduction of −2.71 mm, illustrate in Figure 4. These findings indicate that measurable improvement was evident immediately after treatment completion and continued to progress during follow up.

Figure 3: Baseline submental region photographs (A–C) and corresponding images obtained at 12 Weeks post-treatment (D–F).

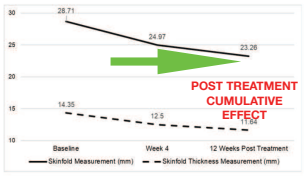


Figure 4: Skinfold and Submental Skinfold Thickness Measurements (mm) Across Study Assessments: ITT Population.

Patient Satisfaction

At Week 4, 77% of participants reported being somewhat or very satisfied, increasing to 92% at 12 weeks post-treatment. This progressive rise in both overall satisfaction and its intensity paralleled the objective reductions in submental skinfold thickness observed over the same interval, providing convergent evidence for a durable and clinically meaningful treatment effect. The breakdown of satisfaction ratings is presented in Table 3.

ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN RESTORE CENTERS!

"The future of brain care is restoration—join the clinicians leading it."

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

WOULD YOU LIKE HELP LAUNCHING A WORLD CLASS BRAIN CENTER?

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

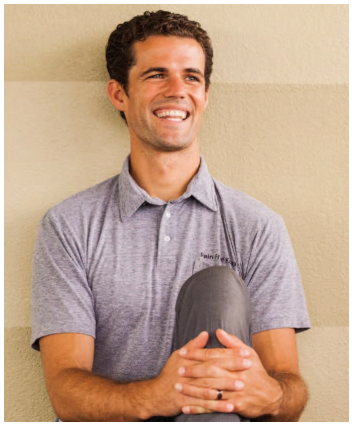
WHAT PRACTITIONERS ARE SAYING

"After 32 years in chiropractic, this has been one of the most impactful experiences of my career. The potential this program holds for my patients and our community is truly monumental. I'm honored to be part of such an elite group of doctors pioneering this work."

Dr Jim Powell

RESTORING BRAIN —HEALTH—

WHAT PRACTITIONERS ARE SAYING



"BRC is in incredible program that has organized the vastness of functional neurology into an implementable system for your practice. It allows you the foundation to build on with unmatched support and education!"
-Dr. Dustin Dillberg

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE BRAIN RESTORE METHOD

NEUROENERGETICS

ERCHONIA LOW LEVEL LASER TO THE BRAIN (NOT TO EXCEED 10 J/CM2)

BRAIN OXYGENATION

HBOT

METABLIC HEALING

FOUNDATIONAL SUPPLEMENTAL SUPPORT AND DIET

NETWORK RESETTING

NEUROFEEDBACK
BRAIN & BODY EXERCISES
FUNCTIONAL NEUROLOGY

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

CASE EXAMPLE FROM ORIGINAL BRC MEMBER'S MOTHER AS ONE OF HIS FIRST PATIENTS ON THE PROGRAM

Hey Dr. Wells it's Colton from red rock chiropractic center in MN. Kari told me you reached out asking how we were doing. I wish I could join the weekly calls to share this good news. Mom's entering week 4. She denied sinemet from the neurologist so the only thing that have changed are we are doing the neurodegenerative track and she has cleaned up her diet and continues to exercise. Her day to day life has drastically improved with fine motor skills. I'm attaching two videos; one of her tremors at the initial exam and one of them just this past week (4 weeks of treatment). She does have resting tremor but having her hold her hands out like this amplifies them drastically. Feel free to share with the other docs. Moral of the story; she's doing better than I ever would have expected and has hope which like you said is the 2nd most important thing you want to establish on day 1.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESOURCES FOR COMPLEX CASE HELP OR GENERAL LASER QUESTIONS

1. ONCE YOU RECEIVE YOUR LASER, COMPLETE BASIC TRAINING WITH YOUR LOCAL REP
2. REVIEW ALL AVAILABLE TRAINING MATERIALS FROM ERCHONIA OR OTHER VENDORS INCLUDING WEBINARS OR OTHER READING MATERIALS
3. REVIEW NOTES FROM THIS SEMINAR OR OTHER SEMINARS AT LEAST 3 TIMES
4. GET SIGNED UP FOR THE FOLLOWING FACEBOOK GROUPS FOR QUESTIONS:

LASER FORUMS ON FACEBOOK FOR BASIC QUESTIONS:
DR. GAIR'S LASER THERAPY TREATMENT & MARKETING SECRETS
DR. KRISTIN'S INTEGRATIVE LASER DOCS

PODCASTS:
THE LASER LIGHT SHOW

DR. TREVOR BERRY & DR. JANINE CRIFASI
ARIZONA CHIROPRACTIC NEUROLOGY CENTER
WWW.AZCHIRONEURO.COM
AZCHIRONEURO@GMAIL.COM

(480) 756-2600

TELEDOC AND COMPLEX CASE CONSULTATIONS
INTENSIVES TO SET UP BASELINE

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

COMPLEX NEUROLOGICAL CONDITIONS

INTENSIVES ARE DESIGNED TO BE THE STEPPING STONE FOR A FOUNDATION OF CARE. ONCE THE PATIENT HAS ESTABLISHED A BASELINE FOR TREATMENT AND OBJECTIVE/ SUBJECTIVE OUTCOMES WE SEND THE PATIENT BACK HOME WITH TREATMENT STRATEGIES FOR ONGOING CARE.
IF YOU AREN'T ALREADY, GET REGISTERED ON ERCHONIA'S DOCTOR LOCATOR LINK. WE REFER PATIENTS TO ERCHONIA DOCTORS IF THEY ARE NOT UNDER LASER CARE WHEN THEY ARE REFERRED TO US!
FOR MORE INFORMATION ABOUT INTEGRATIVE NEUROLOGICAL SEMINARS GO TO ERCHONIA.COM AND GET REGISTERED FOR ONE OF OUR OTHER EVENTS IN 2026

Q: "HOW DO I INTRODUCE MY ERCHONIA DEVICE TO MY PATIENTS?"

A: PRE- AND POST- ASSESSMENTS WITHOUT EVEN TOUCHING THE PATIENT (NEURO SIGNS, ROM ETC)

USE YOUR LASER AS A DIAGNOSTIC TOOL!
IF YOUR PATIENT IS HAVING A DIFFICULT TIME WITH A BEDSIDE EXAM TEST GET THE LASER ON THE INVOLVED REGION AND SEE IF THEY IMPROVE

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LASER CONSIDERATIONS

IF YOUR BASE "SET IT AND FORGET IT APPLICATIONS ARE NOT REACHING YOUR DESIRED TREATMENT GOALS CONSIDER THE FOLLOWING:

1. ALWAYS "SWIM UP STREAM OR DOWNSTREAM". DID YOU DO BRAIN BASED AND/OR VAGAL NERVE AND SPINAL CORD/NERVE ROOT STIMULATION PROTOCOLS?
2. LENGTHEN TREATMENT TIMES
3. SHORTEN TREATMENT TIMES (REMEMBER HORMESIS ESPECIALLY IN HIGH METABOLIC TISSUES LIKE BRAIN)
4. ADD VIOLET WAVELENGTH
5. ADD GREEN WAVELENGTH. DO ALL 3!
6. FX SCANNING VS LINE BEAM. COVER MORE SURFACE.
7. STACK THERAPIES WITH CO-ACTIVATION
8. UTILIZE THE SCANNING FREQUENCY
9. REFER THE PATIENT FOR IMAGING/OTHER TESTING FOR UNDERLYING PATHOLOGY

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PLAN AHEAD FOR 2026!

2026 Erchonia National LASER EXPO

Greenville, SC | September 24-26

The 2025 Erchonia National Laser Expo may be wrapping up, but the excitement doesn't stop here! Start planning now for next year's unforgettable event — filled with world-class education, networking, and fun.

Special Early Registration Offer for Attendees:
Register now for the 2026 Erchonia National Laser Expo & Golf Tournament and get **50% off** your full registration!

How to Claim Your Discount:
1. Go to our registration page.
2. Enter Coupon Code: **JOINUS26** at checkout.
3. Save 50% on the entire event!

SCAN HERE

⏰ Don't want to register right now? No problem! You have until the end of the November to secure your spot at this amazing rate. Just be sure to use **JOINUS26** at checkout for the savings.

After the education wraps up, get ready to kick off the festivities with our Cocktail Party, then enjoy the Golf Tournament with lunch reception to end the weekend perfectly.

Save the Date and Commit Early — you won't want to miss it!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE ENERGY FAILURE MODEL

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

First map of the brain's mitochondria may explain cognitive decline

Compared with white matter, *grey matter contains >50% more mitochondria.* Moreover, the mitochondria in grey matter are biochemically optimized for energy transformation, particularly among recently evolved cortical brain regions. ***(Older regions of the brain such as the brainstem and basal ganglia had fewer mitochondria)***

The dysfunction of brain mitochondria is increasingly viewed as a driver of neurodegenerative disorders; recent reviews tie impaired organelle dynamics to amyloid and tau build-up in Alzheimer's disease.

- **Published: 26 March 2025 NATURE**
- **A human brain map of mitochondrial respiratory capacity and diversity**
- Eugene V. Mosharov,Nature

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Science News

Why Alzheimer's attacks the brain's memory hub first

Fralin Biomedical Research Institute at VTC scientists receive Virginia grant to study how energy failure inside brain cells may damage key memory pathways. Date: **September 2, 2025**

Summary:

Virginia Tech researchers are investigating how overloaded mitochondria in the brain's memory circuits may spark early Alzheimer's damage. Their work focuses on calcium signaling and how it might trigger breakdowns in the entorhinal cortex.

A key focus of their research is mitochondria -- tiny structures inside brain cells that provide the energy needed for a variety of cellular functions in neurons including synaptic transmission. **In Alzheimer's disease, mitochondria stop working properly in the course of the disease.**

Farris and Swanger are investigating whether **mitochondria in a vulnerable memory-related circuit may become overloaded with calcium**, a key signaling chemical for multiple neuronal and synaptic processes. That overload could contribute to the early breakdown of memory circuits.

"The connection between these cells is one of the first to fail in Alzheimer's," Farris said.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

THE ENERGY MODEL

SEPTEMBER 20, 2023

Exploring the mind-mitochondria connection

Picard is a leading proponent of mitochondrial psychobiology (a phrase he coined), an emerging field that examines how psychological states like stress influence mitochondrial functions, which in turn influence mental and physical health."**The powerhouse analogy is outdated and one-dimensional and can impede science** by limiting researchers' perceptions of what mitochondria can do," says Picard, associate professor of behavioral medicine in psychiatry and neurology.

Among other roles, **mitochondria are now known to trigger cell death when needed, synthesize all circulating steroid hormones related to reproduction, and command the nucleus to turn on or turn off genes.**

"**It makes more sense to think of mitochondria as the information processors of the cell,**" he says. "They are equipped with a surprisingly wide variety of receptors to sense what's going on in the cell, they integrate all this information, and they then tell the nucleus and other organelles what to do to maintain the health of the organism."

What keeps people healthy may come down to what keeps their mitochondria healthy, Picard speculates. "The reason we have a heart and lungs is oxygen delivery," he says. "And what needs oxygen? Mitochondria, of course. You could argue that over the eons, mitochondria built an infrastructure—the human body—to feed themselves."

RESTORING BRAIN —HEALTH—

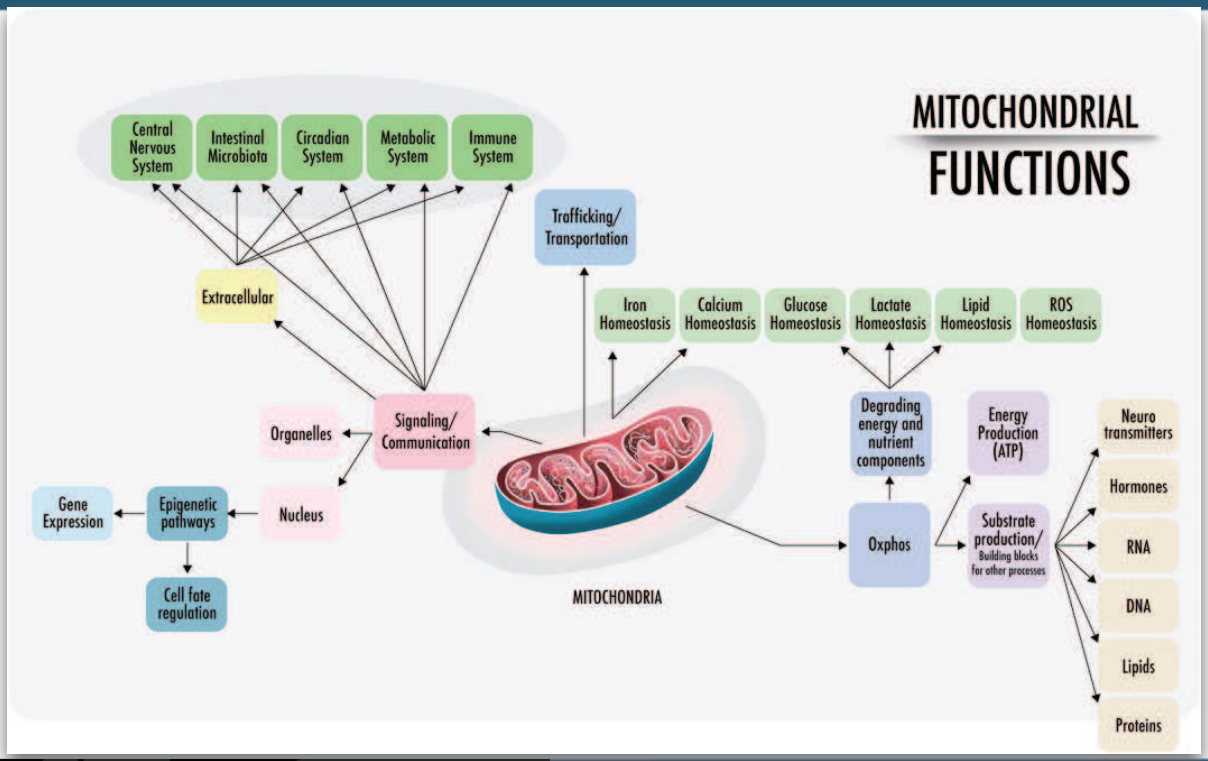
Open Access
Published: 10 August 2023

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Brain mitochondrial diversity and network organization predict anxiety-like behavior in male mice

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MORE THAN JUST THE "POWER HOUSE" OF THE CELL!



FUNCTIONAL NUTRITION FOR BRAIN HEALTH

Mitochondrial Dysfunction May Be a Cause of Age-Related Cognitive Impairment

Previous studies have found that brains lose synapses as they age, and the researchers saw this pattern in their non-human primate model, too. But when they looked at the synapses that remained, **they found evidence of a breakdown in coordination between the size of boutons and the mitochondria they contained.**

By viewing the violation of the ultrastructural size principle and **mitochondria-related failures as the key to age-related cognitive impairment**, the study **ushers in a new era for aging research.**

Front. Aging Neurosci., **12 April 2023**
Sec. Neurocognitive Aging and Behavior
Volume 15 - 2023 | https://doi.org/10.3389/fnagi.2023.1146245

Violation of the ultrastructural size principle in the dorsolateral prefrontal cortex underlies working memory impairment in the aged common marmoset (Callithrix jacchus)

Courtney Glavis-Bloom1*†

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

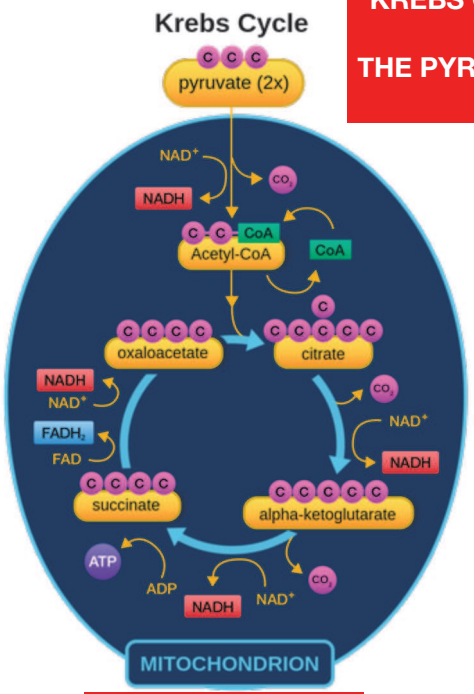
MITOCHONDRIA AND LLLT

Abstract (condensed):

Low-level laser therapy (LLLT), low energy density (0.05–50 J/cm²), significantly increases ATP production and supports mitochondrial function in cell culture models. This enhancement is primarily driven by increased activity of cytochrome c oxidase (COX), which boosts mitochondrial membrane potential (ΔΨm), triggers reactive oxygen species (ROS) signaling, and elevates both intracellular and extracellular ATP. The result is a cascade of intracellular signaling—including MAPK/ERK and calcium flux—promoting mitochondrial biogenesis and long-lasting increases in cellular energy output. **Effects are dose-dependent, following a biphasic ("Arndt–Schulz") curve, meaning optimal laser parameters are essential to maximize biostimulation without inducing adverse effects.**

Rola P. et al. (2022). *Changes in Cell Biology under the Influence of Low-Level Laser Therapy*.

FUNCTIONAL NUTRITION FOR BRAIN HEALTH



NETS ONLY 2 ATP AND 6 NADH

KREBS CYCLE OCCURS IN THE MITOCHONDRIAL MATRIX (VS ETC IN THE INNER MEMBRANE)
THE PYRUVATE AND ACETYL-CoA SHOULD COME FROM GOOD PROTEINS AND FATS RESPECTIVELY

Krebs Cycle vs Electron Transport Chain (ETC)

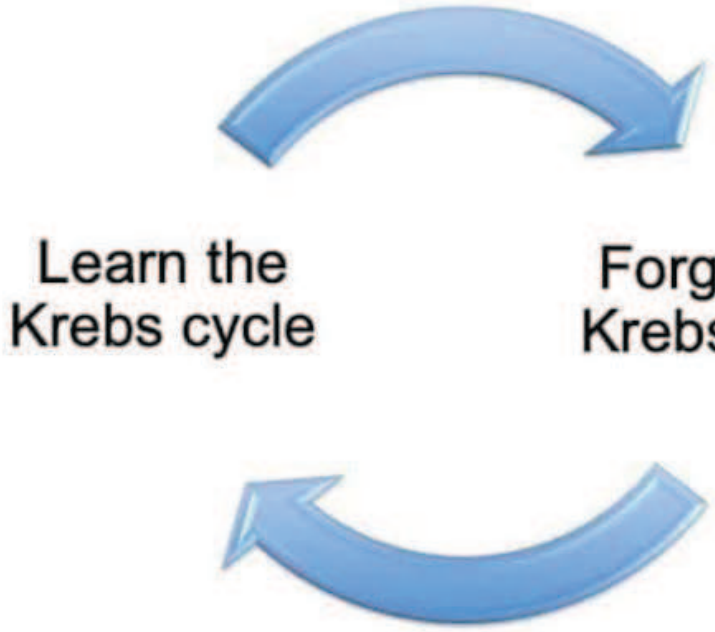
Krebs Cycle	Electron Transport Chain (ETC)
Krebs cycle occurs in the mitochondrial matrix	Electron transport chain occurs in the mitochondrial inner membrane (cristae)
Net gain of ATP per glucose molecule is 2	Net gain of ATP per glucose molecule is 34 (1 NADH=3 ATP & 1 FADH2=2 ATP)
Reduced NAD and FAD are produced as NADH and FADH2 (6 NADH & 2FADH2 per glucose molecule)	NADH and FADH2 are reoxidised to NAD and FAD
The phosphorylation for ATP synthesis is substrate level phosphorylation	The phosphorylation for ATP synthesis is oxidative phosphorylation
CO2 release or decarboxylation occurs at various steps	No decarboxylation
Chemiosmosis; not involved in ATP production	Chemiosmosis (proton gradient formation and associated ATP synthesis) involved in ATP production
Aerobic process, but oxygen not directly involved. It needs byproducts from ETC like NAD and FAD	Aerobic process; oxygen directly involved as terminal electron acceptor
Carbon dioxide is released as a waste product of these reactions.	Water is released as a waste product of these reactions.

RESTORING BRAIN —HEALTH—

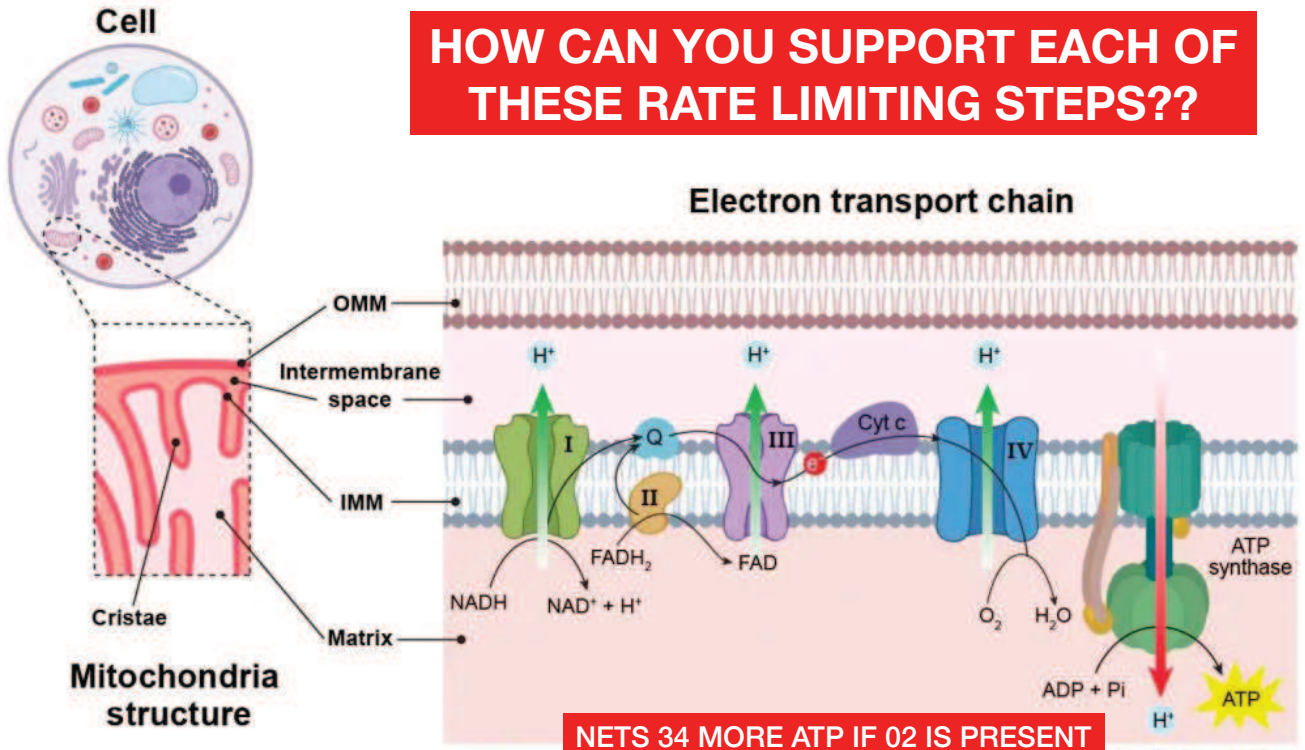
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

The REAL Krebs cycle:



MITOCHONDRIA AND THE ELECTRON TRANSPORT CHAIN



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Lin *et al. BMC Bioinformatics* (2021) 22:369
https://doi.org/10.1186/s12859-021-04285-3

BMC Bioinformatics

RESEARCH Open Access

MitoTox: a comprehensive mitochondrial toxicity database

Yu-Te Lin[1], Ko-Hong Lin[2], Chi-Jung Huang[1] and An-Chi Wei[1,2*]

From The 19th Asia Pacific Bioinformatics Conference (APBC 2021) Tainan, Taiwan. 3-5 February 2021

*Correspondence:
acwei86@ntu.edu.tw
[1] Graduate Institute of Biomedical Electronics and Bioinformatics, National Taiwan University, Taipei, Taiwan
Full list of author information is available at the end of the article

Abstract

Background: Mitochondria play essential roles in regulating cellular functions. Some drug treatments and molecular interventions have been reported to have off-target effects damaging mitochondria and causing severe side effects. The development of a database for the management of mitochondrial toxicity-related molecules and their targets is important for further analyses.

Results: To correlate chemical, biological and mechanistic information on clinically relevant mitochondria-related toxicity, a comprehensive mitochondrial toxicity database (MitoTox) was developed. MitoTox is an electronic repository that integrates comprehensive information about mitochondria-related toxins and their targets. Information and data related to mitochondrial toxicity originate from various sources, including scientific journals and other electronic databases. These resources were manually verified and extracted into MitoTox. The database currently contains over 1400 small-molecule compounds, 870 mitochondrial targets, and more than 4100 mitochondrial toxin-target associations. Each MitoTox data record contains over 30 fields, including biochemical properties, therapeutic classification, target proteins, toxicological data, mechanistic information, clinical side effects, and references.

Conclusions: MitoTox provides a fully searchable database with links to references and other databases. Potential applications of MitoTox include toxicity classification, prediction, reference and education. MitoTox is available online at http://www.mitotox.org.

MITOCHONDRIAL CONDITIONS

Table 1 Examples of mitochondrial toxicity-related drugs in MitoTox

Indication	Drug Name	Mechanism of mitochondrial toxicity	Status	Toxicity	$t_{1/2}$ (h)[a]	Protein binding (%)[a]	LD_{50} (rat)[a]
Antidiabetic	Troglitazone	Inhibition of ETC	Withdrawn, 2000	Hepatotoxicity	16–34	> 99%	1.9768 mol/kg
	Rosiglitazone	Inhibition of ETC	Approved, investigational	Cardiotoxicity	3–4	99.8%	2.4515 mol/kg
	Pioglitazone	Inhibition of ETC	Approved, investigational	Cardiotoxicity	3–7/16–24	> 99%	2.0115 mol/kg
	Ciglitazone	Inhibition of ETC	Discontinued	NA	NA	NA	NA
	Darglitazone	Inhibition of ETC	Discontinued	NA	NA	NA	NA
	Muraglitazar	Inhibition of ETC	Discontinued	NA	NA	NA	NA
	Metformin	Inhibition of complex I; uncoupling; impaired TCA cycle	Approved	Lactic acidosis	6.2	> 90%	1000 mg/kg
Anticancer	Doxorubicin	Increased ROS; mtDNA adduct; iron overload	Approved	Cardiotoxicity	20–48	74–76%	21.8 mg/kg
	Cisplatin	Inhibition of Complex I	Approved	Nephrotoxicity	0.3–0.7	> 90%	2.7612 mol/kg
Hyperlipidemia	Fenofibrate	Inhibition of complex I	Approved	Hepatotoxicity	20	~ 99%	> 2000 mg/kg
	Clofibrate	Inhibition of complex I	Approved	Hepatotoxicity	18–22	95–97%	940 mg/kg
	Ciprofibrate	Inhibition of complex I	Approved	Hepatotoxicity	NA	NA	NA

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MITOCHONDRIAL CONDITIONS

Psycho-tropic	Valproic acid	Inhibition of TCA cycle	Approved	Coma and res-piratory depres-sion	9–16	90%	670 mg/kg
	Clozapine	Inhibition of the ETC	Approved	Metabolic syndrome	8	97%	3.0838 mol/kg
	Fluoxetine	Uncoupler	Approved	CNS, GI effects	1–3 days	94.5%	2.6048 mol/kg
	Nefazodone	Inhibition of Complex I and complex IV	Withdrawn	Hepatotox-icity	2–4	>99%	2.9067 mol/kg
Analgesic	Acetami-nophen	Oxidative stress	Approved	Hepatotox-icity	1–4	25%	1944 mg/kg
	Aspirin	mPTP potentia-tion	Approved	Cardiotoxic-ity, GI effects	0.25	99.5%	920–1480 mg/kg
	Diclofenac	Inhibition of ETC and ATP synthase	Approved	Nephrotox-icity	2	>99%	3.6447 mol/kg
Antibacterial	Imipenem	Oxidative stress	Approved	Nephrotox-icity	1.3–5.1	20%	1.8089 mol/kg
	Chloram-phenicol	Depletion of iron	Approved	Aplastic anemia	1.5–3.5	50–60%	2500 mg/kg
Antiviral	Zidovudine	mtDNA replica-tion (inhibit mtDNA polymerase-γ)	Approved	Myopathy	0.5–2.9	30–38%	NA

293

MITOCHONDRIAL DAMAGE

A major objective of the present study was to investigate whether in vitro the NSAIDs, aspirin, indomethacin, diclofenac, piroxicam and ibuprofen, which feature different chemical structures, are able to inhibit mitochondrial complex I

› Chem Biol Interact. 2012 Jul 30;199(1):18-28. doi: 10.1016/j.cbi.2012.05.006. Epub 2012 May 28.

Inhibition of mitochondrial complex I by various non-steroidal anti-inflammatory drugs and its protection by quercetin via a coenzyme Q-like action

Cristian Sandoval-Acuña [1], Camilo Lopez-Alarcón, Margarita E Aliaga, Hernán Speisky

Affiliations + expand

PMID: 22652335 DOI: 10.1016/j.cbi.2012.05.006

Abstract

Mitochondrial dysfunction plays a major role in the development of oxidative stress and cytotoxicity induced by non-steroidal anti-inflammatory drugs (NSAIDs). A major objective of the present study was to investigate whether in vitro the NSAIDs, aspirin, indomethacin, diclofenac, piroxicam and ibuprofen, which feature different chemical structures, are able to inhibit mitochondrial complex I. All NSAIDs were effective inhibitors when added both, directly to mitochondria isolated from rat duodenum epithelium (50 μM) or to Caco-2 cells (250 μM). In the former system, complex I inhibition was concentration-dependent and susceptible to competition and reversion by the addition of coenzyme Q (32.5-520 μM). Based on reports suggesting a potential gastro-protective activity of quercetin, the ability of this flavonoid to protect isolated mitochondria against NSAIDs-induced complex I inhibition was evaluated. Low micromolar concentrations of quercetin (1-20 μM) protected against such inhibition, in a concentration dependent manner. In the case of aspirin, quercetin (5 μM) increased the IC50 by 10-fold. In addition, the present study shows that quercetin (5-10 μM) can behave as a "coenzyme Q-mimetic" molecule, allowing a normal electron flow along the whole electron transporting chain (complexes I, II, III and IV). The exposed findings reveal that complex I inhibition is a common deleterious effect of NSAIDs at the mitochondrial level, and that such effect is, for all tested agents, susceptible to be prevented by quercetin. Data provided here supports the contention that the protective action of quercetin resides on its, here for first time-shown, ability to behave as a coenzyme Q-like molecule.

Copyright © 2012 Elsevier Ireland Ltd. All rights reserved.

Similar articles

Non-steroidal anti-inflammatory drugs (NSAIDs) inhibit vascular smooth muscle cell proliferation via differential effects on the cell cycle.
Brooks G, Yu XM, Wang Y, Crabbe MJ, Shattock MJ, Harper JV.

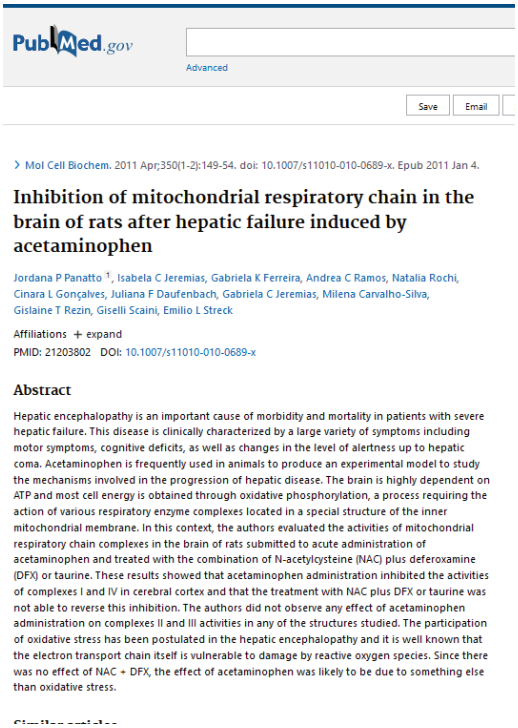
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MITOCHONDRIAL DAMAGE

The brain is highly dependent on ATP and most cell energy is obtained through oxidative phosphorylation, a process requiring the action of various respiratory enzyme complexes located in a special structure of the inner mitochondrial membrane.

These results showed that acetaminophen administration inhibited the activities of complexes I and IV in cerebral cortex



PubMed.gov

Advanced

Save Email

Mol Cell Biochem. 2011 Apr;350(1-2):149-54. doi: 10.1007/s11010-010-0689-x. Epub 2011 Jan 4.

Inhibition of mitochondrial respiratory chain in the brain of rats after hepatic failure induced by acetaminophen

Jordana P Panatto [1], Isabela C Jeremias, Gabriela K Ferreira, Andrea C Ramos, Natalia Rochi, Cinara L Gonçalves, Juliana F Daufenbach, Gabriela C Jeremias, Milena Carvalho-Silva, Gislaine T Rezin, Giselli Scaini, Emilio L Streck

Affiliations + expand

PMID: 21203802 DOI: 10.1007/s11010-010-0689-x

Abstract

Hepatic encephalopathy is an important cause of morbidity and mortality in patients with severe hepatic failure. This disease is clinically characterized by a large variety of symptoms including motor symptoms, cognitive deficits, as well as changes in the level of alertness up to hepatic coma. Acetaminophen is frequently used in animals to produce an experimental model to study the mechanisms involved in the progression of hepatic disease. The brain is highly dependent on ATP and most cell energy is obtained through oxidative phosphorylation, a process requiring the action of various respiratory enzyme complexes located in a special structure of the inner mitochondrial membrane. In this context, the authors evaluated the activities of mitochondrial respiratory chain complexes in the brain of rats submitted to acute administration of acetaminophen and treated with the combination of N-acetylcysteine (NAC) plus deferoxamine (DFX) or taurine. These results showed that acetaminophen administration inhibited the activities of complexes I and IV in cerebral cortex and that the treatment with NAC plus DFX or taurine was not able to reverse this inhibition. The authors did not observe any effect of acetaminophen administration on complexes II and III activities in any of the structures studied. The participation of oxidative stress has been postulated in the hepatic encephalopathy and it is well known that the electron transport chain itself is vulnerable to damage by reactive oxygen species. Since there was no effect of NAC + DFX, the effect of acetaminophen was likely to be due to something else than oxidative stress.

MITOCHONDRIAL DAMAGE

Unlike other organelles, mitochondria contain their own DNA (mtDNA) and must either be tolerated or corrected to preserve mitochondrial function.

Importantly, replication of damaged mtDNA can lead to stalling and introduction of mutations or genetic loss.

Dysfunctional mitochondria survive longer than healthy mitochondria. In this way, some types of dysfunctional mitochondria build up and eventually become more common than healthy ones

Bioscience Reports (2021) 41 BSR20211320
https://doi.org/10.1042/BSR20211320

PORTLAND PRESS

Review Article

Beyond base excision repair: an evolving picture of mitochondrial DNA repair

Kathrin Allkanjari[1] and Robert A. Baldock[2]

[1]Formerly: Solent University Southampton, East Park Terrace, Southampton, SO14 0YN, UK. [2]School of Natural and Social Sciences, University of Gloucestershire, Francis Close Hall, Swindon Road, Cheltenham GL50 4AZ, UK

Correspondence: R.A. Baldock (rbaldock@glos.ac.uk)

OPEN ACCESS

Mitochondria are highly specialised organelles required for key cellular processes including ATP production through cellular respiration and controlling cell death via apoptosis. Unlike other organelles, mitochondria contain their own DNA genome which encodes both protein and RNA required for cellular respiration. Each cell may contain hundreds to thousands of copies of the mitochondrial genome, which is essential for normal cellular function – deviation of mitochondrial DNA (mtDNA) copy number is associated with cellular ageing and disease. Furthermore, mtDNA lesions can arise from both endogenous or exogenous sources and must either be tolerated or corrected to preserve mitochondrial function. Importantly, replication of damaged mtDNA can lead to stalling and introduction of mutations or genetic loss. Mitochondria have adapted mechanisms to repair damaged DNA. These mechanisms rely on nuclear-encoded DNA repair proteins that are translocated into the mitochondria. Despite the presence of many known nuclear DNA repair proteins being found in the mitochondrial proteome, it remains to be established which DNA repair mechanisms are functional in mammalian mitochondria. Here, we summarise the existing and emerging research, alongside examining proteomic evidence, demonstrating that mtDNA damage can be repaired using Base Excision Repair (BER), Homologous Recombination (HR) and Microhomology-mediated End Joining (MMEJ). Critically, these repair mechanisms do not operate in isolation and evidence for interplay between pathways and repair associated with replication is discussed. Importantly, characterising non-canonical functions of key proteins and understanding the bespoke pathways used to tolerate, repair or bypass DNA damage will be fundamental in fully understanding the causes of mitochondrial genome mutations and mitochondrial dysfunction.

Introduction

Mitochondria are highly specialised and dynamic organelles required for fundamental cellular processes including ATP generation via oxidative phosphorylation during cellular respiration and the control of programmed cell death by apoptosis (reviewed in [1]). Unlike other mammalian organelles, mitochondria contain their own 16.5 kb circular DNA genome (often referred to as mitochondrial DNA or mtDNA) comprising 37 genes which in turn encode 13 peptides required for the respiratory chain complexes (I–IV) and ATP synthase [2]. A further 22 transfer RNAs and 2 ribosomal RNAs enable protein synthesis of these proteins within the mitochondria [3]. Compartmentalised in the mitochondrial matrix, each cell is estimated to contain hundreds to thousands of copies of the mitochondrial genome dependent on the cell type and between eight and ten copies per mitochondrion [3]. This genetic material is clustered and organised into distinct nucleoid structures marked predominantly by association with mitochondrial transcription factor A (TFAM) and several other mtDNA-associated proteins [4,5]. Mitochondrial nucleoids are associated with the inner mitochondrial membrane and support mtDNA packaging, replication and mediate signalling (reviewed in [6])

Received: 05 August 2021
Revised: 29 September 2021
Accepted: 04 October 2021

Accepted Manuscript online: 05 October 2021
Version of Record published: 14 October 2021

© 2021 The Author(s). This is an open access article published by Portland Press Limited on behalf of the Biochemical Society and distributed under the Creative Commons Attribution License 4.0 (CC BY).

1

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MITOCHONDRIAL DAMAGE

Mitochondrial disorders comprise a molecular and clinically diverse group of diseases that are associated with mitochondrial dysfunction leading to multi-organ disease.

However, the therapeutic approaches for mitochondrial disorders have lagged behind with treatment options limited mainly to symptom specific therapies and supportive measures.

Contents lists available at ScienceDirect

Molecular Genetics and Metabolism

journal homepage: www.elsevier.com/locate/ymgme

Review article

Clinical trials in mitochondrial disorders, an update

Mohammed Almannai[a], Ayman W. El-Hattab[b], May Ali[c], Claudia Soler-Alfonso[c,d], Fernando Scaglia[c,d,e,*]

[a] Section of Medical Genetics, Children's Hospital, King Fahad Medical City, Riyadh, Saudi Arabia
[b] Department of Clinical Sciences, College of Medicine, University of Sharjah, Sharjah, United Arab Emirates
[c] Department of Molecular and Human Genetics, Baylor College of Medicine, Houston, TX, USA
[d] Texas Children's Hospital, Houston, TX, USA
[e] Joint BCM-CUHK Center of Medical Genetics, Prince of Wales Hospital, Shatin, Hong Kong

ARTICLE INFO

Keywords:
Mitochondria
Randomized clinical trials
Mitochondrial augmentation
Antioxidants
Mitochondrial biogenesis

ABSTRACT

Mitochondrial disorders comprise a molecular and clinically diverse group of diseases that are associated with mitochondrial dysfunction leading to multi-organ disease. With recent advances in molecular technologies, the understanding of the pathomechanisms of a growing list of mitochondrial disorders has been greatly expanded. However, the therapeutic approaches for mitochondrial disorders have lagged behind with treatment options limited mainly to symptom specific therapies and supportive measures. There is an increasing number of clinical trials in mitochondrial disorders aiming for more specific and effective therapies. This review will cover different treatment modalities currently used in mitochondrial disorders, focusing on recent and ongoing clinical trials.

1. Introduction

Primary mitochondrial disorders represent an expanding group of clinically heterogeneous diseases that are associated with mitochondrial dysfunction due to underlying mutations in mitochondrial DNA (mtDNA) or nuclear encoded mitochondrial genes. mtDNA codes for 13 subunits of the oxidative phosphorylation (OXPHOS) complexes, 22 transfer RNAs (tRNA), and 2 ribosomal RNAs (rRNA). Therefore, mutations in mtDNA affect OXPHOS complexes production or mitochondrial translational machinery. The majority of mitochondrial proteins are encoded by nuclear genes, including most of the subunits of OXPHOS complexes, and other proteins involved in OXPHOS complexes assembly, mtDNA maintenance, mtDNA genes expression, mitochondrial fission and fusion, in addition to several cofactors required for proper mitochondrial function [1,2]. As mitochondria are important for energy production through OXPHOS complexes, energy failure contributes to multi-organ involvement usually seen in mitochondrial disorders. Mitochondria also carry other biological functions, including calcium homeostasis, steroid synthesis, and apoptosis and these also likely contribute to the pathophysiology in various mitochondrial disorders [1,3].

The advent of deep, high-throughput, in-parallel DNA sequencing technologies has enabled a better understanding of the bases and mechanisms of a growing list of mitochondrial disorders. In a recent nosology of inborn errors of metabolism, 244 well-characterized disorders are listed under the category of mitochondrial disorders of energy metabolism (http://www.iembase.org/iem-nosology.asp; accessed on 4/18/2020) [4]. Furthermore, a recently published multicenter epidemiological study has provided invaluable data on the natural history of mitochondrial disorders [5] that could lay the foundation for planning randomized clinical trials.

Treatment of mitochondrial disorders has been challenging though. These challenges stem from the ubiquitous nature of mitochondria, genetic complexity with dual genome control, the presence of heteroplasmy associated with most variants in mtDNA, and the heterogeneous clinical presentations associated with multi-organ involvement in various mitochondrial disorders [6]. Therapeutic approaches have been mostly limited to symptom specific therapies and supportive measures including treatment of catabolic stress and infections, exercise, and the use of vitamins, cofactors, and antioxidants [7,8].

Recently, the number of preclinical and clinical trials in mitochondrial disorders has been increasing. These efforts will open venues for more specific and effective treatment modalities for mitochondrial disorders. In this review, various therapeutic modalities currently used or trialed in different mitochondrial disorders are presented, with a focus on recent and ongoing clinical trials.

* Corresponding author at: Department of Molecular and Human Genetics, Baylor College of Medicine and Texas Children's Hospital, Mark Wallace Tower, Suite 1560,6701 Fannin Street, Houston, Texas, 77030, USA Street, Houston, Texas, 77030, USA
E-mail address: fscaglia@bcm.edu (F. Scaglia).

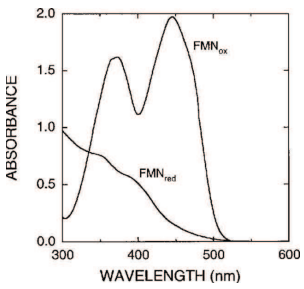
THE MITOCHONDRIAL SOLUTION…

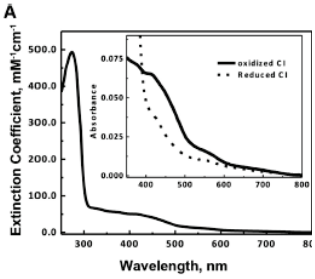
COMPLEX 1 & 2

***FLAVIN MONONUCLEOTIDE* (*FMN*) IS A COMPONENT OF *COMPLEX* I, WHEREAS FLAVIN ADENINE DINUCLEOTIDE (*FAD*) IS PRESENT IN *COMPLEX* II**

Violet and Blue light target Flavoproteins (flavin means blue light-absorbing).

VIOLET LASER COMPLEX 1&2





tion due to the Coulombic repulsion with the additional 5 negative charges distributed over Fe/S clusters. In the donor electronic state (the initial state), the donor tunneling orbital is mostly localized on the donor with an energy at ~3 eV higher than the bridge valence band in the middle of the band gap together with other occupied d orbitals of the donor. Similarly, in the acceptor state (the final state), the acceptor

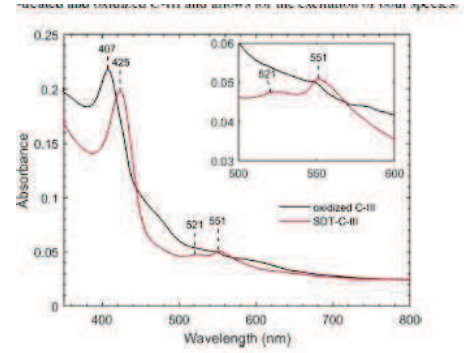
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

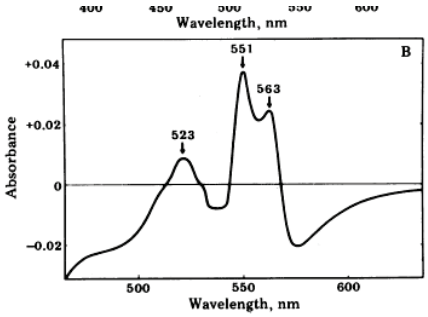
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE MITOCHONDRIAL SOLUTION…

ERCHONIA eCommunity Education

GREEN LASER COMPLEX 3





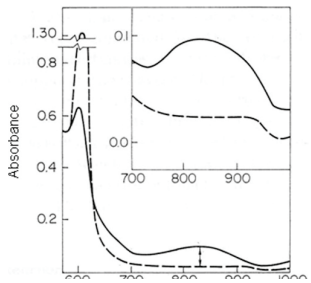
COMPLEX 3

CYTOCHROME BC 1 COMPLEX

THE MITOCHONDRIAL SOLUTION…

ERCHONIA eCommunity Education

RED LASER COMPLEX 4



Biochem Mol Biol Int. 1994 Oct;34(4):817-26.

Increase in <--H+/e- ratio of the cytochrome c oxidase reaction in mitochondria irradiated with helium-neon laser

D Pastore [1], M Greco, V A Petragallo, S Passarella

Affiliations

PMID: 7866309

Abstract

In order to gain a degree of insight into the mitochondrial component/s responsible of the mitochondria-red light interaction, isolated rat liver mitochondria were irradiated with a Helium-Neon laser (energy dose 2 Joules/cm2, light power 10 mW) and measurements made of the activity of cytochrome c oxidase. A low, but statistically significant increase in the oxygen uptake was found, as polarographically measured, in the presence of rotenone and antimycin A, with ascorbate and TMPD used as substrate pair. Measurements were also made both of the electron transfer and of proton pumping activity: as a result of a major stimulation in the proton pumping activity, about 55% increase of <--H+/e- ratio was found in irradiated mitochondria.

Related information

PubChem Compound (MeSH Keyword)

COMPLEX 4

CYTOCHROME C OXIDASE

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EMERGING EVIDENCE OF PBM WAVELENGTHS EFFECTS ON MITOCHONDRIAL COMPLEXES

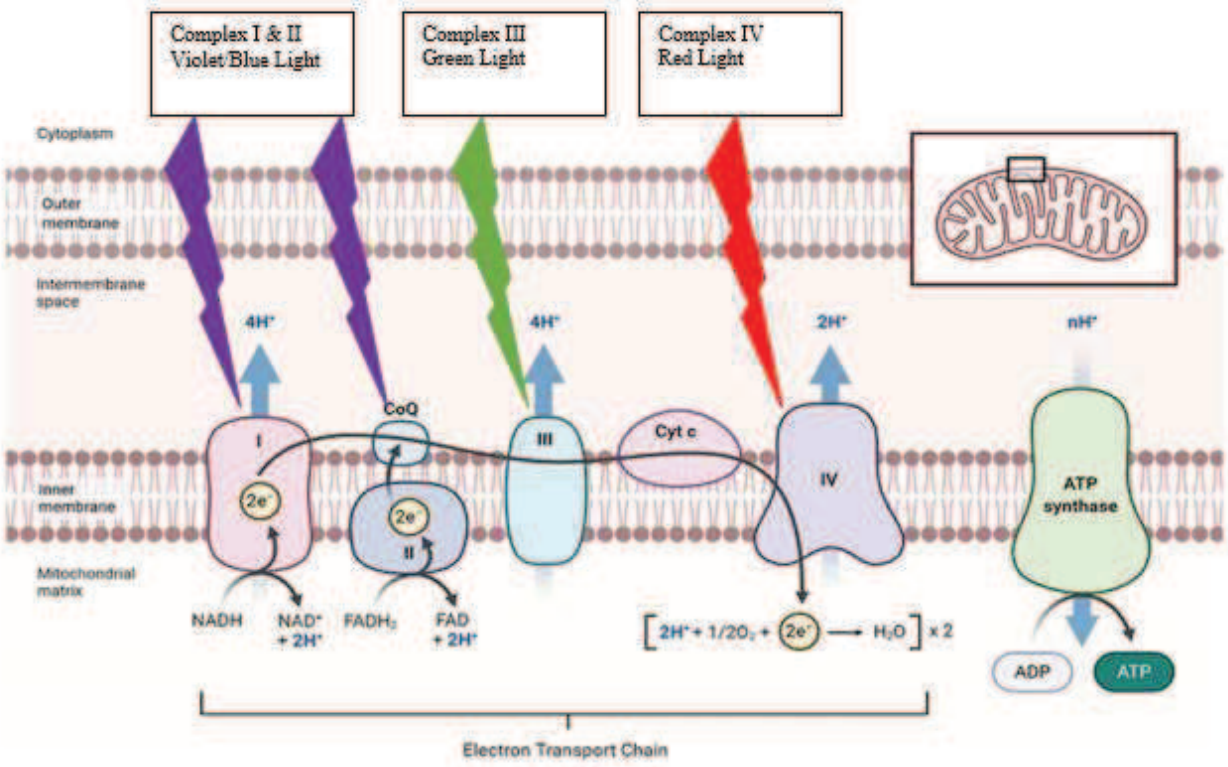
PBM Evidence Table

	Wavelength	Complex / Target	Model	Endpoints	Citation
1	Red (633–635 nm)	Complex IV (CCO)	Cells, isolated mitochondria, retina	↑ATP, ↑ΔΨm, ↑ETC flux, NO photodissociation	Karu 1995; Hamblin 2018; Chaudary 2020; Mezzacappo 2025; Heinig 2020
2	Red (635–670 nm)	AMPK → PGC-1α → NRF-1 → TFAM	SCI, brain models	Mitochondrial biogenesis, transcriptional up-regulation	Zhu 2022; Cheng 2012; Zong 2024
3	Green (520 nm)	Cytochrome c / Complex III overlap	Isolated yeast mitochondria	↑ATP synthesis at 520 & 550 nm	Khmelinskii 2022
4	Green (532 nm)	ΔΨm, proliferation	Human adipose-derived stem cells	↑ΔΨm, ↑cell growth	Anwer 2012; Tamimi 2022
5	Violet (405 nm)	FMN (Complex I), FAD (Complex II)	Platelets, retina, in vitro cells	Metabolic reprogramming, antioxidant enzyme response; hormetic	Jana 2023; Serrage 2019; Heinig 2020; Sommer 2019

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

FUNCTIONAL NUTRITION FOR BRAIN HEALTH



DO NOT EXCEED 10 J/CM2 TO THE BRAIN!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

WHICH LASER SHOULD I GET…

ERCHONIA eCommunity Education

GOOD: A SINGLE ERCHONIA HAND HELD LASER. TARGET THE EVRL IF SO.

BETTER: THE BASE STATION OR BASE STATION BUILD UP. "THE TRIDENT" 1 XLR8, 1 EVRL, 1 GVL

BEST: STACK HAND HELDS WITH FX SCANNING LASERS. DO A RENTAL PROGRAM WITH ONE OF THE BASE STATION DEVICES TO PAY FOR BOTH THE FX AND BASE STATION!

303

Home › Journal of NeuroEngineering and Rehabilitation › Article

Devices used for photobiomodulation of the brain—a comprehensive and systematic review

Review | Open access | Published: 10 April 2024

Volume 21, article number 53, (2024) Cite this article

SYSTEMIC REVIEW OF DEVICES USED IN RESEARCH FOR PHOTOBIOMODULATION OF THE BRAIN. #1 HAND HELD TRUE LASERS!!!

Functional near-infrared spectroscopy (NIRS) studies have shown that PBM can effectively increase cerebral oxygenation, which has a great impact on cognitive tasks, such as memory and attention, among others [8,9,10,11,12,13]. For traumatic brain injury (TBI), there are several case studies in which patients exhibited improvement in symptoms, measured through standard neurological tests and self-assessments [14,15,16,17,18]. PBM has also reduced depression symptoms in patients diagnosed with depression disorder [19,20,21,22]. For neurodegenerative conditions, such as dementia, Alzheimer's disease (AD), and Parkinson's disease (PD), several studies showed improvements in cognition, quality of life, and clinical signs of these conditions [23,24,25,26]. Specifically, a randomized controlled trial on the effects of transcranial PBM in patients diagnosed with PD showed improvements in gait, further establishing the relevance of this therapy for neurodegenerative conditions [27].

Laser handpiece

The most common type of device was the laser handpieces, concerning 14 devices and 39% of all reports (n = 109). This category of devices consists in a handpiece that is manually handled by an operator and can be placed anywhere on the head. These devices are generally of simple construction, requiring only a control unit and a fibre optic cable that connects to the handpiece.

304

ERCHONIA Healing Light. Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LASER PROFIT CENTER IDEA

ERCHONIA eCommunity Education

BASE STATION RENTAL: XLR8 $300/WEEK, EVRL $400/WEEK OR GVL $500/WEEK = COVERS YOUR COST TO PAY OFF A BASE STATION AND FX LASER PURCHASE! GET ONE OF EACH FOR YOUR BASE STATION

© 2022 Erchonia Corporation

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

Mitochondrial Support Supplement Guide

Supplement	Recommended Daily Dose	Suggested Timing
Coenzyme Q10 (Ubiquinol)	100–300 mg	With breakfast or lunch (with fat)
Lipoic Acid (R-ALA)	300–600 mg	Between meals or empty stomach
Omega-3 Fatty Acids (EPA/DHA)	1000–3000 mg (EPA/DHA combined)	With food (preferably with fat)
Pyrroloquinoline Quinone (PQQ)	10–20 mg	Morning (may increase alertness)
N-Acetyl Cysteine (NAC)	600–1200 mg (divided doses)	Empty stomach or between meals
Acetyl-L-Carnitine (ALCAR)	500–2000 mg	Morning or early afternoon
NAD+ Precursors (NR/NMN)	250–500 mg (NR or NMN)	Morning
Quercetin	250–1000 mg	With food
Green Tea Extract (EGCG)	200–500 mg EGCG	With breakfast or lunch
Resveratrol	100–250 mg (up to 500 mg therapeutic)	Morning with food
Creatine Monohydrate	3–5 g	Any time (with carbs enhances absorption)

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

NAD+ IV vs. Methylene Blue: Clinical Summary (BOTH SUPPORT NADH/COMPLEX 1)

Overview

NAD+ and Methylene Blue (MB) are both potent mitochondrial therapies, but they differ in mechanisms, applications, and delivery. This summary provides a comparison of clinical use, dosing, and when to choose one over the other.

NAD+ IV Therapy

Mechanism:

- Coenzyme essential for ATP production, sirtuin activation, DNA repair, and redox balance.
- Declines with age, illness, and neurodegeneration.

Administration:

- Dose: 250–750 mg/infusion (up to 1000 mg in special cases)
- Infusion Duration: 1.5–4 hours
- Frequency: Daily to every other day initially; maintenance weekly to monthly

Indications:

- Fatigue, cognitive decline, addiction recovery, PTSD, depression, longevity

Methylene Blue

Mechanism:

- **Acts as a redox agent in the ETC, bypasses damaged Complex I & III.**
- Enhances ATP, neuroprotection, antioxidant defense, and memory.

Administration:

- Oral Dose: 0.5–2 mg/kg (nootropic/mitochondrial support)
- IV Dose: 1–3 mg/kg (clinical use)
- Frequency: 3–5x/week; avoid daily long-term use unless monitored

Indications:

- Cognitive enhancement, early dementia, mitochondrial dysfunction, post-infectious fatigue, neurodegeneration

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

When to Use Each

Condition	Prefer NAD+ IV	Prefer Methylene Blue
Severe fatigue	Yes Mito support	Yes Oral alternative
Neurodegeneration	Yes DNA/sirtuins	Yes ETC & antioxidants
Brain fog/Concussion	Yes Repair/energy	Yes Oxygenation/focus
Anti-aging/Longevity	Yes Broad support	Yes Synergistic
Cognitive enhancement	Maybe Moderate effect	Yes Strong effect
Addiction recovery	Yes Best choice	Maybe Adjunct only
Depression/Anxiety	Yes Adrenal stress	Caution Avoid w/ SSRIs

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Contraindications of Methylene Blue and NAD+

METHYLENE BLUE (MB)

Absolute Contraindications:

1. G6PD Deficiency - Risk of hemolytic anemia. Always screen before use.
2. Serotonin Syndrome Risk - Potent MAOI. Do NOT use with SSRIs, SNRIs, TCAs, MAOIs, MDMA, etc.
3. Known Hypersensitivity or Allergy to MB.
4. Severe Renal Impairment - Accumulation risk.
5. Pregnancy (especially 2nd/3rd trimester) - Risk of fetal toxicity.

Relative Contraindications / Cautions:

- Liver dysfunction.
- Concurrent MAOI use.
- High doses (>5 mg/kg) increase neurotoxicity risk.
- Elderly or neurologically sensitive individuals.

NAD+ (Nicotinamide Adenine Dinucleotide)

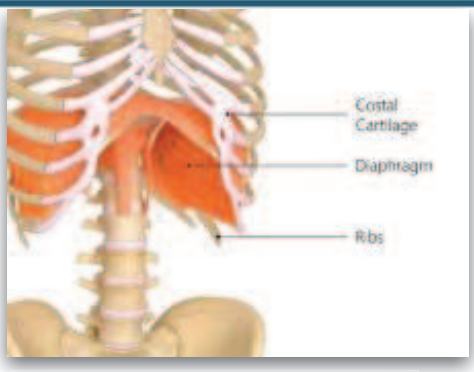
Absolute Contraindications:

1. Known allergy or hypersensitivity.
2. Severe hypotension or bradycardia (especially with rapid infusion).

Relative Contraindications / Cautions:

- Asthma or COPD - Risk of histamine-induced bronchospasm.
- Anxiety or panic disorders - May temporarily worsen symptoms.
- Active cancer (controversial use).
- Methylation SNPs (e.g., MTHFR, COMT).
- Adrenal fatigue or chronic fatigue - Start low and slow.

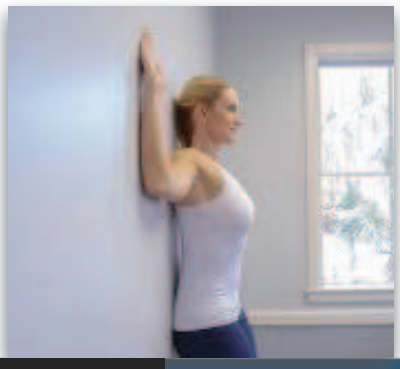
POSTURAL CONSIDERATIONS



WIM HOF
(MORE SYMPATHETIC RESILIENCE)

VS.

BOX BREATHING 448/478
(MORE PARASYMPATHETIC RELAXATION)



ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited

HYPERBARIC OXYGEN THERAPY

1.3 ATA (4.4 PSI)

SOME OF THE BENEFITS OF HBOT:
- INCREASED OXYGEN TO THE BRAIN AND BODY
- PROMOTES DNA SIGNALING (PROMOTING GOOD GENES AND TURNING OFF BAD GENES)
- INCREASED BLOOD FLOW AND CIRCULATION
- NEW BLOOD VESSELS AND PERFUSION TO TISSUES
- BOOST IMMUNE RESPONSES
- HELPS FIGHT INFECTIONS
- WOUND HEALING/BURNS AND OTHER CONNECTIVE TISSUE INJURIES
- PROMOTES NEW BLOOD CELLS AND HELPS WITH ANEMIA
- PROMOTES STEM CELL PRODUCTION AND PROLIFERATION
- STIMULATE MITOCHONDRIAL OUTPUT FOR ENERGY PRODUCTION (ATP)
- REDUCES PAIN AND INFLAMMATION
- PREVENTS REPERFUSION INJURY (EX. POST STROKE)
- PROMOTES ANTI-OXIDANT SYSTEMS
- PROMOTES GREY AND WHITE MATTER STRUCTURES OF THE CNS
- HELPS WITH INJURIES RELATED TO GASES (IE. SCUBA DIVING OR CO/SMOKE INHALATION)
- HELPS WITH INJURIES RELATED TO RADIATION, VACCINE AND CHEMOTHERAPY
- CONDITION SPECIFIC INDICATIONS SUCH AS TBI, STROKE, CHILDHOOD DEVELOPMENT AND MUCH MORE

OTHER OXYGEN SOURCE CONSIDERATIONS:
EWOT
CONCENTRATORS
FNOR COMBO (SEE VIDEO)/POSTURAL THERAPIES
COUPLED RIB CMT
AUTONOMIC REGULATION/VAGAL STIM

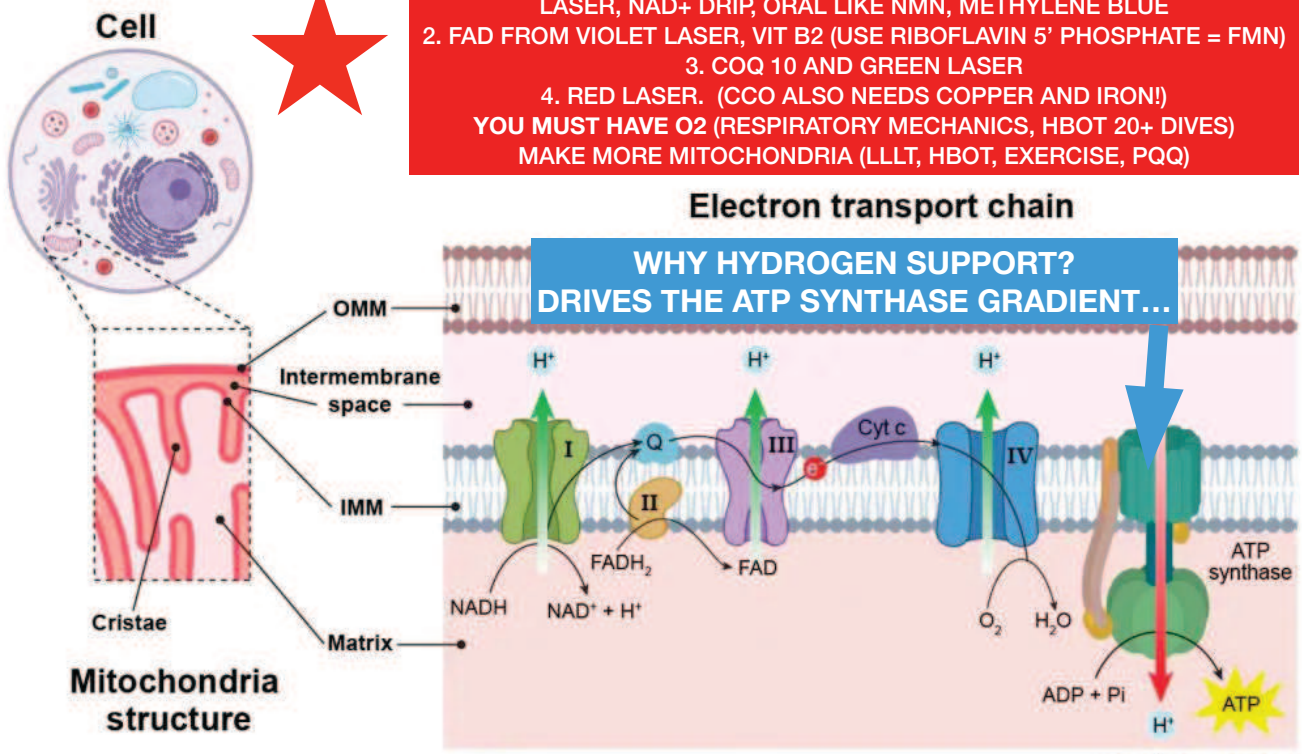
DON'T FORGET TO ADDRESS ANEMIA PATTERNS

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

MITOCHONDRIA AND THE ELECTRON TRANSPORT CHAIN

SUMMARY: 1. NADH FROM KREB'S/GOOD FATS AND PROTEINS, VIOLET LASER, NAD+ DRIP, ORAL LIKE NMN, METHYLENE BLUE
2. FAD FROM VIOLET LASER, VIT B2 (USE RIBOFLAVIN 5' PHOSPHATE = FMN)
3. COQ 10 AND GREEN LASER
4. RED LASER. (CCO ALSO NEEDS COPPER AND IRON!)
YOU MUST HAVE O2 (RESPIRATORY MECHANICS, HBOT 20+ DIVES)
MAKE MORE MITOCHONDRIA (LLLT, HBOT, EXERCISE, PQQ)



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

OPTIMIZATION

INTEGRATIVE CLINICAL NEUROLOGY AND LOW LEVEL LASER IS NOT JUST BRAIN INJURY RECOVERY AND NEURODEGENERATIVE CONDITIONS. OPTIMIZE YOUR BRAIN AND THE BRAINS OF YOUR PATIENTS!

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

LLLT AND BRAIN OPTIMIZATION

J Transl Med
2023 Feb 22;21(1):135. doi: 10.1186/s12967-023-03988-w.

Advances in photobiomodulation for cognitive improvement by near-infrared derived multiple strategies

Conclusion

1 # 1

Cognitive function is an advanced neurological function, which refers to the ability of our brains to form judgement and conclusions from information provided. As a common manifestation of brain diseases, cognitive dysfunction has a causal relationship with pathological changes in neurons and neural networks. To improve cognitive function, direct intervention in targeted encephalic regions or lesion sites may be the most effective therapeutic strategy.

Compared with traditional systemic administrations of drugs, photobiomodulation is a novel and noninvasive physical therapy that utilizes the energy effects of light. Photobiomodulation attracts much attention due to its benefits, ranging from safety, flexibility and operability.

However, the combined therapy of photosensitive nanoparticles and lasers, which can accurately regulate neural activity and improve cognitive function, is a new direction in drug delivery development. Presently, it is far from clinical use, but undoubtedly, ongoing further research will make these strategies to be clinically available in the foreseeable future.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

TRANSCRANIAL LIGHT THERAPIES

Rev Neurosci

2023 Mar 17. doi: 10.1515/revneuro-2023-0003. Online ahead of print.

A systematic review of the effects of transcranial photobiomodulation on brain activity in humans

Marjorie Dole [1], Vincent Auboiroux [2], Lilia Langar [3], John Mitrofanis [1 4]

Abstract

In recent years, transcranial photobiomodulation (tPBM) has been developing as a promising method to protect and repair brain tissues against damages. The aim of our systematic review is to examine the results available in the literature concerning the efficacy of tPBM in changing brain activity in humans, either in healthy individuals, or in patients with neurological diseases. Four databases were screened for references containing terms encompassing photobiomodulation, brain activity, brain imaging, and human. We also analysed the quality of the included studies using validated tools. **Results in healthy subjects showed that even after a single session, tPBM can be effective in influencing brain activity**. In particular, the different transcranial approaches - using a focal stimulation or helmet for global brain stimulation - seemed to act at both the vascular level by increasing regional cerebral blood flow (rCBF) and at the neural level by changing the activity of the neurons. In addition, studies also showed that even a **focal stimulation was sufficient to induce a global change in functional connectivity across brain networks.** Results in patients with neurological disease were sparser; nevertheless, they indicated that tPBM could improve rCBF and functional connectivity in several regions. Our systematic review also highlighted the heterogeneity in the methods and results generated, together with the need for more randomised controlled trials in patients with neurological diseases. In summary, tPBM could be a promising method to act on brain function, but more consistency is needed in order appreciate fully the underlying mechanisms and the precise outcomes.

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
—HEALTH—

Abstract

LASERS AND OPTIMIZATION

The parameters used were categorized into those with positive effects or no effects on muscle performance and recovery. Randomized controlled trials and case-control studies in both healthy trained and untrained participants, and elite athletes were retrieved from MEDLINE up to 2016. **Performance metrics included fatigue, number of repetitions, torque, hypertrophy; measures of muscle damage and recovery such as creatine kinase and delayed onset muscle soreness.** Searches retrieved 533 studies, of which 46 were included in the review (n = 1045 participants). Studies used single laser probes, cluster of laser diodes, LED clusters, mixed clusters (lasers and LEDs), and flexible LED arrays. Both red, NIR, and red/NIR mixtures were used. **PBM can increase muscle mass gained after training, and decrease inflammation and oxidative stress in muscle biopsies. We raise the question of whether PBM should be permitted in athletic competition by international regulatory authorities.**

J Biophotonics

2016 Dec;9(11-12):1273-1299. doi: 10.1002/jbio.201600176. Epub 2016 Nov 22.

Photobiomodulation in human muscle tissue: an advantage in sports performance?

KIRK GAIR SEMINAR
FOR SPORTS RECOVERY
AND PERFORMANCE

Cleber Ferraresi [1 2 3], Ying-Ying Huang [1 2], Michael R Hamblin [1 2 4]

Affiliations expand

PMID: 27874264 PMCID: PMC5167494 DOI: 10.1002/jbio.201600176

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN OPTIMIZATION, INJURY CARE, RECOVERY AND MORE…

WORLD CHAMPION AND OLYMPIC GOLD MEDALIST

HELEN MAROULIS

AND NOW THE FIRST EVER FEMALE USA WRESTLER TO QUALIFY AND MEDAL AT 3 OLYMPICS AND WIN 3 WORLD CHAMPIONSHIPS!

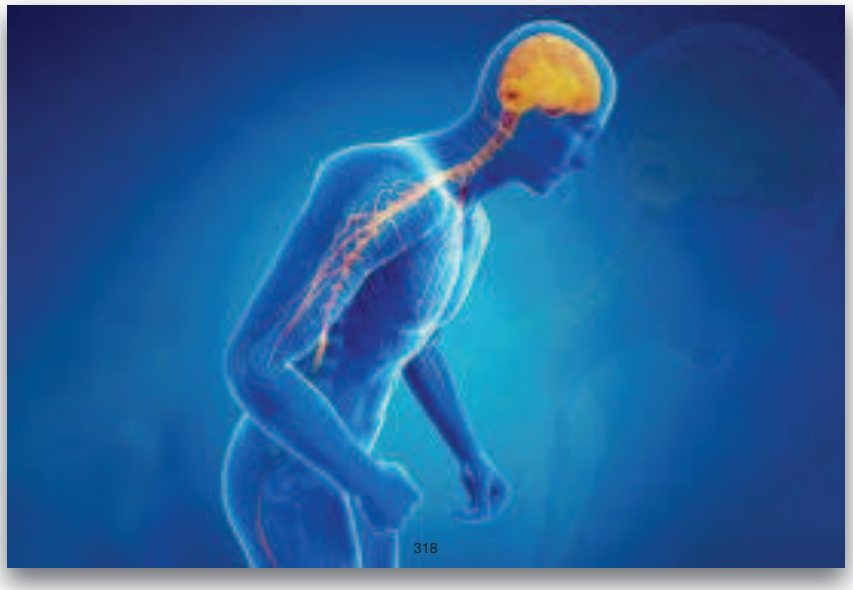
ALWAYS CONSIDER "STACKING" YOUR LASER WITH OTHER THERAPIES AND MODALITIES TO OPTIMIZE METABOLIC CAPACITY AND MAXIMIZE OUTCOMES BOTH IN OFFICE AND INCREASING THEIR BRAIN AND BODY FUNCTION FOR THEIR TARGET GOALS AND DAILY ACTIVITY

ERCHONIA® Healing Light, Perfected.

317

RESTORING BRAIN —HEALTH—

PARKINSON'S DISEASE



318

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PARKINSON'S EARLY WARNING SIGNS

Although Parkinson's disease is often thought of as someone with tremors, rigidity and slow movement, those are actually more typical in later stages when there has been over 50% dopamine neuron production loss (like bone density loss on an X-ray) and an overwhelmed energetic crisis over reaction in the basal ganglia. Here is a constellation of symptoms often present in earlier "warning signs" of Parkinson's:

- CONSTIPATION
- LOSS OF SMELL/OLFACTORY CHANGES
- MICROGRAPHIA (SMALLER HAND WRITING. LESS LEGIBLE)
- ANXIETY
- DEPRESSION
- ASYMMETRIC/UNRESOLVING SHOULDER PAIN AND STIFFNESS
- RAPID EYE MOVEMENT (REM) SLEEP BEHAVIOR DISORDER (CAN'T INHIBIT DREAM MOTOR FUNCTION SO IT GETS "ACTED OUT"
AND MAY HARM YOURSELF OR PARTNER)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Journal of Clinical Medicine
MDPI

J Clin Med. 2023 Apr 13;12(8):2846. doi: 10.3390/jcm12082846

A Randomized Placebo-Controlled Study of a Transcranial Photobiomodulation Helmet in Parkinson's Disease: Post-Hoc Analysis of Motor Outcomes

Claire McGee [1], Ann Liebert [2,3,4], Brian Bicknell [4], Vincent Pang [4], Vivian Isaac [5], Craig S McLachlan [6], Hosen Kiat [4,6,7,8,9], Geoffrey Herkes [8,10,*]

Abstract

Emerging evidence is increasingly supporting the use of transcranial photobiomodulation (tPBM) to improve symptoms of neurodegenerative diseases, including Parkinson's disease (PD). The objective of this study was to analyse the safety and efficacy of tPBM for PD motor symptoms. The study was a triple blind, randomized placebo-controlled trial with 40 idiopathic PD patients receiving either active tPBM (635 nm and 810 nm diodes) or sham tPBM for 24 min per day (56.88J), six days per week, for 12 weeks. **The primary outcome measures were treatment safety and a 37-item MDS-UPDRS-III (motor domain) assessed at baseline and 12 weeks.** Individual MDS-UPDRS-III items were clustered into sub-score domains (facial, upper-limb, lower-limb, gait, and tremor). **The treatment produced no safety concerns or adverse events,** apart from occasional temporary and minor dizziness. There was no significant difference in total MDS-UPDRS-III scores between groups, presumably due to the placebo effect. Additional analyses demonstrated that facial and lower-limb sub-scores significantly improved with active treatment, while gait and lower-limb sub-scores significantly improved with sham treatment. **Approximately 70% of participants responded to active treatment (≥5 decrease in MDS-UPDRS-III score) and improved in all sub-scores, while sham responders improved in lower-limb sub-scores only. tPBM appears to be a safe treatment and improved several PD motor symptoms in patients that responded to treatment. tPBM is proving to be increasingly attractive as a possible non-pharmaceutical adjunct therapy.**

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PARKINSON'S (PD) AND LLLT

EXCELLENT REVIEW ARTICLE (FREE). SOME HIGHLIGHTS INCLUDE: NO LIGHT DEVICE HAS BEEN SHOWN TO "PENETRATE" TO THE SUBSTANTIA NIGRA AND THAT ABSCOPAL STUDIES HAVE BEEN SHOWN TO BE AS EFFECTIVE AS TCT IN PD PATIENTS. DOSE PARAMETERS ARE MENTIONED INLCUDING THE NEED FOR "NON-THERMAL" PHOTOBIOMODULATION AND BELOW 500 mW DEVICES

Abstract

Parkinson's disease is the second most common neurodegenerative disease and is increasing in incidence. The combination of motor and non-motor symptoms makes this a devastating disease for people with Parkinson's disease and their care givers. Parkinson's disease is characterised by mitochondrial dysfunction and neuronal death in the substantia nigra, a reduction in dopamine, accumulation of α-synuclein aggregates and neuroinflammation. The microbiome–gut–brain axis is also important in Parkinson's disease, involved in the spread of inflammation and aggregated α-synuclein. The mainstay of Parkinson's disease treatment is dopamine replacement therapy, which can reduce some of the motor signs. There is a need for additional treatment options to supplement available medications. Photobiomodulation (PBM) is a form of light therapy that has been shown to have multiple clinical benefits due to its enhancement of the mitochondrial electron transport chain and the subsequent increase in mitochondrial membrane potential and ATP production. PBM also modulates cellular signalling and has been shown to reduce inflammation. Clinically, PBM has been used for decades to improve wound healing, treat pain, reduce swelling and heal deep tissues. Pre-clinical experiments have indicated that PBM has the potential to improve the clinical signs of Parkinson's disease and to provide neuroprotection. This effect is seen whether the PBM is directed to the head of the animal or to other parts of the body (remotely). A small number of clinical trials has given weight to the possibility that using PBM can improve both motor and non-motor clinical signs and symptoms of Parkinson's disease and may potentially slow its progression.

J Pers Med. **2024 Jan 19**;14(1):112. doi: 10.3390/jpm14010112

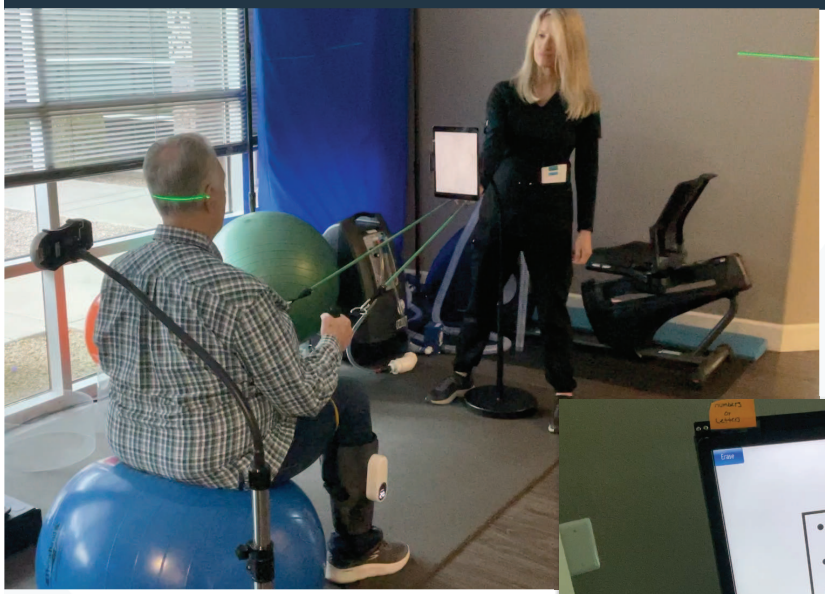
Parkinson's Disease and Photobiomodulation: Potential for Treatment

Brian Bicknell [1,]

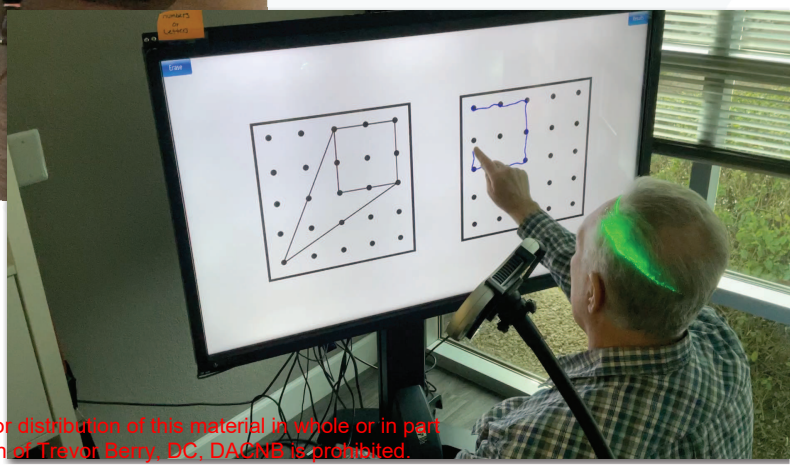
RESTORING BRAIN —HEALTH—

STACKING MODALITIES

ERCHONIA eCommunity Education



DICK S. 1/7/25
RIGHT HANDED MALE
WORKING DX:
ESSENTIAL TREMORS
PARKINSON'S LEFT BG/RIGHT UE



ON DAY 3 OF HIS 1 WEEK INTENSIVE HE CAME IN AND SAID HE WAS ABLE TO EAT AND BRUSH HIS TEETH WITH HIS RIGHT HAND AGAIN AND HE ACTUALLY BALANCED HIS CHECK BOOK LEGIBLY

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

PARKINSON'S DISEASE

FIX BARRIER SYSTEMS/LEAKY GUT PROTOCOL/VAGAL NERVE STIMULATION

ADDRESS DOPAMINE NEUROCHEMISTRY/CO-FACTORS/MICRONUTRIENTS

LLLT GI AND SCM vagal setting (10-10-10-10). VAGAL PROTOCOL (2min SCM while percussor gut then 2 min gut). Also consider SCM stem with laser while doing motor exercises/rehab/"unfreezing

LLLT MASTER BRAIN SETTING 1-10-40-60 OR BRAIN RESTORE SETTING (10-40-10-40) (BIAS DIODES TO FRONTAL CORTEX AND CONTRALATERAL CEREBELLUM USING FX) Avoid Beta Hz oscillations (around 13-30Hz). Consider co-activation with targeting saccades (SVI/Ipad tapping/ISOMETRICS/LINEAR PROGRESSION/ANY TARGET/ BOXING GYMS/VIDEO GAMES).

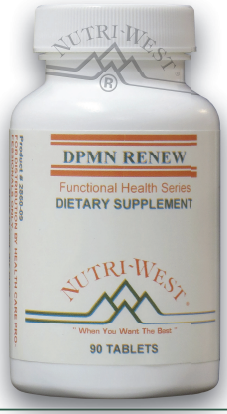
Initial treatment trial = 3x/week 4 weeks MINIMUM, monitor progress and phase down.

BIAS TREATMENTS TO FX 405 OR EVRL BUT STACK WITH GVL WHEN AVAILABLE

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PARKINSON'S DISEASE

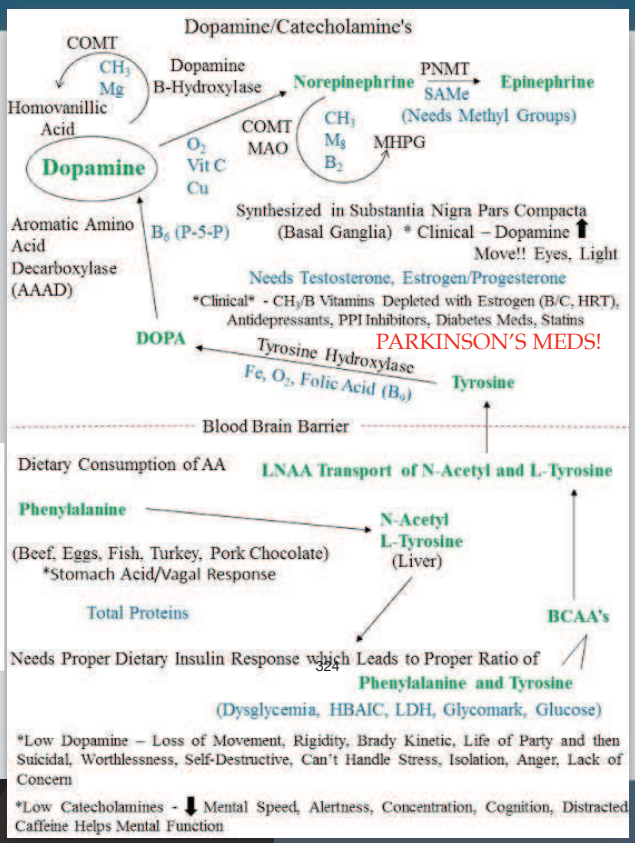


Supplement Facts
Serving Size 1 TABLET

Amount Per Serving	%DV
Proprietary blend 500 mg	*

Mucuna Pruriens Extract (plant), N-Acetyl L-Tyrosine, Organic Blueberry (fruit), G.A.B.A (gamma aminobutyric acid), Ginkgo Biloba Extract (leaf), Berberine HCL (bark), Cytidine 5 Diphosphocholine.

TAKE PRN AND PRIOR TO PHYSICAL REHAB!



CoQ10 (ubiquinol): 200–400 mg/day

Alpha-lipoic acid: 300–600 mg/day

NAD⁺ support (NMN or NR): 250–500 mg/day

Omega-3s (EPA/DHA): 2–4 g/day

Curcumin (bioavailable): 500–1,000 mg/day

RUN LABS
MICRONUTRIENT PROFILES

OTHER NUTRIENT SUPPORT
MUCUNA
IBOGAINE/MUSHROOMS

FIX LEAKY GUT
ADDRESS AUTOIMMUNITY

FALL PREVENTION/ SECONDARY INJURY

IMPROVEMENTS IN ADL…
IT'S NOT A CURE

SLOW PROGRESSION? DBS?
REGENERATIVE MEDICINE?

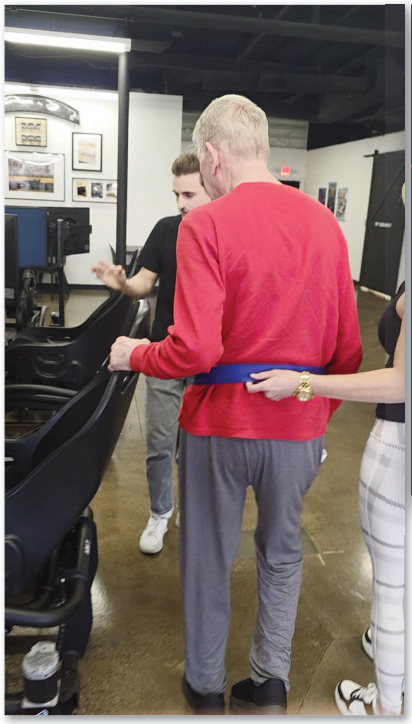
ERCHONIA
Healing Light, Perfected.

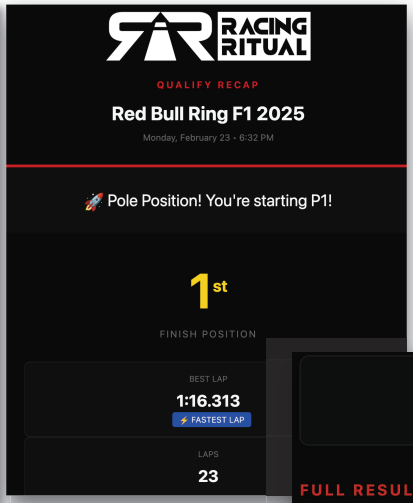
RESTORING BRAIN —HEALTH—

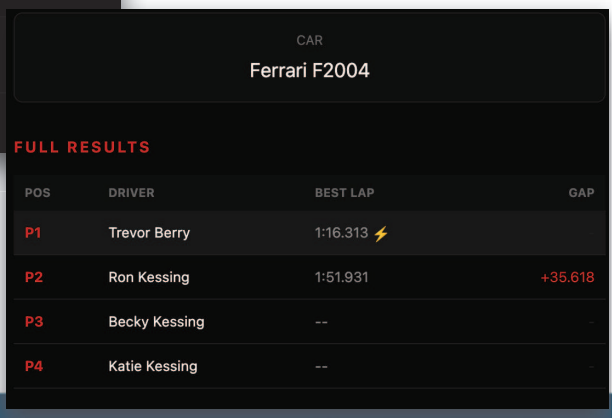
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RON K. PARKINSON'S







THE FAMILY JUST RECEIVED THEIR GVL THEY PURCHASED FOR HOME USE!

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PARKINSON'S DISEASE

1. Neuroprotection First

Target mitochondrial dysfunction, oxidative stress, and neuroinflammation to preserve dopaminergic neurons and slow progression. **STACK ERCHONIA LOW LEVEL LASER THERAPY, HBOT, NEUROFEEDBACK AND BRAIN-BODY EXERCISES** (energy restoration > dopamine replacement alone)

2. Network Re-Training Through Movement

Actively retrain basal ganglia–cortical–cerebellar circuits using task-specific gait, balance, rhythm, and sensory-motor integration
(plasticity is treatment)

3. Gut–Autonomic–Vagal Restoration

Address early and ongoing autonomic dysfunction—gut health, vagal tone, sleep, and inflammation—to improve both motor and non-motor symptoms
(Parkinson's is not just a motor disorder)

Low-Level Laser / Photobiomodulation Research in Parkinson's Disease

- **Neuroprotection of dopaminergic neurons:** PBM improves mitochondrial function, reduces oxidative stress, and preserves dopaminergic neurons in Parkinsonian animal models.
  Hamblin MR. BBA Bioenergetics, 2016.
- **Motor improvement in human PD:** Transcranial and remote PBM demonstrated improvements in mobility, balance, and motor scores in Parkinson's patients with good safety.
  Liebert et al. Journal of Clinical Medicine, 2021.
- **Feasibility & safety established:** Human feasibility trials show PBM is safe, well tolerated, and capable of modulating motor symptoms without adverse neurological effects.
  Hamilton et al. EClinicalMedicine (Lancet), 2023.
- **Disease-modifying mechanisms:** PBM modulates neuroinflammation, microglial activation, cerebral blood flow, and mitochondrial Complex IV activity relevant to Parkinson's pathology.
  Johnstone et al. Neuroscience & Biobehavioral Reviews, 2016.

- **Remote & systemic PBM effects:** Peripheral or remote PBM can induce central neuroprotective effects via systemic immune and mitochondrial signaling pathways.
  Mitrofanis et al. Journal of Neural Transmission, 2017.
- **Long-term treatment benefit:** Extended PBM treatment shows sustained symptom improvement and potential slowing of functional decline.
  Liebert et al. Photobiomodulation, Photomedicine, and Laser Surgery, 2022.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION
Journal of Affective Disorders
Volume 393, Part B, 15 January 2026, 120413

Review Article

Effect of photobiomodulation intervention for depressive symptoms: A systematic review and meta-analysis

Ji-Woo Seok a

Results

PBM showed a moderate overall effect in reducing depressive symptoms (Hedges' g = −0.47, 95 % CI: −0.73 to −0.22). ***Importantly, systemic PBM (sPBM) demonstrated significantly larger effects than those of transcranial PBM (tPBM), underscoring their distinct mechanistic and methodological profiles***. **Laser-based PBM was more effective than LED-based PBM**

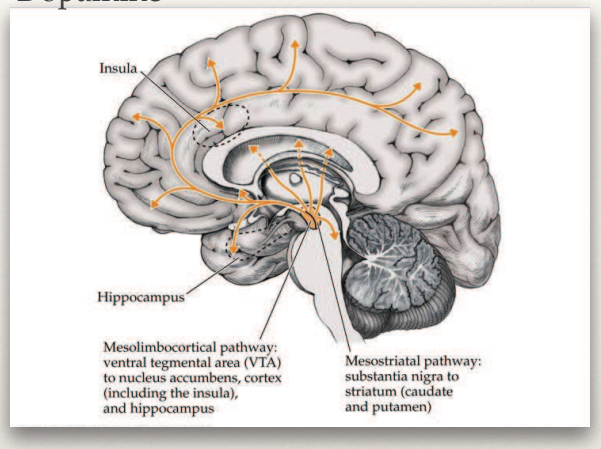
4.4. Key factors modulating PBM's therapeutic effects: Treatment duration

Meta-regression analysis revealed that a longer overall treatment duration (in weeks) was substantially linked to larger reductions in depression symptoms (β = 0.105, *p* = .004). This implies that PBM's anti-inflammatory and neuroprotective benefits can gradually increase with continued use. However, simply raising treatment frequency or intensity does not always result in improved therapeutic success. *This is because PBM follows a biphasic dose-response, where excessive intensity can diminish its neuroprotective effects.*

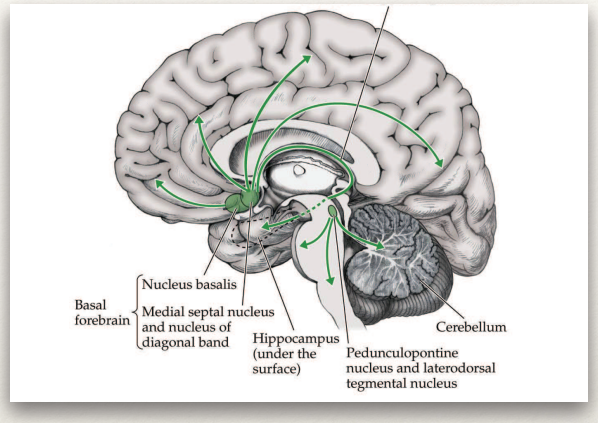
As shown in previous studies, while proper PBM intensity augments mitochondrial function and promotes neuroprotection, excessive intensity can lead to increased oxidative stress and diminished cellular metabolism (Huang et al., 2011; Lanzafame et al., 2007). This is consistent with our finding that neither weekly treatment frequency (β = −0.01, *p* = .694) nor PBM irradiance (β = −4.059 × 10−4, *p* = .685) significantly influenced the amelioration of depressive symptoms. Accordingly, augmenting these parameters does not inherently improve therapeutic results, highlighting the necessity to optimize them (Ji et al., 2024; Zhang et al., 2021).

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

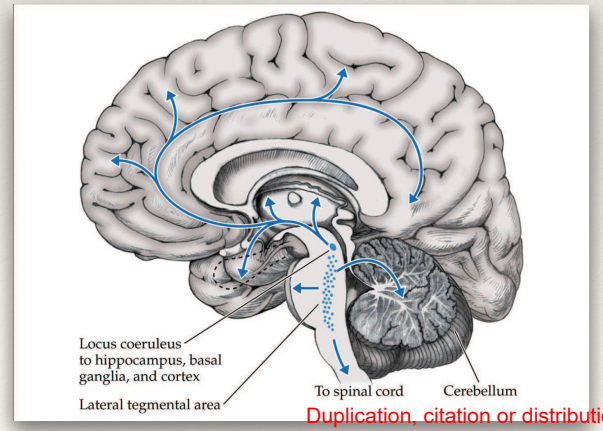
Dopamine



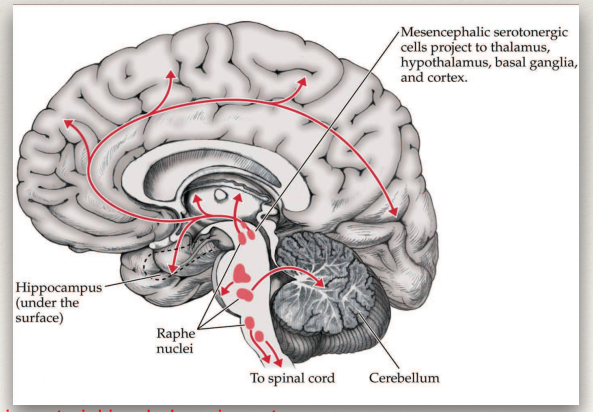
Acetylcholine



Norepinephrine



Serotonin



Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION: CLINICAL SUPPORT SUMMARY. (SUPPORT SLIDES IN THE BACK OF SLIDE DECK)

1. MOVE! EXERCISE, ESPECIALLY IN SUNLIGHT AND IN A "GROUNDING" ENVIRONMENT. LIGHT THERAPIES
2. MAKE SURE THEY HAVE THE RIGHT BUILDING BLOCKS FOR NEUROCHEMISTRY. EAT A GOOD FIBER/VEGGIE, HIGH IN GOOD FATS AND PROTEINS. RUN LABS FROM BASE CBC/CHEM SCREENS, HORMONE TESTING (DUTCH OR VIBRANT), MICRONUTRIENT PROFILE AND INFLAMMATION SOURCES (FOOD SENSITIVITIES, PATHOGENS, HBAIC)
3. A HEALTHY GUT MICROBIOME (GUT ZOOMER 3.0)
4. ERCHONIA LOW LEVEL LASER THERAPY (RIGHT PREFRONTAL CORTEX 6-9-10-12. STACK WITH 5 MIN MEDITATION OF GRATITUDE/5 MIN CLEARING OF MIND). VAGAL NERVE STIM PROTOCOL (10HZ)
5. TRANSCRANIAL MAGNETIC STIMULATION. PEMF. NEUROFEEDBACK
6. CBD PRODUCTS AND CLASSIC PSYCHEDELICS (MICRO OR MACRO DOSING. WATCH LEGAL MATTERS ON DMT -> DMN THERAPIES - SCHEDULE 1 SUBSTANCES)

J Stud Alcohol Drugs

2025 Dec 11:10.15288/jsad.25-00346. doi: 10.15288/jsad.25-00346. Online ahead of print.

Lifetime Cannabis Use Is Associated with Brain Volume and Cognitive Function in Middle-Aged and Older Adults

Anika Guha [1], Zening Fu [2], Vince Calhoun [2], Kent E Hutchison [1]

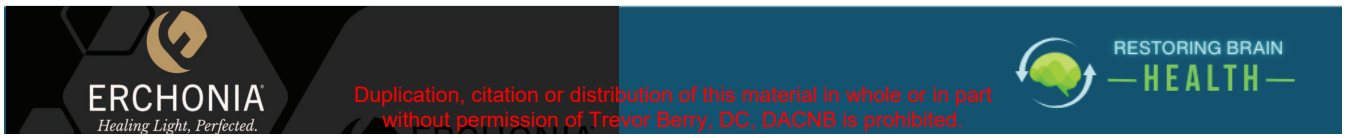
Abstract

Objective: Cannabis use has increased among older adults, yet the neurocognitive effects in this demographic remain unclear. Prior work has suggested cannabis may increase brain volume in areas rich in cannabinoid (CB1) receptors, though negative effects are often reported in adolescents. This study sought to clarify the relationship between cannabis use and brain health among middle-aged and older adults.

Method: Using data from the UK Biobank, which includes health information from over **500,000 adults, associations between cannabis use, regional brain volume, and cognition in participants aged 40-70 years (mean age = 54.5) were evaluated.**

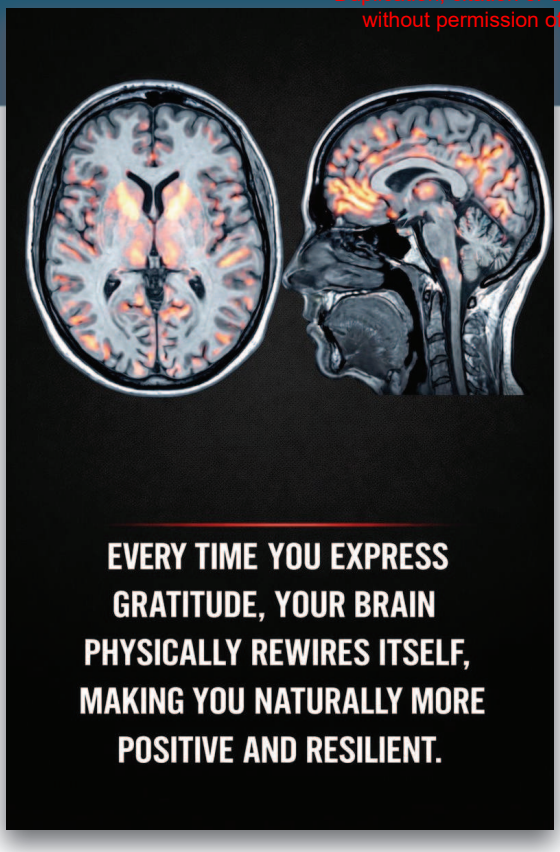
Results: **Lifetime cannabis use was positively associated with regional brain volume in CB1-rich regions, including the caudate, putamen, hippocampus, and amygdala. Greater lifetime use was also linked to better performance in learning, processing speed, and short-term memory. Individuals reporting use limited to adolescence also showed larger regional volumes and better cognitive performance than non-users. Sex differences in cannabis effects on brain volume and cognition were also observed.**

Conclusions: Results highlight that cannabis may influence brain health differently across the lifespan, potentially offering protective effects in older age while posing risks earlier in development. Protective effects may result from endocannabinoid-mediated modulation of inflammation, immune function, and neurodegeneration. Observed sex differences likely reflect variation in the endocannabinoid system and underscore the importance of considering sex as a biological variable in studies of cannabis and brain health.



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



Research Article

Strengthening the heart by means of a gratitude intervention?

Andreas R. Schwerdtfeger,Claudia Traunmüller,Bernhard Weber &Christian Rominger

Received 29 Jul 2025, Accepted 22 Sep 2025, Published online: 19 Oct 2025

Individual differences in posterior cortical volume correlate with proneness to pride and gratitude

Free

Roland Zahn , Griselda Garrido , Jorge Moll , Jordan Grafman

Social Cognitive and Affective Neuroscience, Volume 9, Issue 11, November 2014, Pages 1676–1683, https://doi.org/10.1093/scan/nst158

Published: 28 October 2013

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
—HEALTH—

DR. BERRYS MASTER LASER
SELF CARE

STACK WITH MEDITATION GRATITUDE THEN PURE MEDITATION

RESTORING BRAIN
—HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

MUSHROOMS

MUSHROOMS FOR THE BRAIN

I AM A HUGE FAN OF PAUL STAMETS. HE IS THE MUSHROOM GURU. IT SHOULD BE NOTED HIS LINE "HOST DEFENSE" IS MYCELIUM BASED

MYCELIUM BASED IS GROWN WITH BROWN RICE SO EVERY CAP HAS OVER 50% BROWN RICE

ONE OF THE MOST IMPORTANT ACTIVE INGREDIENTS OF MUSHROOMS IS BETA GLUCANS WHICH IS A POLYSACCHARIDE. HOST DEFENSE HAS LESS THAN 20% BETA GLUCANS

MOST COMPANIES DON'T PASS SOURCE INSPECTION OF QUALITY AND DOSE OF BETA GLUCANS

ONE OF BETA GLUCAN'S PRIMARY FUNCTIONS IS TO TAG PATHOGENS AND BAD CELLS FOR COMPLIMENT SYSTEMS TO RECOGNIZE, ATTACK AND KILL THEM. BETA GLUCANS ALSO HELP WITH SCFA'S AND THE GUT MICROBIOME, WHICH CAN INFLUENCE NEUROTRANSMITTERS IMPACTING MOOD AND COGNITION.

MUSHROOMS ARE A GOOD SOURCE OF VITAMINS INCLUDE B AND D VITAMINS. THEY ARE ALSO A GOOD SOURCE OF ERGOTHIONEINE (A NATURAL AMINO ACID AND POTENT ANTIOXIDANT) HIGHEST IN OYSTER, MAITAKE AND SHIITAKE MUSHROOMS

MUSHROOMS

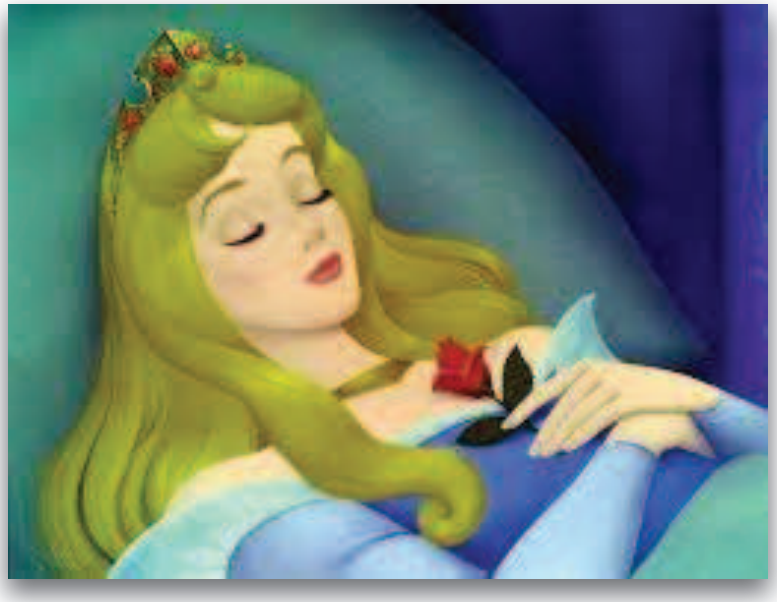
BEST MUSHROOM TYPES FOR THE BRAIN:

1. **LION'S MANE (HERICIUM ERINACEUS). HIGH IN HERICENONES AND ERINACINES WHICH STIMULATE NERVE GROWTH FACTORS, MAY HELP MEMORY AND COGNITIVE FUNCTION AND PROTECT AGAINST NEURODEGENERATION**
2. **CORDYCEPS. CONTAINS CORDYCEPIN, ADENOSINE AND POLYSACCHARIDES (LIKE BETA GLUCANS). HELPS BLOOD FLOW TO THE BRAIN, REDUCES BRAIN FATIGUE AND IMPROVES MENTAL PERFORMANCE**
3. **SHIITAKE. RICH IN VITAMINS, MINERALS AND LENTINANS GOOD FOR OVERALL BRAIN HEALTH, IMPROVE MEMORY AND PROTECT AGAINST DEGENERATION. ALSO HIGH IN ERGOTHIENINE**
4. **REISHI. HIGH IN TRITERPENES AND POLYSACCHARIDES LIKE BETA GLUCANS. HELPS WITH REDUCING INFLAMMATION AND OXIDATIVE STRESS AS WELL AS REGULATING NEUROTRANSMITTERS AND MOOD**
5. **CHAGA. HIGH IN POLYPHENOLS AND MELANIN. GOOD ANTI-OXIDANT AND ANTI-INFLAMMATORY PROPERTIES**
6. **PSILOCYBIN!!! (WITH STATE AND NATIONAL LEGALITY CONSIDERATIONS)**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP SUMMARY (SUPPORTING SLIDES IN THE BACK OF THE DECK)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

SLEEP: NEW GUIDELINES

Dementia: Early bedtime, sleeping over 8 hours linked to increase risk

The dementia risk was 69% greater for individuals who slept over 8 hours, versus 7-8 hours. The risk was also twice as high for those who went to sleep before 9:00 p.m., versus 10:00 p.m. or later (but go before midnight)

CONVERSELY, In a long-term study, Harvard Medical School followed 2,800 individuals ages 65 and older. **Researchers found that individuals who slept under five hours per night were twice as likely to develop dementia compared to those who slept six to eight hours per night. A different study of nearly 8,000 participants found a 30% increase in dementia risk was associated with sleeping six hours or less at age 50, 60 and 70, compared to a normal sleep duration of seven hours.**

SUMMARY: GO TO BED BETWEEN 10-11 PM AND SLEEP 7.5 HOURS. HOWEVER…

Associations of sleep timing and time in bed with dementia and cognitive decline among Chinese older adults: A cohort study

Rui Liu MD, Yifei Ren MD, Tingting Hou MD, PhD, Xiaoyan Liang MD, Yi Dong MD, Yongxiang Wang MD, PhD, Lin Cong MD, PhD, Xiang Wang MD, PhD, Yu Qin MD … **See all authors**

First published: 21 September 2022

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP

- POLYSOMNOGRAPHY (CENTRAL VS OBSTRUCTIVE APNEA VS BODY MOVEMENTS)
- C-PAP/BI-PAP, BREATHING STRIPS, BED/MATTRESS POSITION/PILLOW
- TONE OF ORAL-PHARYNGEAL REGION (VAGAL STIM/DIRECT SOFT PALATE STIM) MOUTH TAPING WITH BREATHE RIGHT NASAL STRIPS
- BASE LAB CHEMISTY (ANEMIA PATTERNS, DYSGLYCEMIA, THYROID, SEX HORMONES…HP AXIS)
- ADVANCED LAB MARKERS: MICRONUTRIENT (CO-FACTORS FOR MELATONIN SYNTHESIS)
- ASI/CORTISOL CURVE AND GLYCOGEN STORES. CORTISOL VS MELATONIN
- CYTOKINES AND MESENCEPHALIC RETICULAR ACTIVATING SYSTEM. FOOD SENSITIVITY TESTING, INFECTIOUS DISEASE MANAGEMENT
- PAIN MANAGEMENT
- BASAL GANGLIA ACTIVATION/DISINHIBITION
- QUALITY OF PILLOW, MATTRESS (AGE ETC)
- ELECTRONICS AT NIGHT, AVOID BLUE WAVELENGTHS/NIGHT SETTING, MINIMIZE VISUAL STIMULATION THROUGHOUT DAY/COMPUTER FILTERS
- QEEG/NEUROFEEDBACK
- CBD/MEDICAL MARIJUANA. MELATONIN CR, PROBIOTICS, GABA

LOW LEVEL LASER TO CNS…

RESTORING BRAIN —HEALTH—

SLEEP

Front Hum Neurosci. 2018; 12: 387.
Published online 2018 Sep 24. doi: 10.3389/fnhum.2018.00387

A Novel Insight of Effects of a 3-Hz Binaural Beat on Sleep Stages During Sleep

Nantawachara Jirakittayakorn* and Yodchanan Wongsawat*

These findings suggested that the 3-Hz binaural beat on a 250-Hz carrier tone can be used to modulate sleep stage by decreasing the latency to the N3 stage, **extending the N3 duration, and reducing the N2 duration without sleep disturbance and sleep fragmentation, while increasing the quality of sleep in this study is also associated slow wave sleep in which is related to memory consolidation and other regulation of the body; and the 3-Hz binaural beat can enhance power of delta activity during sleep.**

J Family Med Prim Care. 2019 Dec; 8(12): 3915–3919.
Published online 2019 Dec 10. doi: 10.4103/jfmpc.jfmpc_575_19

Effect of music of specific frequency upon the sleep architecture and electroencephalographic pattern of individuals with delayed sleep latency: A daytime nap study

Pramita Dubey,1 Yogesh Kumar,1 Ramji Singh,1 Kamlesh Jha,1 and Rajesh Kumar1

Conclusion:

It was concluded that 432 Hz music has some significant calming effect as reflected by increased alpha activities without any significant effect upon the sleep latency in the daytime naps.

Nature
. 2006 Nov 30;444(71

Boosting slo

Lisa Marshall 1, Halla

Here we show that in … potentials (0.75 Hz) during e… wave sleep, enhances the… ries in healthy huma… e sleep, endogenous cortical …

BASE SLEEP SETTING 1 - 2 - 3 - 432
RED XLR8
ALSO CONSIDER "REST AND DIGEST"
VAGAL STIM IN THE PM (1-10-1-10)
COMBO SETTING (1-3-10-432)
10 MIN PROTOCOL BEFORE BED
(8 MIN EACH PRE-FRONTAL CORTEX
2 MIN VAGAL STIM COMBINATION)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THE ADVANCED ASD NEUROLOGY PROTOCOL FOR AUTISM

TREATMENT TIME: 30 SECONDS PER YEAR OF AGE

DELTA AND ALPHA BANDS
MASTER AUTISM FREQUENCIES
1-10-1-10
GVL RIGHT PREFRONTAL CORTEX
WITH TEMPORAL LOBE OVERLAP
THEN LEFT CEREBELLUM/TEMPORAL LOBE
STACKING WITH MODALITIES
FINISH WITH XLR8 OR FX 635 IF AVAILABLE - PASSIVE REST

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

AUTISM TREATMENT CONSIDERATIONS SUMMARY

- SLOW COMPLEX LEFT UPPER EXTREMITY AND LOWER EXTREMITY MOVEMENTS
- INTERACTIVE METRONOME IF AVAILABLE
- CROSS BODY (INTERACTIVE) METRONOME
- BASIC MARCHING CROSS-CRAWL
- MIRROR MOVEMENTS (HAVE THEM BIAS TOWARDS THEIR LEFT HAND/FOOT IF RIGHT HEMISPHERISTIC DEFICIT)
- DEEP PRESSURE SENSORY OR PERCUSSOR VIBRATION
- SACCADES WITH LEFTWARD BIAS (RIGHT FRONTAL LOBE)...ANTI-SACCADES
- GO-NO GO EXERCISES
- PRIMITIVE REFLEX INTEGRATION (CONSIDER DR. MELILLO'S CHILDHOOD NEUROBEHAVIORAL DISORDERS CERTIFICATION COURSE)

COMPLETE THE ADVANCED AUTISM PROTOCOL AS DESCRIBED ABOVE.

CONSIDER THE NEW GVL FOR OPTIMAL OUTCOMES.

12 VISIT TREATMENT TRIAL RECOMMENDED WITH A MINIMUM OF 6 VISITS FOR BEHAVIORAL MODIFICATION ASSESSMENT

-ALWAYS STACK YOUR THERAPIES WITH YOUR ERCHONIA LLLT BRAIN APPLICATIONS

ERCHONIA
Healing Light, Perfected.

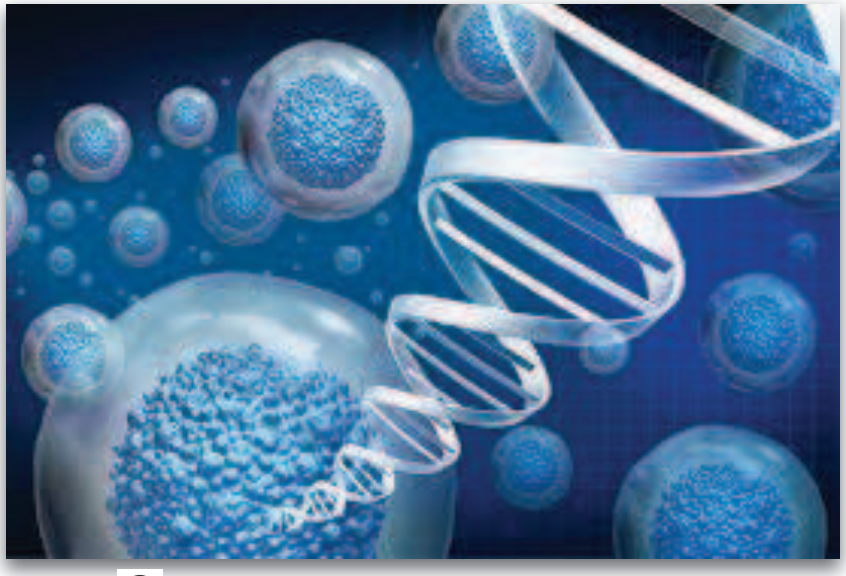
SEE SUPPORTING SLIDES FOR NUTRITIONAL CONSIDERATIONS

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REGENERATIVE MEDICINE FOR CNS AND LOW LEVEL LASER THERAPY



© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

PEPTIDES FOR BRAIN HEALTH

1. Cerebrolysin

- **Mechanism**: A mixture of neuropeptides and amino acids with neurotrophic effects. It mimics endogenous neurotrophic factors (like BDNF) and supports neurogenesis, synaptogenesis, and protection against excitotoxicity.
- **Dosing**:
  - **IV**: 5 mL to 10 mL daily or every other day for 10–20 sessions.
  - **IM**: 1–5 mL daily or every other day for maintenance.
- **Delivery**: Injectable (IV or IM).
- **Sourcing (licensed providers)**:
  - **International**: Licensed pharmacies in Germany, Austria, or online via CerebrolysinShop.com (requires diligence for quality assurance).
  - **U.S. Providers**: Special order via compounding pharmacies with international import (legal but limited).
- **Notes**: Shown benefit in stroke recovery, TBI, Alzheimer's, Parkinson's, and even depression.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

2. Dihexa (N-hexanoic-Tyr-Ile-(6) aminohexanoic amide)

- **Mechanism**: A nootropic peptide derived from angiotensin IV; penetrates the BBB and enhances synaptogenesis and BDNF activity.
- **Dosing**:
  - **Oral**: 2–10 mg daily (titrated slowly).
  - **Sublingual**: 1–5 mg/day for faster onset and bypassing first-pass metabolism.
- **Delivery**: Oral capsule, sublingual troche.
- **Sourcing**: Compounding pharmacies (e.g., Tailor Made, Empower, Wells Pharmacy).
- **Clinical Use**: Early cognitive decline, TBI, long COVID neurocognitive symptoms, Parkinson's.

PEPTIDES FOR BRAIN HEALTH

3. Selank / Semax

- **Mechanism**:
  - **Selank**: Anxiolytic and nootropic via GABAergic and serotonergic modulation.
  - **Semax**: Increases BDNF, modulates ACTH/MCR pathways; neuroprotective and anti-inflammatory.
- **Dosing**:
  - **Selank**: 250–500 mcg intranasally 1–2x/day.
  - **Semax**: 300–600 mcg intranasally 1–2x/day.
- **Delivery**: Intranasal spray (fast CNS delivery).
- **Sourcing**:
  - **Licensed**: Russian pharmaceutical brands (e.g., PeptGenLab).
  - **U.S. Compounding**: Some functional med providers offer compounded versions (verify quality).
- **Use Cases**: Anxiety with cognitive impairment, stroke rehab, ADHD, neuroinflammatory conditions.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Safety & Regulatory Notes

- These peptides are generally **prescription-only** in the U.S. or available through **research compounders**.
- Work with **licensed compounding pharmacies** or **verified international peptide labs**.
- Always follow **clinical supervision** protocols, especially with injectable agents like Cerebrolysin.
- Consider baseline labs (CBC, CMP, inflammatory markers, cognitive assessments) and repeat for tracking.

Clinical Application Tip

Use **multimodal protocols** with:

- **Peptides + LLLT (FX 405 over cortex)**
- **HBOT** for oxygenation
- **NAD+ IV or oral**
- **Anti-inflammatory diet + mitochondrial support (CoQ10, PQQ, Alpha-GPC)**

PBM + Stem Cells: Synergistic Regenerative Effects
PBM Enhances Survival and Differentiation of Neural Stem Cells
Citation:
Rhee, Y. H., et al. (2015). Enhancement of neural differentiation using photobiomodulation in human neural stem cells. **Scientific Reports, 5, 14437.** https://doi.org/10.1038/srep14437
Findings: PBM enhanced neural stem cell proliferation and differentiation—key for targeting brain repair.

***Takeaway:** PBM makes the local environment more conducive to peptide/ protein activity and stem cell integration.*

PEPTIDES FOR BRAIN HEALTH

Photobiomodulation Enhancing Stem Cell Homing & Migration

1. LLLT Attracts Stem Cells via Chemotactic Signaling

Citation:
Ma, Z., et al. (2017). *Photobiomodulation enhances mesenchymal stem cell migration via the CXCR4/SDF-1α axis*. **Lasers in Medical Science, 32(3), 641–647.**
https://doi.org/10.1007/s10103-017-2156-y

- **Findings**: PBM increased **CXCR4 expression** on mesenchymal stem cells (MSCs) and upregulated **SDF-1α** in injured tissue—enhancing **MSC homing** to damaged areas.
  Mechanism: This is the same axis used in bone marrow homing and repair.

2. LLLT Enhances Engraftment of Transplanted Stem Cells

Citation:
Zhou, T., et al. (2016). *Photobiomodulation preconditioning enhances the therapeutic efficacy of mesenchymal stem cells in myocardial infarction*. **Cell Transplantation, 25(5), 1083–1093.**
https://doi.org/10.3727/096368916X690668

- **Findings**: PBM pretreatment increased **survival and homing** of transplanted stem cells to ischemic heart tissue.
- **Outcome**: Improved cardiac function and infarct size reduction.

TREATMENT SUGGESTION: STACK YOUR LOW LEVEL LASER USING 10-40-10-40 OVER THE TARGET REGION OF THE BRAIN OR SPINAL CORD WITH CO-ACTIVATION (IE. SENSORY STIMULATION IF ITS A PARIETAL LOBE LESION). MINIMUM 4 VISITS POST REGENERATIVE TREATMENT. 12 VISIT TRIAL

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HAIR PROTOCOL

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

HAIR PROTOCOL

(9)-25-100-727

NEW FX 9-25, 25-100,100-727

(HAND HELDS MAY BE USED BUT RECOMMEND WANDING THE LINE BEAM FOR BETTER SURFACE COVERAGE)

10 MINUTES
2X/WEEK FOR 6 WEEKS
BASE MINIMUM TRIAL
CONSIDER YEAR LONG PROGRAM (SEE SLIDES AHEAD

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Cutis. 2019 Jul;104(1):17-24.

Nonsurgical hair restoration treatment.

Nazarian RS1, Farberg AS1, Hashim PW1, Goldenberg G2.

Author information Abstract
Patterned hair loss is common and can negatively impact quality of life. Patients often seek nonsurgical treatment options as a first-line measure to avoid undue risks and expense associated with surgery. This article discusses these noninvasive treatment options, with a focus on minoxidil, finasteride, dutasteride, spironolactone,

low-level laser therapy (LLLT), platelet-rich plasma (PRP), microneedling, and oral supplements.

Dermatol Ther. 2019 Sep;32(5):e13059. doi: 10.1111/dth.13059. Epub 2019 Aug 20.

Physiopathology and current treatments of androgenetic alopecia: Going beyond androgens and anti-androgens.

Katzer T1, Leite Junior A2, Beck R3, da Silva C1.

Author information
Androgenetic alopecia (AGA) is the most diagnosed hair loss dysfunction. Its physiopathology comprises a genetic predisposition affording an exacerbated response of the hair follicles cells to androgens aggravated by scalp inflammation and extrinsic factors. This paper presents a review of the mechanisms and extrinsic factors involved in the AGA physiopathology as well as its conventional and emerging treatments. The research focused on reports regarding AGA physiopathology and treatments published between January 2001 and July 2019 in medical and related journals. The most used medical treatments for AGA-*minoxidil and finasteride-present non satisfactory results in some cases*. **Currently, the low-level laser therapy is recognized as a safe and effective treatment for AGA. Some minimally invasive techniques-mesotherapy, microneedling, carboxytherapy, and platelet-rich plasma-are also used to stimulate hair growth.** Pharmaceutical substances with mechanisms differing from the anti-androgen activity are under current investigation and many of them have botanical origins; however, formulations with higher performance are required, and the hair follicles ability of being a drug and nanoparticle reservoir has been researched. **The association of different strategies, that is, substances with synergic mechanisms and the use of advantageous technologies associated with lifestyle changes could improve the treatment outcomes.**

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

HAIR PROTOCOL

STEP 1.
Refer to patient calendar.
Beginning the following day after procedure, inject .25mL Thymosin Beta 4 daily for 9 consecutive days.
Stop the injections for 10 days.
Resume injections for an additional 10 days for a total of 20 days.

STEP 2.
Apply topical foam GHK for 60 days – 2 pumps nightly except on derma roller days
Apply topical spray of ZN-Thymulin for 60 days - 2 sprays nightly except on derma roller days.

STEP 3.
Begin derma roller weekly treatments (2x per day) exactly two weeks following initial procedure.

Instructions for derma roller. Firmly roll .5mm of the device over the treatment areas and immediately spray the PTD-DBM and Valproic Acid products onto the scalp – once in the morning and once in the evening. This process should be repeated on days 14, 21 and 28.

IMPORTANT: Do not use the ZN-Thymulin and GHK on these derma roller days.

Aging Pathobiol Ther. 2020 Mar 27; 2(1): 58–61.
doi: 10.31491/apt.2020.03.014

The potential of GHK as an anti-aging peptide

Yan Dou,a Amanda Lee,a Lida Zhu,a John Morton,a and Warren Ladigesa,*

XYMOGEN NUTRITIONS SUPPLEMENTS:
(SYSTEMIC CONDITIONS HAVE BEEN RULED OUT LIKE THYROID DYSFUNCTION, ANEMIA PATTERNS, AUTOIMMUNITY ETC)

- **CONSIDER 6 DAY DETOX KIT AT ONSET**
- **DIM PRODUCTS (FEMQUIL, HORMONE PROTECT OR DIMENSION 3)**
- **REGENEMAX PLUS (1 DAILY AS SUGGESTED)**

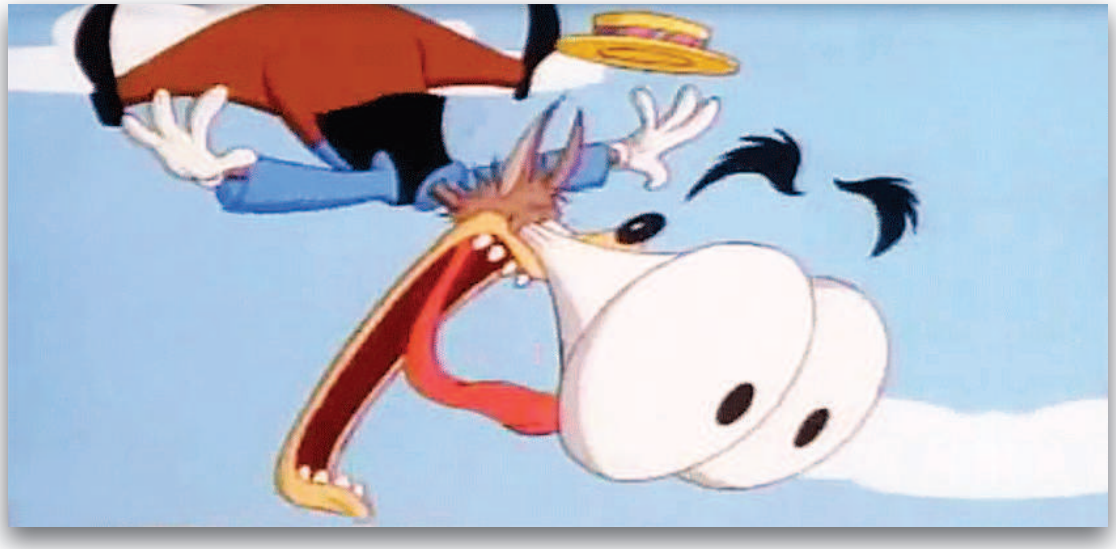
REGENEMAX PLUS CONTAINS ch-OSA (choline-stabilized orthosilicic acid) to nourish the body's "beauty proteins" collagen, elastin and keratin by facilitating collagen generating cell enzymes. It also contains BIOTIN (watch anti-seizure meds like carbamazepine, phenobarbital, phenytoin and primidone)

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RETINAL/VISUAL APPLICATIONS



WARNING: DO NOT USE HIGHER POWERED DEVICES FOR THESE APPLICATIONS!

Adv Exp Med Biol
2026:1487:59-62. doi: 10.1007/978-3-032-03398-7_7.

Visual Perception Changes After Transcranial Photobiomodulation: Preliminary Eye-Tracking Study

Alex O Trofimov 1

Abstract

Purpose: The aim was to study visual perception changes based on eye-tracking data assessments in response to transcranial photobiomodulation (t-PBM) in young, healthy subjects.

Material and methods: Our non-randomized single-center study involving 53 young, healthy volunteers (22 men and 31 women). The eye tracking procedure (EyeTracker, BVG Software Group LLC, CA, USA) was carried out before and immediately after t-PBM (Elmedlife H, RF). Data were expressed as a median [interquartile range]. Statistical analysis was performed using the T- criterion Wilcoxon. The significance level was preset $p < 0.05$.

Results: Vertical vergence reactivity indexes (VRx) after t-PBM were significantly higher than before 0.881 [0.841; 0.914] vs. 0.784 [0.711; 0.832], $p < 0.05$, respectively. Horizontal VRx before t-PBM were significantly lower than after t-PBM 0.893 [0.822; 0.935] vs. 0.920 [0.917; 0.929], $p < 0.05$.

Conclusion: Transcranial PBM improves visual perception in young, healthy volunteers, as reflected by a significant increase in both vergence reactivity indices.

SEE SUPPORT SLIDES IN THE BACK OF THE DECK FOR OPTIC AND OCULOMOTOR APPLICATIONS AND RESEARCH INVOLVING LIGHT THERAPIES AND FUNCTIONAL NEUROLOGY

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RETINAL/OCULAR APPLICATIONS

*** WARNING THIS IS AN OFF LABEL APPLICATION. LASER CLASSIFICATION IS BASED ON RETINAL DAMAGE CAPACITY. DO NOT USE CLASS 3+, CLASS 4 OR HIGHER DEVICES**

RECOMMENDATION: HAND HELD RED BEAM ADVANCED OPTIC SETTING:

10-40-10-40 (UTILIZE VEP 40 HZ AMYLOID CLEARING WITH VAGAL/PERFUSION FREQUENCIES)

APPLICATIONS (PICK ONE OF THESE 3):

- **2 MINUTES SIDE APPLICATION TEMPORAL REGION**
- **30 SECONDS EYES CLOSED "WISPING" OVER EYELID**
- **30 SECONDS EYES OPEN "WISPING" DIRECTLY INTO PUPIL EYES OPEN**
- **5 MINUTES OCCIPITAL LOBE, 2 MINUTES SCM/VAGUS**

TREATMENTS RANGE FROM 2-4 VISITS ACUTE PHASE/ INJURY/POST-OP TO 12 VISIT TRIAL FOR MORE COMPLEX CONDITIONS LIKE MACULAR DEGENERATION

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

DECEMBER 2019 354
"BUDDY"
PRIOR TO INITIAL EVALUATION

IDEXX LABORATORIES

IDEXX Telemedicine Consultants
radiology | cardiology | specialty services

Radiology Report (5159980-15/Radiographs Only - Stat)
Completed 12/20/19 02:17 PM

Patient Name: Buddy Berry (265A)

Requesting Doctor:	Shereen Masaih, DVM	**Species:**	Canine	**Age:**	5Yr 9Mo
Gender:	Unknown	**Breed:**	Unknown	**Weight:**	7.00 Kg

PRESENTING COMPLAINT: Neurological deficits

HISTORY: Buddy is suffering from not bearing weight in both hind limb for last two weeks... need assistant to go outside.. still controlling defecation/ urination. apatite slow down this morning, examining both hid limb reveal motor, sensory response but weak. while taking xray buddy was extremely arching his back. Tucking his hind limb under his body all time

THORAX and ABDOMEN/THORACOLUMBAR SPINE SURVEY December 20, 2019: 6 views are available for interpretation.

FINDINGS:
The cardiovascular structures are within normal limits. The lungs are also normal, with no focal changes to suggest pulmonary edema, pneumonia, or pulmonary metastatic disease seen. No pleural effusion is noted, and no mediastinal abnormalities are suspected. The visible trachea is also within normal limits. There is no significant abnormality such as an aggressive bony lesion or fracture suspected in the thoracic spine, with narrowing of the intervertebral disc space at T10-T11 considered anatomic, at the anticlinal space. There is however, mineralization in situ at multiple disc locations, including T11-T12, much less at L1-L2 and L2-L3. There is significant particulate mineral superimposed with the intervertebral foramen dorsal to the mineralized disc at L3-L4, and a small focus located dorsal to L4-L5. There is also subjective narrowing of L2-L5, compared to the surrounding disc spaces.

A mild amount of heterogenous soft tissue and gas opacity resembling normal ingesta is seen within the stomach. Similar opacity material represents a mild amount of normal feces within the colon. None of the small intestinal loops are distended beyond normal limits, and have normal shape, and pattern. The liver, spleen, left kidney, urinary bladder, and abdominal serosal detail are normal. The right kidney is not well identified, due to superimposition with the liver and gastrointestinal structures. There lines of enthesophytes paralleling the physeal scar on both femoral necks.

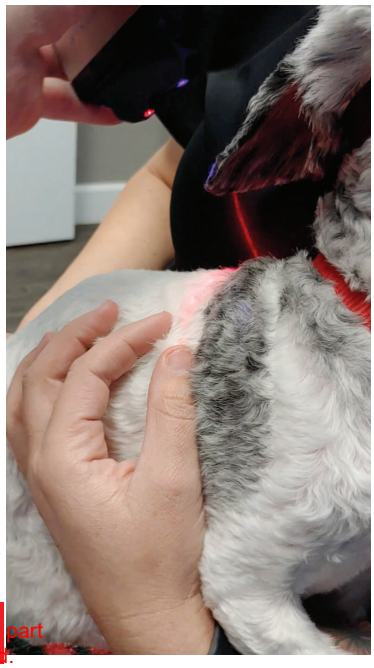
CONCLUSIONS:
--There is evidence of multifocal intervertebral disc disease, and the superimposition of the mineral opacities with the intravertebral foramen is concerning for extrusion into the canal, dorsal to L3-L4, though this could also be lateral to the spinal cord. Mineralization in situ does not correlate well with regions of spinal cord compression, and advanced imaging. Unfortunately, plain films cannot determine any degree of spinal cord or spinal nerve root compression associated with bony changes.
--The thorax and abdominal cavities are considered within normal limits.

Requested By:
Shereen Masaih, DVM
Dignity Veterinary Hospital
4980 S. Alma School Road
Chandler, AZ 85248
P: 480-508-5515
F: 480-508-5514
Re: Buddy Berry (265A)

Do you have feedback on this report? We want to

Report provided by:

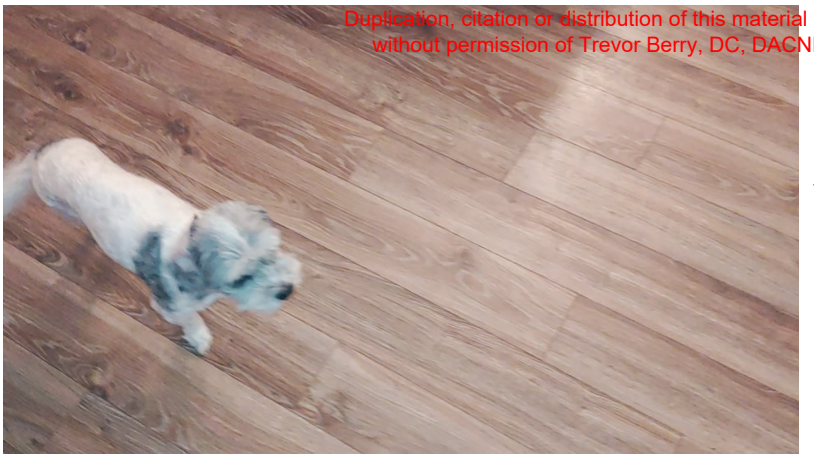
INITIAL TREATMENT
ERCHONIA EVRL LASER
10-16-40-60
FULL SPINE/BRAIN 4 MIN
IMPULSE ADJUSTER T/L JCT TO L/S REGION



DON'T FORGET TO TREAT YOUR PETS!

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



BUDDY POST 2ND TREATMENT

BUDDY POST 3RD TREATMENT

ERCHONIA
World Leaders in Low Level Laser Technology

BUDDY OCTOBER 10, 2020

ERCHONIA
World Leaders in Low Level Laser Technology

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

OTHER PROTOCOLS/ SLIDES BELOW!

SEE PROTOCOL SECTION AND OTHER SLIDES BELOW FOR MISCELLANEOUS SLIDES AND PROTOCOLS THAT MAY NOT HAVE BEEN COVERED DURING THE BODY OF THE SEMINAR THIS WEEKEND.

EXAMPLES INCLUDE:
HP AXIS DYSFUNCTION (THYROID, GONADAL SUPPORT/ ERECTILE DYSFUNCTION, FERTILITY)
IMMUNOLOGY AND PATHOGENS (HERPETIC FAMILY, EBV/ MONO AND OTHERS)
SPECIFIC CARDIOVASCULAR SUPPORT
OTHER SUPPORTING SLIDES SUCH AS RESEARCH VALIDATION AND MORE!

© 2026 DR. TREVOR BERRY ALL RIGHTS RESERVED

GOALS FOR THE SEMINAR!

LEARN 3 THINGS YOU CAN DO TO PROTECT AND REPAIR YOUR BRAIN IN EACH OF THE FOLLOWING AREAS:

DIET

TESTS (LAB/NEURO/BEDSIDE ETC)

SUPPLEMENTS

EXERCISE/FUNCTIONAL NEUROLOGY

LIFESTYLE

AVOIDANCE

OXYGEN DELIVERY

MIND STIMULATION

LOW LEVEL LASER THERAPY APPLICATIONS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PROTECT YOUR BRAIN!

RULE OF 3'S

CAN YOU THINK OF 3 THINGS YOU CAN DO TO PROTECT AND REPAIR YOUR BRAIN IN EACH OF THE FOLLOWING AREAS:

DIET
MIND, MODIFIED PALEO, KETOGENIC = (KALEO), AIP/IMMUNE NEUTRAL, CARNIVORE, "MOTHER NATURE", FASTING(?)

TESTS
MOCA/MMSE, BASE CHEM/CBC WITH DIFF, MICRONUTRIENT/INTRACELLULAR OMEGA 6:3, AUTOIMMUNE, CDP BALANCE
RHOMBERG'S/CEREBELLAR BALANCING

SUPPLEMENTS
OMEGA 3'S, MAGNESIUM, MULTI WITH TRAACS/4TH GENERATION -CH3, VITAMIN D, OTHER SPECIALTY NEEDS

EXERCISE
HIIT, RESISTANCE, COMPLEX/CHANGING PATTERNS, ANY MOVEMENT! FNOR COMBO EXERCISE

LIFESTYLE
STAY SOCIALLY ENGAGED, REDUCE STRESS, GET OUT WITH NATURE (SUN, GROUNDING, FRACTALS)
SLEEP HYGIENE, RESTING DIGESTING VAGAL NERVE STIMULATION

AVOIDANCE
GLUTAMATE (MSG/ARTIFICIAL SWEETENERS), INORGANIC GENETICALLY ENGINEERED FOODS, SUGAR/CARBS
STRESS, IMMUNE TRIGGERING FOODS (GF, DF, SF), ULTRAPROCESSED FOODS

OXYGEN DELIVERY
WATCH BLOOD PRESSURE (HIGH OR LOW)/DYSAUTONOMIA, ANEMIA PATTERNS, MITOCHONDRIA/ENERGY MODEL
HBOT, EWOT, CONCENTRATOR, BOX BREATHING (4-4-8)

MIND STIMULATION AND TRAINING
APPS LIKE BRAIN HQ, ELEVATE, LUMOSITY, LEARN! LANGUAGE, INSTRUMENT, CEU'S. MEDITATE
GAMES LIKE SUDOKU, PUZZLES, CROSSWORDS, QEEG/NEUROFEEDBACK

LOW LEVEL LASER THERAPY BELOW 10 J/CM2 TO THE BRAIN WITH MULTIPLE WAVELENGTHS!
BASE BRAIN PROTOCOL (10-40-10-40), VAGAL PROTOCOL, BRAIN BALANCING

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

2026 FOUNDATIONAL PROTOCOLS FOR YOUR ERCHONIA DEVICES

MOST COMMONLY USED BRAIN FREQUENCIES:

BRAIN RESTORE **10-40 FOR ALL DIODES (HAND HELDS OR FX'S)**

MASTER **1-10-40-60 (ADD 10s AND 40s ON FX DEVICES FOR THE REMAINING DIODES)**

BASE IT ON QEEG MAPPING SUGGESTIONS AND LOCATION (EX. SMR 12-15 UP OVER MIDLINE)

FIRST PRINCIPLES…ARE YOU DEALING WITH HIGH ANXIETY, OCD ETC = SLOW IT DOWN WITH A REST-DIGEST TYPE OF 1-10-1-10
IF THEY ARE BOGGED DOWN LIKE STUCK IN DELTA/THETA POST CONCUSSION USE MORE 40s

DEFAULT TIME PER REGION FOR DIFFERENT ERCHONIA HAND HELD DEVICES:

2 MINUTES PER REGION GVL
3 MINUTES PRE REGION EVRL
4 MINUTES PER REGION XLR8
MINIMUM 6 MINUTES FX 405 (SPLIT 3 AND 3 SAGITTAL/CORONAL)
AND 10 MINUTES WITH FX 635 (SPLIT 5 AND 5)

DO NOT GET ANALYSIS PARALYSIS! YOU CAN LEAVE THE FX ON SOMEONE'S HEAD FOR THE FULL 20 MINUTES WITHOUT GOING NEAR EXCESS DOSE PARAMETERS! AND THEY AREN'T GOING TO GROW A LIVER IN THEIR BRAIN IF YOU ACCIDENTALLY LEAVE IT ON THE LIVER SETTING AND PUT IT ON SOMEONE'S HEAD!

A DAILY STACK FOR BRAIN HEALTHY, LONGEVITY AND OVERALL FUNCTION AND PERFORMANCE

AM

1. AFTER A GOOD QUALITY SLEEP TAKE
2. GO OUTSIDE AND GROUND (BARE FEET ON GRASS), SUNLIGHT IN EYES, RESILIENCE BREATHING (IE. WIM HOF),
3. TAKE YOUR FOUNDATIONAL (AND TARGET SPECIFIC) SUPPLEMENTS. MULTI WITH NOOTROPICS, OMEGA 3'S, MG, D
4. CONSUME YOUR MINIMAL PROTEIN INTAKE FOR BREAKFAST (MINIMUM 30 GRAMS)
5. HEAD TO THE GYM OR HOME/OFFICE WORKOUT (REVIEW EXERCISE TYPES)

PROTEIN AT EVERY MEAL WITH MINIMUM 1.2 g/kg/day (INSTEAD OF JUST WITH DINNER)
AND AT LEAST 2 GRAMS OF OMEGA 3 FATTY ACIDS DAILY
PERFORM 5 MINUTES OF MEDITATION OF GRATITUDE/PRAYER AND 5 MINUTES PURE MEDITATION (RIGHT PREFRONTAL HANDHELD OR LEFT CEREBELLUM RIGHT FRONTAL, APICAL FX LASER)
CORTEX ERCHONIA 10-40-10-40 DEFAULT SETTING)

PM
AFTER DINNER CONSIDER THE FOLLOWING:

1. TARGET SLEEP START BETWEEN 10-11 PM (7.5 HRS)
2. TAKE SUPPLEMENT SUPPORT (MG, L-THEANINE, GABA, KAVA, VALERIAN, CORTISOL ADAPTOGENS)
3. WHEN AND WHY USE CBD AND MELATONIN?
4. VAGAL STACK - ERCHONIA LASER THE VAGUS NERVE (10 HZ) AND GUT MIN 2 MINUTES EACH, MASSAGE GUT, BOX BREATHING, GARGLE WITH NIGHT TOOTH BRUSHING
5. AROMATHERAPY
6. SLEEP BINAURALS OR BRAIN TAP (BRAIN HQ DURING ACTIVE REHAB) WHILE LASERING THE TEMPORAL LOBE ABOVE THE EAR 5 MINUTES EACH SIDE USING 1-10-40-10 SETTING (REVIEW WHY LASER IS SO IMPORTANT)
7. IF TRYING TO LEARN SOMETHING, MAKE THAT YOUR LAST READ BEFORE BED (NO BLUE LIGHT AND USE NIGHT SETTING IF ELECTRONIC)

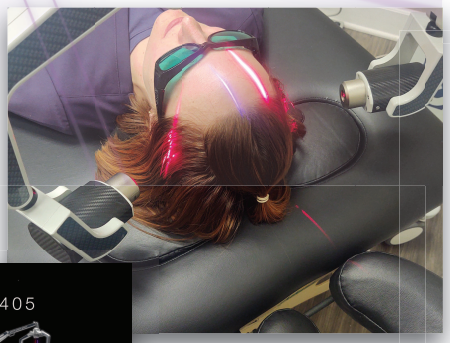
ERCHONIA
Healing Light, Perfected.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE ULTIMATE ERCHONIA PRACTICE FLOW



ALL PATIENTS GET:

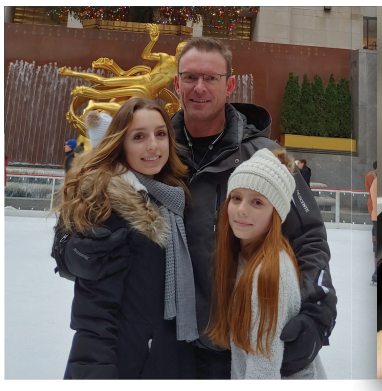
10 MINUTES VZ8 (ZERONA/EMERALD) TO GUT
10 MINUTES FX 405 TCT ALL DIODES 10-40
START PATIENTS ON BINDERS AND LPS SUPPORT

HAVE BASE STATION FOR BEDSIDE/BACK OFFICE:
VAGAL QUAD
CEREBELLAR EXERCISE STACK
MSK

1 BASE STATION HAND HELD
FOR WEEKLY RENTAL

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—



“You only have one brain—but you are not trapped by its past.
The brain can heal and it can be restored.
Laser therapy, combined with proper nutrition and lifestyle, gives us a research-backed way to support real change.
Don’t wait for decline to force action.
Choose to invest in your brain—because you’re worth it.”

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

"PROTOCOLS" (SUMMARY SECTION)

THE HEALTHCARE PROVIDER SHOULD MAKE EVERY ATTEMPT TO SUPPORT COMPLEX NEUROLOGICAL CASES WITH INDIVIDUALIZED CARE. FOR EXAMPLE, ALZHEIMER'S LIKE DEMENTIA MAY HAVE 6 DIFFERENT GROUPINGS WITH HUNDREDS OF VARIATIONS OF UNDERLYING CAUSES. WITH THAT SAID, I HAVE ATTEMPTED TO GIVE FOUNDATIONAL PROTOCOLS IN MOST OF THE SECTIONS OF THESE SEMINAR NOTES FOR YOU TO EASILY REFERENCE. LOOK FOR THIS SYMBOL:

NOTE: A SUMMARY SECTION OF THE PRIMARY "PROTOCOL" PAGES HAS BEEN ADDED IN AT THE END OF THE PRIMARY SEMINAR SLIDES, BEFORE THE MISCELLANEOUS SUPPORT SLIDE SECTION FOR EASY REFERENCE

DO NOT BE CONCERNED ABOUT CONTENT THAT IS COVERED BY DROP DOWN BOXES OF TEXT COVERING UNDERLYING TEXT. I HAVE MADE SURE TO NOT COVER ANY PERTINENT OR NECESSARY INFORMATION WHEN YOU GO BACK TO REVIEW YOUR NOTES

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EXERCISE FOR THE BRAIN: WHICH IS BEST??

TECHNICALLY ANY FORM OF MOVEMENT IS BETTER THAN NONE FOR BRAIN HEALTH. WITH THAT SAID HERE ARE THE "TOP 4" FORMS:

1. **FOR OVERALL NEUROGENESIS AND BDNF FACTORS, ESPECIALLY IN THE HIPPOCAMPUS, CONSIDER HIIT/INTERVAL TRAINING (1 MIN MAX, 1 MIN REST X3)**

2. **FOR FRONTAL INHIBITORY CONTROL, EXECUTIVE FUNCTION AND PROCESSING SPEED, CONSIDER RESISTANCE TRAINING**

3. **FOR HP AXIS/STRESS REDUCTION, ATTENTION AND NETWORK EFFICIENCY CONSIDER COMPLEX AEROBIC LIKE YOGA AND TAI CHI**

4. **THE NORWEGIAN 4X4 (4 MINUTES NEAR MAX HR ~90% EFFORT - 3 MINUTES REST X4)**

A SOLID OVERALL WEEKLY WORKOUT ROUTINE TO TARGET WOULD BE THE FOLLOWING:

MINIMUM 2X/WEEK HIIT TRAINING (BASE OR 4X4)…DO DAILY BURSTS!
MINIMUM 2X/WEEK RESISTANCE TRAINING
1X/WEEK YOGA OR TAI CHI (SOME CULTURES DO THIS DAILY)

LASER YOUR BRAIN DAILY! STACK IT WITH EXERCISES (PRE OR DURING). GIVE DR. BERRY EXAMPLES OF WHERE AND WHY…

NEURODEGENERATION SUPPORT PARAMETERS

HOW LONG SHOULD THE PATIENT IMPLEMENT LIFESTYLE AND TREATMENT PARAMETERS IN NEURODEGENERATIVE CONDITIONS TO SEE CHANGES?

A: 6 MONTHS
DIET, LIFESTYLE, NUTRITIONAL SUPPORT AND OTHER RECOMMENDATIONS OF THE BREDESEN PROTOCOL SHOULD BE IMPLEMENTED FOR 6 MONTHS BEFORE DECIDING IF THEY WILL BE A VIABLE SOLUTION FOR THE PATIENT.
THIS TIMEFRAME SHOULD ALSO BE CONSIDERED FOR FOOD ELIMINATION PROTOCOLS.

HOW LONG SHOULD SUPPLEMENTS/MEDICATIONS BE TAKEN FOR SHIFTS IN MICRONUTRIENT AND CHEMISTRY PROFILE CHANGES?

A: 3 MONTHS MINIMUM

HOW MANY IN-OFFICE LASER AND BRAIN BASED TREATMENTS SHOULD BE UTILIZED TO SEE IF ANY OBJECTIVE AND SUBJECTIVE MARKERS IMPROVE (TO ANY DEGREE)?

A: 12 VISITS MINIMUM (OVER A 4-6 WEEK PERIOD)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

2026 FOUNDATIONAL PROTOCOLS FOR YOUR ERCHONIA DEVICES

MOST COMMONLY USED BRAIN FREQUENCIES:

BRAIN RESTORE **10-40 FOR ALL DIODES (HAND HELDS OR FX'S)**

MASTER **1-10-40-60 (ADD 10s AND 40s ON FX DEVICES FOR THE REMAINING DIODES)**

BASE IT ON QEEG MAPPING SUGGESTIONS AND LOCATION (EX. SMR 12-15 UP OVER MIDLINE)

FIRST PRINCIPLES…ARE YOU DEALING WITH HIGH ANXIETY, OCD ETC = SLOW IT DOWN WITH A REST-DIGEST TYPE OF 1-10-1-10
IF THEY ARE BOGGED DOWN LIKE STUCK IN DELTA/THETA POST CONCUSSION USE MORE 40s

DEFAULT TIME PER REGION FOR DIFFERENT ERCHONIA HAND HELD DEVICES:

2 MINUTES PER REGION GVL
3 MINUTES PRE REGION EVRL
4 MINUTES PER REGION XLR8
MINIMUM 6 MINUTES FX 405 (SPLIT 3 AND 3 SAGITTAL/CORONAL)
AND 10 MINUTES WITH FX 635 (SPLIT 5 AND 5)

369

DO NOT GET ANALYSIS PARALYSIS! YOU CAN LEAVE THE FX ON SOMEONE'S HEAD FOR THE FULL 20 MINUTES WITHOUT GOING NEAR EXCESS DOSE PARAMETERS! AND THEY AREN'T GOING TO GROW A LIVER IN THEIR BRAIN IF YOU ACCIDENTALLY LEAVE IT ON THE LIVER SETTING AND PUT IT ON SOMEONE'S HEAD!

2026 MINI MASTER PROTOCOL

ALL HAND HELDS SET FOR 10-40-10-40
2 MINUTES EACH REGION FOR GVL
3 MINUTES FOR EVRL
4 MINUTES FOR XLR8

DLPF/TEMPORAL/ PARIETAL

STACK WITH CEREBELLAR COMPLEX

BRAINSTEM/ CEREBELLUM/ VAGUS

370

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

SOME OF DR BERRYS FAVORITE FREQUENCY SETTINGS

MASTER BRAIN 1-10-40-60 MASTER GAMMA 40-40-40-40

VAGUS 10-10-10-10 BRAIN RESTORE 10-40-10-40

REST DIGEST 1-10-1-10 DELTA 1-1-1-1

MASTER LOVE 1-10-40-528

IMMUNE BOOST 20-73-465-728

DEFAULT VIRUS (ACUTE) 20-73-625-787

DEFAULT BACTERIA (ACUTE) 20-664-787-1560

DEFAULT MUSCULOSKELETAL 9-16-42-53

PAIN 4-60-5000-10000

ACUTE INJURY (GAIR) 8-25-42-125

CHRONIC INJURY (GAIR) 8-25-42-279

CLINICAL GEM: ADD 10 HZ AND 40 HZ TO JEROME'S BASE 4 FREQUENCY PLATFORM FOR FX LASERS

REFER TO JEROME RERUCHA'S LASER A-Z PROTOCOL BOOK!

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

SOMETIMES IT'S BEST JUST TO TRY GETTING THE LASER ON THE BRAIN!

MANISH PATEL (A P.T.): HIS FATHERS PATIENT (ON THE LEFT) HAD A STROKE AND HAD NOT OPENED HIS HAND IN THE 2 YEARS SINCE. HE GOT HIS GVL, HEARD ME SAY "JUST GET THE LASER ON THE HEAD EVEN IF YOU DON'T FULLY UNDERSTAND NEUROLOGY. BIG THINGS CAN HAPPEN WHEN THE BRAIN HAS MORE ENERGY!"
FOLLOWED BY THIS MOST RECENT VIDEO SENT ON 9/17/25. MSA PATIENT (MULTIPLE SYSTEM ATROPHY)

ERCHONIA® Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

BRAIN HEALTH APPS

APPS FOR BRAIN TRAINING AND OPTIMIZATION:

- FOCUS BUILDER (NEEDS DOCTOR RX INSTRUCTIONS)
- LUMOSITY
- NEURONATION
- **BRAIN HQ**
- ELEVATE (WIDE RANGE OF MENTAL SKILLS)
- COGNIFIT
- PERSONAL ZEN (ANTI-ANXIETY TRAINING)
- HAPPIFY
- RELIEFLINK (SUICIDE PREVENTION AND MOOD TRACKER)
- BRAIN TAP

CONSIDER A SECOND LANGUAGE APP:

- BABBEL
- DUOLINGO
- ROSETTA STONE

CONSIDER AI TRAINING. NOT IN A CO-DEPENDENT WAY BUT RATHER BY PROMPTING DAILY EXERCISES

THE BRAIN-MEMORY STACK

AFTER DINNER CONSIDER THE FOLLOWING:

1. TARGET SLEEP START BETWEEN 10-11 PM (7.5 HRS)
2. TAKE SUPPLEMENT SUPPORT (MG, L-THEANINE, GABA, KAVA, VALERIAN, CORTISOL ADAPTOGENS)
3. ACETYLCHOLINE (ALPHA GPC) AND MG L-THREONATE SUPPORT
4. WHEN AND WHY USE CBD AND MELATONIN?
5. VAGAL STACK - ERCHONIA LASER THE VAGUS NERVE (10 HZ) AND GUT MIN 2 MINUTES EACH, MASSAGE GUT, BOX BREATHING, GARGLE WITH NIGHT TOOTH BRUSHING
6. AROMATHERAPY (PLEASANT SMELL UNDER NOSTRIL OR DIFFUSER)
7. SLEEP BINAURALS, SOLFEGGIO, OR BRAIN TAP (BRAIN HQ DURING ACTIVE REHAB) WHILE LASERING THE TEMPORAL LOBE ABOVE THE EAR 5 MINUTES EACH SIDE USING 1-10-40-10 SETTING (REVIEW WHY LASER IS SO IMPORTANT. REVIEW MUSIC SUMMATION)
8. IF TRYING TO LEARN SOMETHING, MAKE THAT YOUR LAST READ BEFORE BED (NO BLUE LIGHT AND USE NIGHT SETTING IF ELECTRONIC)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CONSENSUS PROTEIN INTAKE SUMMARY

Daily Protein Intake at 1.2 g/kg

Adult Females

Body Weight	Weight (kg)	Protein / Day
120 lb	54.5 kg	**65 g**
130 lb	59 kg	**71 g**
140 lb	63.6 kg	**76 g**
150 lb	68 kg	**82 g**
160 lb	72.7 kg	**87 g**
170 lb	77 kg	**92 g**

Adult Males

Body Weight	Weight (kg)	Protein / Day
150 lb	68 kg	**82 g**
160 lb	72.7 kg	**87 g**
170 lb	77 kg	**92 g**
180 lb	81.8 kg	**98 g**
190 lb	86.4 kg	**104 g**
200 lb	90.9 kg	**109 g**

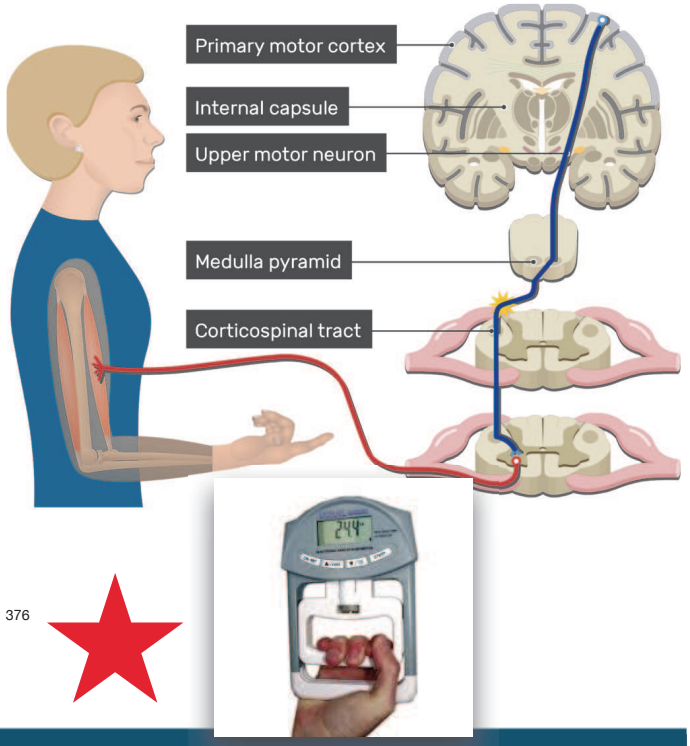
375

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH

DOWNSTREAM DYNAMOMETER PROTOCOL

HOW TO UPREGULATE MOTOR SYSTEMS FROM CENTRAL TO END TARGET

WITH CROSSOVER INTO PAIN MANAGEMENT



376

ERCHONIA
Healing Light, Perfected.
RESTORING BRAIN HEALTH
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

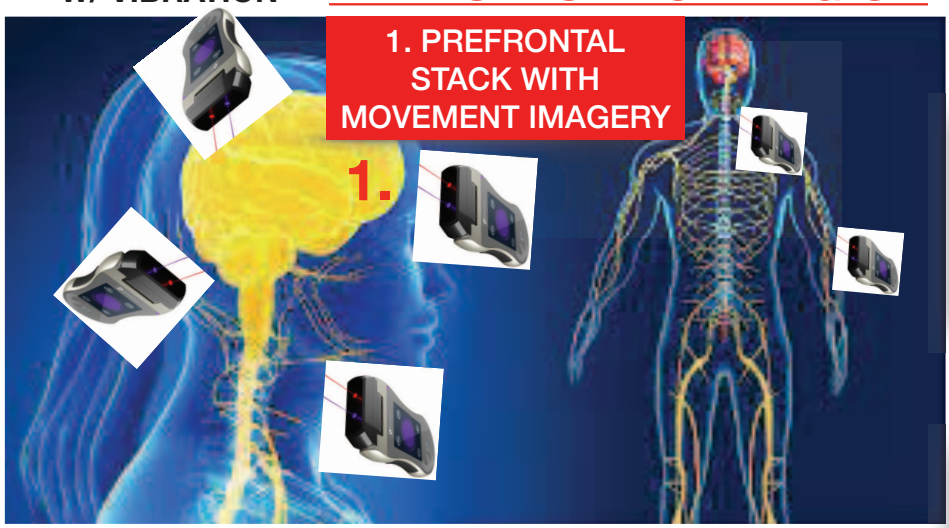
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THE "DOWNSTREAM" MOTOR PROTOCOL

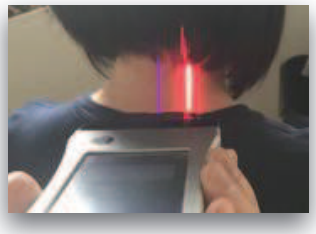
(FOR EVEN BETTER RESULTS, PERFORM AFTER BRAIN BALANCE)

SUMMARY OVERVIEW
2 MINUTES EACH REGION

2. PARIETAL LOBE W/ VIBRATION



5. SPINAL CORD/ NERVE ROOT



6. TARGET MUSCLE/ DISTAL TISSUE

3. IPSI CEREBELLUM STACK WITH MOVEMENT/ISOMETRICS

4. VAGAL NERVE STIMULATION IPSILATERAL TO INVOLVED SIDE

USE 10-40-10-40

MOTOR AND SENSORY MIRROR THERAPIES

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SET IT AND FORGET IT

STANDARD BASE POSITION LOW BACK PAIN. MAX 20 MINUTES. 10 MINUTES AVERAGE

ERCHONIA
Healing Light, Perfected.

379

LOW BACK BACK PAIN PROTOCOLS

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

380

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LASERS A-Z MANUAL

JEROME RERUCHA

BASE MUSCULOSKELETAL/PAIN DEFAULT UPREGULATION

9-16-42-53

LIGAMENT
5-9-125-2720

CARTILAGE
20-690-787-2720

BONE
20-45-30594-8687

BURSITIS
9-16-1442-6568

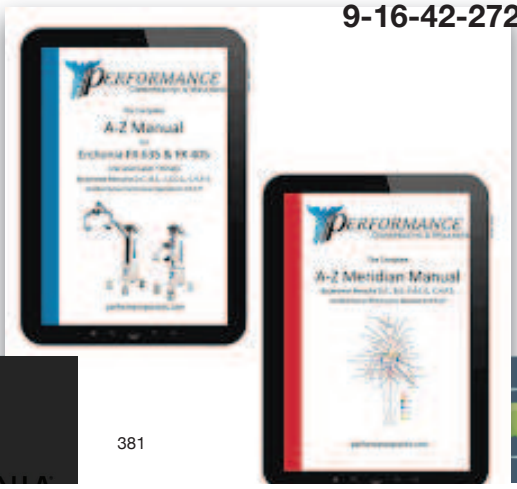
EDEMA
21-33-43-5000

TENDONITIS
1-21-250-2720

NEUROGENIC INFLAMMATION/FASCIITIS
9-16-33-36

WOUNDS
3-16-24-111

INFLAMMATION
9-16-42-2720

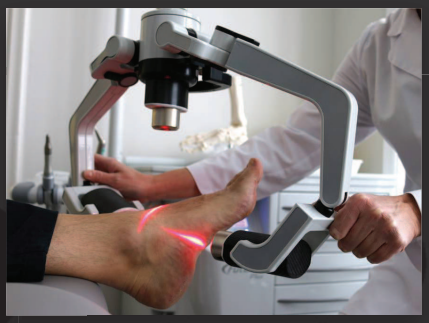


ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
HEALTH

FDA STUDY 6 VISITS. SET IT AND FORGET IT
SWIM UP STREAM (USE FX OR LINE BEAM TO TARGET
FOOT POPLITEAL/BIFURCATION AND
LOW BACK NERVE ROOT/SPINAL CORD ALL
AT ONCE. 9-16-42-53 (ADD 2HZ TO FX MODELS)

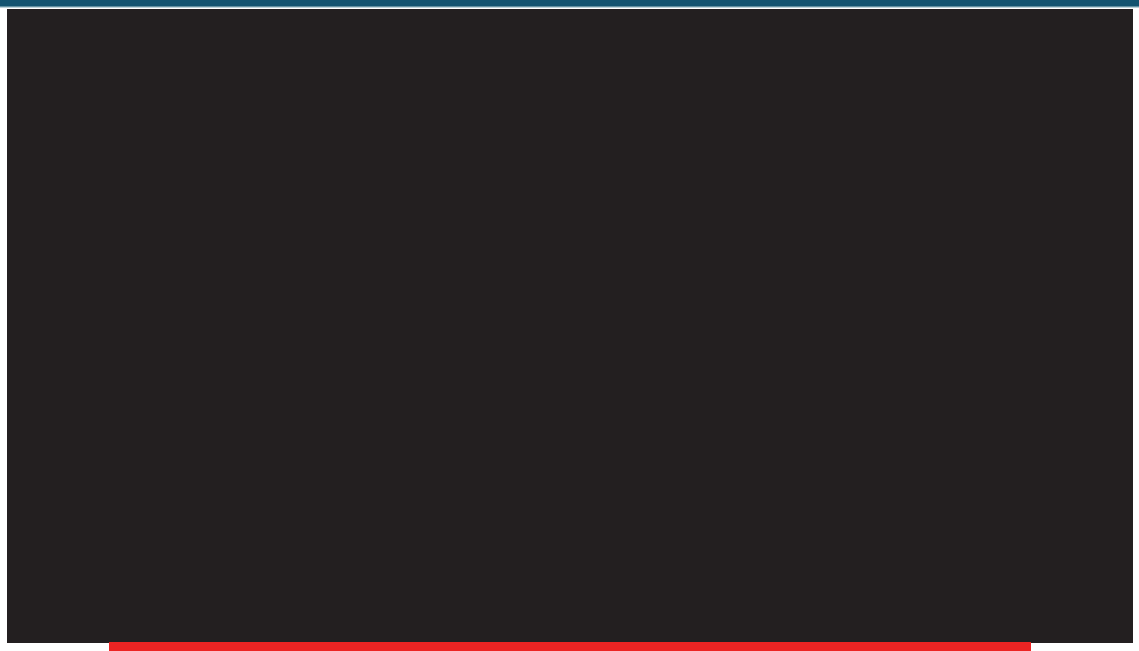
ERCHONIA®
Healing Light, Perfected.



Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FX LE PROTOCOL BASIC



CONSIDER 10 MINUTES EACH LEG/NERVE DISTRIBUTION
L/S PLEXUS, POPLITEAL BIFURCATION AND COMMON PERONEAL
TARSAL TUNNEL/PLANTAR REGION

Case Report ISSN 2768-6647
Medical and Clinical Case Reports

Impact of Red and Violet Laser Therapy on Peripheral Arterial Disease: Case Series

Andrzej Eberhardt[1] and Travis Sammons[2]*

[1]General surgery and Vascular, Vascular Surgery and Phlebology in Femmed Clinic, Warsaw, Poland.

[2]Erchonia Corporation, Melbourne, FL, USA.

*Correspondence:
Travis M. Sammons, Erchonia Corporation, 112 Southchase Blvd., Fountain Inn, SC 29644, Tel: 888-242-0571, Fax: 321.473.1608.

Received: 29 Nov 2023; Accepted: 26 Dec 2023; Published: 02 Jan 2024

Citation: Andrzej Eberhardt, Travis Sammons. Impact of Red and Violet Laser Therapy on Peripheral Arterial Disease: Case Series. Med Clin Case Rep. 2024; 4(1): 1-4.

Keywords
Peripheral arterial diseases, Red and violet Laser therapy, Surgery.

Introduction

Peripheral arterial disease (PAD) is a manifestation of atherosclerotic disease impacting the lower section of the aorta, along with its primary offshoots and the arteries that provide blood to the limbs. The range of clinical manifestations associated with PAD encompasses individuals with symptomless conditions, those who undergo intermittent claudication (IC), and those exhibiting more serious signs of critical limb ischemia. PAD is a common condition that affects a large proportion of the adult population worldwide. The condition occurs predominantly in individuals between the ages of 45 and 70 years and present much more frequently in males than in females [1]. The estimated prevalence of claudication ranges from 0.4% to 14.4%. The occurrence of symptomless conditions identified through noninvasive examinations is notably greater, varying from 0.9% to 22%. The proportion of symptomatic cases to symptomless cases ranges from 1:0.9 to 1:6.

Factors that elevate the risk of atherosclerosis, such as advancing age, smoking, diabetes, dyslipidemia, and hypertension, enhance the probability of developing PAD in the lower extremities [2]. Individuals often report experiencing limb pain consistent with intermittent claudication, along with walking difficulties. Eventually, they may also encounter rest pain, notably at night, which can be described as sensations of coldness, burning, heightened sensitivity, and tingling [3].

Low-level laser therapy (LLLT) is a promising non-invasive physiotherapeutic method. The primary objective of this research was to investigate the impact of low-level laser therapy on the blood circulation to the lower extremities.

Material and Methods

Thirty-six (n=36) individuals treated in Warsaw Outpatient Clinics of Vascular Surgery were qualified for the study. All individuals had symptoms of intermittent claudication. The claudication distance was not less than 200 meters (Fontaine's stages II B). Segmental or total occlusion of the superficial femoral artery was found in ultrasound examination in all individuals. The duration of symptoms ranged from 6 to 27 months. All individuals had pharmacological therapy in accordance with the TASC II guidelines and walking training (3 times a week from 3-6 kilometers). All individuals underwent a Doppler ultrasound examination of the popliteal artery with flow measured in cm/sec performed once a week for 12 weeks. Each individuals received an application with Erchonia EVRL device, which emits a red laser (635 nm) and blue/violet laser (405 nm) both at 7.5mW. The laser procedure was performed once a week for 15 minutes. The area on the thigh was irradiated to the height of the occluded artery. Each individual provided written informed consent prior to participating in any study-related activity.

Results

All thirty-six (n=36) individuals completed the study. The age ranges from 41–80 years, with 24 men (67%) and 12 women. Among the respondents, there were 21 finger smokers, which constituted 58% of the study group and 15 people with diabetes, which constituted 42% of the study group. 10 subjects (28%) were both smokers and had diabetes.

Page 1 / 4

ERCHONIA RESEARCH 2025

36 PATIENTS WITH PERIPHERAL ARTERY DISEASE FROM ATHEROSCLEROSIS. PRIMARY OBJECTIVE MEASUREMENT WAS DOPPLER FLOW RATE TO THE POPLITEAL ARTERY (MEASURED IN CM/SEC). THIS STUDY INCLUDED DIABETICS, SMOKERS AND MORE WITH NO CHANGE IN THEIR MEDICATION OR EXERCISE SCHEDULES. ALL PARTICIPANTS HAD FULL OR PARTIAL OCCLUSION OF THE SUPERFICIAL FEMORAL ARTERY BASED ON ULTRASOUND AND ALL HAD CLAUDICATION TO THE LOWER EXTREMITIES AT <200 METERS.

DEVICE AND TREATMENT:
ERCHONIA RED/VIOLET
15 MINUTES OVER THE
THIGH TO THE HEIGHT OF THE
OCCLUDED ARTERY
1X/WEEK FOR 12 WEEKS

MEAN DOPPLER FLOW RATE IMPROVED FROM:
32.86 CM/SEC
TO
79.31 CM/SEC (WHICH IS CONSIDERED WNL!)
OVER DOUBLE FLOW IMPROVEMENT
(46.44 CM/SEC)
USING ONLY ERCHONIA RED/VIOLET LASER.
NO OTHER LIFESTYLE OR MEDICATION CHANGES!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

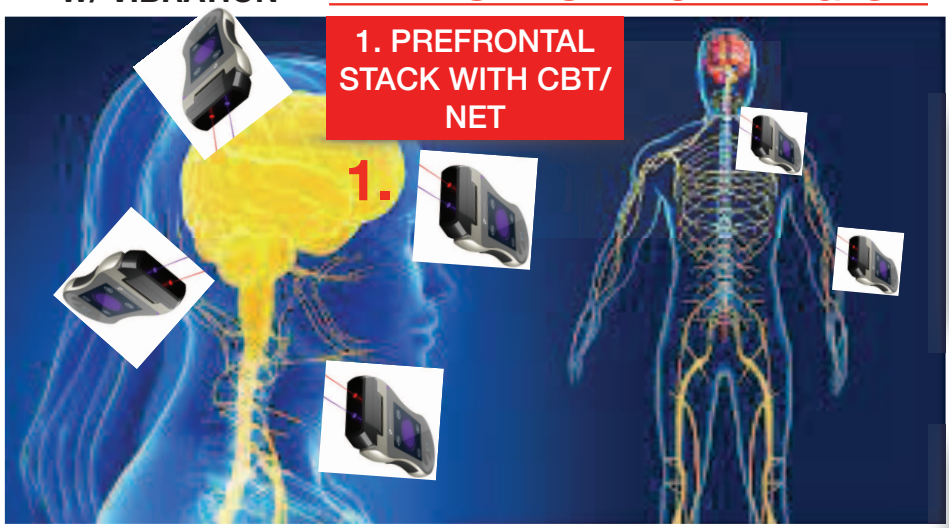
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE DOWNSTREAM PROTOCOL

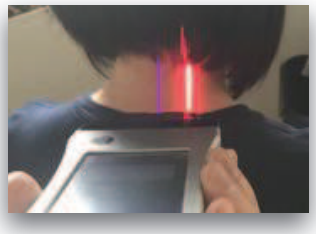
(FOR EVEN BETTER RESULTS, PERFORM AFTER BRAIN BALANCE)

SUMMARY OVERVIEW
2 MINUTES EACH REGION

2. PARIETAL LOBE W/ VIBRATION



5. SPINAL CORD/ NERVE ROOT



6. INVOLVED AREA/ DISTAL TISSUE

3. IPSI CEREBELLUM STACK WITH MOVEMENT

4. VAGAL NERVE STIMULATION IPSILATERAL TO INVOLVED SIDE

USE 10-40-10-40

ERCHONIA'S FDA CLEARANCE FOR POST-SURGICAL PAIN

FDA Grants 510(k) Market Clearance for Whole Body Postoperative Pain to World Leader in Low Level Laser Technology Randomized and controlled peer reviewed published clinical trials on 635 nm red laser prove effectiveness for treatment As the World Leader in Low Level Laser Technology, we have always believed we need to continue expanding the science of this amazing technology. This latest U.S. FDA 510(k) statement adds to our commitment. MELBOURNE, Fla. (PRWEB) November 01, 2021 Erchonia, the World Leader in Low Level laser Technology announces that on October 22, 2021, they have received their latest 510(k) statement from the U.S. FDA based on level 1 blinded and controlled clinical trials. 510(k) #211186 The new FDA 510(k) statement for whole body treatment of postoperative pain was based on the company's previously granted 510(k) statements from 2004 and 2008 along with data from seven randomized clinical trials on coronary artery bypass graft surgery, coronary bypass surgery with internal mammary artery grafts, tibial fracture surgery, cesarean section, endodontic surgery and tonsillectomy. This groundbreaking FDA market clearance provides a drug free & safe treatment option for postoperative pain. Based on clinical data, subjects who received Erchonia laser treatments

AVERAGE PAIN REDUCTION POST-OP >50%
REDUCTION IN NARCOTICS USE
RED LASER 635 NM 7.5 MW DIODES
5 MINUTES PRE-SURGICAL
5 MINUTES POST-SURGICAL

ERCHONIA
Healing Light, Perfected.

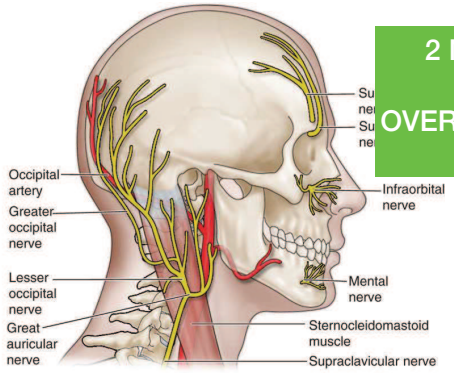
RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

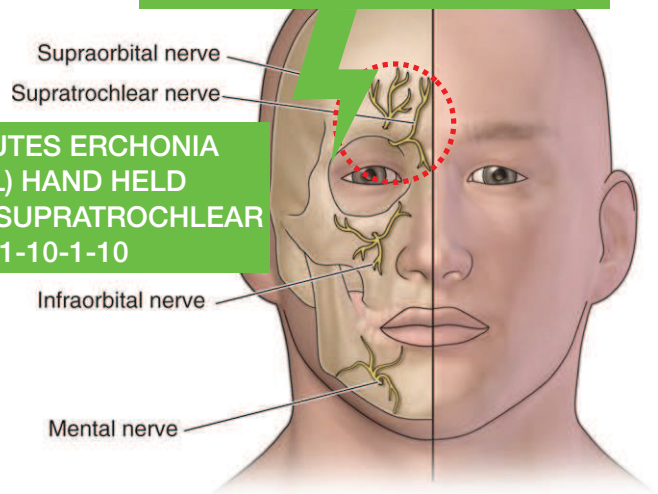
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SUPRAORBITAL NERVE

30 SECONDS OCULAR WISPING
(GREEN DIODE)
1-10-1-10
EYES OPEN VS CLOSED



2 MINUTES ERCHONIA
(GVL) HAND HELD
OVER V1/SUPRATROCHLEAR
1-10-1-10

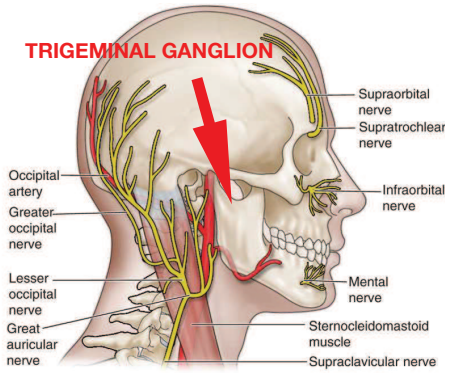


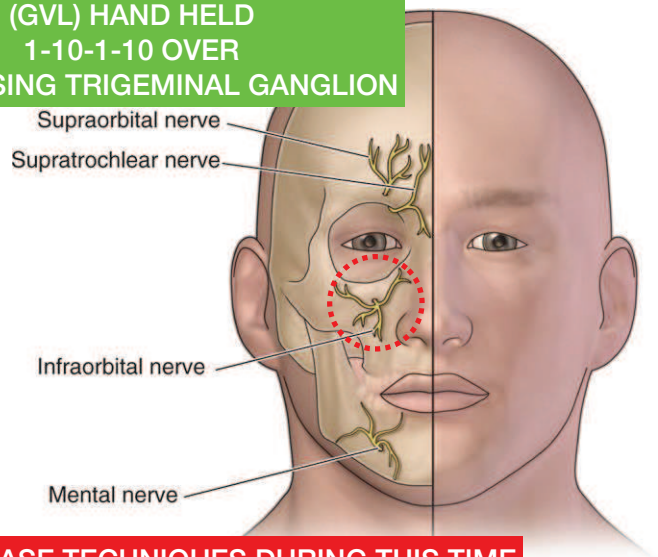
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
HEALTH

INFRATROCHLEAR NERVE AND TRIGEMINAL GANGLIA

2 MINUTES ERCHONIA
(GVL) HAND HELD
1-10-1-10 OVER
V2 CROSSING TRIGEMINAL GANGLION





CONSIDER MASTICATION RELEASE TECHNIQUES DURING THIS TIME
(INTERNAL AND EXTERNAL)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN
HEALTH

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

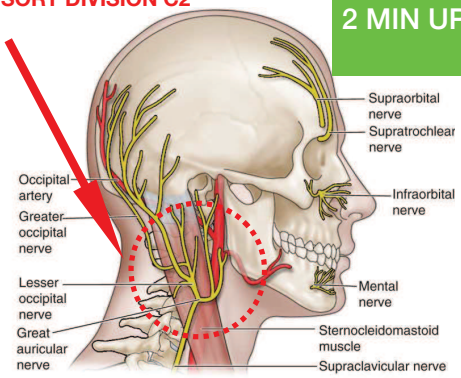
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

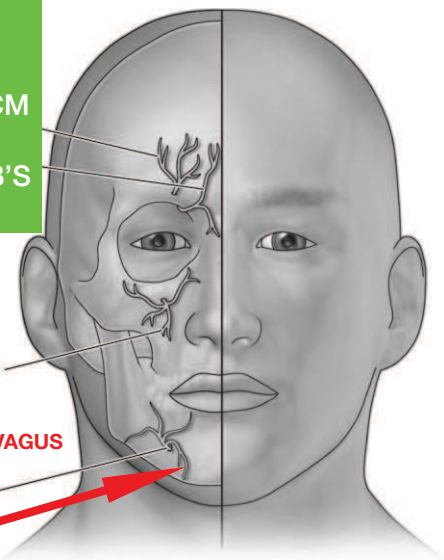
ERB'S POINT

2 MINUTES ERCHONIA
(GVL) HAND HELD
1-10-1-10
V3/MENTAL OVER VAGUS/SCM

2 MIN UPPER C REGION/ERB'S

UPPER CERVICAL COMPLEX
SENSORY DIVISION C2





CLINICAL GEM: ONCE BASE HEADACHE PROTOCOLS AND FOUNDATIONAL FUNC MED DIFFERENTIALS (IE. ANEMIA PATTERNS, MICRONUTRIENT DEPLETION ETC) HAVE BEEN RULED OUT, CONSIDER GUT MICROBIOME, HISTAMINES AND DYSAUTONOMIA FOR MIGRAINES INSUFFLATION TECHNIQUE

HEADACHE STACKING

STACKING THE BOX!

1. MSK CORRECTION ONCE BUCKET IS BIG ENOUGH TO TOLERATE (COUPLED CMT, OCULOMOTOR CORRECTION VERGENCE, MASTICATION/CRANIAL FASCIA - TIME PERMITTING DEMONS)
2. VAGAL QUAD WITH INSUFFLATION (DURING MIGRAINES LASER THE UPPER C/SCM, TO PREVENT MIGRAINES LASER THE AURICULAR BRANCH)
3. HEADACHE LLLT PROTOCOL (GVL IF AVAILABLE)
4. FX 405 SCAN TO CN5 DISTRIBUTION
5. TOPICAL APPLICATION (OFFICE FINISH AND HOME) PROLOGEL, CBD WITH MG, GLUTATHIONE
6. SUPPLEMENT SUPPORT (MQ-7 PREVENTION,)

ERCHONIA
Healing Light, Perfected.
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.
RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

TINNITUS
J Speech Lang Hear Res
2021 Jan 14;64(1):279-298. doi: 10.1044/2020_JSLHR-20-00066. Epub 2020 Dec 29.

Effects of Low-Level Laser Therapy as a Therapeutic Strategy for Patients With Tinnitus: A Systematic Review

Maria Carolina Ferreira [1], Izabella Lima de Matos [1], Isabela Porto de Toledo [2], Heitor Marques Honório [3], Maria Fernanda Capoani Garcia Mondelli [4]

All the seven selected studies found different degrees of significant results regarding tinnitus severity; however, there was no consensus among the results. Conclusion Even though the LLLT showed positive effects in the tinnitus severity in some studies, it is not possible yet to make any recommendation over its uses for the treatment of tinnitus severity.

CLINICAL CONSIDERATIONS:
LLLT - VAGAL PROTOCOLS (SCM AND AURICULAR/EAM)
TEMPORAL LOBE APPLICATIONS WITH STACKING 2+ MIN EACH MASTER 1-10-40-60
VESTIBULAR CO-ACTIVATION/TONE GENERATOR/TONGUE STIM (SEE NEXT SLIDE)
HEARING AIDS? UPPER C ADJUSTING
TMD THERAPIES/MASTICATION/EAR ADJUSTING/NASAL SPECIFIC (MURPHY)

OTHER CLINICAL CONSIDERATIONS:
OTC PAIN MEDS, NOISE POLLUTION, ANEMIA PATTERNS, METALS, Mg DEFICIENCY, LOW SODIUM DIET, (MICRONUTRIENTS PANEL), SOURCES OF CYOTOKINES TO TECTAL SITES (RUN FOOD SENSITIVITIES. CHRONIC PATHOGENS/MOLD, PAIN), THYROID DYSFUNCTION

TINNITUS

- Open access
- **Published: 19 August 2024**

Combining sound with tongue stimulation for the treatment of tinnitus: a multi-site single-arm controlled pivotal trial

- Michael Boedts,

Abstract

Bimodal neuromodulation is emerging as a nonsurgical treatment for tinnitus. **Bimodal treatment combining sound therapy with electrical tongue stimulation using the Lenire device is evaluated in a controlled pivotal trial** (TENT-A3, NCT05227365) consisting of 6-weeks of sound-only stimulation (Stage 1) followed by 6-weeks of bimodal treatment (Stage 2) with 112 participants serving as their own control. The primary endpoint compares the responder rate observed in Stage 2 versus Stage 1, where a responder exceeds 7 points in the Tinnitus Handicap Inventory. In participants with moderate or more severe tinnitus, there is a clinically superior performance of bimodal treatment (58.6%; 95% CI: 43.5%, 73.6%; $p = 0.022$) compared to sound therapy alone (43.2%; 95% CI: 29.7%, 57.8%), which is not observed in the full cohort across all severity groups. Consistent results are observed for the secondary endpoint based on the Tinnitus Functional Index (bimodal treatment: 45.5%; 95% CI: 31.7%, 59.9%; sound-only stimulation: 29.6%; 95% CI: 18.2%, 44.2%; $p = 0.010$), where a responder exceeds 13 points. There are no device related serious adverse events. These positive outcomes led to FDA De Novo approval of the Lenire device for tinnitus treatment.

ADVANCED TREATMENT STRATEGY: LENIRE DEVICE. MINISTIM OR STIMPOD TO TONGUE WITH AUDITORY THERAPIES LIKE TONE GENERATOR STACKED WITH LOW LEVEL LASER PROTOCOLS DESCRIBED IN PRIOR SLIDE

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN HEALTH

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

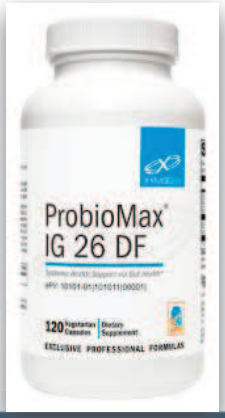
VAGAL NERVE STIMULATION PROTOCOL

CONSIDER THE SACRAL PLEXUS!

RESTORING BRAIN —HEALTH—

INFLAMMATION AND CAUSES OF DEATH

CLINICAL GUIDELINES FOR LPS INDUCED INFLAMMATION REDUCTION AND ENDOTOXEMIA:
1. FIX THE LEAKY GUT. (GI BALANCE XYMOGEN, REPAIR AND CLEAR APEX, TOTAL LEAKY GUT NUTRIWEST). RUN TESTS LIKE VIBRANT WHEAT ZOOMER AND FOOD SENSITIVITIES TESTS
2. BRAIN GUT AXIS. VAGAL NERVE STIMULATION PROTOCOL, PREFRONTAL INHIBITION/GABA
3. IMMUNOGLOBULIN THERAPIES (IGG/IGY, IVIG, COLOSTRUM)
4. LOW LEVEL LASER PROTOCOLS (FOCUS ON MASTER BRAIN, VAGAL/BRAIN-GUT AXIS, AND GLOBAL BODY PROTOCOLS
5. TARGET 3 MONTHS OF GUT REPAIR, FOOD ELIMINATION, SUPPLEMENT SUPPORT AND LLLT
6. AVOID GLUTEN/WESTERN WHEAT



Front. Nutr., 21 June 2018
Sec. Nutritional Immunology
Volume 5 - 2018 | https://doi.org/10.3389/fnut.2018.00052

Effects of Bovine Immunoglobulins on Immune Function, Allergy, and Infection

.2018 Apr;49(4):474-482. doi: 10.1097/SHK.0000000000000937.

Protection Against Lipopolysaccharide-Induced Immunosuppression by IgG and IgM

Christiana Kyvelidou 1, Dimitris Sotiriou, Ioanna Zerva, Irene Astrakianakis

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

Supplement Facts

Serving Size: 2 Scoops (about 13.7 g)
Servings Per Container: 14

	Amount Per Serving	%DV
Calories	40	
Total Carbohydrate	7 g	3%†
Dietary Fiber	2 g	7%
Total Sugars	1 g	**
Zinc (as zinc L-carnosine)	32.25 mg	293%
L-Glutamine	4 g	**
Slippery Elm (*Ulmus fulva*)(bark)	2 g	**
N-Acetyl-D-Glucosamine	2 g	**
Aloe Vera 200:1 Extract (*Aloe barbadensis*)(leaf gel)	600 mg	**
Porcine Gastric Mucin	400 mg	**
Green Kiwifruit (*Actinidia deliciosa*)(fruit)(62,500 AU actinidin)[S1]	400 mg	**
Marshmallow (*Althaea officinalis*)(root)	400 mg	**
Licorice Extract (*Glycyrrhiza glabra*)(root)(3.5% glabridin)[S2]	300 mg	**
Quercetin (as quercetin dihydrate)	200 mg	**
Prune (*Prunus domestica*)(fruit)	200 mg	**
Chamomile Extract (*Matricaria recutita*)(flowering head)(1% apigenin-7-glucoside)	200 mg	**
Cat's Claw (*Uncaria tomentosa*)(bark)	200 mg	**
Okra (*Hibiscus esculentus*)(pod)	200 mg	**

† Percent Daily Values are based on a 2,000 calorie diet.
** Daily Value (DV) not established.

Other Ingredients: Guar gum, natural flavors, and stevia leaf extract.

395

EXAMPLE OF A BROAD SPECTRUM GI AND BARRIER SYSTEM REPAIR PRODUCT

NOTE: There is No Evidence of Glutamate Toxicity with Oral Doses
Clinical studies using **5–30 g/day of glutamine** in gut healing, cancer, and trauma patients **do not show increased neurotoxicity.**

In fact, glutamine often reduces inflammation and enhances gut–brain axis stability.

Reference:
Cury-Boaventura, M. F., et al. (2006). Effect of oral glutamine on gut permeability and plasma cytokines in severely ill patients. **JPEN Journal of Parenteral and Enteral Nutrition**, 30(5), 480–486. https://07106030005480

RESTORING BRAIN —HEALTH—

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

TURMERIC
BCM-95 VS PIPERINE
DOSE: BASELINE 500 MG DAILY
CHRONIC INFLAMMATORY CONDITIONS AND NEURODEGENERATIVE CONDITIONS
MIN 1000 MG DAILY (500 MG BID OR TID)
BEST TAKEN WITH FOOD FOR ABSORPTION
INHIBITS NF-KB, IL-6, TNF-ALPHA

OMEGA 3 FATTY ACIDS
MIN 2G, TARGET 3G/DAY
(2G EPA/ 1G DHA)
WITH MEALS
INHIBITS PGE2
SPM/PRMS?

RESVERATROL (ACTIVATED TRANS-RESVERO)
MINIMUM 200 MG BUT TARGET UP TO 1,000 MG (500 MG BID)
SYNERGISTIC EFFECT WHEN COMBINED WITH TURMERIC
ALSO GOOD FOR BARRIERS

1. Choi, J. Y., Kim, Y. H., & Lee, J. (2010).
"Synergistic neuroprotective effects of resveratrol and curcumin against oxidative stress in rat primary cortical neurons."
Journal of Medicinal Food, *13*(3), 608–614.
https://doi.org/10.1089/jmf.2009.1271
• Conclusion: Co-treatment more effectively reduced markers of oxidative stress and neuronal damage compared to single treatment.

2. Wang, H., Liu, Y., Briesacher, J. A., et al. (2018).
"Synergistic anti-inflammatory effects of curcumin and resveratrol in astrocyte cultures."
Neurochemical Research, *43*(5), 1025–1033.
• Demonstrated that the combination significantly reduced proinflammatory cytokine release (TNF-α, IL-1β) from activated glial cells more effectively than either agent alone.

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

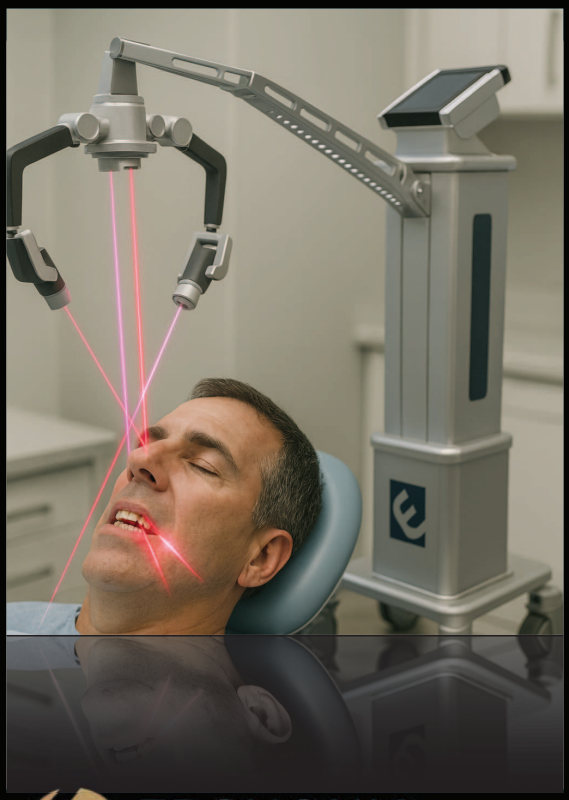
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA eCommunity Education

EVRL 405 NM LASER

"BACTERIA SETTING" 20-664-787-1560
4 MINUTES
1 MINUTE NASAL, 1 MINUTE THROAT
1 MINUTE MAXILLARY GUM LINE
1 MINUTE MANDIBULAR GUM LINE

OTHER CONSIDERATIONS:
REGULAR FLOSSING, BRUSHING AND DENTAL HYGIENE
LOW SUGAR/CARB DIET
"OIL PULLING" WITH COCONUT OIL
ARGENTYN SILVER



ERCHONIA
Healing Light, Perfected.

LASERS AND LONG LASTING SMILES:
THE ULTIMATE DENTAL STACK

PROGRAM YOUR "MASTER DENTAL" SETTING ON AN FX 405 LASER TO COMBINE BRAIN FREQUENCIES WITH ANTI-MICROBIAL

10-20
40-664
10-787
40-1560

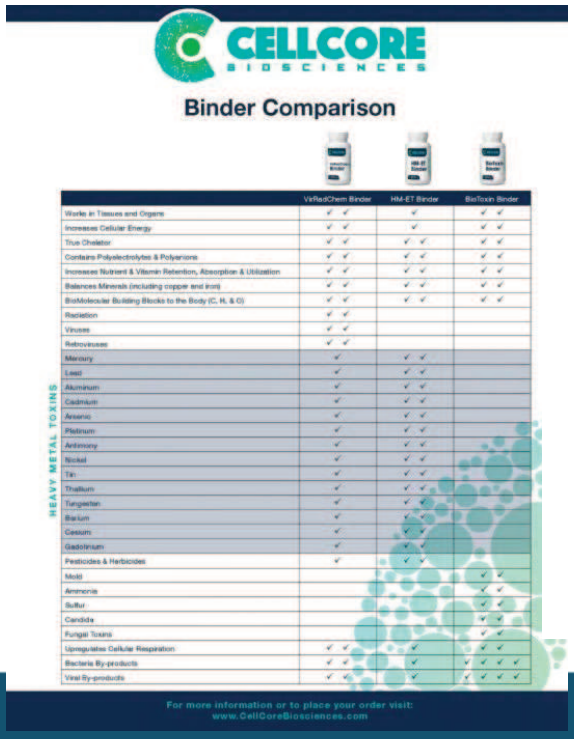
10 MINUTES OVER THE HEAD AND FACE AREA DURING YOUR PROCEDURES

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

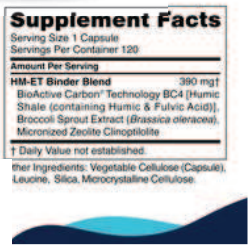
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CHELATION

WHAT SHOULD BE CONSIDERED BEFORE ENGAGING IN HEAVY CHELATION SCHEDULES?



INTACT BARRIER SYSTEMS
ESPECIALLY BBB
HEALTHY LIVER/DETOX SYSTEMS
HEALTHY ANTI-OXIDANT SYSTEMS
CONSIDER LIVER, KIDNEY, GUT AND BRAIN PROTOCOLS



RESTORING BRAIN —HEALTH—

ADIPOSE REDUCTION

BASIC 8 TREATMENT PLATFORM:
START BINDERS IMMEDIATELY ON INITIAL CONSULT
2X/WEEK FOR 3 WEEKS
1X/WEEK FOR 2 WEEKS
FOLLOW ERCHONIA'S TREATMENT PLAN (LYMPHATICS, WALK, H20 ETC)
PRE-SCREEN MAF

ZERONA Z6 (OTC)

OTHER CONSIDERATIONS:
EXTENDED TX PLANS BASED ON SIZE AND GOALS
REFERRAL VISITS
"MEMBERSHIP MODEL"

THE NEW VZ8

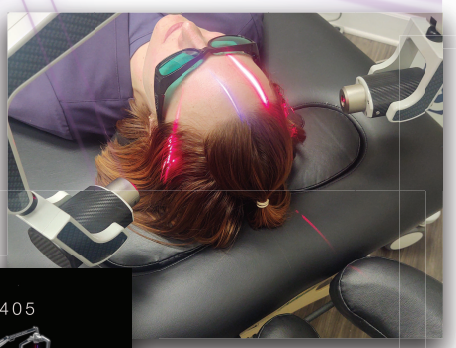
ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE ULTIMATE ERCHONIA PRACTICE FLOW



ALL PATIENTS GET:

10 MINUTES VZ8 (ZERONA/EMERALD) TO GUT
10 MINUTES FX 405 TCT ALL DIODES 10-40
START PATIENTS ON BINDERS AND LPS SUPPORT

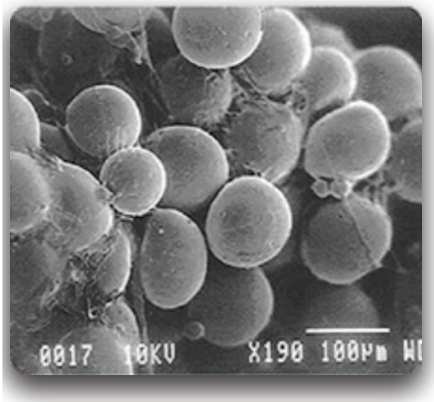
HAVE BASE STATION FOR BEDSIDE/BACK OFFICE:
VAGAL QUAD
CEREBELLAR EXERCISE STACK
MSK

1 BASE STATION HAND HELD
FOR WEEKLY RENTAL

401

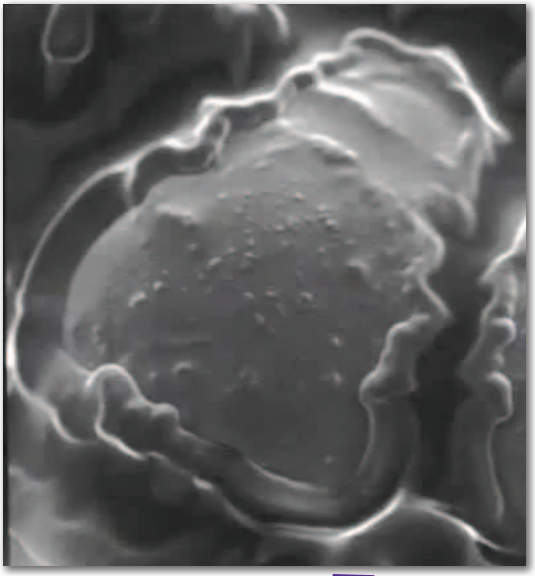
ERCHONIA
Healing Light, Perfected.

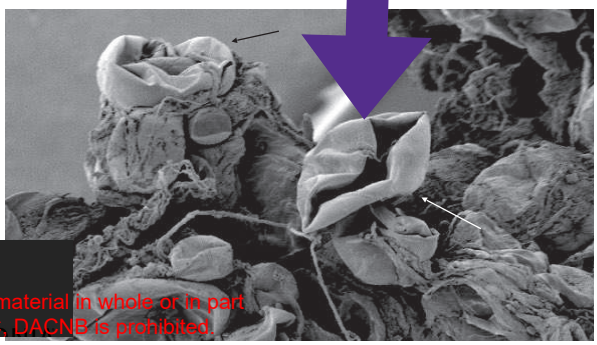
RESTORING BRAIN HEALTH



ZERONA
AND
VZ8
LASERS

cAMP &
HSL





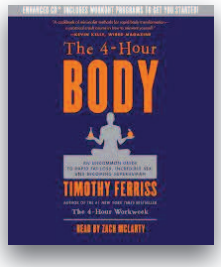
ERCHONIA
Healing Light, Perfected.

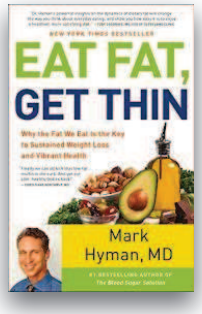
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

NUTRITIONAL SUPPORT FOR ADIPOSE REDUCTION

THE 30-30-30 FAT MELTING WORKOUT ROUTINE





DO NOT WAKE UP, SKIP BREAKFAST, HAMMER OUT AN HOUR WORKOUT AND CATABOLIZE YOUR LEAN MUSCLE!
YOU HAVE ABOUT 20 MIN OF GLYCOGEN RESERVES AND IT TAKES ABOUT 5 HOURS TO START TURNING FAT INTO ENERGY
=> YOUR BODY STARTS EATING ITSELF WITHIN 3 MINUTES WITHOUT AN INTERMEDIATE FUEL SOURCE

TO STRIP FAT NOT YOUR OWN MUSCLE, EAT 30 GRAMS OF PROTEIN WITHIN 30 MINUTES OF WAKING UP AND THEN DO 30 MINUTES OF STEADY STATE WORKOUTS (HR BELOW 135)

Q: "HOW DO I INTRODUCE MY ERCHONIA DEVICE TO MY PATIENTS?"

A: PRE- AND POST- ASSESSMENTS WITHOUT EVEN TOUCHING THE PATIENT (NEURO SIGNS, ROM ETC)

USE YOUR LASER AS A DIAGNOSTIC TOOL!
IF YOUR PATIENT IS HAVING A DIFFICULT TIME WITH A BEDSIDE EXAM TEST GET THE LASER ON THE INVOLVED REGION AND SEE IF THEY IMPROVE

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

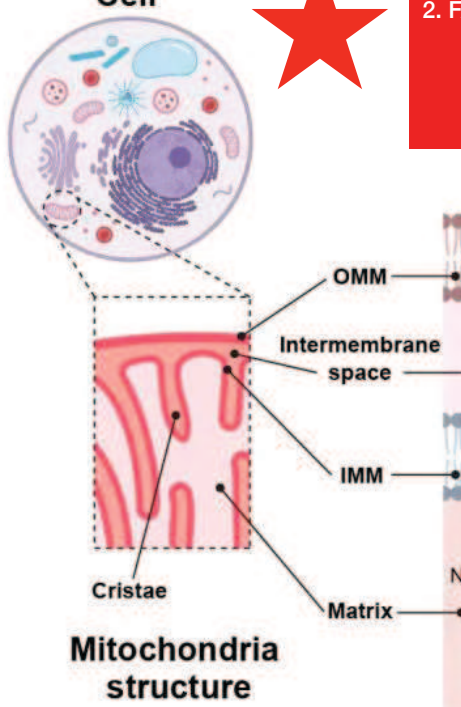
LASER CONSIDERATIONS

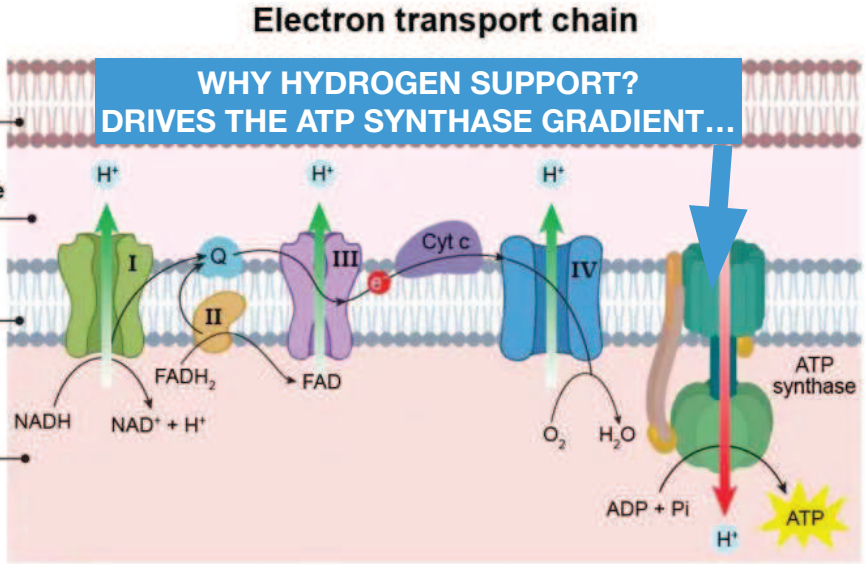
IF YOUR BASE "SET IT AND FORGET IT APPLICATIONS ARE NOT REACHING YOUR DESIRED TREATMENT GOALS CONSIDER THE FOLLOWING:

1. ALWAYS "SWIM UP STREAM OR DOWNSTREAM". DID YOU DO BRAIN BASED AND/OR VAGAL NERVE AND SPINAL CORD/NERVE ROOT STIMULATION PROTOCOLS?
2. LENGTHEN TREATMENT TIMES
3. SHORTEN TREATMENT TIMES (REMEMBER HORMESIS ESPECIALLY IN HIGH METABOLIC TISSUES LIKE BRAIN)
4. ADD VIOLET WAVELENGTH
5. ADD GREEN WAVELENGTH
6. FX SCANNING VS LINE BEAM. COVER MORE SURFACE.
7. STACK THERAPIES WITH CO-ACTIVATION
8. UTILIZE THE SCANNING FREQUENCY
9. REFER THE PATIENT FOR IMAGING/OTHER TESTING FOR UNDERLYING PATHOLOGY

MITOCHONDRIA AND THE ELECTRON TRANSPORT CHAIN

SUMMARY: 1. NADH FROM KREB'S/GOOD FATS AND PROTEINS, VIOLET LASER, NAD+ DRIP, ORAL LIKE NMN, METHYLENE BLUE
2. FAD FROM VIOLET LASER, VIT B2 (USE RIBOFLAVIN 5' PHOSPHATE = FMN)
3. COQ 10 AND GREEN LASER
4. RED LASER. (CCO ALSO NEEDS COPPER AND IRON!)
YOU MUST HAVE O2 (RESPIRATORY MECHANICS, HBOT 20+ DIVES)
MAKE MORE MITOCHONDRIA (LLLT, HBOT, EXERCISE, PQQ)





Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

PARKINSON'S DISEASE

FIX BARRIER SYSTEMS/LEAKY GUT PROTOCOL/VAGAL NERVE STIMULATION

ADDRESS DOPAMINE NEUROCHEMISTRY/CO-FACTORS/MICRONUTRIENTS

LLLT GI AND SCM vagal setting (10-10-10-10). VAGAL PROTOCOL (2min SCM while percussor gut then 2 min gut). Also consider SCM stem with laser while doing motor exercises/rehab/"unfreezing

LLLT MASTER BRAIN SETTING 1-10-40-60 OR BRAIN RESTORE SETTING (10-40-10-40) (BIAS DIODES TO FRONTAL CORTEX AND CONTRALATERAL CEREBELLUM USING FX) Avoid Beta Hz oscillations (around 13-30Hz). Consider co-activation with targeting saccades (SVI/Ipad tapping/ISOMETRICS/LINEAR PROGRESSION/ANY TARGET/ BOXING GYMS/VIDEO GAMES).

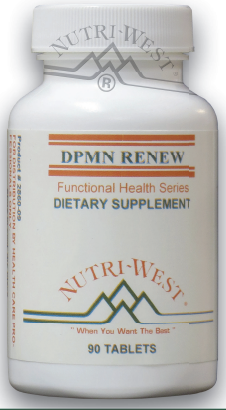
Initial treatment trial = 3x/week 4 weeks, monitor progress and phase down.

BIAS TREATMENTS TO FX 405 OR EVRL BUT STACK WITH GVL WHEN AVAILABLE

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

PARKINSON'S DISEASE

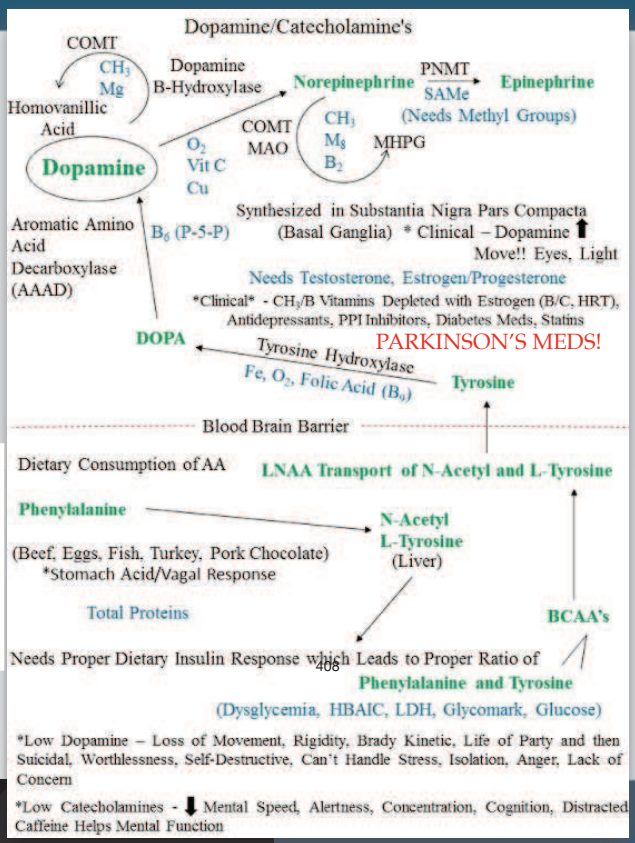


Supplement Facts
Serving Size 1 TABLET

Amount Per Serving		%DV
Proprietary blend	500 mg	*

Mucuna Pruriens Extract (plant), N-Acetyl L-Tyrosine, Organic Blueberry (fruit), G.A.B.A (gamma aminobutyric acid), Ginkgo Biloba Extract (leaf), Berberine HCL (bark), Cytidine 5 Diphosphocholine.

TAKE PRN AND PRIOR TO PHYSICAL REHAB!



CoQ10 (ubiquinol): 200–400 mg/day

Alpha-lipoic acid: 300–600 mg/day

NAD⁺ support (NMN or NR): 250–500 mg/day

Omega-3s (EPA/DHA): 2–4 g/day

Curcumin (bioavailable): 500–1,000 mg/day

RUN LABS
MICRONUTRIENT PROFILES

OTHER NUTRIENT SUPPORT
MUCUNA
IBOGAINE/MUSHROOMS

FIX LEAKY GUT
ADDRESS AUTOIMMUNITY

FALL PREVENTION/ SECONDARY INJURY

IMPROVEMENTS IN ADL…
IT'S NOT A CURE

SLOW PROGRESSION? DBS?
REGENERATIVE MEDICINE?

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION: CLINICAL SUPPORT SUMMARY. (SUPPORT SLIDES IN THE BACK OF SLIDE DECK)

1. MOVE! EXERCISE, ESPECIALLY IN SUNLIGHT AND IN A "GROUNDING" ENVIRONMENT. LIGHT THERAPIES
2. MAKE SURE THEY HAVE THE RIGHT BUILDING BLOCKS FOR NEUROCHEMISTRY. EAT A GOOD FIBER/VEGGIE, HIGH IN GOOD FATS AND PROTEINS. RUN LABS FROM BASE CBC/CHEM SCREENS, HORMONE TESTING (DUTCH OR VIBRANT), MICRONUTRIENT PROFILE AND INFLAMMATION SOURCES (FOOD SENSITIVITIES, PATHOGENS, HBAIC)
3. A HEALTHY GUT MICROBIOME (GUT ZOOMER 3.0)
4. ERCHONIA LOW LEVEL LASER THERAPY (RIGHT PREFRONTAL CORTEX 6-9-10-12. STACK WITH 5 MIN MEDITATION OF GRATITUDE/5 MIN CLEARING OF MIND). VAGAL NERVE STIM PROTOCOL (10HZ)
5. TRANSCRANIAL MAGNETIC STIMULATION. PEMF. NEUROFEEDBACK
6. CBD PRODUCTS AND CLASSIC PSYCHEDELICS (MICRO OR MACRO DOSING. WATCH LEGAL MATTERS ON DMT -> DMN THERAPIES - SCHEDULE 1 SUBSTANCES)

ERCHONIA
Healing Light, Perfected.

RESTORING BRAIN HEALTH

PEPTIDES FOR BRAIN HEALTH

Photobiomodulation Enhancing Stem Cell Homing & Migration

1. LLLT Attracts Stem Cells via Chemotactic Signaling

Citation:
Ma, Z., et al. (2017). *Photobiomodulation enhances mesenchymal stem cell migration via the CXCR4/SDF-1α axis*. **Lasers in Medical Science, 32(3), 641–647.**
https://doi.org/10.1007/s10103-017-2156-y

- **Findings**: PBM increased **CXCR4 expression** on mesenchymal stem cells (MSCs) and upregulated **SDF-1α** in injured tissue—enhancing **MSC homing** to damaged areas.
  Mechanism: This is the same axis used in bone marrow homing and repair.

2. LLLT Enhances Engraftment of Transplanted Stem Cells

Citation:
Zhou, T., et al. (2016). *Photobiomodulation preconditioning enhances the therapeutic efficacy of mesenchymal stem cells in myocardial infarction*. **Cell Transplantation, 25(5), 1083–1093.**
https://doi.org/10.3727/096368916X690668

- **Findings**: PBM pretreatment increased **survival and homing** of transplanted stem cells to ischemic heart tissue.
- **Outcome**: Improved cardiac function and infarct size reduction.

TREATMENT SUGGESTION: STACK YOUR LOW LEVEL LASER USING 10-40-10-40
OVER THE TARGET REGION OF THE BRAIN OR SPINAL CORD WITH CO-ACTIVATION (IE. SENSORY STIMULATION IF ITS A PARIETAL LOBE LESION). MINIMUM 4 VISITS POST REGENERATIVE TREATMENT. 12 VISIT TRIAL

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP

- POLYSOMNOGRAPHY (CENTRAL VS OBSTRUCTIVE APNEA VS BODY MOVEMENTS)
- C-PAP/BI-PAP, BREATHING STRIPS, BED/MATTRESS POSITION/PILLOW
- TONE OF ORAL-PHARYNGEAL REGION (VAGAL STIM/DIRECT SOFT PALATE STIM) MOUTH TAPING WITH BREATHE RIGHT NASAL STRIPS
- BASE LAB CHEMISTY (ANEMIA PATTERNS, DYSGLYCEMIA, THYROID, SEX HORMONES…HP AXIS)
- ADVANCED LAB MARKERS: MICRONUTRIENT (CO-FACTORS FOR MELATONIN SYNTHESIS)
- ASI/CORTISOL CURVE AND GLYCOGEN STORES. CORTISOL VS MELATONIN
- CYTOKINES AND MESENCEPHALIC RETICULAR ACTIVATING SYSTEM. FOOD SENSITIVITY TESTING, INFECTIOUS DISEASE MANAGEMENT
- PAIN MANAGEMENT
- BASAL GANGLIA ACTIVATION/DISINHIBITION
- QUALITY OF PILLOW, MATTRESS (AGE ETC)
- ELECTRONICS AT NIGHT, AVOID BLUE WAVELENGTHS/NIGHT SETTING, MINIMIZE VISUAL STIMULATION THROUGHOUT DAY/COMPUTER FILTERS
- QEEG/NEUROFEEDBACK
- CBD/MEDICAL MARIJUANA. MELATONIN CR, PROBIOTICS, GABA

LOW LEVEL LASER TO CNS…

SLEEP

Front Hum Neurosci. 2018; 12: 387.
Published online 2018 Sep 24. doi: 10.3389/fnhum.2018.00387

A Novel Insight of Effects of a 3-Hz Binaural Beat on Sleep Stages During Sleep

Nantawachara Jirakittayakorn* and Yodchanan Wongsawat*

These findings suggested that the 3-Hz binaural beat on a 250-Hz carrier tone can be used to modulate sleep stage by decreasing the latency to the N3 stage, **extending the N3 duration, and reducing the N2 duration without sleep disturbance and sleep fragmentation, while increasing the quality of sleep in this study is also associated slow wave sleep in which is related to memory consolidation and other regulation of the body; and the 3-Hz binaural beat can enhance power of delta activity during sleep.**

J Family Med Prim Care. 2019 Dec; 8(12): 3915–3919.
Published online 2019 Dec 10. doi: 10.4103/jfmpc.jfmpc_575_19

Effect of music of specific frequency upon the sleep architecture and electroencephalographic pattern of individuals with delayed sleep latency: A daytime nap study

Pramita Dubey,1 Yogesh Kumar,1 Ramji Singh,1 Kamlesh Jha,1 and Rajesh Kumar1

Conclusion:

It was concluded that 432 Hz music has some significant calming effect as reflected by increased alpha activities … naps.

Nature
. 2006 Nov 30;444(71

Boosting slo

Lisa Marshall 1 Halla

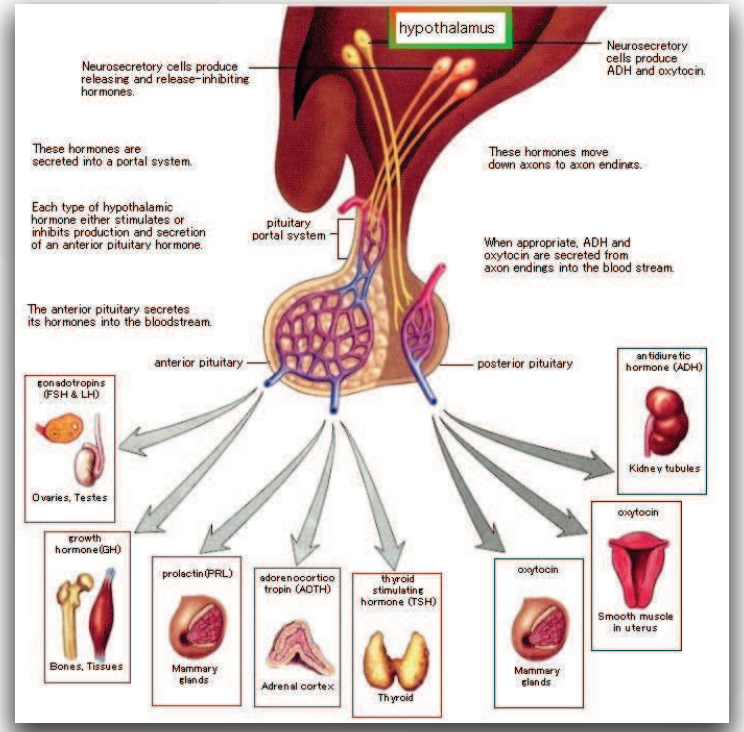
Here we show that in … g potentials (0.75 Hz) during … w wave sleep, enhances the … ories in healthy huma … ve sleep, endogenous cortical …

BASE SLEEP SETTING 1 - 2 - 3 - 432
RED XLR8
ALSO CONSIDER "REST AND DIGEST"
VAGAL STIM IN THE PM (1-10-1-10)
COMBO SETTING (1-3-10-432)
10 MIN PROTOCOL BEFORE BED
(8 MIN EACH PRE-FRONTAL CORTEX
2 MIN VAGAL STIM SCM REGION)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

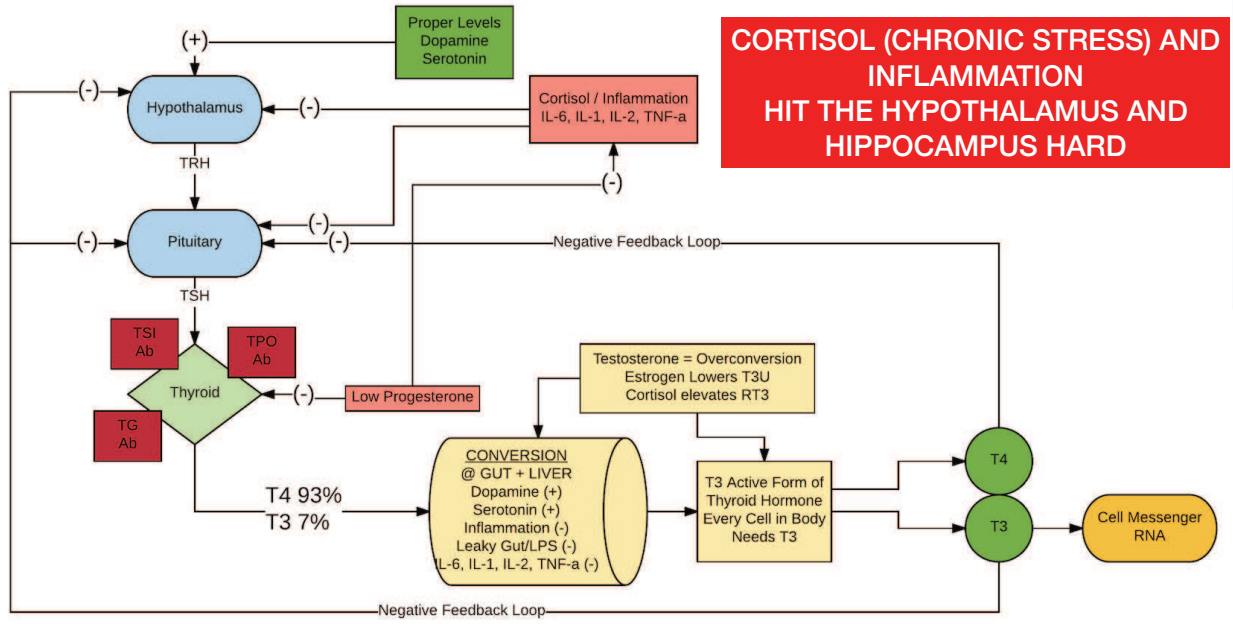
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HP AXIS: 2025 MATERIAL BELOW



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

THYROID



CORTISOL (CHRONIC STRESS) AND INFLAMMATION HIT THE HYPOTHALAMUS AND HIPPOCAMPUS HARD

THYROID DYSFUNCTION COULD BE A DIFFERENTIAL FOR ALMOST EVERY CONDITION OF THE HUMAN BODY INCLUDING BRAIN DEGENERATION, GUT ISSUES LIKE GERD AND SO ON. EVERY CELL IN THE HUMAN BODY NEEDS THYROID HORMONE!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THYROID LAB TESTING
RUN A COMPLETE PANEL

- **T4, Total** – T4 (thyroxine) is a hormone produced by the thyroid gland. This test measures the amount of T4 in your blood.
- **T4, Free** – Most of the T4 in your body binds to protein, the T4 that does not is called T4 and circulates unbound in your blood. This test measures these T4 hormones.
- **T3, Total** – T3 (triiodothyronine) is a thyroid hormone and is important for the body's control of metabolism. This test measures the amount of T3 in your blood.
- **T3, Free** – Most of the T3 in your body binds to protein, the T3 that does not is called T3 and circulates unbound in your blood. This test measures these T3 hormones. ACTIVE FORM OF THYROID
- **T3, Reverse** – Reverse T3 is converted by the liver from the stored hormone T4, it is the body's way of getting rid of unneeded T4 every day. Sometimes the body can make too much reverse T3 from a number of causes including chronic emotional, physical, or biological stress, chronic sickness, after surgery, diabetes, aging, or an acute injury. This test measures for levels of reverse T3.
- **Thyroid Stimulating Hormone (TSH)** – TSH causes the thyroid gland to make two hormones, T3 and T4, which help control your body's metabolism. This test is done to find out whether the thyroid gland is working properly.
- **Thyroglobulin Antibody** – Thyroglobulin is a protein produced and used by the thyroid gland to make the hormones T3 and T4. Antibodies are the proteins made by the immune system to fight toxins, bacteria, and viruses. High levels of antibodies suggest the immune system is mistakenly attacking the thyroid gland. This test measures the blood levels of antibodies the body has made against the compound thyroglobulin.
- **Thyroid Peroxidase Antibody** – Thyroid peroxidase (TPO) is an enzyme made in the thyroid gland and is important in producing the thyroid hormone. TPO converts to thyroid hormone T4 to T3. High levels of antibodies suggest the immune system is mistakenly attacking the thyroid gland. This test measures the level of antibody the body has made against thyroid peroxidase.

THYROID AND LLLT

Low-level laser in the treatment of patients with hypothyroidism induced by chronic autoimmune thyroiditis: A randomized, placebo-controlled clinical trial

- **June 2012 Lasers in Medical Science 28(3)**

These findings suggest that LLLT was effective at improving thyroid function, promoting reduced TPOAb-mediated autoimmunity and increasing thyroid echogenicity in patients with CAT hypothyroidism.

Volume 2012 | Article ID 126720 | https://doi.org/10.5402/2012/126720
Show citation

Assessment of the Effects of Low-Level Laser Therapy on the Thyroid Vascularization of Patients with Autoimmune Hypothyroidism by Color Doppler Ultrasound

Abstract

Background. Chronic autoimmune thyroiditis (CAT) frequently alters thyroid vascularization, likely as a result of the autoimmune process. *Objective*. To evaluate the effects of low-level laser therapy (LLLT) on the thyroid vascularization of patients with hypothyroidism induced by CAT using color Doppler ultrasound parameters. *Methods*. In this randomized clinical trial, 43 patients who underwent levothyroxine replacement for CAT-induced hypothyroidism were randomly assigned to receive either 10 sessions of LLLT (L group,) or 10 sessions of a placebo treatment (P group,). Color Doppler ultrasounds were performed before and 30 days after interventions. To verify the vascularity of the thyroid parenchyma, power Doppler was performed. *Results*. The frequency of normal vascularization of the thyroid lobes observed in the postintervention power Doppler examination was significantly higher in the L than in the P group (). The pulsed Doppler examination revealed an increase in the SPV of the ITA in the L group compared with the P group (). No significant differences in the SPV of the STA and in the RI were found between the groups. ***Conclusion*. These results suggest that LLLT can ameliorate thyroid parenchyma vascularization and increase the SPV of the ITA of patients with hypothyroidism caused by CAT.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Researchers in Brazil have been studying the effects of low level laser therapy on the thyroid gland in Hashimoto's thyroiditis, and the results have been astonishing!

Most significantly, they found that all patients who received the therapy were able to reduce their levothyroxine dose, while 47% were able to discontinue levothyroxine and have normal thyroid function during the 9-month follow-up.

Additionally, LLLT can increase Transforming Growth Factor B (TGF-B), which is a cytokine that helps to induce and maintain the tolerance of self thus, also reduce thyroid autoimmunity.

- **a reduction of levothyroxine dose from a mean of 93 mcg to 38 mcg (with 95.7% of treatment group being able to reduce or stop medications…47.8% no longer needing thyroid medications)**
- **a reduction in TPOAb, mean 1289 to 656 (around 50% reduction)**
- **a reduction in TG antibodies, 720 to 656**
- **a normalization of thyroid volume (in 66%)**
- **and less infiltration of the thyroid gland on ultrasounds (meaning fewer inflammatory cells were present). Echogenicity index on thyroid ultrasound was improved in 95% of the study group, meaning that their thyroid gland had less damage and fewer white blood cells on ultrasound.**

THYROID

International Journal of Endocrinology
Volume 2018, Article ID 8387530, 9 pages
https://doi.org/10.1155/2018/8387530
Clinical Study

Safety and Efficacy of Low-Level Laser Therapy in Autoimmune Thyroiditis: Long-Term Follow-Up Study

Danilo Bianchini Höfling,[1] Maria Cristina Chavantes,[2] Carlos Alberto Buchpiguel,[3] Giovanni Guido Cerri,[3] Suemi Marui [4] Paulo Campos Carneiro,[5] and Maria Cristina Chammas

Introduction. A randomized clinical trial (RCT) was performed to evaluate the efficacy of low-level laser therapy (LLLT) for hypothyroidism induced by chronic autoimmune thyroiditis (CAT). *Objective*. The objective was to assess the safety and actions of LLLT 6 years after completion of the RCT. *Materials and Methods*. Forty-three participants were invited to participate in this study 6 years after completion of the RCT. Twenty-five were subjected to LLLT (group L), and 18 were subjected to placebo (group P). Primary outcome measure: frequency of thyroid nodules, which were subjected to fine-needle aspiration biopsy. Secondary outcome measures: dose of levothyroxine required to treat hypothyroidism, thyroid peroxidase antibodies (anti-TPO), and anti-thyroglobulin antibodies (anti-Tg). *Results*. In group L, a nodule was observed in three patients, who all had a Bethesda II classification. In group P, a nodule was also observed in three patients, with two classified as Bethesda II and one as Bethesda III. The levothyroxine dose required by group L was significantly lower than that required by group P (). The anti-TPO and anti-Tg levels did not differ between the groups. ***Conclusion*. LLLT, by the methods described, has been shown to be safe for the treatment of hypothyroidism resulting from Chronic Autoimmune Thyroid.** This trial is registered with ClinicalTrials.gov Identifier: NCT02240563.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

BASIC THYROID LASER PROTOCOL
COURTESY OF KIRK GAIR

BASIC THYROID PROTOCOL

Step 1: Laser over the cervical spine and thoracic spine down to the adrenals for 2-3 minutes with the basic frequencies of 9, 16, 42, and 53 hz.

Step 2: You can also use the thyroid specific frequencies of 20, 59, 802, 3884 hz over the thyroid or near it for 2-3 minutes *(if there is a history of thyroid cancer, although there is research that supports the use of laser for cancers, I would recommend not using it directly over the thyroid until there is more of a consensus from the research)*

Step 3: Laser 2-3 minutes over the liver using the frequencies of 53, 55, 537, 751 hz.

Step 4: Laser 2-3 minutes over thymus using the frequencies of 20, 73, 465, 728 hz.

NOTE: If at any time... lasts. If you have a... very much stimulat... ...te how long it ...able to tolerate

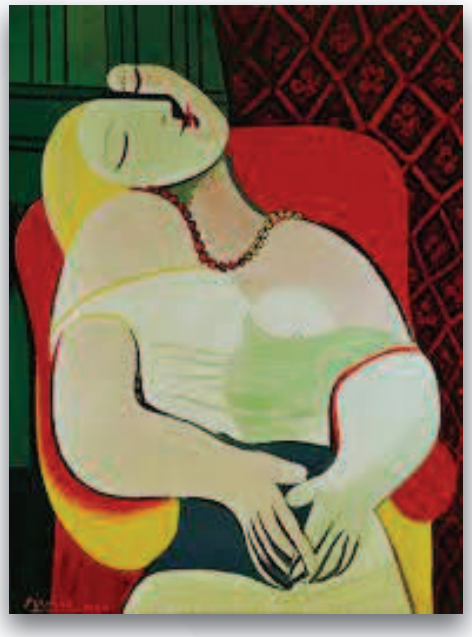
Additional note: I S... contacted by pati... and an ER visit with... laser use in the ma... ...cols. I have been ...thyroid storm ...port that type of

62

BROCK PROTOCOL
HYPOTHYROID 20-59-822-3884
HYPERTHYROID 3-20-59-160

USE GVL OR EVRL IF AVAILABLE

REPRODUCTIVE SYSTEM



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

"DOWNSTREAM PROTOCOL"

COMBINE WITH VAGUS PROTOCOL AND THE SACRAL PLEXUS!

REPRODUCTIVE SYSTEM: LLLT AND FEMALE INFERTILITY

Background and Aims: The rapidly graying population in Japan is being compounded by the rapidly-dropping birth rate. The latter is mostly due to the later ages at which women are giving birth as the marriage age has also been increasing. Giving birth at a later stage is associated with problems for both mother and child, but for older would-be mothers the greatest problem is infertility, sometimes severe. The present article will show how the application of low level laser therapy (LLLT) is a potentially effective treatment for severe infertility. **Subjects and Methods:** Seventy-four females (average age 39.28 yr) with severe infertility in whom assisted reproductive technology (ART) had been unsuccessful (average of 9.13 yr) participated in the first part of a study from October 1996 — April 2000. LLLT was applied (830 nm, CW, GaAlAs **60 mW diode LLLT) in Ohshiro's proximal priority technique (average 21.08 sessions)** with or without other ART approaches. Based on successful outcomes, the study was then extended to March 2012, amassing a final total of 701 patients. **Results:** Pregnancy was achieved in the first part of the trial in 16 patients (21.7% of 74) of whom 11 (68%) achieved successful live delivery. In the extended trial, pregnancy was achieved in 156 (22.3% of 701) with 79 live deliveries (50.1%). **Conclusions:** The use of 830 nm LLLT in the proximal priority technique at the parameters used in the present study, on its own or as an adjunct to other techniques, **resulted in successful induction of pregnancy in just over 21% of severely infertile females, with a substantial number of these achieving live births. No adverse events were noted in any patient. LLLT is a pain-free and sideeffect free modality which could give hope to the increasing numbers of older females with infertility in Japan and potentially worldwide. Multinational studies are warranted.**

Laser Ther. 2012 Jul 3; 21(2): 97–103.
doi: 10.5978/islsm.12-OR-05

PMCID: PMC3944482
PMID: 24610987

Personal Overview of the Application of LLLT in Severely Infertile Japanese Females

Toshio Ohshiro, MD PhD

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REPRODUCTIVE SYSTEM: LLLT AND MALE INFERTILITY

In half of the cases, the infertility of the couple is due to the disorder of the male fertility. The leading factors that cause male infertility are urogenital infections, disorders of the immune system, testicular and prostate pathology, as well as endocrine disorders.

Low level laser therapy (LLLT) is a very effective physical therapy method, used in many areas of medicine, including obstetrics and gynaecology, andrology and urology; and it is recommended as an integral part of the complex treatment of infertility.

The literature review showed that LLLT is beneficial in treating male infertility. **Laser can significantly improve the survival, motility and speed of movement of spermatozoa**. Laser therapy of patients with prostatitis and vesiculitis can eliminate infiltrative-exudative changes, improve reproductive and copulatory functions. Local illumination of red (635 nm) and infrared (904 nm) spectra should be combined with intravenous laser blood illumination (ILBI) of red (635 nm) and ultraviolet (UV) (365 nm) spectra.

Biomedicine (Taipei). 2018 Jun; 8(2): 7.
Published online 2018 May 28. doi: 10.1051/bmdcn/2018080207

PMCID: PMC5992952
PMID: 29806585

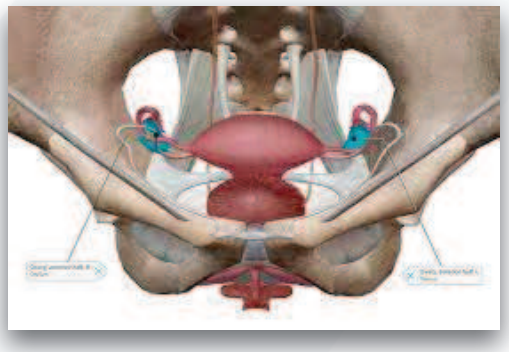
Effectiveness of low level laser therapy for treating male infertility

Sergey Vladimirovich Moskvin[1,*] and Oleg Ivanovich Apolikhin[2]

REPRODUCTIVE SYSTEM: CLINICAL LASER PROTOCOLS

1. ALWAYS CONSIDER "UPSTREAM" HP-AXIS AND CNS FUNCTION
2. ALWAYS CONSIDER HORMONE METABOLISM CLEARANCE, LIVER DETOX
3. ALWAYS CONSIDER DIET ESPECIALLY SUGAR (AROMATASING/17-20 LYASE) AND A.I. CROSS REACTIVITY
4. ALWAYS CONSIDER BUILDING BLOCKS (NEEDS CHOLESTEROL, PREGNENOLONE STEAL)

FOR OVARIES, HAVE PATIENT PLACE HANDS ON PUBIC SYMPHYSIS AND PLACE BEAMS ABOUT 2" LATERAL AND 2" ROSTRAL



ERCHONIA LLLT:

2-10-246-417 (SOLFEGGIO FREQUENCIES INFERTILITY/ SACRAL CHAKRA)

2 MINUTES DIRECTLY OVER EACH GONAD
2 MINUTES SACRAL PLEXUS
2 MINUTES VAGAL NERVE/SCM
2-5 MINUTES PREFRONTAL CORTEX

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Mol Cell Endocrinol

2021 Jul 1;531:111318. doi: 10.1016/j.mce.2021.111318. Epub 2021 May 12.

Local application of low level laser therapy in mice ameliorates ovarian damage induced by cyclophosphamide

Gonzalo Oubiña [1],

Abstract

The aim of the present study is to assess whether low level laser therapy (LLLT) can protect ovaries from chemotherapy-induced gonadotoxicity using a mice model of premature ovarian failure induced by cyclophosphamide (CTX). LLLT (64 J/cm2) increased the number of antral follicles whilst decreasing the number of atretic follicles compared to CTX alone. LLLT increased the number of primordial follicles compared with those in the CTX group but they did not differ from those in the control group. LLLT treatment increased the number of AMH-positive follicles compared to CTX alone. LLLT application increased ovarian weight, serum progesterone concentration and P450scc protein levels compared to CTX alone. LLLT reduced the apoptosis in antral follicles and the BAX/BCL-2 ratio compared to CTX alone. Vascular morphology, analysed by CD31 and α-SMA immunostaining, was restored in LLLT-treated ovaries compared to CTX alone. **In conclusion, application of LLLT prior to CTX might serve as a promising and novel protocol to preserve female fertility in cancer survivors.**

REPRODUCTIVE SYSTEM

Abstract

It is known that LLLT has beneficial effects on several pathological conditions including wound healing, pain and inflammation. LLLT modulates biological processes, including cell proliferation, apoptosis and angiogenesis. In the present study, we examined the effect of local **application of LLLT on follicular dynamics, ovarian reserve, AMH expression, progesterone levels, apoptosis, angiogenesis, and reproductive outcome** in adult mice. LLLT (200 J/cm2) increased the percentage of primary and preantral follicles, whilst decreasing the percentage of corpora lutea compared to control ovaries. **LLLT-treated ovaries did not exhibit any changes regarding the number of primordial follicles. We observed a higher percentage of AMH-positive follicles (in early stages of development) in LLLT-treated ovaries compared to control ovaries. LLLT reduced the P_4 concentration and the apoptosis in early antral follicles compared to control ones. LLLT caused a reduction in the endothelial cell area and an increase in the periendothelial cell area in the ovary. Additionally, LLLT was able to improve oocyte quality.** Our findings suggest that local application of LLLT modulates follicular dynamics by regulating apoptosis and the vascular stability in mouse ovary. In conclusion, **these data indicate that LLLT might become a novel and useful tool in the treatment of several pathologies, including female reproductive disorders.**

Prog Biophys Mol Biol

2019 Aug;145:10-18. doi: 10.1016/j.pbiomolbio.2018.11.010. Epub 2018 Nov 27.

Low level laser therapy (LLLT) modulates ovarian function in mature female mice

Gonzalo Oubiña [1],

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REPRODUCTIVE HORMONES

DO NOT FORGET TO ADDRESS REPRODUCTIVE HORMONES. HYPOGONADISM IS COMMON IN POST-CONCUSSION SYNDROME BUT SEX HORMONES ARE ALSO NEEDED TO MAINTAIN AND PROTECT THE BRAIN IN AGING!

ALZHEIMER'S DISEASE

Women are at greater risk to suffer from Alzheimer's - here's why, claims study

The researchers discovered that the brains of women who had died from the condition had substantially higher amounts of complement C3, an inflammatory immune protein, than those of men who had died from the sickness.

A hint to the molecular aetiology of Alzheimer's disease has been discovered by researchers at MIT and Scripps Research; this clue may also explain why women are more susceptible to the condition. In **the study published on December 14, 2022, in Science Advances,** the researchers discovered that the **brains of women who had died from the condition had substantially higher amounts of complement C3, an inflammatory immune protein, than those of men who had died from the sickness**. Additionally, **they demonstrated how regularly occurring estrogen, whose synthesis declines during menopause, prevents the formation of this form of complement C3.**

LOW PROGESTERONE SIGNS

5 SIGNS OF LOW PROGESTERONE IN FEMALES:

1.TROUBLE SLEEPING

2.ANXIETY

3.DEPRESSION

4.BLOATING

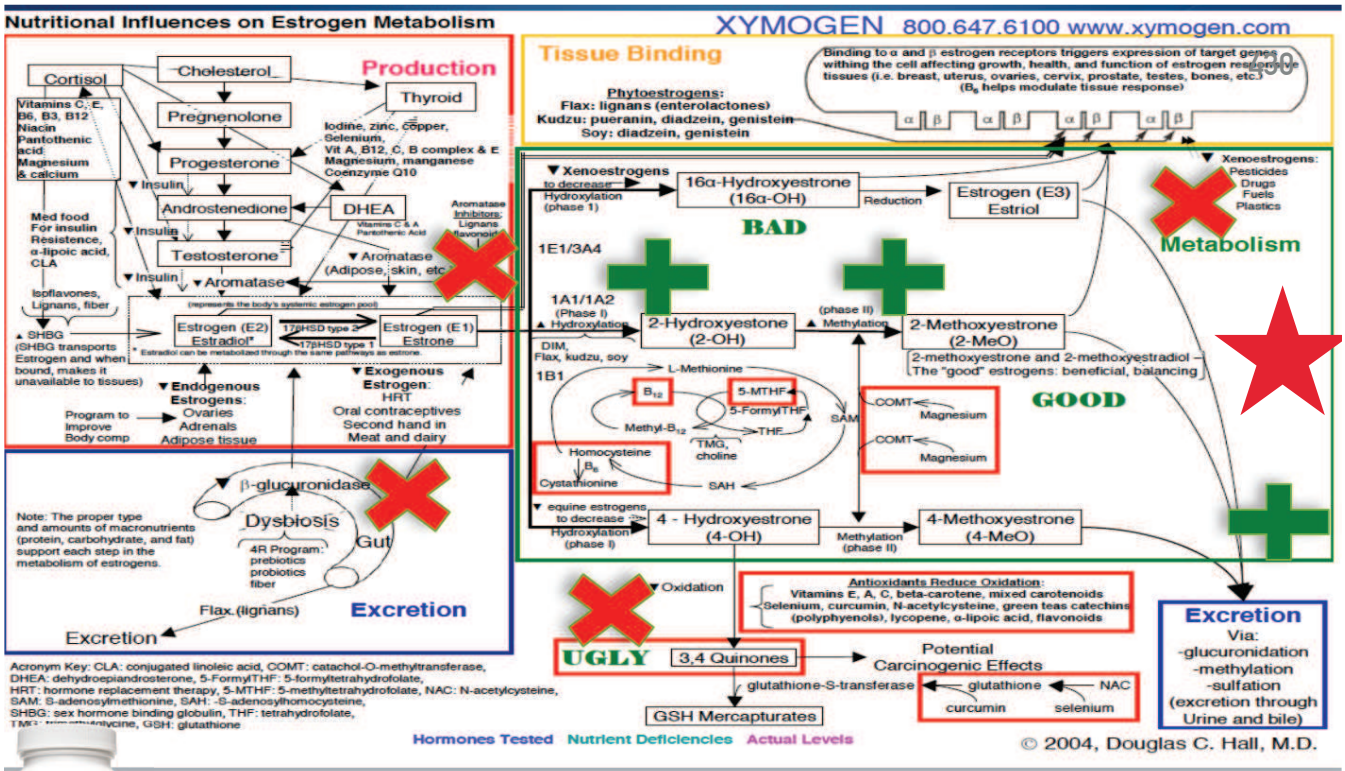
5.HEAVY PERIODS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MISCARRIAGE DIFFERENTIALS

- LOW PROGESTERONE
- DYSGLYCEMIA
- ANTI-PHOSPHOLIPID ANTIBODIES -> LUPUS AI
- LOW THYROID/AUTOIMMUNE THYROID
- SMOKING, ALCOHOL OR DRUG USE
- CONGENITAL DEFECT/CHROMOSOMAL DISORDER
- CERVIX INSTABILITY/UTERINE FIBROIDS
- INFECTIOUS DISEASE (IE. CMV OR STD'S)



Femquil® delivers biologically active folate and other key methylation vitamins in combination with a targeted blend of ingredients to encourage hormone balance, help modify xenoestrogen activity, and restore tranquility. Vitex and black cohosh provide traditional hormone- balancing support; DIM, calcium D-glucarate, and 8-prenylnaringenin (from hops extract) promote estrogen detoxification; and rosemary, resveratrol, grape seed extract, and green tea extract provide antioxidant activity. 1-3 daily as directed

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited

ERECTILE DYSFUNCTION PROTOCOL

ERCHONIA eCommunity Education

- THE CANARY IN THE COAL MINE - BLOOD FLOW AND 02 DELIVERY COMPROMISE (CV DISEASE, DIABETES, ANEMIA ETC)
- CHOLESTEROL IS NEEDED!
- PROCREATION VS SURVIVAL
- OTHER LOW T FACTORS (AROMATIZATION?)
- ARGININE VS VINPOCETINE NON-ADRENERGIC NON-CHOLINERGIC FIBERS (NANC) USE NITRIC OXIDE (NO) AS PRIMARY NEUROTRANSMITTER. FM TESTOSTERONE SUPPORT
- ERECTION IS PARASYMPATHETIC. EJACULATION IS

CIRCULATION SETTING (DAN MURPHY)
20-42-43-53 ABOVE PUBIC BONE
SHOCKWAVE THERAPY. IE THE PHOENIX

THE ED LASER ALGORITHM LASER 2 MINUTES EACH REGION
1-10-1-10 SETTING
1. LOCAL TISSUE EXPOSURE
2. SACRAL PLEXUS/SPINAL CORD (PUDENDAL NERVE S2-3-4)
3. RIGHT VAGAL NERVE STIMULATION
4. THE "RIGHT" MIND SET - PREFRONTAL CORTEX/INHIBITION RIGHT ORBITOFRONTAL/ANTERIOR INSULAR (5 MIN). STACK WITH LEFT VISUAL FIELD CO-ACTIVATION! (NOT ACTIVE OR PASSIVE ROM)
5. FIND A NEW SPOUSE

ALZHEIMER'S AND VIAGRA

ERCHONIA eCommunity Education

Viagra associated with reduced risk of Alzheimer's disease

Those who took sildenafil were 69% less likely to develop AD over 6 years than those who did not take the drug.

At a Glance

- People who took the drug sildenafil were less likely to develop
- In cultur owth and redu
- The find nafil as a possibl

COMMON MEDICATIONS THAT CAN CAUSE ED:
NARCOTICS (RX OR RECREATIONAL)
ANTI-DEPRESSANTS
ANTI-HISTAMINES/H2 BLOCKERS
BLOOD PRESSURE MEDICATIONS
PARKINSON'S MEDICATIONS
CANCER/HORMONE MEDICATIONS
NSAIDS
AND MANY MORE!

A team of res nic developed a c gs that might be effective against AD. NIH's National Institute of Aging (NIA) supported the study. Results appeared in Nature Aging on December 6, 2021.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

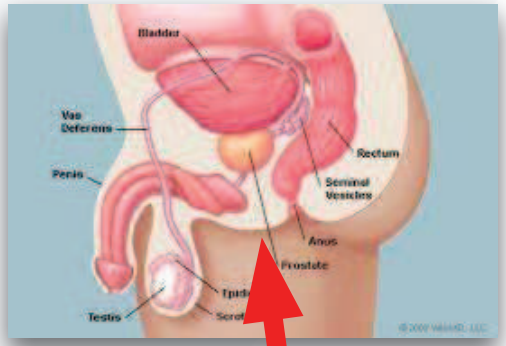
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LLLT AND PROSTATE CANCER

ERCHONIA eCommunity Education

in-LLLT combination led to a significant increase in the MDA t as the product of membrane lipid peroxidation. Analyzing the LDH activity under the effect of cisplatin-LLL combined treatment showed a remarkable increase in the enzyme activity. We conclude that applying the cisplatin-LLL combination therapy is promising as an effective anti-cancer treatment. This novel combination has a potential to attenuate adverse side effects of earlier monotherapy strategies.

TREATMENT PARAMETERS:
LOW DOSE ERCHONIA HAND HELD LASER
SETTINGS 9-10-33-100
2 MINUTES TO PERINEUM PRE AND POST
CISPLATIN TREATMENT (6+ HOUR IV DRIP
EVERY 4 WEEKS)



Lasers Med Sci
2022 Mar;37(2):1283-1288. doi: 10.1007/s10103-021-03386-y. Epub 2021 Aug 10.

The effect of cisplatin-low-level laser therapy on cell viability and death of LNCaP prostate cancer cell line

433

Jaber Zafari [1], Fatemeh Javani Jouni [2]

UTI'S/INTERSTITIAL CYSTITIS

ERCHONIA eCommunity Education

Evid Based Complement Alternat Med
2021 Jun 23;2021:9976299. doi: 10.1155/2021/9976299. eCollection 2021.

Pycnogenol® Supplementation Prevents Recurrent Urinary Tract Infections/Inflammation and Interstitial Cystitis

A Ledda [1]

Abstract

This open pilot registry study aimed to evaluate and compare the prophylactic effects of Pycnogenol® or cranberry extract in subjects with previous, **recurrent urinary tract infections (UTI) or interstitial cystitis (IC).** *Methods*. Inclusion criteria were recurrent UTI or IC. **One subject group was supplemented with 150 mg/day Pycnogenol®, another with 400 mg/day cranberry extract, and a group served as a control in a 2-month open follow-up.**

***Conclusions*. This pilot registry suggests that *60 days of Pycnogenol®* supplementation possibly decrease the occurrence of UTIs and IC without side effects and with an efficacy superior to cranberry.**

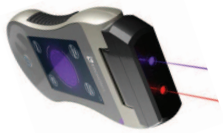
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

UTI'S/INTERSTITIAL CYSTITIS

SUPPLEMENT IDEAS:

- PYCNOGENOL
- D MANNOSE
- ARGENTYN SILVER (INGEST AND LOCAL)
- CRANBERRY CONCENTRATE



LASER APPLICATIONS:
UTI FREQUENCIES (EVRL OR GVL)
24-43-2008-7498
4 minutes over bladder-urethra/opening

UPSTREAM NEUROGENIC CONSIDERATIONS:
1-10-40-60
2 MINUTES NERVI ERIGENTES (PELVIC SPLANCHNIC PLEXUS)
2 MINUTES VAGUS NERVE (SCM)
4 MINUTES MEDIAL PREFRONTAL CORTEX AND PONS (MICTURITION CENTERS)

LLLT AND OVERACTIVE BLADDER

Patients were instructed to apply an LLLT device or a sham device at home three times daily for 12 weeks. The primary outcome was the change in the mean daily number of urge urinary incontinence (UUI) episodes between baseline and 12 weeks. The secondary outcomes were the mean changes in incontinence, voiding, and nocturia episodes from baseline and the likelihood of achieving a > 50% reduction in UUI and incontinence episodes after 12 weeks

Results: **Compared with those in the sham group, the numbers of UUI and urinary incontinence episodes in the LLLT group were significantly decreased at week 12** (UUI, (-1.0 ± 1.7 vs. -0.4 ± 2.5, P = 0.003; urinary

TREATMENT RECOMMENDATIONS:

1. CNS MASTER BRAIN SETTING (1-10-40-60) TO MICTURITION CENTERS (MEDIAL ORBITAL FRONTAL AND PONTINE) AS WELL AS VAGAL NERVE AND SACRAL PLEXUS STIMULATION WHILE DOING PELVIC FLOOR/KEGEL EXERCISES
2. DIRECT BLADDER LOW LEVEL LASER EXPOSURE FOR 2 MINUTES (SETTING 24-43-2008-7498)IF AVAILABLE USE EVRL OR FX 405 AS MANY OAB CONDITIONS ARE ASSOCIATED WITH BACTERIA AND BIOFILMS LIKE INTERSTITIAL CYSTITIS
3. LASER RENTAL FOR DAILY USE UNTIL SYMPTOMS SUBSIDE. IN OFFICE 3X/WEEK FOR 4 WEEK MINIMUM TREATMENT IS SUGGESTED FOR A TREATMENT TRIAL

Efficacy and safety of skin-adhesive low-level light therapy for overactive bladder: a Phase III study

Woo Yeon Hwang [1,2], Yong Beom Kim [3,4], Sei Ra Lee [5], Dong Hoon Suh [1,2], Kidong Kim [1,2], Jae Hong No [1,2]

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

The oral-brain axis: New research uncovers surprising links between the bacteria in your mouth and mental health symptoms

Participants with symptoms of posttraumatic stress disorder and depression exhibited higher levels of certain bacteria, such as *Prevotella histicola*. This bacterium, associated with oral inflammation, was also linked to lower interpersonal quality of life scores

Another key finding was the association between oral microbiota and early life trauma. For instance, **higher levels of *Streptococcus mutans*, a bacterium often linked to dental decay, were found in individuals who reported experiencing childhood emotional neglect.**

- Open access
- Published: **05 October 2024**

Probing the oral-brain connection: oral microbiome patterns in a large community cohort with anxiety, depression, and trauma symptoms, and periodontal outcomes

- Stefanie Malan-Müller, Rebeca Vidal, Esther O'Shea, Eduardo Montero, Elena Figueras-Plaza, Zamira, Javier Sánchez Díaz, Adolfo

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Int J Dent Hyg

2018 May;16(2):e120-e127. doi: 10.1111/idh.12328. Epub 2018 Jan 12.

Clinical, microbiological and cytomorphometric evaluation of low-level laser therapy as an adjunct to periodontal therapy in patients with chronic periodontitis

Results: Evaluation using clinical parameters showed better results in LLLT group. **A statistically significant decrease in the prevalence of bacteria after treatment in LLLT group was observed for the following:** T. forsythensis and T. denticola ($P < .001$), P. gingivalis ($P < .01$), A. actinomycetemcomitans and **P(REVOTELLA). intermedia** ($P < .05$). The values of nuclear area, perimeter and Ferret's diameter were significantly lower in both studied groups after treatment, but statistical significance was higher in LLLT group ($P < .001$) than in the SRP therapy group ($P < .05$).

Lasers Med Sci. 2018 Aug 25;34(1):129–137. doi: 10.1007/s10103-018-2622-6

Effects of Nd:YAG laser irradiation on the growth of *Candida albicans* and *Streptococcus mutans*: in vitro study

Kinga Grzech-Leśniak [1]

Macroscopic and microscopic analyses revealed a reduction in cell number and a significant decrease of cell metabolism after laser application for both *C. albicans* and *S. mutans*.

Research

Volume 12, Issue 3, May–June 2022, Pages 396-400

Effect of low-level diode laser on *streptococcus mutans* and *lactobacillus acidophilus* growth: An invitro study

Author links open overlay panel

Maryam Robati

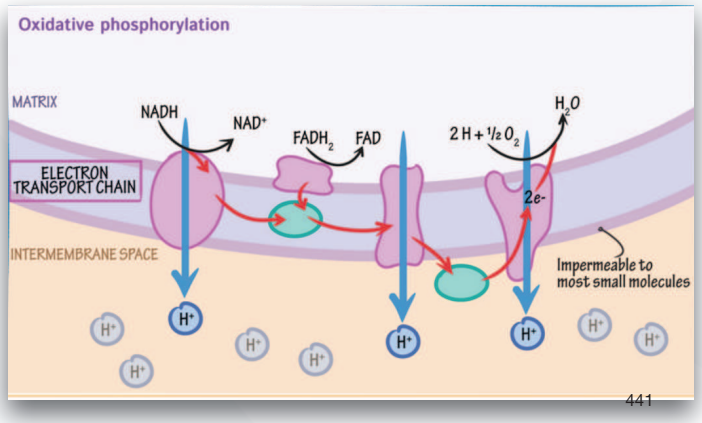
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LLLT AND IMMUNE PROPERTIES

QUESTION: HOW DOES LOW LEVEL LASER KNOW HOW TO TARGET CERTAIN CELLS OVER OTHERS IN DISEASED STATES?

ANSWER: TECHNICALLY IT DOESN'T SPECIFICALLY TARGET UNHEALTHY CELLS PER SE. A BIG FACTOR IN UNHEALTHY CELLS IS THE PH OF THE CELL. LLLT WILL "TARGET" UNHEALTHY CELLS IN AN ACIDIC LOW PH "REDOX" STATE BY INDUCING THE ELECTRON TRANSPORT CHAIN AND DONATING HYDROGEN MOLECULES TO RESTORE THE DISEASED CELL TO A HEALTHY "NORMAL" PH. IN HEALTHY CELLS, LLLT WILL PROMOTE OTHER FACTORS TO FURTHER OPTIMIZE THE HEALTH OF THE CELL (EX. ATP SYNTHESIS, MITOCHONDRIAL BIOGENESIS ETC)



CANCER CELLS DO NOT EXHIBIT THE SAME LIGHT PROPERTIES AS HEALTHY CELLS (BIOPHOTONICS). THIS MAY BE ANOTHER MECHANISM HOW COHERENT LLLT MAY EFFECTIVELY TARGETS DISEASED CELLS

CLINICAL PEARL WHEN TREATING SEVERE CHRONIC PATHOGEN AND CO-MORBIDITY PATIENTS:

- START LOW, SLOW AND DISTAL (ABSCOPAL EFFECT) TRY LIMB EXPOSURE FIRST
- GI AND RESPIRATORY SYSTEM SECOND
- VAGAL STIM THIRD
- TRANSCRANIAL LAST
- 2 MINUTES EACH THEN WORK UP TO FULL TRANSCRANIAL MASTER PROTOCOLS
- CONSIDER RED LASER ONLY
- DOSE DOWN TIME ON KIDS 1 MINUTE PER YEAR WITH RED, 30 SECONDS GVL/EVRL

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

IMMUNOLOGY AND FUNCTIONAL NEUROLOGY

CLINICAL PEARL:

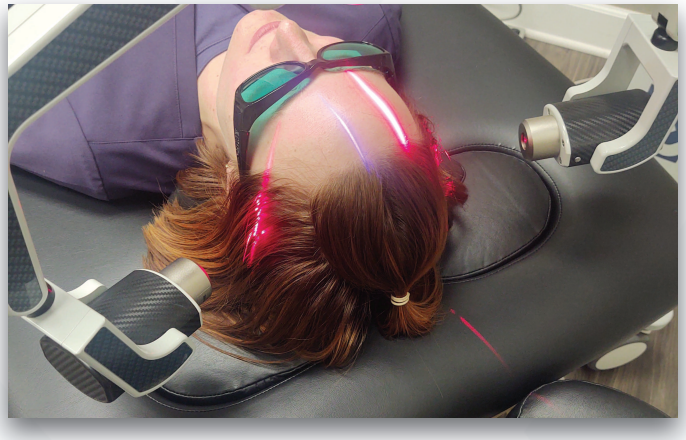
IF YOU DON'T HAVE A HEALTHY BRAIN, BRAINSTEM AND BRAIN-GUT AXIS (ANS REGULATION), IMMUNOLOGICAL FUNCTION AND GUT HEALTH WILL BE COMPROMISED. USE YOUR ERCHONIA LASER TO IMPROVE FUNCTIONAL NEUROLOGY WITH BRAIN, VAGUS, GUT AND OTHER REGIONAL APPLICATIONS TO OPTIMIZE RESULTS WITH IMMUNOLOGY AND OTHER FUNCTIONAL MEDICINE THERAPIES

LEFT BRAIN THINK TH1 "ATTACK", RIGHT BRAIN THINK TH2 AND "RESOLVE"

THE PRIMARY IMMUNE SETTING POSITION 1 FX

-IMMUNE: 20-73-465-728 (HANDHELDS)

-20-10-73-40-465-728 (FX 635 OR 405)



5 MINUTES TRANSCRANIAL THEN SWITCH 5 MINUTES TO A VERTICAL "GUT SHOT" (NEXT SLIDE)

HANDHELDS: 5 MINUTES TRANSCRANIAL
2 MINUTES VAGUS NERVE/SCM/THROAT
2 MINUTES LUNG FIELD
2 MINUTES GUT
USE THE HANDHELD STAND OR EVEN HAVE PATIENT WAND THEMSELVES

RENTAL???

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE PRIMARY IMMUNE PROTOCOL
2ND PART: "GUT SHOT"

EVRL FOR ORAL PATHOGENS

EVRL 405 NM LASER

"BACTERIA SETTING" 20-664-787-1560
4 MINUTES
1 MINUTE NASAL, 1 MINUTE THROAT
1 MINUTE MAXILLARY GUM LINE
1 MINUTE MANDIBULAR GUM LINE

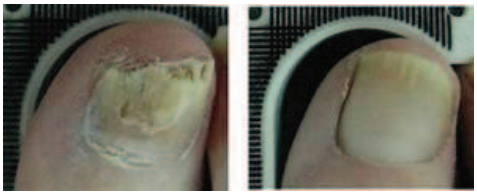
OTHER CONSIDERATIONS:
REGULAR FLOSSING, BRUSHING AND DENTAL HYGIENE
LOW SUGAR/CARB DIET
"OIL PULLING" WITH COCONUT OIL
ARGENTYN SILVER

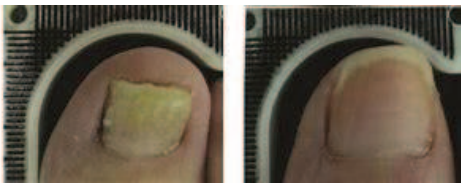
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

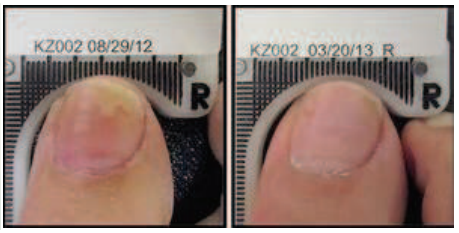
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FINALLY A SOLUTION FOR TOENAIL FUNGUS (ONYCHOMYCOSIS)

- 405 NM LUNULA WAS THE FIRST AND ONLY NON-THERMAL COLD LASER TO RECEIVE FDA 510(K) MARKET CLEARANCE FOR ONYCHOMYCOSIS
- 10% OF THE POPULATION SUFFERS FROM IT
- "SET IT AND FORGET IT". 1 TREATMENT PER WEEK FOR 4 WEEKS. 12 MINUTES
- 90% PATIENT RESPONSE, PAINLESS, NO DOWN TIME AND NO SIDE-EFFECTS







IMMUNE FUNCTION AND PATHOGENS: COMMON SETTINGS

ERCHONIA eCommunity Education

- STREPTOCOCCUS: 6 MINUTES. 465-662-785-2003. 2 MINUTES THROAT, 4 MINUTES FRONTAL/BASAL GANGLIA (2 MINUTES EACH SIDE)
- STAPH/MARCONS: 5 MINUTES. 424-453-634-2600. 2 MINUTES EACH NOSTRIL, 1 MINUTE THROAT
- HERPES SIMPLEX 1 VIRUS: 322-468-647-664
- HERPES ZOSTER/CHICKEN POX: 20-304-787-1865
- LYME: 306-312-525-534
- CANDIDA: 21-762-880-1146
- EBV: 27-73-274-728
- CMV: 126-629-2145-8856
- COLD/FLU COMMON: 20-125-10,000-13,011
- HIV: 73-238-683-2420
- DEFAULT VIRUS: 20-73-625-787
- DEFAULT BACTERIA: 20-664-787-1560
- *IMMUNE: 20-73-465-728*

FX 405 SCANNING 6 MIN TRANSCRANIAL (3 MIN SAGITTAL 3 MIN CORONAL)
EVRL 6 MIN (3 MIN EACH HEMISPHERE)
FX 635 OR XRL8 BASE BRAIN PROTOCOLS
ALWAYS DO VAGAL STIM PROTOCOL WITH ANY OF THE LASERS!

ACUTE PHASE ACTIVE CARE 3X/WEEK (OR DAILY IF POSSIBLE-RENTAL FOR MINIMUM 6 VISITS. MAY TAKE MONTHS FOR CHRONIC INFECTIOUS DISEASE)
ADD TO PREVENTATIVE/MAINTENANCE CARE ON ANY FOLLOW UP VISITS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

IMMUNE FUNCTION AND PATHOGENS

ERCHONIA eCommunity Education

- Corpus ID: 74396187

HERPES VIRUS INFECTION LOW LEVEL LASER THERAPY (LLLT)- PHOTOBIOSTIMULATION APPLIED AS MONO THERAPY IN TREATMENT OF HUMAN PATHOGEN HERPES

TREATMENT PLANS: RED ERCHONIA LLLT SETTING 322-468-647-664
HSV OR 20-304-787-1865 ZOSTER
HERPES SIMPLEX 1 (LABIAL) 2 MIN DAILY FOR 4-5 DAYS
HERPES SIMPLEX 2 (GENITAL) 4 MIN DAILY FOR 8 DAYS
HERPES ZOSTER (SHINGLES) 5 MINUTES DAILY FOR 10 DAYS
DON'T FORGET DRG AND CMT TO SEGMENT FOR ZOSTER!

treated n=20 cases of herpes labialis with *LLLT HeNe 632,8 nm (2J/cm2 for 3-5 days).* In pharmacological group n=20 cases are treated with pharmacological products, virostatic effect (acyclovir), treated at least 8 days. There were treated n=24 cases of genital herpes with LLLT combination of HeNe and IR therapy (6J/cm2 on skin and 3J/cm2 on mucosa, 4x a week/2 weeks). In clinical research we treated n=30 cases of herpes zoster with LLLT:IR (8-16 J/cm2, 5x week/2 weeks). Results Low level laser therapy (HeNe, IR) application proves with all virus diseases less relapse and very good and with 100% curable healing results as monotherapy (virocide effect). Pharmacological products (acyclovir,zovirax,virulex,ehinacin etc.)available for treatment of herpes simplex type HSV 1 , HSV 2 and herpes zoster have only virostatic effect

MULTIPLE SCLEROSIS & EBV

ERCHONIA eCommunity Education

EBV Maybe Treatable with Low Light Laser

posted: Feb. 07, 2019.

Journal of Investigative Dermatology

Low-intensity laser therapy is an effective treatment for recurrent herpes simplex infection. Results from a randomized double-blind placebo-controlled study.

Schindl

Author in

Depart

Abstract

TREATMENT PLAN: ACUTE EBV 10 TREATMENTS TOTAL (DAILY)
12 MINUTES TOTAL ERCHONIA SETTING 28-73-274-728
6 MINUTES TRANSCRANIAL
6 MINUTES RESPIRATOR SYSTEM AND GI
CONTINUE TREATMENT APPLICATIONS ALL FOLLOW-UP VISITS
MONITOR BRAIN AUTOIMMUNE WITH VIBRANT NEURAL ZOOMER PLUS
OTHER CONSIDERATIONS FOR ANTI-VIRAL SUPPORT:
VIRAGRAPHIS, LAURICIDIN, BIOCIDIN, ARGENTYN SILVER

Recurr ...een **introdu** ...d in pain **therapy** ...the **influen** ...) in 50 **patient** **random** ...ions for **2 wk, w** ...tment, **patients were asked to return to the Department of Dermatology, University of Vienna Medical School at the time of recurrence. All except two patients completed the study and were monitored for 52 wk. The median recurrence-free interval in the laser-treated group was 37.5 wk (range: 2-52 wk) and in the placebo group 3 wk (range: 1-20 wk). This difference was found to be statistically significant ($p < 0.0001$; Wilcoxon's Rank Sum Test). In conclusion, we demonstrated that a total of 10 irradiations with low-intensity laser therapy significantly lowers the incidence of local recurrence of herpes simplex infection. Since this athermic phototherapeutic modality represents a safe, noninvasive treatment, it might be considered as an alternative to established therapeutic regimens in this indication.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MULTIPLE SCLEROSIS SUMMARY

ERCHONIA eCommunity Education

- ASSESS AND REPAIR LEAKY BARRIERS WITH WHEAT ZOOMER AND NEURAL ZOOMER PLUS, PATHOGEN AI?

LEAKY GUT AND LEAKY BRAIN PROTOCOL

-FOOD ELIMINATION: GLUTEN, AQUAPORINS AND DAIRY FREE

WAHL'S PROTOCOL, KETOGENIC DIET

- VIRAGRAPHIS, IMMUNE ESSENTIALS XYMOGEN

- LAURICIDIN, SILVER HYDROSOL

-PATHOGENS: YOU MUST ADDRESS EPSTEIN-BARREARLY AND OFTEN DURING "MONO" AND REGULAR TRANSCRANIAL TREATMENTS FROM ADOLESCENCE ON

- MONITOR AUTOIMMUNE PROFILE WITH VIBRANT'S NEURAL ZOOMER PLUS

NEW ERCHONIA LLLT MASTER PROTOCOL FOR M.S.:

BEST DEVICE IS FX 405 SCANNING TRANSCRANIAL

1-28 PFC

40-73 APICAL
40-274 APICAL

10-728 BRAINSTEM

(HYBRID OF EBV AND MASTER BRAIN SETTINGS)

10 MINUTES TOTAL
8 MIN GLOBAL TCT (4+4)
4 MIN BRAIN/GUT VAGAL

EBV FOR HAND HELDS: 28-73-274-728

SILVER HYDROSOL

ERCHONIA eCommunity Education

Account set up: Call customer care line at 888-328-8840 or email our customer care team at orders@n-icorp.com

MENTION THE DISCOUNT CODE
BERRYAG

Or go to the link:
https://sovereignsilver.com/discount/DRB20

DRB20

ARGENTYN 23®
SAFE.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
Without permission of Trevor Berry, DC, DACNB is prohibited.

CARDIOVASCULAR DISEASE AND LLLT

Research Article ISSN 2771-9057

Recent Advances in Clinical Trials

Exploring A Novel Approach Using Red and Violet Laser Therapy For Carotid Artery Stenosis - A Case Series

Andrzej Eberhardt[1] and Travis Sammons[2*]

[1]General surgery and Vascular, Vascular Surgery and Phlebology in Femmed Clinic, Warsaw, Poland.

[2]Erchonia Corporation, Melbourne, FL, USA.

*Correspondence:
Travis M. Sammons, Erchonia Corporation, 112 Southchase Bvld., Fountain Inn, SC 29644, Tel: 888-242-0571, Fax: 321.473.1608.

Received: 20 Mar 2024; Accepted: 26 Apr 2024; Published: 03 May 2024

Citation: Eberhardt A, Sammons T. Exploring A Novel Approach Using Red and Violet Laser Therapy For Carotid Artery Stenosis - A Case Series. Recent Adv Clin Trials. 2024; 4(2); 1-6.

Keywords
Carotid artery stenosis, Violet laser therapy, Ischemic stroke, Novel therapeutic approaches.

Introduction

Carotid artery stenosis (CS), characterized by atherosclerotic plaque accumulation within the carotid arteries, is a prominent risk factor for ischemic stroke [1]. The World Health Organization recognizes ischemic stroke as the second leading cause of mortality worldwide [2]. Complications arising from unstable atherosclerotic plaque, primarily through embolization (blood clot formation and travel), are responsible for approximately 75-80% of ischemic strokes [3]. However, reduced blood flow (hypoperfusion) due to severe stenosis also plays a role, potentially contributing to an estimated 20-25% of cases. This established link between CS and stroke necessitates the exploration of novel therapeutic approaches.

Current management strategies for CS primarily focus on lifestyle modifications, such as a healthy diet, exercise, and smoking cessation, which aim to improve overall cardiovascular health and potentially slow atherosclerotic plaque progression [4]. Antiplatelet medications are a mainstay of medical therapy [5], while these drugs cannot reverse arterial narrowing, they play a crucial role in reducing platelet activity and consequently the risk of stroke. However, long-term use of antiplatelet drugs comes with its own set of side effects, including stomach ulcers, kidney problems, high blood pressure, and tinnitus. For symptomatic patients with severe stenosis (70% to 99%), carotid endarterectomy, a surgical procedure to remove plaque, is often recommended [6].

Recent studies investigating the effects of low-level laser therapy (LLLT) on vascular health have shown promising results, and may provide a non-invasive, and non- pharmacological therapeutic option for CS. LLLT appears to influence factors like vascular endothelial growth factor (VEGF) and may potentially reduce inflammation within the vessel wall and plaque structure [7,8]. This study aims to contribute to the expanding body of research by evaluating the potential therapeutic effects of LLLT on both plaque size and morphology in patients diagnosed with CS.

Material and Methods

This study enrolled a total of 30 patients (17 male, 13 female) receiving treatment at a vascular surgery Femmed clinic in Warsaw, Poland. All participants were on established pharmacological therapy regimens, adhering to the 2023 Clinical Practice Guidelines on the Management of Atherosclerotic Carotid and Vertebral Artery Disease set forth by the European Society for Vascular Surgery. Throughout the 12-week study period, each patient underwent bi-weekly ultrasound examinations to monitor their condition. Both the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria and the Gray-Scale Median (GSM) scale were employed to assess the severity of carotid artery stenosis and atherosclerotic plaque instability, respectively. The GSM value is one of the most widespread methods of studying atherosclerotic plaques and it is important to comprehensively standardize image post-processing. Higher GSM values indicate denser, more calcified plaques, which are generally considered more stable. A lower GSM value suggest softer plaques with a higher lipid or fluid content, potentially indicating a more unstable plaque and a worse prognosis for CS.

All participants received laser therapy using the Erchonia EVRL device, which emits a combination of red (635 nm) and blue-violet (405 nm) lasers, each delivering a power output of 7.5

Conclusions

Our investigation explored the application of **low-level laser therapy with red and violet/blue wavelengths in patients with carotid artery stenosis. The results demonstrated a significant reduction in stenotic changes, along with enhanced stability of atherosclerotic plaque and reduced inflammation. Notably, these positive outcomes were independent of factors such as gender, smoking history, or diabetic status. These findings suggest that low-level laser therapy holds promise as a therapeutic strategy to not only impede atherosclerotic progression but also potentially mitigate its complications, including ischemic stroke.** Further exploration through multicenter trials is crucial to elucidate the underlying mechanisms of action and firmly establish this modality as a novel and efficacious treatment for vascular diseases.

(12 WEEKS OF TREATMENT. BI-WEEKLY 15 MINUTES EVRL)

CARDIOVASCULAR SUPPORT

OPTIMAL XYMOGEN MEDPAX FOR CARDIOVASCULAR SUPPORT:

DIOVASC = DIOSMIN THINK STRONG BLOOD VESSELS, IMPROVED MICROCIRCULATION AND LYMPHATICS, GLUCOSE METABOLISM AND ANTIOXIDANT SUPPORT. MAY ALSO HELP WITH S2 SPIKE AND CAPILLARY STABILITY WHICH CAN LEAD TO END ORGAN FAILURE IN COVID-19

BERGACOR PLUS = TARGETS POOR CARDIOMETABOLIC PROFILE ALONG WITH PERIPHERAL INSULIN RESISTANCE FOR HEALTHY LIPID AND GLUCOSE LEVELS. CONTAINS THE HIGHEST DOSE OF BERGAMOT ON THE MARKET ALONG WITH THE SUPER FRUIT AMLA. WILL ALSO SUPPORT HIGH HS-CRP

NIAVASC = NIACIN FOR HEALTHY LIPIDS AND TUNICA INTIMA-MEDIA

ENDOCALYX SUPPORTS AND MAINTAINS A HEALTHY GLYCOCALYX WHICH IS A LIKE A MICRO-THIN SHIELD PROTECTING THE BLOOD VESSELS INCLUDING CORONARY ARTERIES AND MICROCIRCULATION LIKE CAPILLARY BEDS. IT KEEPS BLOOD VESSELS SLICK, SMOOTH AND PROTECTED WHILE OPTIMIZING BLOOD FLOW, GAS AND NUTRIENT EXCHANGE

* ALWAYS ADDRESS UNDERLYING CO-MORBIDITIES, ELEVATED HOMOCYSTEINE, SAD DIET AND OTHER SOURCES OF INFLAMMATION, SEDENTARY LIFESTYLE ETC.

Duplication, citation or distribution of this material in whole or in part
Without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

S2 SPIKE PROTEIN

Published online 2022 Aug 24. doi: 10.3390/molecules27175405

PMCID: PMC9458005
PMID: 36080170

Degradative Effect of Nattokinase on Spike Protein of SARS-CoV-2

Takashi Tanikawa,

Abstract

The coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged as a pandemic and has inflicted enormous damage on the lives of the people and economy of many countries worldwide. However, therapeutic agents against SARS-CoV-2 remain unclear. SARS-CoV-2 has a spike protein (S protein), and cleavage of the S protein is essential for viral entry. Nattokinase is produced by *Bacillus subtilis* var. *natto* and is beneficial to human health. In this study, we examined the effect of nattokinase on the S protein of SARS-CoV-2. When cell lysates transfected with S protein were incubated with nattokinase, the S protein was degraded in a dose- and time-dependent manner. Immunofluorescence analysis showed that S protein on the cell surface was degraded when nattokinase was added to the culture medium.

Thus, our findings suggest that nattokinase exhibits potential for the inhibition of SARS-CoV-2 infection via S protein degradation.

GERD

Nutrients. 2020 Mar; 12(3): 665.
Published online 2020 Feb 29. doi: 10.3390/nu12030665

A Review of Zinc-L-Carnosine and Its Positive Effects on Oral Mucositis, Taste Disorders, and Gastrointestinal Disorders

Susan Hewlings1,

Evidence supports the safety and efficacy of ZnC for the maintenance, prevention, and treatment of the mucosal lining and other epithelial tissues. The research supports its use for gastric ulcers (approved in Japan) and conditions of the upper GI and suggests other applications, particularly for oral mucositis.

ERCHONIA LLLT:
VAGAL (10HZ) + LINE BEAM LINEAR DOWN ESOPHOGEAL/ EPIGASTRIC REGION
2 MINUTES/ VISIT

FOR AT LEAST 1 WEEK PRIOR TO PPI/H2 BLOCKER WITHDRAWAL CONSUME ZINC L-CARNITINE (XYMOGEN PEPCIX). IF CHEWABLE TABLET, CHEW AND SWALLOW TO TARGET ENTIRE GI LINING. DURING ACTIVE WITHDRAWAL PHASE USE 1 PEPCIX AND 1 HISTDAO PRE-MEAL(S) AND 1 AM XYMOZYME HCL

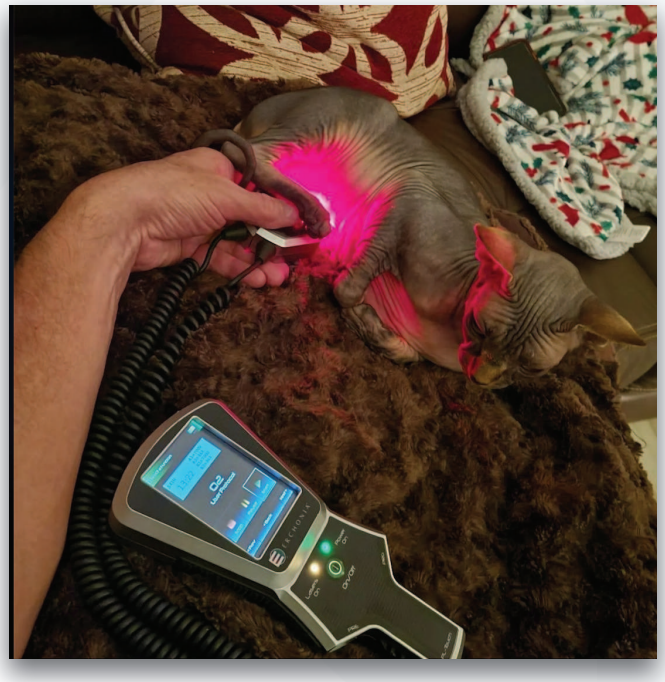
VIBRANT LAB CONSIDERATIONS:
FOOD ALLERGY
FOOD SENSITIVITY
LECTIN ZOOMER
FOOD ZOOMERS

OTHER CLINICAL AND LIFESTYLE CONSIDERATIONS:
SLOW AND RELAXED EATING/CHEW FOOD
HOME VAGAL STIM
AM APPLE CIDER VINEGAR 2 TBSP (DO CHALLENGE FIRST)
BEFORE BED ALOE JUICE 2 TBSP AND ANY TIME OF PAIN POST MEAL
CHIROPRACTIC/MSK REGIONAL T SPINE/RIB/PEC AND GI RELEASE

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HAIR PROTOCOL



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

HAIR PROTOCOL

(9)-25-100-727

NEW FX 9-25, 25-100,100-727

(HAND HELDS MAY BE USED BUT RECOMMEND WANDING THE LINE BEAM FOR BETTER SURFACE COVERAGE)

10 MINUTES
2X/WEEK FOR 6 WEEKS
BASE MINIMUM TRIAL
CONSIDER YEAR LONG PROGRAM (SEE SLIDES AHEAD

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HAIR PROTOCOL

Cutis. 2019 Jul;104(1):17-24.

Nonsurgical hair restoration treatment.

Nazarian RS1, Farberg AS1, Hashim PW1, Goldenberg G2.

Author information Abstract

Patterned hair loss is common and can negatively impact quality of life. Patients often seek nonsurgical treatment options as a first-line measure to avoid undue risks and expense associated with surgery. This article discusses these noninvasive treatment options, with a focus on minoxidil, finasteride, dutasteride, spironolactone,

low-level laser therapy (LLLT), platelet-rich plasma (PRP), microneedling, and oral supplements.

Dermatol Ther. 2019 Sep;32(5):e13059. doi: 10.1111/dth.13059. Epub 2019 Aug 26.

Physiopathology and current treatments of androgenetic alopecia: Going beyond androgens and anti-androgens.

Katzer T1, Leite Junior A2, Beck R3, da Silva C1.

Author information

Androgenetic alopecia (AGA) is the most diagnosed hair loss dysfunction. Its physiopathology comprises a genetic predisposition affording an exacerbated response of the hair follicles cells to androgens aggravated by scalp inflammation and extrinsic factors. This paper presents a review of the mechanisms and extrinsic factors involved in the AGA physiopathology as well as its conventional and emerging treatments. The research focused on reports regarding AGA physiopathology and treatments published between January 2001 and July 2019 in medical and related journals. The most used medical treatments for AGA-*minoxidil and finasteride-present non satisfactory results in some cases*. **Currently, the low-level laser therapy is recognized as a safe and effective treatment for AGA. Some minimally invasive techniques-mesotherapy, microneedling, carboxytherapy, and platelet-rich plasma-are also used to stimulate hair growth.** Pharmaceutical substances with mechanisms differing from the anti-androgen activity are under current investigation and many of them have botanical origins; however, formulations with higher performance are required, and the hair follicles ability of being a drug and nanoparticle reservoir has been researched. **The association of different strategies, that is, substances with synergic mechanisms and the use of advantageous technologies associated with lifestyle changes could improve the treatment outcomes.**

HAIR PROTOCOL

STEP 1.
Refer to patient calendar.
Beginning the following day after procedure, inject .25mL Thymosin Beta 4 daily for 9 consecutive days.
Stop the injections for 10 days.
Resume injections for an additional 10 days for a total of 20 days.

STEP 2.
Apply topical foam GHK for 60 days – 2 pumps nightly except on derma roller days
Apply topical spray of ZN-Thymulin for 60 days - 2 sprays nightly except on derma roller days.

STEP 3.
Begin derma roller weekly treatments (2x per day) exactly two weeks following initial procedure.

Instructions for derma roller. Firmly roll .5mm of the device over the treatment areas and immediately spray the PTD-DBM and Valproic Acid products onto the scalp – once in the morning and once in the evening. This process should be repeated on days 14, 21 and 28.

IMPORTANT: Do not use the ZN-Thymulin and GHK on these derma roller days.

Aging Pathobiol Ther. 2020 Mar 27; 2(1): 58–61.
doi: 10.31491/apt.2020.03.014

The potential of GHK as an anti-aging peptide

Yan Dou,a Amanda Lee,a Lida Zhu,a John Morton,a and Warren Ladigesa,*

XYMOGEN NUTRITIONS SUPPLEMENTS:
(SYSTEMIC CONDITIONS HAVE BEEN RULED OUT LIKE THYROID DYSFUNCTION, ANEMIA PATTERNS, AUTOIMMUNITY ETC)

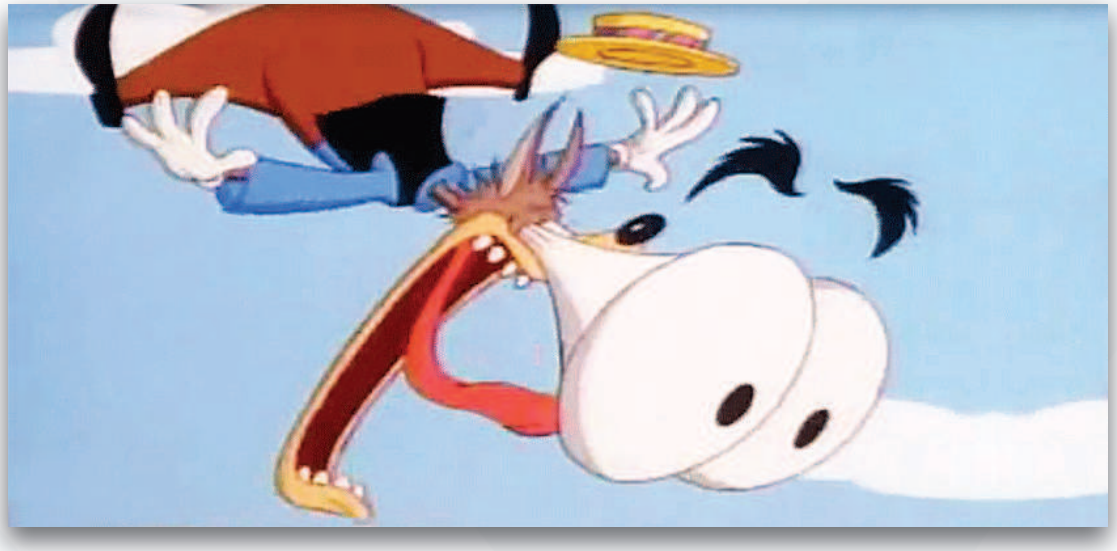
- **CONSIDER 6 DAY DETOX KIT AT ONSET**
- **DIM PRODUCTS (FEMQUIL, HORMONE PROTECT OR DIMENSION 3)**
- **REGENEMAX PLUS (1 DAILY AS SUGGESTED)**

REGENEMAX PLUS CONTAINS ch-OSA (choline-stabilized orthosilicic acid) to nourish the body's "beauty proteins" collagen, elastin and keratin by facilitating collagen generating cell enzymes. It also contains BIOTIN (watch anti-seizure meds like carbamazepine, phenobarbital, phenytoin and primidone)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RETINAL/VISUAL APPLICATIONS



WARNING: DO NOT USE HIGHER POWERED DEVICES FOR THESE APPLICATIONS!

RETINAL/VISUAL APPLICATIONS

*** WARNING THIS IS AN OFF LABEL APPLICATION. LASER CLASSIFICATION IS BASED ON RETINAL DAMAGE CAPACITY. DO NOT USE CLASS 3+, CLASS 4 OR HIGHER DEVICES**

RECOMMENDATION: HAND HELD RED BEAM ADVANCED OPTIC SETTING:

10-40-10-40 (UTILIZE VEP 40 HZ AMYLOID CLEARING WITH VAGAL/PERFUSION FREQUENCIES)

APPLICATIONS (PICK ONE OF THESE 3):

- **2 MINUTES SIDE APPLICATION TEMPORAL REGION**
- **30 SECONDS EYES CLOSED "WISPING" OVER EYELID**
- **30 SECONDS EYES OPEN "WISPING" DIRECTLY INTO PUPIL EYES OPEN**
- **5 MINUTES OCCIPITAL LOBE, 2 MINUTES SCM/VAGUS**

TREATMENTS RANGE FROM 2-4 VISITS ACUTE PHASE/ INJURY/POST-OP TO 12 VISIT TRIAL FOR MORE COMPLEX CONDITIONS LIKE MACULAR DEGENERATION

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

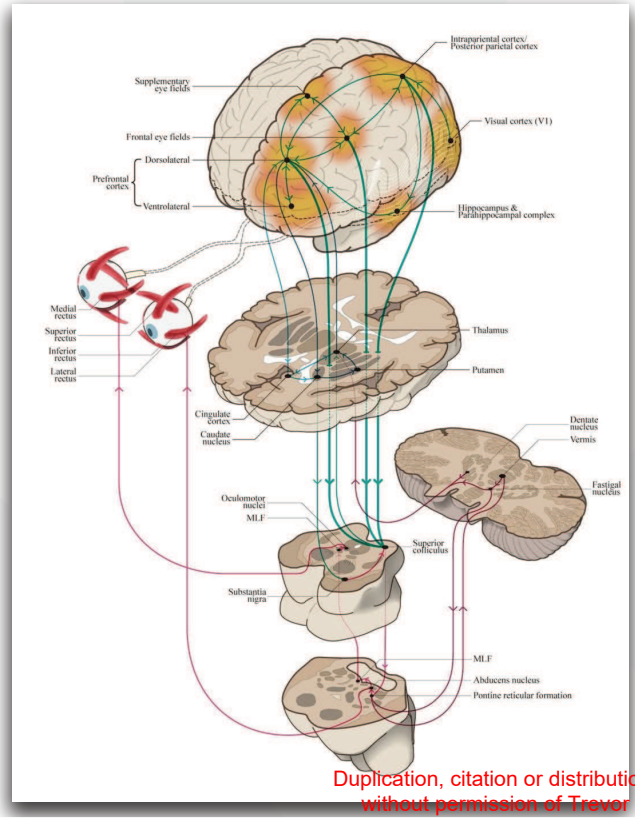
ERCHONIA®
Healing Light, Perfected.

ERCHO
Healing Light, Perf

THE FOLLOWING SLIDES ARE FROM SEMINAR STACKS FROM PRIOR YEARS. DR. BERRY MAY REFERENCE THEM WITHIN THE BODY OF HIS CURRENT SEMINAR, BUT ARE ALSO FOR YOUR REFERENCE TO REVIEW. *PLEASE NOTE, SOME PROTOCOLS MAY BE UPDATED SINCE THE UTILIZATION OF THESE SLIDES.

NEUROLOGY OF EYE MOVEMENTS

ERCHONIA eCommunity Education



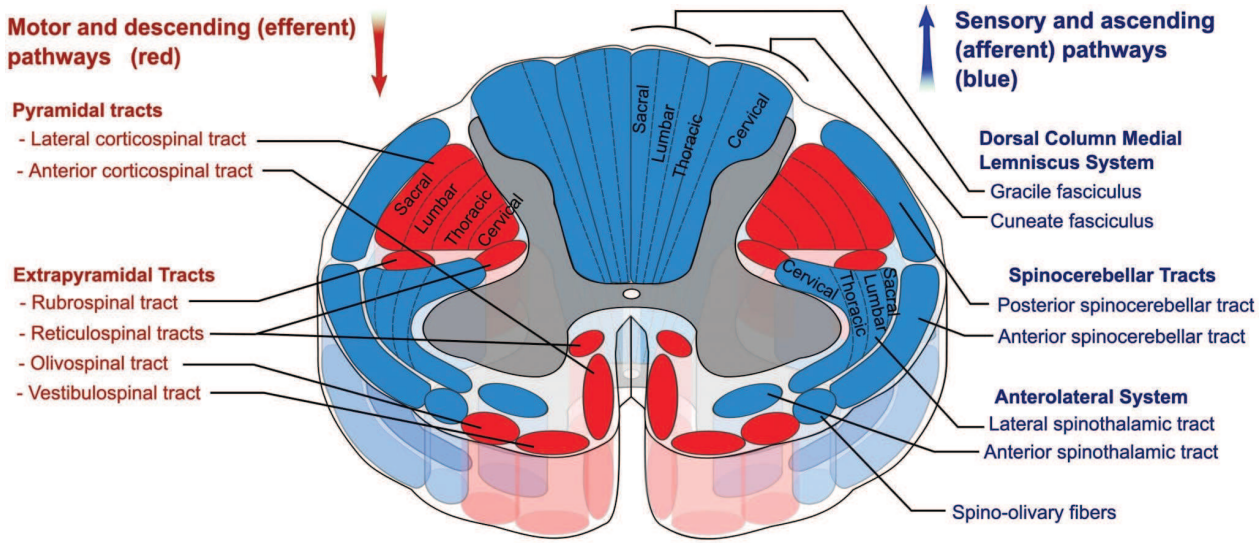
THE EYES ARE A DIAGNOSTIC AND THERAPEUTIC WINDOW FOR A MAJOR PORTION OF THE CENTRAL NERVOUS SYSTEM!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

NEUROLOGY OF EYE MOVEMENTS

Eye Movement Pattern	Primary Brain Region Activated	Clinical Relevance
Horizontal Saccades (L-R)	Frontal Eye Fields (Frontal Lobe), Parietal Lobe	Enhances cortical integration, improves attention and processing speed
Vertical Saccades (Up-Down)	Superior Colliculus, Frontal Eye Fields	Improves vertical gaze control, useful in brainstem rehab
Pursuits (Following moving target)	Parieto-Occipital Junction, Cerebellum	Enhances smooth pursuit pathways, useful for reading and tracking
Convergence (Focusing on near object)	Midbrain, Brainstem (Oculomotor Nucleus)	Stimulates accommodation and convergence reflexes, supports focus
Divergence (Focusing on far object)	Midbrain, Brainstem (Abducens Nucleus)	Supports gaze stability and distance vision control
Diagonal Saccades	Frontal Eye Fields, Parietal Cortex	Challenges complex gaze control networks, aids spatial awareness
Circular Eye Movements	Cerebellum, Brainstem Integrative Centers	Improves coordination and vestibulo-ocular integration
Near-Far Switching	Occipital Lobe, Frontal Eye Fields	Stimulates depth perception and dynamic focusing ability
Left Gaze Holding	Right Frontal Eye Field, Right Parietal Cortex	Facilitates right hemisphere control of gaze and attention
Right Gaze Holding	Left Frontal Eye Field, Left Parietal Cortex	Facilitates left hemisphere control of gaze and attention
Leftward Saccades	Right Frontal Eye Field, Left Pons (PPRF)	Activates right hemisphere, supports language and logical processing
Rightward Saccades	Left Frontal Eye Field, Right Pons (PPRF)	Activates left hemisphere, supports spatial awareness and emotion
Left Cerebellar Activation (Rightward Smooth Pursuits)	Right Parietal Cortex, Left Cerebellum (Flocculus/Nodulus)	Rehab strategy for left cerebellar dysfunction affecting right gaze
Right Cerebellar Activation (Leftward Smooth Pursuits)	Left Parietal Cortex, Right Cerebellum (Flocculus/Nodulus)	Rehab strategy for right cerebellar dysfunction affecting left gaze



CORTICOSPINAL TRACTS: VOLUNTARY MOVEMENT FROM THE CONTRALATERAL FRONTAL LOBE. LATERAL IS FOR OPPOSITE EXTREMITIES WHEREAS ANTERIOR IS MORE TRUNK AND AXIAL MOTOR CONTROL
RUBROSPINAL TRACT: FRONTAL AND CEREBELLAR OUTPUT TO MOTOR NEURONS. MOSTLY FOR FLEXOR MUSCLE STIMULATION
RETICULOSPINAL TRACT: MOSTLY TO FIRE TO MUSCLES OF POSTURAL CONTROL/AXIAL AND LOCOMOTION
OLIVOSPINAL TRACT: INTEGRATES CEREBELLUM AND SPINAL CORD FOR LEARNING OF MOVEMENTS AND OTHER BRAINSTEM/ CEREBELLAR SYSTEMS
VESTIBULOSPINAL TRACT: INTEGRATES BALANCE CENTERS, POSTURAL AND EXTENSOR MUSCLES

LATERAL SPINOTHALAMIC TRACT: RELAYS PAIN AND TEMPERATURE
ANTERIOR SPINOTHALAMIC TRACT: RELAYS CRUDE TOUCH
SPINOCEREBELLAR SYSTEMS: UNCONCIOUS PROPRIOCEPTION FROM MUSCLE SYSTEMS
DORSAL COLUMN MEDIAL LEMNISCUS: FINE/ACCURATE TOUCH, 2 POINT DISCRIMINATION, PROPRIOCEPTION AND VIBRATION.
GRACILE = LOWER BODY/LIMBS, CUNEATE = UPPER BODY/LIMBES
SPINO-OLIVARY SYSTEM: UNCONSCIOUS PROPRIOCEPTION AND BALANCE (SKIN/CUTANEOUS, MUSCLES AND TENDONS TO THE OLIVES OF THE MEDULLA AND 3RD ORDER NEURONS INTO THE CEREBELLUM VIA THE INFERIOR CEREBELLAR PEDUNCLE)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE MASTER PROTOCOL
NEUROMUSCULOSKELETAL

ERCHONIA eCommunity Education

1. ASSESS THE BALANCE OF THE CENTRAL NERVOUS SYSTEM. A COMPREHENSIVE DIAGNOSTIC AND BEDSIDE WORK-UP IS ALWAYS RECOMMENDED (SEE NEUROLOGICAL EXAM AT BACK OF NOTES). WITH THAT SAID, FOCUS ON CENTER OF PRESSURE/PERCEIVED CENTER OF PRESSURE, BRAINSTEM AND CEREBELLAR BALANCE. PERFORM THE FOLLOWING TESTS:

RHOMBERG'S
TANDEM STANCE LE, FINGER TO NOSE UE. SECONDARY TESTS SUCH AS DDK AND HEEL TO SHIN ARE RECOMMENDED
FAKUDA STEP TEST (EYES CLOSED, ARMS RAISED, MARCH IN PLACE 30+ STEPS)

WITH ANY OF THE 10-40 PRE-SETS (IE. MASTER BRAIN 1-10-40-60, OR OPTIMIZE 10-40-10-40) LASER THE SIDE OF WEAKNESS OR DEFICIT. EX. IF THEY ARE SWAYING TO THE RIGHT ON RHOMBERG'S LASER THE OPPOSITE DIRECTION ON THE RIGHT BRAINSTEM. IF THE RIGHT CEREBELLUM IS SHOWING DEFICIT (DYSMETRIA, RIGHT FOOT TANDEM FALL RIGHTWARD ETC) LASER THAT CEREBELLUM. USE THE RULE OF 2 MINUTES UPREGULATION. RECHECK. IF THE PATIENT IS BALANCED MOVE ON TO THE "DOWNSTREAM" ALGORITHM BELOW. IF THEY ARE NOT BALANCED YET GO BACK TO LASER FOR LONGER AND STACK WITH CO-ACTIVATION (IE. CEREBELLAR COMPLEX MOVEMENTS TO THAT SIDE, VIBRATION TO THE LOWER EXTREMITIES WITH RHOMBERG'S ETC). TRY TO OBTAIN IMPROVEMENT IN THEIR SIGNS BEFORE MOVING DOWN STREAM.

A CENTRAL BALANCE WILL IMPROVE OUTCOMES WITH YOUR THERAPIES TO THE BODY, PREVENT INJURY AND HELP TREATMENTS HOLD AND IMPROVE FASTER!

2. THE "DOWNSTREAM" PROTOCOL: USING THE SAME SETTING USE THE FOLLOWING SEQUENCE, AGAIN USING 2 MINUTES TO EACH REGION DESCRIBED.

A) WHILE LASERING THE PREFRONTAL CORTEX (CAN BE OPPOSITE TO THE INVOLVED PROBLEM AREA A), HAVE THE PATIENT VISUALIZE MOVEMENTS AND ACTIVITIES WITHOUT PAIN OR STRESS, VISUALIZE DOING ACTIVITIES HAPPY AND FREE OF PAIN, EVEN IF THEY HAVE TO GO BACK TO CHILDHOOD ACTIVITIES OR MEMORIES.
B) MOVE THE LASER TO THE PARIETAL LOBE (CAN BE BILATERAL OR OPPOSITE) AND VIBRATE OVER THE INVOLVED REGION OF PAIN/DYSFUNCTION (ASSUMING NO CONTRAINDICATIONS)
C) MOVE THE LASER TO THE IPSILATERAL CEREBELLUM AND PERFORM MOVEMENT PATTERNS (COMPLEX, LINEAR OR ISOMETRIC. MAY BE PASSIVE OR ACTIVE. MAY OR MAY NOT OVERLAP THE PHASE 1 CEREBELLAR BALANCING).
D) MOVE THE LASER TO THE IPSILATERAL VAGUS NERVE/SCM ALONG THE NECK WITH 2 MINUTES OF BREATHING AND RELAXATION
E) MOVE THE LASER TO THE SPINAL CORD AND NERVE ROOTS OF THE INVOLVED REGION (DO VERTICALLY AND USE THE VIOLET ON THE CORD IF AVAILABLE WITH THE RED OR GREEN TOWARDS THE INVOLVED NERVE ROOTS)
F) FINISH WITH THE FINAL 2 MINUTES DIRECTLY OVER/AROUND/ON THE INVOLVED REGION

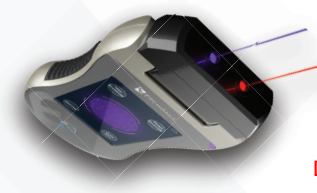
"THE LITTLE BRAIN"

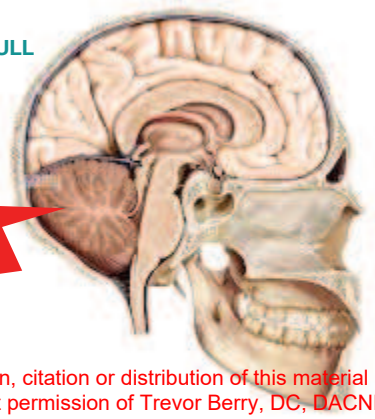
ERCHONIA eCommunity Education

DO NOT UNDERESTIMATE THE IMPORTANCE OF THE CEREBELLUM!
ESTIMATES OF THE NUMBER OF NEURONS IN THE BRAIN RANGE FROM 80 BILLION TO NEWER MODELS OF 150 BILLION
OF THOSE, IT IS ESTIMATED THE CEREBELLUM CONTAINS ABOUT 80 BILLION NEURONS!
NEWER QEEG STUDIES SHOW IT HAS IT'S OWN HUBS SIMILAR TO THE NEOCORTEX AND BROADMAN'S AREAS
IT GOES FAR BEYOND TONE AND BALANCE…
IT INTEGRATES WITH THE FRONTAL LOBE INCLUDING PRE-PLANNING, SPEECH CO-ORDINATION AND MORE
IT MODULATES BRAINSTEM ACTIVITY INCLUDING AUTONOMIC FUNCTION
IT IS INVOLVED IN PAIN ADAPTATION
IT IS VERY METABOLICALLY DEMANDING THEREFORE HEAVILY AFFECTED BY ETOH, ANEMIA, SUGARS ETC
IT'S MOST COMMON AUTOIMMUNE TRIGGER IS GLIADIN
IT IS ALSO A TARGET FOR PATHOGENS LIKE MOLD AND OTHER ENVIRONMENTAL BURDENS SUCH AS METALS
WATCH FOR METASTATIC SPREAD!

IT LIKES HIGH AND LOW FREQUENCIES AS WELL AS ALPHA WAVE FORMS (MASTER BRAIN SETTING 1-10-40-60)

MINIMUM 2 MINUTES BASE OF THE SKULL
CONSIDER STACKING THERAPIES:
COMPLEX MOVEMENTS
ISOMETRICS
BALANCE DEVICES
PLAYING AN INSTRUMENT





ARBORVITAE
"TREE OF LIFE"
FRACTAL PATTERNS
MANDELBROT SET
AYA

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PROTECT YOUR BRAIN!

Study Links Thumb Length to Brain Size and Cognition

FeaturedNeuroscience·August 26, 2025

Thumbs linked to thinking, not movement

The scientists made a surprising discovery about which part of the brain grows alongside longer thumbs. They expected longer thumbs to be linked to the cerebellum because it is the region of the brain that controls movement and coordination.

Instead, longer thumbs were connected to the neocortex (a complex layered region comprising approximately half the volume of the human brain), which processes sensory information and handles cognition and consciousness. (Think Parietal lobe integration).

It was a surprise that only one of the two major brain regions they thought would be involved actually was.

CLINICAL TAKE HOME: CONSIDER SENSORY INTEGRATION USING DIGITS TO FURTHER PROMOTE BRAIN HEALTH AND GROWTH SUCH AS PLAYING INSTRUMENTS LIKE THE GUITAR…OR GROW LONGER THUMBS!

Original Research: Open access.
"Human dexterity and brains evolved hand in hand" by Joanna Baker et al. *Communications Biology*

APPLIED FUNCTIONAL NEUROLOGY

OCULOMOTOR ALGORITHM

ERCHONIA eCommunity Education

WHEN ASSESSING EYE MOVEMENTS SUCH AS VNG (VIDEONYSTAGMOGRAPHY) OR BEDSIDE TESTING CONSIDER THE FOLLOWING ORDER OF ASSESSMENT AND TREATMENT:

1. RULE OUT NYSTAGMUS (BOTH IN NEUTRAL SEATED AND IN CANAL CHALLENGE POSITIONS LIKE DIX-HALLPIKE)
2. HOLDING GAZE. CENTRAL GAZE HOLD THEN ASSESS AND TREAT ALL QUADRANTS
3. VOR AND VHIT. CAN THE PATIENT MAINTAIN GAZE WHILE THE HEAD IS IN MOTION. THERE ARE vHIT (VIDEO HEAD IMPULSE TESTS) BUT CONSIDER HALMAGYI'S HEAD THRUST BEDSIDE
4. SACCADES. FAST EYE MOVEMENTS. MOST TESTING DEVICES ASSESS HORIZONTAL AND VERTICAL BUT NEWER DEVICES ASSESS ANGULAR AS WELL. ASSESS FOR ACCURACY, LATENCY AND VELOCITY
5. OPTOKINETICS. HORIZONTAL AND VERTICAL
6. SMOOTH PURSUITS. HORIZONTAL AND VERTICAL

*IF THEY CAN'T MAINTAIN GAZE HOLD DUE TO NYSTAGMUS OR INJURY TO GAZE HOLDING CENTERS, IT WILL BE VERY DIFFICULT TO BE SUCCESSFUL WITH TREATMENT OUTCOMES. AS A GENERAL CLINICAL SUMMARY, HORIZONTAL EYE AND HEAD ACTIVITY IS A PRODUCT OF THE PONTOMEDULLARY REGION AND VERTICAL EYE ACTIVITY IS MORE MIDBRAIN (MESENCEPHALON) RELATED.

A GOOD REFERENCE IS "THE NEUROLOGY OF EYE MOVEMENTS BY LEIGH AND ZEE"
FOR ADVANCE MODULE TRAINING, CONSIDER CARRICK INSTITUTE COURSES OR WORK WITH A CARRICK CERTIFIED DIPLOMATE OF NEUROLOGY

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

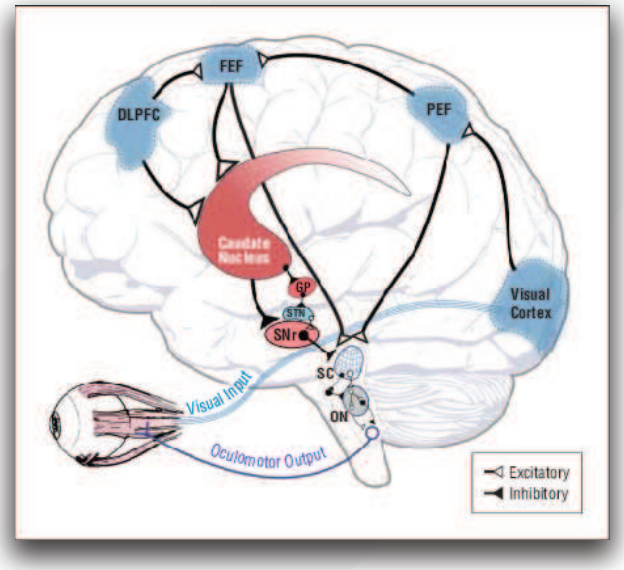
EYE MOVEMENTS FOR ASSESSING AND TREATING THE BRAIN

ERCHONIA eCommunity Education

Brain region that controls eye movements found to also play important role in higher cognitive functions

In the study, published in *Nature Neuroscience*, scientists measured the information contained in patterns of brain cell activity across multiple brain regions involved in visual category decisions. The researchers monitored activity in the superior colliculus (SC) and part of the posterior parietal cortex (PPC), a region of the cerebral cortex that is important for visual categorical decisions.

To their surprise, they saw that activity in the SC was even more involved than the PPC in guiding the subjects' category decisions, suggesting that it helps coordinate higher-order cognitive processes traditionally thought to take place in the neocortex.



Barbara Peysakhovich et al, Primate superior colliculus is causally engaged in abstract higher-order cognition, ***Nature Neuroscience* (2024)**. DOI: 10.1038/s41593-024-01744-x

EYE MOVEMENTS FOR ASSESSING AND TREATING THE BRAIN

ERCHONIA eCommunity Education

Brain region that controls eye movements found to also play important role in higher cognitive functions

"Some of this data might be telling us is that the reason we're making these kinds of spatial gestures and eye movements is because the spatial parts of the brain are getting recruited into helping us perform these non-spatial cognitive functions," said first author Barbara Peysakhovich, Ph.D., a former graduate student in Freedman's lab now a postdoctoral researcher at Harvard. Or, **we've all had the experience of struggling to understand something written in text—like a long press release about a neuroscience study—but having it instantly click into place when the same information is presented in a graphic.**"They say a picture is worth 1,000 words—even a very simple spatial diagram can rapidly convey so much more information than you can possibly describe," Freedman said. "It's like the brain has created this beautiful mental graph paper which it can use to solve both spatial and non-spatial problems."

Freedman pointed out the kind of eye movements and hand gestures that humans make when we're asked to recall something or make decisions. If someone asks what you had for dinner last night, for example, your eyes often drift upward, as if the answer were written on the ceiling. Or when weighing a decision between two choices, you might move your hands up and down like two sides of a balance scale.

Barbara Peysakhovich et al, Primate superior colliculus is causally engaged in abstract higher-order cognition, ***Nature Neuroscience* (2024)**. DOI: 10.1038/s41593-024-01744-x

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

EYE MOVEMENTS FOR ASSESSING AND TREATING THE BRAIN

ERCHONIA eCommunity Education

Front Neurol. 2016 Jan 22;7:3. doi: 10.3389/fneur.2016.00003

Eye-Movement Training Results in Changes in qEEG and NIH Stroke Scale in Subjects Suffering from Acute Middle Cerebral Artery Ischemic Stroke: A Randomized Control Trial

Frederick Robert Carrick [1,2,3,4,*], Elena Oggero [1,5], Guido Pagnacco [1,5], Cameron H G Wright [1,5], Calixto Machado [1]

Results

There was strong statistical and substantive significant improvement in all outcome measures for the group of stroke patients undergoing EMT. Such improvement was not observed for the "control" group, and there were no adverse effects.

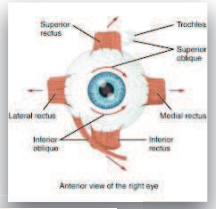
Conclusion

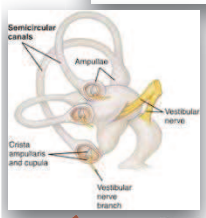
The addition of EMT to a MCA ischemic stroke treatment paradigm has demonstrated statistically significant changes in outcome measures and is a low cost, safe, and effective complement to standard treatment.

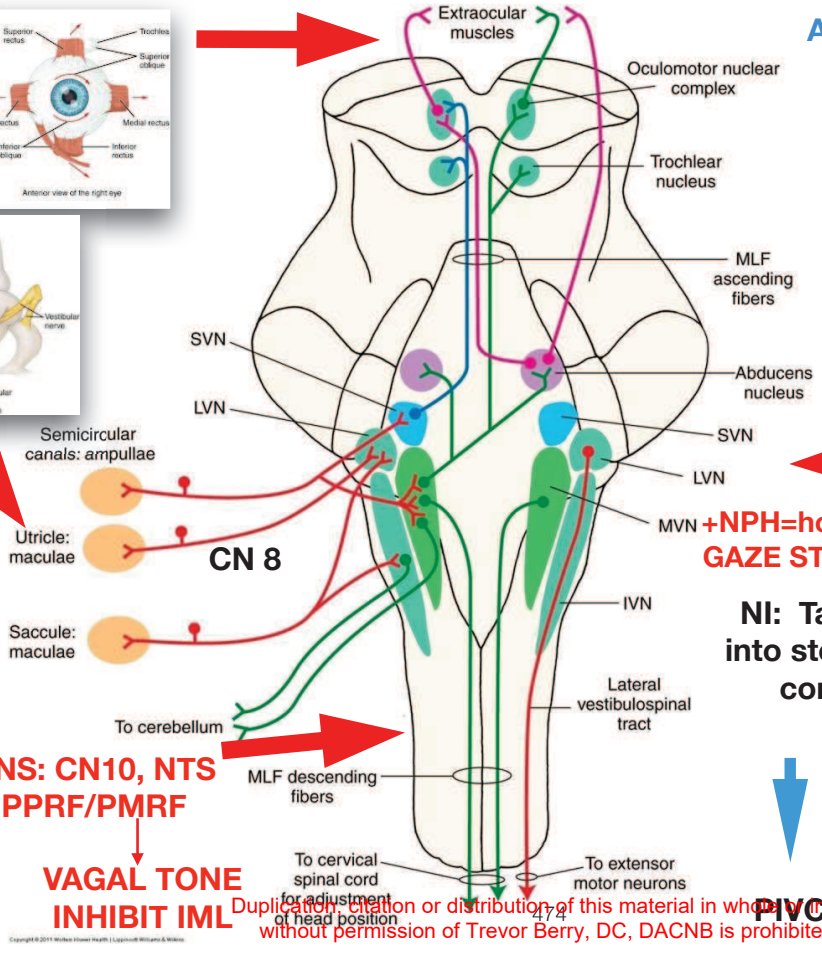
3 repetitions of canal/hemisphere bias combination of 2 second gap saccades (for left brain that is down and to the patient right, down and left for right brain) followed by smooth pursuits the opposite direction back to starting point. In the study this was done for 3 reps (about 3 minutes worth of therapy between activation and rest in between each), 3x/day. Pre and post EEG studies done at 7 days

HOW DO YOU KNOW WHICH SIDE? (IMAGING, BEDSIDE EXAM/SUBJECTIVE)
HOW DO YOU PERFORM THE EXERCISES? (BEDSIDE, FOCUS BUILDER APP)
WHERE WOULD YOU "STACK THE LASER"?

"THE EYES HAVE IT"







Vestibular/Sensory Mismatch?

Are our visual, auditory, vestibular and proprioceptive/LDA systems matching

Filtered through a common neural integrator

"Y"

Cerebellum

+NPH=horizontal GAZE STABILITY

"Leaky NI"

NI: Takes the pulse command into step command or a velocity command into a position command

ANS: CN10, NTS PPRF/PMRF

VAGAL TONE INHIBIT IML

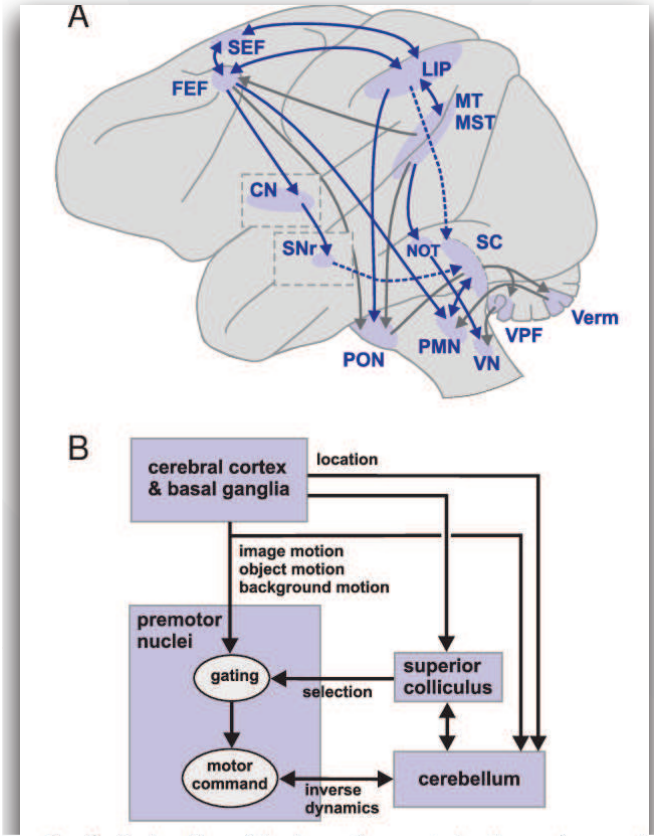
PVC

BALANCE TRACKING SYSTEMS

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE NEUROLOGY OF EYE MOVEMENTS



SMOOTH PURSUITS

475

LOW LEVEL LASER AND TBI

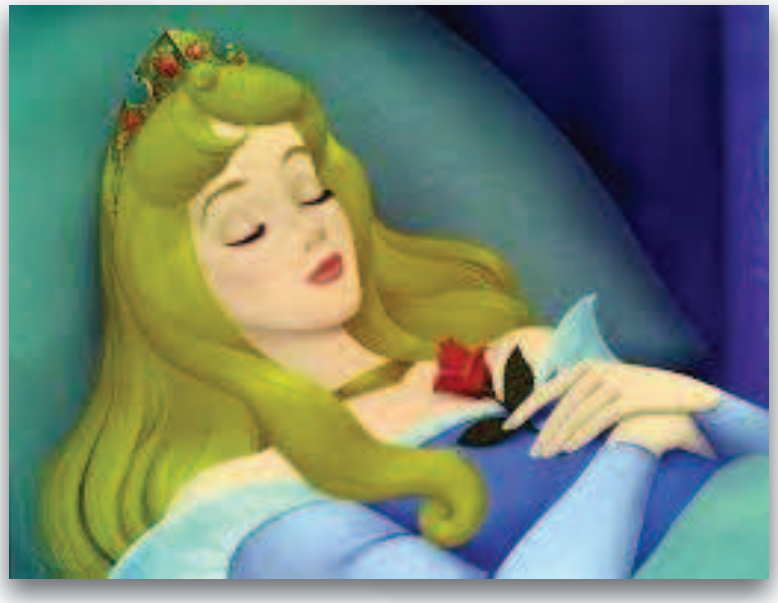
USING FIRST PRINCIPLES, WHAT AREAS OF THE BRAIN MIGHT THE CLINICIAN "UP REGULATE" WITH LOW LEVEL LASER THERAPY FOR THE FOLLOWING EYE MOVEMENTS:

- GAZE HOLDING (NEUTRAL, HORIZONTAL OR VERTICAL)
- FAST EYE MOVEMENTS (SACCADES)
- SMOOTH PURSUITS
- CANAL BALANCING (IN ABSENCE OF LITHIASIS)

HINT: ALWAYS LASER THE CEREBELLUM!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Dementia: Early bedtime, sleeping over 8 hours linked to increase risk

The dementia risk was 69% greater for individuals who slept over 8 hours, versus 7-8 hours. The risk was also twice as high for those who went to sleep before 9:00 p.m., versus 10:00 p.m. or later (but go before midnight)

CONVERSELY, In a long-term study, Harvard Medical School followed 2,800 individuals ages 65 and older. **Researchers found that individuals who slept under five hours per night were twice as likely to develop dementia compared to those who slept six to eight hours per night. A different study of nearly 8,000 participants found a 30% increase in dementia risk was associated with sleeping six hours or less at age 50, 60 and 70, compared to a normal sleep duration of seven hours.**

SUMMARY: GO TO BED BETWEEN 10-11 PM AND SLEEP 7.5 HOURS. HOWEVER…

Associations of sleep timing and time in bed with dementia and cognitive decline among Chinese older adults: A cohort study

Rui Liu MD, Yifei Ren MD, Tingting Hou MD, PhD, Xiaoyan Liang MD, Yi Dong MD, Yongxiang Wang MD, PhD, Lin Cong MD, PhD, Xiang Wang MD, PhD, Yu Qin MD … See all authors

First published: 21 September 2022

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP HYGIENE

Why Do Some People Thrive on So Little Sleep?

Short sleepers cruise by on four to six hours a night and don't seem to suffer ill effects

SUMMARY:

- SHORT SLEEPERS MAY FUNCTION OPTIMALLY AND NOT BE SUBJECT TO HARMFUL EFFECTS OF SLEEP "DEPRIVATION" IN RELATION TO THE STANDARD NORMS
- GENE MUTATIONS MAY BE THE CAUSE OF SHORT SLEEPERS. PARTICULARLY OF SNP's *DEC2, ADRB1, GRM1 AND NPSR1*
- THESE GENETIC MUTATIONS ALTER OREXIN LEVELS (OREXIN DEFICIENCY IS THE PRIMARY CAUSE OF NARCOLEPSY). SHORT SLEEPER MUTATIONS CREATE HIGHER LEVELS OF OREXIN
- IN ANIMAL STUDIES WITH THESE MUTATIONS, NOT ONLY DO THE SHORT SLEEPER GENE SNP's PERFORM WELL, ARE AMBITIOUS, ENERGETIC AND OPTIMISTIC, THEY ARE MORE RESILIENT AGAINST STRESS, HAVE HIGHER PAIN THRESHOLDS AND MAY ACTUALLY LIVE LONGER!
- SHORT SLEEPER GENES HAVE EVEN BEEN SHOWN TO BE MORE EFFICIENT AT REMOVING BAD PROTEINS LIKE AMYLOID FORMS AND TAU THAN REGULAR SLEEPERS

ANNUAL REVIEW OF GENOMICS AND HUMAN GENETICS Volume 25, 2024

Review Article Open Access

The Genetics of Human Sleep and Sleep Disorders

- Xianlin Zou1, Louis J. Ptáček1,2,3,4, and Ying-Hui Fu1,2,3,4
- View Affiliations

olume 25, Issue 4103964April 15, 2022*Open Access*

Familial natural short sleep mutations reduce Alzheimer pathology in mice

Qing Dong1,6

SLEEP CLINICAL SUMMARY

- SLEEP IS THE NUMBER 1 DETOX SYSTEM FOR BRAIN AND NEURODEGENERATION. WE FLUSH THE TOILET NIGHTLY WITH CSF LIKE A FAUCET TURNING ON. CELLS ACTUALLY SHRINK IS SIZE UP TO 60% ALLOWING A CSF BATH TO BE WASHED AWAY VIA THE GLYMPHATIC WASTE SYSTEM…RECENT SLYM LAYER TIED DIRECTLY INTO LYMPHATIC SYSTEM AND GLIAL CELLS. THE IMMUNE SYSTEM OF THE BRAIN HOVERS IN THIS MENINGEAL LAYER. LLLT AT NIGHT CLEARS OUT EVEN MORE AB AND TAU PROTEINS.
- IDEAL SLEEP CYCLE FOR ADULTS BETWEEN 7-8 HOURS (IDEAL IS 7.5 FOR 90 MINUTE CYCLE) WITH APPROX. 9:30-11:30 START TIME (NOT BEFORE 9 OR AFTER MIDNIGHT). HOWEVER, GENETIC AND OTHER VARIABLES FACTOR INTO THIS CYCLE OPTIMIZATION
- USE CIRCADIAN RYTHMS. WAKE UP AT SUNRISE AND GO OUT FOR A WALK TO GET DIRECT SUNLIGHT EXPOSURE/GROUNDING
- AVOID BLUE LIGHT ESPECIALLY AFTER DARK GETTING CLOSER TO BEDTIME, USE COMPUTER FILTERS, NIGHT SETTING ETC
- PRECURSORS AND CO-FACTORS FOR SLEEP. SEROTONIN (FROM TRYPTOPHAN), GUT HEALTH/PROBIOTIC
- PROBIOTIC STRAINS SLEEP SPECIFIC INCLUDE LACTOBACILLUS HELVETICUS, ACIDOPHILUS AND REUTERI NK33, ADOLESCENTIS NK 98, LACHNOSPIRACEAE UCG004, BIFIDO INFANTIS/BREVE/LONGUM, AND ODORIBACTER. BAD FOR INSOMINA = SELENOMONADALES AND NEGATIVICUTES
- MELATONIN AND CBD HELP GET INTO A RELAXED ALPHA WAVE STATE BUT NOT NECESSARILY DEEP DELTA/REM
- MELATONIN USE IN TRAVEL, POST-CONCUSSION, COVID/LONG COVID AND SOME NEURODEVELOPMENTAL LIKE AUTISM.
- USE CR IF YOU DO
- AVOID IN TH1 DOMINANT AUTOIMMUNE CONDITIONS SINCE MELATONIN PROMOTES TH1 SO WATCH IN CONDITIONS LIKE MS, IBD/CHRON'S/CELIAC, HASHIMOTO'S, RHEUMATOID
- WATCH GENE SNPS FOR NTS MTHFR AND COMT
- CO FACTOR SUPPLEMENTATION (SEE SLIDE)
- MAGNESIUMS FOR SLEEP #1 LYSINATE/GLYCINATE, #2 L THREONATE
- FASTING? DYSGLYCEMIA, PREGNENOLONE
- INFLAMMATION SOURCES AND MRA
- SOUND DEVICES: PINK VS WHITE VS BROWN NOISE. BRAIN TAP. SOLFEGGIO (432 HZ). BINAURAL BEATS (10 HZ TO WIND DOWN, 1-4 HZ DELTA FOR DEEP SLEEP AND 4-8 HZ THETA FOR REM SLEEP) MOST OF THESE CAN BE FOUND FOR FREE.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP HYGIENE

The truth about magnesium glycinate's effect on your sleep quality

A landmark 2012 study published in the Journal of Research in Medical Sciences found that **magnesium supplementation significantly improved sleep efficiency by 16%, increased sleep time by 47 minutes,** and reduced early morning awakening in elderly participants with insomnia. More recent studies in 2023 have corroborated these findings across different age groups, suggesting broad applicability for sleep support.

Its primary action involves GABA receptor activation and melatonin regulation, while simultaneously supporting cortisol reduction and muscle relaxation.

DOSE: MINIMUM 200 MG 1 HOUR BEFORE BED. STACK WITH 1 GRAM OF MAGNESIUM L-THREONATE

481

MAGNESIUM

MAGNESIUM: EVERY CELL IN THE HUMAN BODY NEEDS MAGNESIUM. INVOLVED IN OVER 300 PROCESSES IN THE HUMAN BODY AND IS THE 4TH MOST ABUNDANT MINERAL IN HUMANS. 60% IS STORED IN BONES AND THE REMAINDER IN OTHER TISSUES. GOOD FOOD SOURCES INCLUDE GREEN LEAFY VEGETABLES, NUTS, LEGUMES, MEAT, POTATOES, RICE AND WHOLE GRAINS*. OVER 1/2 OF AMERICANS ARE MAGNESIUM DEFICIENT.

MAGNESIUM L-THREONATE (MAGTEIN): CROSSES THE BLOOD BRAIN BARRIER, PROTECTS AND REPAIRS THE BBB. NMDA "PLUG" PROTECTS AGAINST GLUTAMATE EXCITOTOXICITY. PROMOTES HEALTHY NEUROPLASTICITY/SYNAPTIC FUNCTION. PROMOTES HEALTHY COGNITION AND MEMORY, AND MITIGATES COGNITIVE DECLINE, DEPRESSION AND OTHER NEUROLOGICAL DISORDERS.

MAGNESIUM MALATE (DI-MAGNESIUM): MG AND MALATE HIGH IN FRUIT AND WINE. ATP SYNTHESIS/ENERGY AND PAIN RELIEF. GENTLE ALUMINUM CHELATOR. HIGH ABSORPTION RATE AND LOW SIDE EFFECTS.

MAGNESIUM LYSINATE/GLYCINATE: MG AND AA'S LYSINE AND GLYCINE. = RELAXATION AND CALMING. USED IN ANXIETY AND INSOMNIA.

MAGNESIUM CITRATE: MG AND CITRIC ACID = LAXATIVE. HIGH LEVEL OF SIDE EFFECTS AND DRUG INTERACTIONS. CHEST PAINS, DIARRHEA, NAUSEA/VOMITING, HEART REGULATION AND MORE. HIGH IN CITRUS FOODS AND OFTEN USED IN FOODS AND SUPPLEMENTS.

MAGNESIUM OXIDE: MG AND OXYGEN ALSO = LAXATIVE (IE MILK OF MAGNESIA) ALSO USED IN HEARTBURN AND INDIGESTION. POOR ABSORPTION (APPROX. 4%) VS OTHER MG FORMS. MOST COMMON FORM OF MG USED IN SUPPLEMENTS.

MAGNESIUM CHLORIDE: SIMILAR FUNCTION AS OXIDE WITH SLIGHTLY HIGHER ABSORPTION. ALSO OFTEN USED IN TOPICALS FOR MUSCLE RELAXATION/SORENESS.

MAGNESIUM LACTATE: MG AND LACTIC ACID. GENTLE ON THE STOMACH VS FORMS LIKE CITRATE. OFTEN USED IN FOOD ADDITIVES.

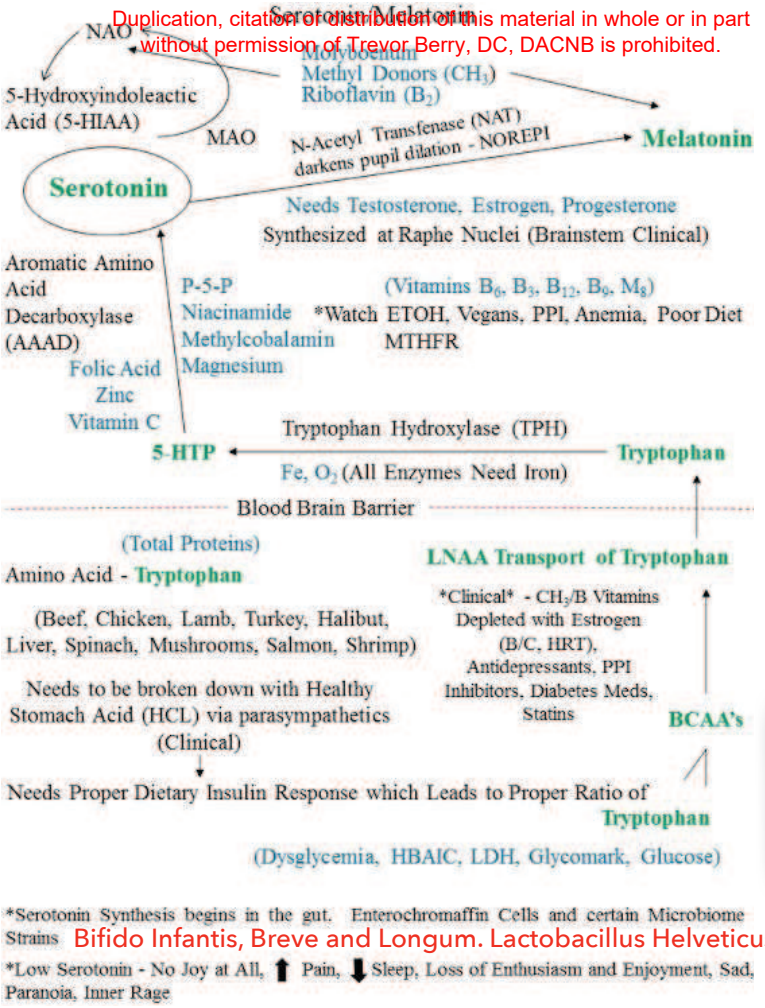
MAGNESIUM SULFATE: MG, SULFUR AND OXYGEN. = EPSOM SALT. TOPICAL/BATHS FOR SORE MUSCLES/RELAXATION. HARD ON STOMACH.

MAGNESIUM OROTATE: MG AND OROTIC ACID. CARDIOVASCULAR SYSTEMS BENEFITS.

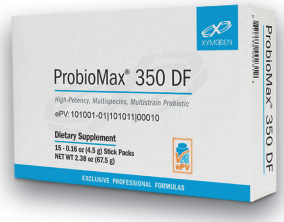
Open Access Review
Magnesium in Prevention and Therapy by **Uwe Gröber**, **Joachim Schmidt** and **Klaus Kisters** 482
Nutrients **2015**, 7(9), 8199-8226; **https://doi.org/10.3390/nu7095388**

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



MICRONUTRIENT PANEL!



SLEEP

ERCHONIA eCommunity Education

- POLYSOMNOGRAPHY (CENTRAL VS OBSTRUCTIVE APNEA VS BODY MOVEMENTS)
- C-PAP/BI-PAP, BREATHING STRIPS, BED/MATTRESS POSITION/PILLOW
- TONE OF ORAL-PHARYNGEAL REGION (VAGAL STIM/DIRECT SOFT PALATE STIM) MOUTH TAPING WITH BREATHE RIGHT NASAL STRIPS
- BASE LAB CHEMISTY (ANEMIA PATTERNS, DYSGLYCEMIA, THYROID, SEX HORMONES…HP AXIS)
- ADVANCED LAB MARKERS: MICRONUTRIENT (CO-FACTORS FOR MELATONIN SYNTHESIS)
- ASI/CORTISOL CURVE AND GLYCOGEN STORES. CORTISOL VS MELATONIN
- CYTOKINES AND MESENCEPHALIC RETICULAR ACTIVATING SYSTEM. FOOD SENSITIVITY TESTING, INFECTIOUS DISEASE MANAGEMENT
- PAIN MANAGEMENT
- BASAL GANGLIA ACTIVATION/DISINHIBITION
- QUALITY OF PILLOW, MATTRESS (AGE ETC)
- ELECTRONICS AT NIGHT, AVOID BLUE WAVELENGTHS/NIGHT SETTING, MINIMIZE VISUAL STIMULATION THROUGHOUT DAY/COMPUTER FILTERS
- QEEG/NEUROFEEDBACK
- CBD/MEDICAL MARIJUANA. MELATONIN CR, PROBIOTICS, GABA

LOW LEVEL LASER TONS…

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP

Front Hum Neurosci. 2018; 12: 387.
Published online 2018 Sep 24. doi: 10.3389/fnhum.2018.00387

A Novel Insight of Effects of a 3-Hz Binaural Beat on Sleep Stages During Sleep

Nantawachara Jirakittayakorn* and Yodchanan Wongsawat*

These findings suggested that the 3-Hz binaural beat on a 250-Hz carrier tone can be used to modulate sleep stage by decreasing the latency to the N3 stage, **extending the N3 duration, and reducing the N2 duration without sleep disturbance and sleep fragmentation, while increasing the quality of sleep in this study is also associated slow wave sleep in which is related to memory consolidation and other regulation of the body; and the 3-Hz binaural beat can enhance power of delta activity during sleep.**

Family Med Prim Care. 2019 Dec; 8(12): 3915–3919.
Published online 2019 Dec 10. doi: 10.4103/jfmpc.jfmpc_575_19

Effect of music of specific frequency upon the sleep architecture and electroencephalographic pattern of individuals with delayed sleep latency: A daytime nap study

Pramita Dubey,1 Yogesh Kumar,1 Ramji Singh,1 Kamlesh Jha,1 and Rajesh Kumar1

Conclusion:

It was concluded that 432 Hz music has some significant calming effect as reflected by increased alpha activities … naps.

Nature
. 2006 Nov 30;444(71

Boosting slo

Lisa Marshall 1, Halla

Here we show that in … g potentials (0.75 Hz) during … w wave sleep, enhances the … ories in healthy huma … ve sleep, endogenous cortical

BASE SLEEP SETTING 1 - 2 - 3 - 432
RED XLR8
ALSO CONSIDER "REST AND DIGEST"
VAGAL STIM IN THE PM (1-10-1-10)
COMBO SETTING (1-3-10-432)
10 MIN PROTOCOL BEFORE BED
(8 MIN EACH PRE-FRONTAL CORTEX
2 MIN VAGAL STIM SCM REGION)

LOW LEVEL LASER THERAPY AND ALZHEIMER'S

Light therapy helps the brain clear out toxic Alzheimer's proteins

Despite their tireless efforts, researchers have been unable to develop a safe and effective way of treating Alzheimer's disease (AD) using pharmaceuticals, which has meant turning to non-pharmaceutical methods. A new study has demonstrated the therapeutic potential of light therapy, or phototherapy, in treating AD, showing promising results in mice that the researchers hope can be just as effective in humans.

Measuring the level of beta-amyloid in the hippocampus following PBM, the researchers found that levels were lower whether it was applied during wakefulness or sleep but that PBM during sleep produced a greater reduction in beta-amyloid. **They concluded that PBM stimulated the evacuation of beta-amyloid from the hippocampus even more effectively during sleep than wakefulness.**

THE "AFTER HOURS" ALZHEIMER'S ADD ON:

ALBEIT NOT AS PRACTICAL, USING LLLT DURING SLEEP SHOULD BE CONSIDERED IF A DEVICE IS AVAILABLE AT HOME. IF A HAND HELD LASER IS AVAILABLE AT HOME (OWNERSHIP OR LASER RENTAL PROGRAM, CONSIDER THE "AFTER HOURS" SETTING FROM A SPOUSE OR PARTNER DURING SLEEP CYCLES)

1-10-1-40

They say that this non-pharmaceutical, non-invasive treatment could be used in people with AD and other conditions that involve the brain's lymphatic system. "Since pharmacological therapy of AD have failed to show effectiveness or safety, PBM as a non-invasive and safe approach has the high prospects for implementation in clinical practice for the treatment of brain diseases with lymphatic disorders, such as AD, Parkinson's disease, glioma, traumatic brain injury, intracranial hemorrhages," the researchers said.

Oxana, SG., Alexander, S., Inna, B. *et al.* Mechanisms of phototherapy of Alzheimer's disease during sleep and wakefulness: the role of the meningeal lymphatics. *Front. Optoelectron.* **16**, 22 (2023). **Published18 September 2023**

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

GREEN FOR SLEEP?

Here, we show that blue light (470 nm) causes behavioural arousal, elevating corticosterone and delaying sleep onset. By contrast, green light (530 nm) produces rapid sleep induction.

Melanopsin Regulates Both Sleep-Promoting and Arousal-Promoting Responses to Light

- Violetta Pilorz, Published: June 8, 2016

Researchers have found that green light promotes sleep while blue light delays it. In a study conducted by Oxford University, green light produced rapid sleep onset in mice - between 1 and 3 minutes.

Lighting color affects sleep, wakefulness

Green light promotes sleep while blue light delays it, find researchers

Date:

June 8, 2016

GVL CONSIDERATION: GREEN BEAM 2 MIN APPLICATION RETINAL BEFORE BEDTIME

MATTRESS RECOMMENDATIONS

Best Mattresses of 2024 (SOURCE SLEEPOPOLIS):

- Helix – Best Mattress Overall
- Nectar – Best Value Mattress
- DreamCloud – Best Mattress for Support
- Saatva Classic – Best Hybrid Mattress
- Helix Midnight Luxe – Best Luxury Mattress
- Plank Firm – Best Firm Mattress
- Saatva Rx – Best Mattress for Back Pain
- Nectar Premier – Best Mattress for Side Sleepers
- Titan Plus – Best Mattress for Heavy People
- DreamCloud Premier – Best Cooling Mattress

ANOTHER CONSIDERATION: THE SS500 COMFORT PREMIUM BASE "SUPERIOR SLEEP EXPERIENCE" COOL GEL ADJUSTABLE BED

WARNING: CONSUMER REPORTS CONTINUES TO LIST THE AVOCADO GREEN AS THEIR TOP RECOMMENDED MATTRESS YEAR AFTER YEAR. THEY MAY BE RIGHT FOR SOME ESPECIALLY BACK SLEEPERS, BUT THEY TEND TO BE TOO FIRM FOR SIDE SLEEPERS!

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

DEPRESSION AND BRAIN INFLAMMATION

Front Neurosci. 2019; 13: 213.
Published online 2019 Mar 15. doi: 10.3389/fnins.2019.00213

Brain Microglial Activation in Chronic Pain-Associated Affective Disorder

MOST CHRONIC PAIN AND NEUROPSYCH DISORDERS HAVE A GLIAL/ INFLAMMATION COMPONENT OR CAUSE. ALWAYS ADDRESS CNS ANTI-INFLAMMATION TREATMENTS (LASER BRAIN, VAGUS NERVE, NUTRITION ETC) AND ELIMINATE SOURCES LIKE LEAKY GUT, SAD DIET, PATHOGENS AND SO ON…

injury. In the present study, we characterized microglial activation in brain sub-regions of CX3CR1GFP/+ mice after chronic constriction injury (CCI) of the sciatic nerve, including observations at delayed time points when affective brain dysfunctions such as depressive-like behaviors typically develop. **Mice manifested chronic mechanical hypersensitivity immediately after CCI and developed depressive-like behaviors 8 weeks post-injury. Concurrently, significant increases of soma size and microglial cell number were observed in the medial prefrontal cortex (mPFC), hippocampus, and amygdala 8 weeks post-injury. Transcripts of CD11b, and TNF-α, genes associated with microglial activation or depressive-like behaviors, are correspondingly upregulated in these brain areas. Our results demonstrate that microglia are activated in specific brain sub-regions after CCI at delayed time points and imply that *brain microglial activation plays a role in chronic pain-associated affective disorders.***

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION AND SEROTONIN

ERCHONIA eCommunity Education

Serotonin Deficiency Directly Linked With Depression in Groundbreaking Study

TOPICS: DepressionPopularPsychiatrySerotonin

By ELSEVIER DECEMBER 27, 2022

Researchers have postulated since the 1960s that major depression stems from disruptions in the serotonin neurotransmitter system. However, the evidence for that idea, though plentiful, was indirect. **In fact, a recent comprehensive analysis of existing studies concluded that there was not strong evidence to support the "serotonin hypothesis." In its wake, some in the field have called for a reexamination of the hypothesis. Not so fast, says a new study that provides direct evidence of disrupted serotonin release in the brains of individuals with depression.**

"This study used a new and more direct method to measure serotonin in the living human brain, and the results suggest reduced serotonin (release) functioning in depression."

Participants with depression and healthy controls underwent **PET scanning with [^{11}C]Cimbi-36 to measure 5-HT2A receptor availability in the frontal cortex; the two groups did not differ significantly at baseline.** Both groups then received a dose of d-amphetamine, a stimulant drug that works to increase 5-HT concentration outside of neurons, where it interacts with 5-HT2A receptors and reduces the binding of [^{11}C]Cimbi-36. In a second scanning session three hours after drug administration, **healthy control participants had significantly reduced 5-HT2A receptor availability, indicating an increase in serotonin levels. Participants with depression, however, did not show a significant decrease in binding potential, suggesting they had a blunted serotonin release capacity in key brain regions.**

Of note, all patients were free of antidepressant medication, and 11 out of the 17 had never received antidepressant treatment, indicating that low serotonin release capacity is a feature of depression rather than a result of antidepressant treatment.

Reference: "Brain Serotonin Release Is Reduced in Patients With Depression: A [11C]Cimbi-36 Pet Study With a D-Amphetamine Challenge" by David Erritzoe, Beata R. Godlewska, Gaia Rizzo, Graham E. Searle, Claudio Agnorelli, Yvonne Lewis, Abhishekh H. Ashok, Alessandro Colasanti, Iro Boura, Chloe Farrell, Hollie Parfit, Oliver Howes, Jan Passchier, Roger N. Gunn, David J. Nutt, Philip J. Cowen, Gitte Knudsen and Eugenii A. Rabiner, 28 October 2022, *Biological Psychiatry*.

DOI: 10.1016/j.biopsych.2022.10.012

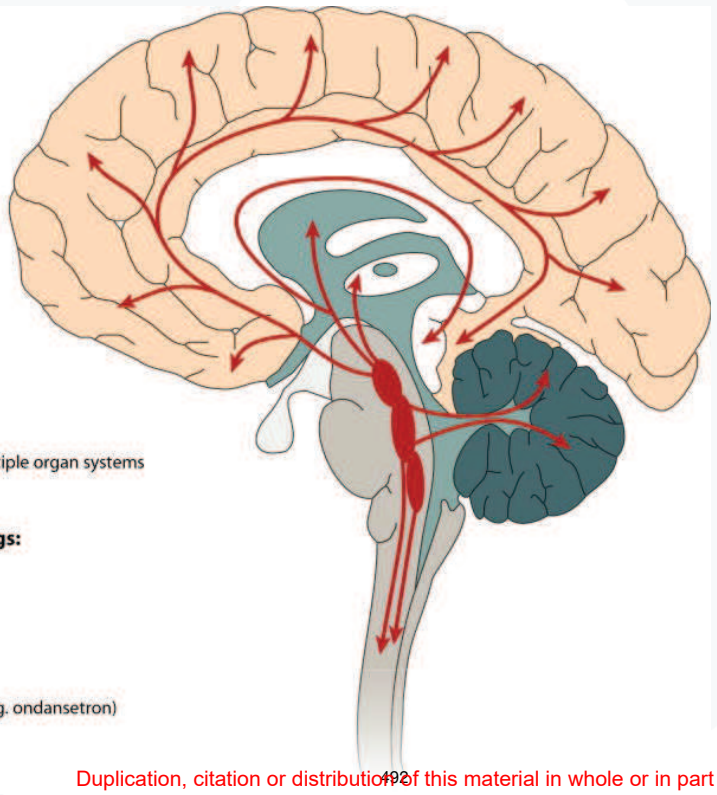
SEROTONIN PRODUCTION IN THE BRAIN

ERCHONIA eCommunity Education

Behavioral effects:
Mood
Perception
Memory
Anger
Aggression
Fear
Stress responses
Appetite
Addiction
Sexuality

Other CNS effects:
Motor control
Cerebellar regulation
Sleep/circadian rhythms
CNS vascular tone
Emesis
Respiratory drive
Body temperature
Descending regulation of multiple organ systems

Central serotonergic drugs:
SSRIs
Tricyclic antidepressants
MAOIs
Other antidepressants
Buspirone
Atypical antipsychotics
Triptans
5-HT$_3$ receptor antagonists (e.g. ondansetron)
Fenfluramine
Ergotamine/methysergide
Hallucinogens



MANUFACTURED IN THE RAPHE NUCLEI LOCATED IN THE BRAINSTEM

MADE FROM TRYPTOPHAN, AN ESSENTIAL AMINO ACID

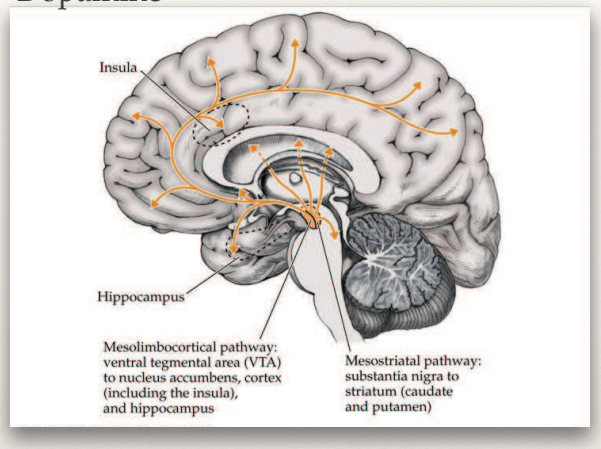
YOU ALSO NEED CO-FACTORS AND TRANSPORT SYSTEMS

A HEALTHY GUT AND MICROBIOME ARE ALSO CRITICAL FACTORS (UP TO 90% OF THE BODY'S SEROTONIN IS MADE IN THE GUT

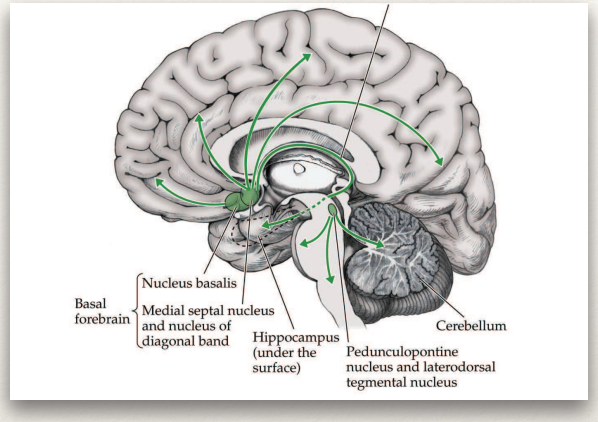
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

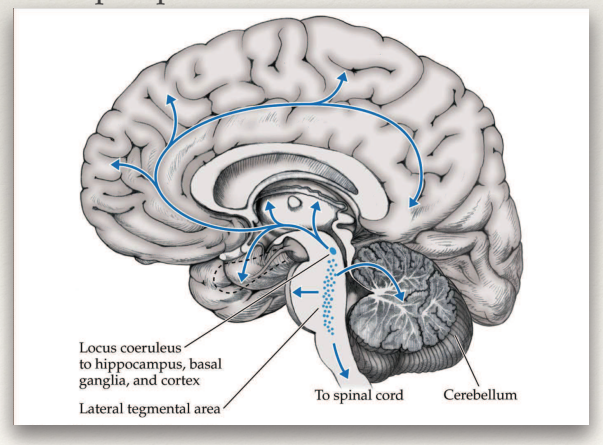
Dopamine



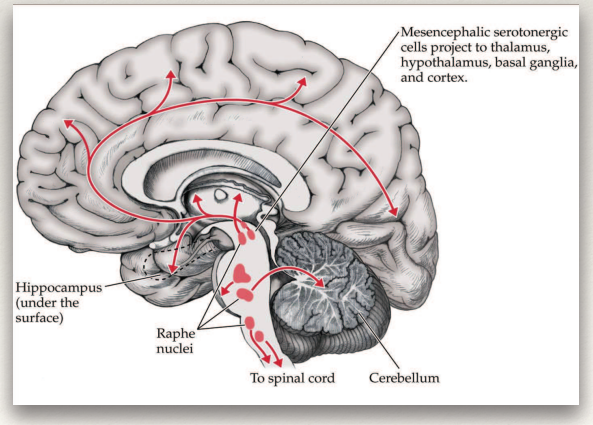
Acetylcholine



Norepinephrine



Serotonin



DEPRESSION

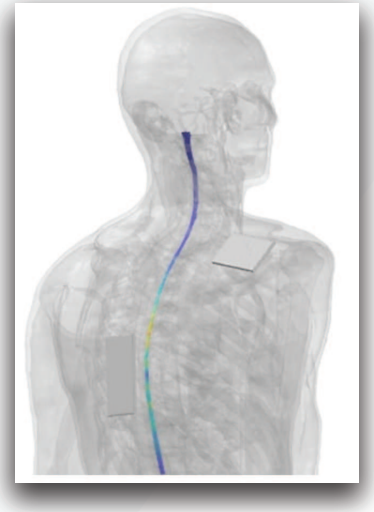
ERCHONIA eCommunity Education

The Spinal Cord Could Provide a Radical New Way to Treat Depression

HEALTH

13 January 2024

A team led by researchers at the University of Cincinnati College of Medicine devised a pilot clinical trial in which a little black box was placed on the spinal cord of **20 volunteers with depression, with one electrode on the back and one on the right shoulder.**



The box then delivered a specially customized, low-level electric buzz to half of the volunteers, for **three sessions per week over eight weeks**. This was shown to have a greater effect on depressive symptoms than the different, ' placebo' charge administered to the other half of the volunteers.

"We think that the connection between the brain and the body is essential for psychiatric disorders," says Romo-Nava.

- Open access MOLECULAR PSYCHIATRY
- **Published: 20 December 2023**

Effect of non-invasive spinal cord stimulation in unmedicated adults with major depressive disorder: a pilot randomized controlled trial and induced current flow pattern

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

- Francisco Romo-Nava

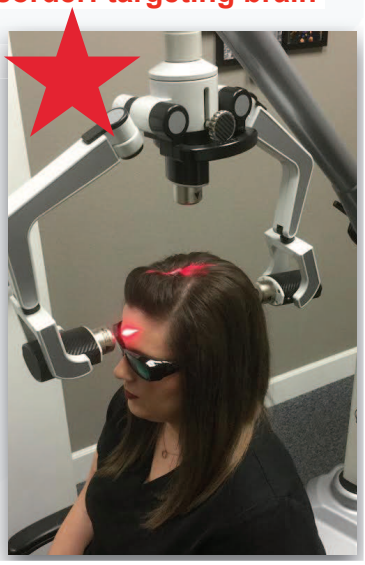
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION AND LLLT

"Depression: let's talk" says WHO, as depression tops list of causes of ill health 30 MARCH 2017 | GENEVA - Depression is the leading cause of ill health and disability worldwide. According to the latest estimates from WHO, more than 300 million people are now living with depression, an increase of more than 18% between 2005 and 2015.

Neurophotonics. 2016 Jul;3(3):031404. doi: 10.1117/1.NPh.3.3.031404. Epub 2016 Mar 4.
Review of transcranial photobiomodulation for major depressive disorder: targeting brain metabolism, inflammation, oxidative stress, and neurogenesis.
Cassano P1, Petrie SR2, Hamblin MR3, Henderson TA4, Iosifescu DV5.

Neuroscience. 2013 Jan 29;230:13-23. doi: 10.1016/j.neuroscience.2012.11.016. Epub 2012 Nov 27.
Transcranial infrared laser stimulation produces beneficial cognitive and emotional effects in humans. **Barrett DW1, Gonzalez-Lima F.**



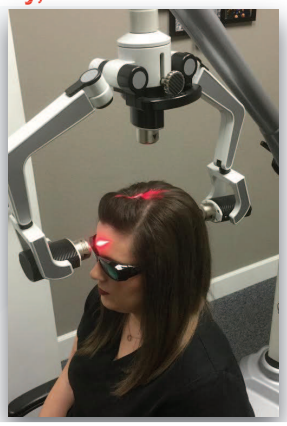
Theta to Alpha (frontal to parietal)6-9-10-12 setting . Mid-Sagittal plane (Mohawk) 10 minutes 2-3x/week minimum 6 visits.

CO-ACTIVATION CONSIDERATIONS: PEMF, MEDITATION, DEEP BREATHING 4:4:8, VEPs, BINAURAL THERAPIES

DEPRESSION AND LLLT

J Affect Disord. **2019 Jan 15**;243:262-273. doi: 10.1016/j.jad.2018.09.048. Epub 2018 Sep 17.
Transcranial and systemic photobiomodulation for major depressive disorder: A systematic review of efficacy, tolerability and biological mechanisms.
Caldieraro MA1, Cassano P2.

Author information Abstract
BACKGROUND:
Photobiomodulation (PBM) with red and near-infrared light (NIR) -also known as Low-Level Light Therapy-is a low risk, inexpensive treatment-based on non-retinal exposure-under study for several neuropsychiatric conditions. The aim of this paper is to discuss the proposed mechanism of action and to perform a systematic review of pre-clinical and clinical studies on PBM for major depressive disorder (MDD).
RESULTS:
Red and NIR light penetrate the skull and modulate brain cortex; an indirect effect of red and NIR light, when delivered non-transcranially, is also postulated. The main proposed mechanism for PBM is the enhancement of mitochondrial metabolism after absorption of NIR energy by the cytochrome C oxidase; however, actions on other pathways relevant to MDD are also reported. Studies on animal models indicate a benefit from PBM that is comparable to antidepressant medications. Clinical studies also indicate a significant antidepressant effect and good tolerability.
LIMITATIONS:
Clinical studies are heterogeneous for population and treatment parameters, and most lack an appropriate control.
CONCLUSIONS:
Preliminary evidence supports the potential of non-retinal PBM as a novel treatment for MDD. Future studies should clarify the ideal stimulation parameters as well as the overall efficacy, effectiveness and safety profile of this treatment.



Theta to Alpha (frontal to parietal)6-9-10-12 setting . Mid-Sagittal plane (Mohawk) 10 minutes 2-3x/week minimum 6 visits.

CO-ACTIVATION CONSIDERATIONS: PEMF, MEDITATION, DEEP BREATHING 4:4:8, VEPs, BINAURAL THERAPIES, NO-NO VOR'S, TMS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION AND LLLT

Randomized Controlled Trial
Brain Stimul
. Sep-Oct 2016;9(5):780-787. doi: 10.1016/j.brs.2016.05.009. Epub 2016 May 24.

Transcranial Laser Stimulation as Neuroenhancement for Attention Bias Modification in Adults with Elevated Depression Symptoms

Seth G Disner 1 , Christopher G Beevers 2 , Francisco Gonzalez-Lima 3

ADVANCED CLINICAL APPLICATION: ERCHONIA LLLT TO THE RIGHT PREFRONTAL CORTEX WITH ABM IE. MEDITATION OF GRATITUDE COMBINED WITH THE "HAPPY" FREQUENCY = 6HZ
ADVANCED "HAPPY" SETTING 6-9-10-12 (THETA-ALPHA-VAGAL) 5 MINUTES RIGHT PREFRONTAL CORTEX

Methods: Fifty-one adult participants with elevated symptoms of depression received ABM before and after laser stimulation and were randomized to one of three conditions: right forehead, left forehead, or sham. Participants repeated LLLT two days later and were assessed for depression symptoms one and two weeks later.

Results: A significant three-way interaction between LLLT condition, ABM response, and time indicated that **right LLLT led to greater symptom improvement among participants whose attention was responsive to ABM (i.e., attention was directed away from negative stimuli). Minimal change in depression was observed in the left and sham LLLT.**

Conclusions: The beneficial effects of ABM on depression symptoms may be enhanced when paired with adjunctive interventions such as right prefrontal LLLT; however, cognitive response to ABM likely moderates the impact of neuroenhancement. The results suggest that larger clinical trials examining the efficacy of using photoneuromodulation to augment cognitive training are warranted.

THE IMPORTANCE OF MITOCHONDRIA

Mitochondria Linked to Major Depression in Older Adults

Conclusion: Our results suggest that depression in late life is associated with GDF-15, a marker of amplified age-related biological changes. GDF-15 is a novel and potentially targetable biological pathway between depression and accelerated aging, including cognitive aging.

"This is the largest study to date providing a link between accelerated mitochondrial aging and depression in older adults…"

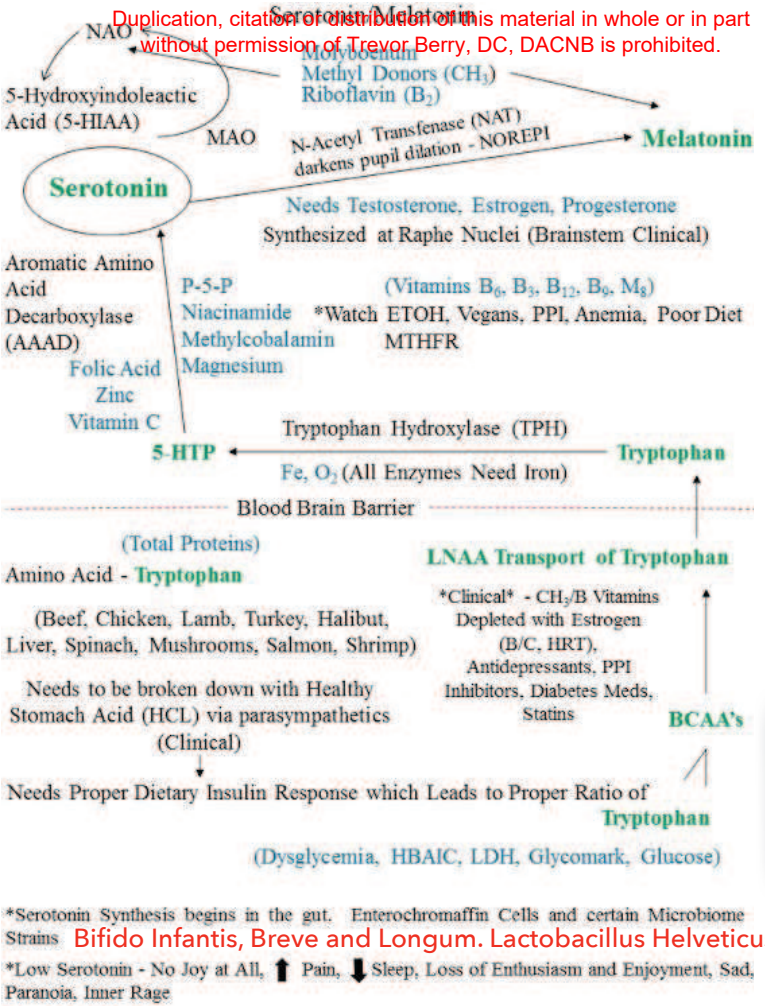
Am J Geriatr Psychiatry
2023 Jan;31(1):1-9. doi: 10.1016/j.jagp.2022.08.003. Epub 2022 Aug 26.

Late-Life Depression is Associated With Increased Levels of GDF-15, a Pro-Aging Mitokine

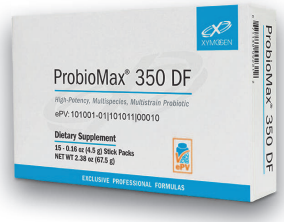
Emma Mastrobattista 1

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



MICRONUTRIENT PANEL!



DEPRESSION, HISTAMINES AND INFLAMMATION

ERCHONIA eCommunity Education

Inflammation-Induced Histamine Impairs the Capacity of Escitalopram to Increase Hippocampal Extracellular Serotonin

Melinda Hersey, Srimal Samaranayake, Shane N. Berger, Navid Tavakoli, Sergio Mena, H. Frederik Nijhout, Michael C. Reed, Janet Best, Randy D. Blakely, Lawrence P. Reagan and Parastoo Hashemi

Journal of Neuroscience **28 July 2021**, 41 (30) 6564-6577;

The researchers discovered it was increased levels of histamine in the brain, generated as part of the animals' inflammatory response, that was causing the reduction in serotonin by, "acting on inhibitory histamine H3 heteroreceptors on serotonin terminals."

In fact, it was discovered that the antidepressant's effect was blunted because it hindered the brain's ability to clear histamine, interfering with the regulation of serotonin levels.

Finally, to confirm this potential mechanism, the researchers treated the animals with histamine-reducing drugs alongside the antidepressant. And this combination of drugs saw serotonin levels rise again in the inflammation-induced mice.

"Our work shines a spotlight on histamine as a potential key player in depression," says lead author on the study, Parastoo Hashemi. "This, and its interactions with the 'feel-good molecule' serotonin, may thus be a crucial new avenue in improving serotonin-based treatments for depression."

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

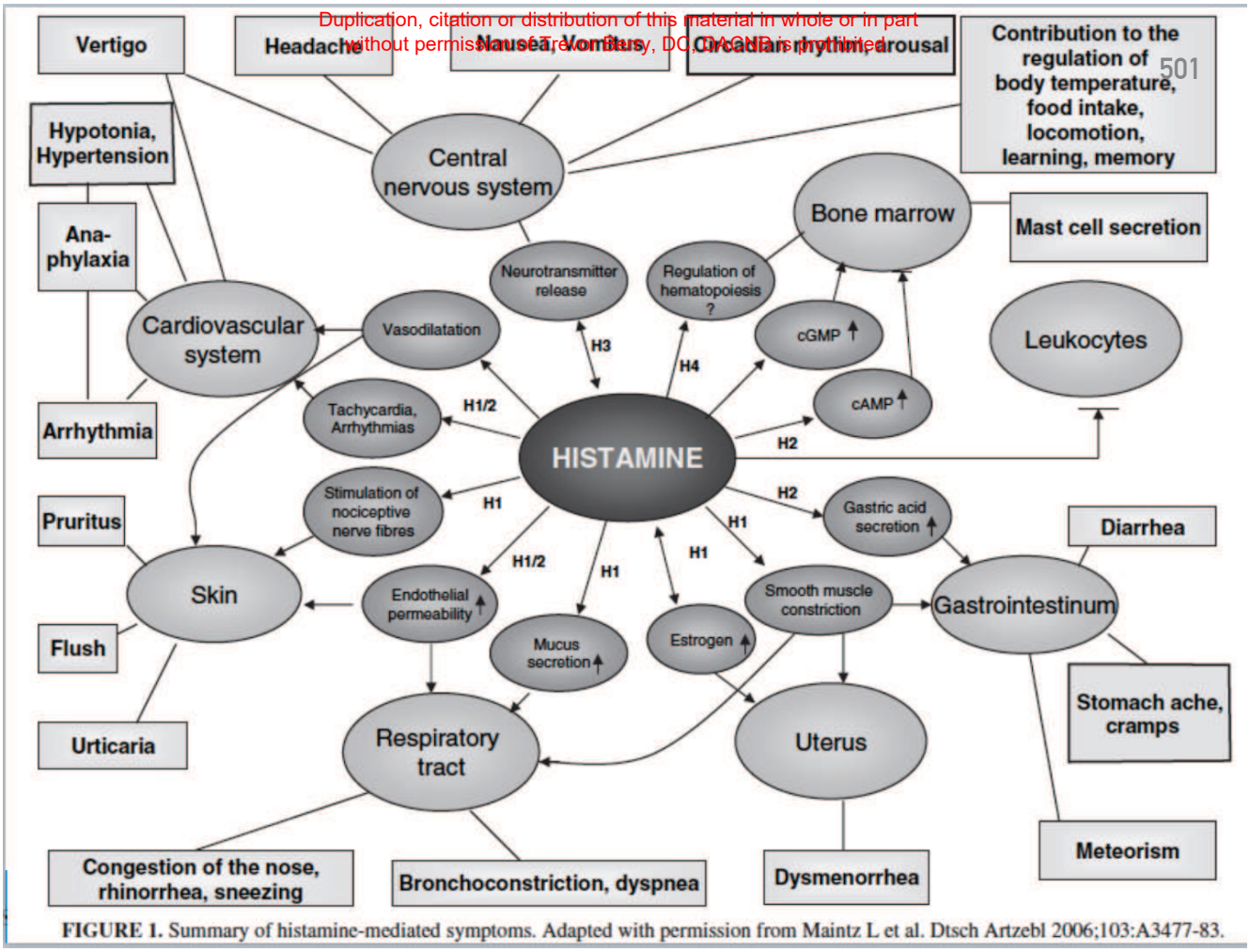
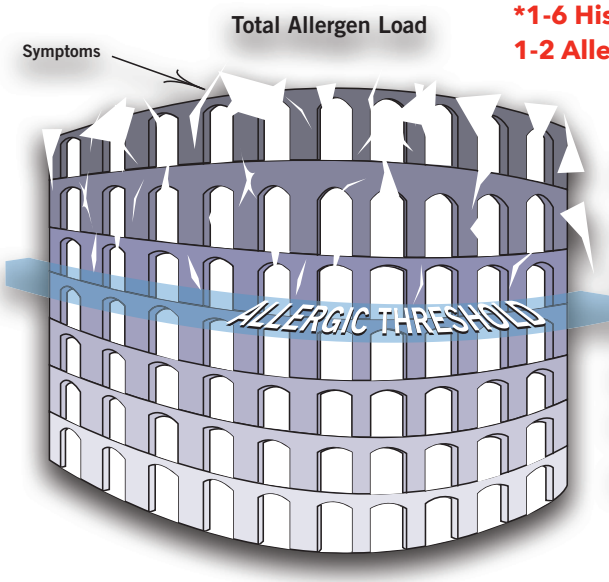


FIGURE 1. Summary of histamine-mediated symptoms. Adapted with permission from Maintz L et al. Dtsch Artzebl 2006;103:A3477-83.

CLINICAL CONSIDERATIONS FOR REDUCING HISTAMINES



***1-6 HistDAO per day (use pre meal)**
1-2 AllerDHQ daily combo

The enzyme diamine oxidase (DAO) degrades histamine by converting it from 2-(4-imidazolyl)-ethylamine to the inactive metabolite imidazole acetaldehyde.

Supports Healthy Degradation of Food-Derived Histamine
Enhances the Presence of Diamine Oxidase in the Digestive Tract

WATCH PATHOGEN CO-MORBIDITIES LIKE MOLD. LOW HISTAMINE DIET!

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

HISTAMINES

Histamine-rich foods are:

- alcohol and other fermented beverages
- fermented foods and dairy products, such as yogurt and sauerkraut
- dried fruits
- eggplant
- spinach
- processed or smoked meats
- shellfish
- aged cheese

There are also a number of foods that trigger histamine release in the body, such as:

- alcohol
- bananas
- tomatoes
- wheat germ
- beans
- papaya
- chocolate
- citrus fruits
- nuts, specifically walnuts, cashews and peanuts
- food dyes and other additives

FOODS THAT PROMOTE DAO PRODUCTION: ORGAN MEATS ESPECIALLY KIDNEY GREEN PEA SPROUTS FATS: OLIVE OIL OMEGA 3'S

Foods that block DAO production include:

- alcohol
- black tea
- mate tea
- green tea
- energy drinks

HISTAMINES IN THE CNS AND INTRACELLULAR ARE BROKEN DOWN BY HISTAMINE N-METHYLTRANSFERASE ENZYME WHICH REQUIRES PROPER METHYLATION OF B12/FOLATE, VITAMINS A&D, MINERALS MOLYBDENUM, COPPER, SELENIUM AND ZINC

LASERS AND ALLERGIES

Results: **We identified increased expression of immunomodulatory factors, such as HGF, TGF-β, and PGE, in T-MSCs-CM treated with LLLT**, compared to T-MSCs-CM without LLLT. **Our animal study demonstrated reduced allergic symptoms and lower expression of total IgE and OVA-specific IgE in the LLLT-treated** T-MSCs-CM group compared to the AR group and T-MSCs-CM alone. Moreover, ***we found that T-MSCs-CM treated with LLLT showed significantly decreased infiltration of eosinophils, neutrophils, and IL-17 cells in the nasal mucosa and reduced IL-4, IL-17, and IFN-γ expression*** in OVA-incubated splenocytes compared to the AR group.

Conclusions: The present study suggests that T-MSCs-CM treated with **LLLT may provide an improved therapeutic effect against nasal allergic inflammation** than T-MSCs-CM

Am J Rhinol Allergy **2022 Mar**;36(2):261-268. doi: 10.1177/19458924211053762. Epub 2021 Nov 5.

Increased Anti-Allergic Effects of Secretome of Low-Level Light Treated Tonsil-Derived Mesenchymal Stem Cells in Allergic Rhinitis Mouse Model

CONSIDER SINUSITIS SETTING: 20-60-160-400 WITH HANDHELDS. 2 MINUTES PFC 5 MINUTES SINUSES, 2 MINUTES VAGAL

In-Su Park 1, Dong-Kyu Kim 2 3, Ji Hye Kim 4 5

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION CLINICAL CONSIDERARTION SUMMARY

1. MOVE! EXERCISE, ESPECIALLY IN SUNLIGHT AND IN A "GROUNDING" ENVIRONMENT. LIGHT THERAPIES
2. MAKE SURE THEY HAVE THE RIGHT BUILDING BLOCKS FOR NEUROCHEMISTRY. EAT A GOOD FIBER/VEGGIE, HIGH IN GOOD FATS AND PROTEINS. RUN LABS FROM BASE CBC/CHEM SCREENS, HORMONE TESTING (DUTCH OR VIBRANT), MICRONUTRIENT PROFILE AND INFLAMMATION SOURCES (FOOD SENSITIVITIES, PATHOGENS, HBAIC)
3. A HEALTHY GUT MICROBIOME (GUT ZOOMER 3.0)
4. ERCHONIA LOW LEVEL LASER THERAPY (RIGHT PREFRONTAL CORTEX 6-9-10-12. STACK WITH 5 MIN MEDITATION OF GRATITUDE/5 MIN CLEARING OF MIND). VAGAL NERVE STIM PROTOCOL (10HZ)
5. TRANSCRANIAL MAGNETIC STIMULATION. PEMF
6. CBD PRODUCTS AND CLASSIC PSYCHEDELICS (MICRO OR MACRO DOSING. WATCH LEGAL MATTERS ON DMT -> DMN THERAPIES - SCHEDULE 1 SUBSTANCES)

AUTISM

© 2024 DR. TREVOR BERRY ALL RIGHTS RESERVED

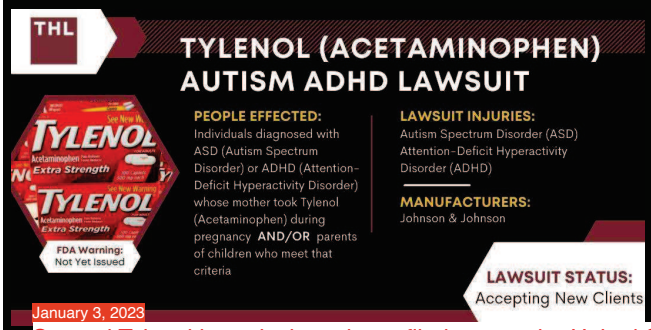
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

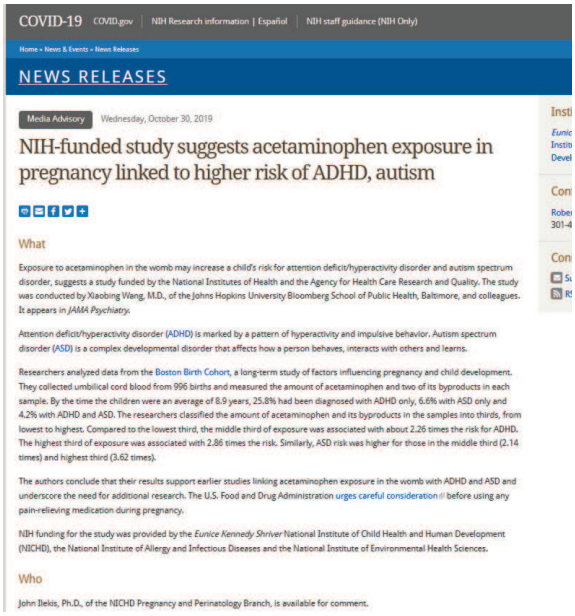
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MITOCHONDRIAL DAMAGE, TYLENOL AND AUTISM

ERCHONIA eCommunity Education

They collected umbilical cord blood from 996 births and measured the amount of acetaminophen and two of its byproducts in each sample. By the time the children were an average of 8.9 years, 25.8% had been diagnosed with ADHD only, 6.6% with ASD only and 4.2% with ADHD and ASD




COVID-19 COVID.gov | NIH Research information | Español | NIH staff guidance (NIH Only)

NEWS RELEASES

Media Advisory Wednesday, October 30, 2019

NIH-funded study suggests acetaminophen exposure in pregnancy linked to higher risk of ADHD, autism

What

Exposure to acetaminophen in the womb may increase a child's risk for attention deficit/hyperactivity disorder and autism spectrum disorder, suggests a study funded by the National Institutes of Health and the Agency for Health Care Research and Quality. The study was conducted by Xiaobing Wang, M.D., of the Johns Hopkins University Bloomberg School of Public Health, Baltimore, and colleagues. It appears in *JAMA Psychiatry*.

Attention deficit/hyperactivity disorder (ADHD) is marked by a pattern of hyperactivity and impulsive behavior. Autism spectrum disorder (ASD) is a complex developmental disorder that affects how a person behaves, interacts with others and learns.

Researchers analyzed data from the Boston Birth Cohort, a long-term study of factors influencing pregnancy and child development. They collected umbilical cord blood from 996 births and measured the amount of acetaminophen and two of its byproducts in each sample. By the time the children were an average of 8.9 years, 25.8% had been diagnosed with ADHD only, 6.6% with ASD only and 4.2% with ADHD and ASD. The researchers classified the amount of acetaminophen and its byproducts in the samples into thirds, from lowest to highest. Compared to the lowest third, the middle third of exposure was associated with about 2.26 times the risk for ADHD. The highest third of exposure was associated with 2.86 times the risk. Similarly, ASD risk was higher for those in the middle third (2.14 times) and highest third (3.62 times).

The authors conclude that their results support earlier studies linking acetaminophen exposure in the womb with ADHD and ASD and underscore the need for additional research. The U.S. Food and Drug Administration urges careful consideration before using any pain-relieving medication during pregnancy.

NIH funding for the study was provided by the *Eunice Kennedy Shriver* National Institute of Child Health and Human Development (NICHD), the National Institute of Allergy and Infectious Diseases and the National Institute of Environmental Health Sciences.

Who

John Ilekis, Ph.D., of the NICHD Pregnancy and Perinatology Branch, is available for comment.

January 3, 2023

Several Tylenol lawsuits have been filed across the United States in Federal Courts alleging that acetaminophen manufacturers and distributors failed to warn consumers of an elevated risk of Autism and ADHD developing in babies of mothers who use the product during pregnancy. These cases have been consolidated into the Tylenol MDL in the U.S. District Court for the Southern District of New York, MDL 3043. If you used Tylenol or generic acetaminophen regularly during pregnancy and your child developed Autism or ADHD, you may have a claim for compensation.

RISKS OF TYLENOL

ERCHONIA eCommunity Education

Acetaminophen use during pregnancy is associated with neurobehavioral problems in 3-year-old offspring

Discussion

The present study found that in utero exposure to acetaminophen predicted sleep and attention problems in offspring at 3 years of age, both of which indicate problems with child self-regulation. Indeed, the current study found the predictive association between acetaminophen use and adverse child behavioral outcomes was mitigated but not eliminated after controlling for stress, suggesting that both stress and acetaminophen use may independently affect child behavioral outcomes. Consistent with the present study, **several previous studies found that acetaminophen use during pregnancy was associated with attention problems in offspring [1–3], but the present study is the first to find predictive association between prenatal acetaminophen use and preschool-age sleep problems.** The fact that neurological dysfunction underlies both sleep and attentional issues in children [40], suggests that an important mechanism of influence may be the impact of acetaminophen on prenatal neurology, which may impact the regulation of attention and sleep in the preschool period. On this note, acetaminophen has been found to be associated with reduced brain connectivity of the amygdala, a part of the brain responsible for self-regulation [41].

Maternal use of acetaminophen during pregnancy and neurobehavioral problems in offspring at 3 years: A prospective cohort study

- Kristin K. Sznajder ,
- Douglas M. Teti,
- Kristen H. Kjerulff. Published:

September 28, 2022 PLOS ONE

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM: THE MITOCHONDRIAL SOLUTION…

In the frontal cortex of Group A, a lower level of ETC complexes was observed in a subset of autism cases, that is, 60% (3/5) for complexes I, II, and V, and 40% (2/5) for complexes III and IV.

THE ETC COMPLEXES WERE LOWER IN THE FRONTAL AND CEREBELLAR REGIONS. 1, 2 AND 5 (VIOLET) WERE PRIMARILY FRONTAL DEFICIENT. COMPLEX 3 (GREEN) WAS DEFICIENT IN FRONTAL, TEMPORAL AND CEREBELLAR REGIONS.

Author Manuscript
Author Manuscript
Author Manuscript
Author Manuscript

HHS Public Access
Author manuscript
J Neurochem. Author manuscript; available in PMC 2016 April 21.

Published in final edited form as:
J Neurochem. 2011 April ; 117(2): 209–220. doi:10.1111/j.1471-4159.2011.07189.x.

Brain region-specific deficit in mitochondrial electron transport chain complexes in children with autism

Abha Chauhan, Feng Gu, Musthafa M. Essa, Jerzy Wegiel, Kulbir Kaur, William Ted Brown, and Ved Chauhan
NYS Institute for Basic Research in Developmental Disabilities, Staten Island, NY, USA

Abstract

Mitochondria play important roles in generation of free radicals, ATP formation, and in apoptosis. We studied the levels of mitochondrial electron transport chain (ETC) complexes, that is, complexes I, II, III, IV, and V, in brain tissue samples from the cerebellum and the frontal, parietal, occipital, and temporal cortices of subjects with autism and age-matched control subjects. The subjects were divided into two groups according to their ages: Group A (children, ages 4–10 years) and Group B (adults, ages 14–39 years). In Group A, we observed significantly lower levels of complexes III and V in the cerebellum ($p < 0.05$), of complex I in the frontal cortex ($p < 0.05$), and of complexes II ($p < 0.01$), III ($p<0.01$), and V ($p < 0.05$) in the temporal cortex of children with autism as compared to age-matched control subjects, while none of the five ETC complexes was affected in the parietal and occipital cortices in subjects with autism. In the cerebellum and temporal cortex, no overlap was observed in the levels of these ETC complexes between subjects with autism and control subjects. In the frontal cortex of Group A, a lower level of ETC complexes was observed in a subset of autism cases, that is, 60% (3/5) for complexes I, II, and V, and 40% (2/5) for complexes III and IV. A striking observation was that the levels of ETC complexes were similar in adult subjects with autism and control subjects (Group B). A significant increase in the levels of lipid hydroperoxides, an oxidative stress marker, was also observed in the cerebellum and temporal cortex in the children with autism. These results suggest that the expression of ETC complexes is decreased in the cerebellum and the frontal and temporal regions of the brain in children with autism, which may lead to abnormal energy metabolism and oxidative stress. The deficits observed in the levels of ETC complexes in children with autism may readjust to normal levels by adulthood.

Keywords

autism; electron transport chain complexes; energy; mitochondria; oxidative stress

Address correspondence and reprint requests to Abha Chauhan, NYS Institute for Basic Research in Developmental Disabilities, 1050 Forest Hill Road, Staten Island, NY 10314, USA. abha.chauhan@opwdd.ny.gov.

Supporting information

Additional supporting information may be found in the online version of this article.

Table S1. Case history of autism and control brain samples.

As a service to our authors and readers, this journal provides supporting information supplied by the authors. Such materials are peer-reviewed and may be re-organized for online delivery, but are not copy-edited or typeset. Technical support issues arising from supporting information (other than missing files) should be addressed to the authors.

© 2022 Erchonia Corporation

AUTISM

Biological Psychology
Volume 175, **November 2022,** 108448

Right anterior theta connectivity predicts autistic social traits in typically developing children

Author links open overlay panel
Aron T.Hill[ab] Jodie Van Der Els[a] Felicity J.Bigelow[a] Jarrad A.G.Lum[a] Peter G.Enticott[a]

Abstract

Growing evidence supports functional network alterations in autism spectrum disorder, however much less is known about the neural mechanisms underlying autistic traits in typically developing children. Using resting-state electroencephalographic (EEG) recordings, we examined whether functional connectivity could predict autistic trait expression in 127 children aged between 4 and 12 years. Regression models showed that **right anterior theta connectivity was a significant predictor of autistic traits (p = 0.013), with increased connectivity in this region associated with greater autistic trait expression. These results corroborate similar recent findings in adults,** extending this observation to a cohort of children spanning early-to-middle childhood. These findings further highlight EEG-derived functional connectivity as a sensitive physiological correlate of autistic trait expression in typically developing children.

Highlights

Right anterior theta connectivity was a significant predictor of autistic traits.

Increased theta (4-8HZ) connectivity was associated with greater autistic trait expression.

These findings highlight EEG functional connectivity as a physiological correlate of autistic trait expression.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Discussion

Summary of the main observations reported in the reviewed literature

-Abnormal lateralization of activity in resting-state and in specific tasks seems to be typical, with a very systematic report of *elevated left-over-right* EEG and MEG functional connectivity ratio in ASD

-Underconnectivity can most reliably be observed in lower frequency bands

PLoS One. 2017; 12(5): e0175870.
Published online 2017 May 3. doi: 10.1371/journal.pone.0175870

PMCID: PMC5414938
PMID: 28467487

Is functional brain connectivity atypical in autism? A systematic review of EEG and MEG studies

Christian O'Reilly,1,2,¤* John D. Lewis,3 and Mayada Elsabbagh1,2

Sci Rep. 2017 Nov 24;7(1):16253. doi: 10.1038/s41598-017-16440-z.

Disrupted Brain Network in Children with Autism Spectrum Disorder.

Zeng K1, Kang J2, Ouyang G3, Li J4, Han J1, Wang Y1, Sokhadze EM5, Casanova MF5, Li X6.
Author information

Abstract

Alterations in brain connectivity have been extensively reported in autism spectrum disorder (ASD), while their effects on the topology of brain network are still unclear. This study investigated whether and how the brain networks in children with ASD were abnormally organized with resting state EEG. Temporal synchronization analysis was first applied to capture the aberrant brain connectivity. Then brain network topology was characterized by three graph analysis methods including the commonly-used weighted and binary graph, as well as minimum spanning tree (MST). **Whole brain connectivity in ASD group was found to be significantly reduced in theta and alpha band compared to typically development children (TD).** Weighted graph found significantly decreased path length together with marginally significantly decreased clustering coefficient in ASD in alpha band, indicating a loss of small-world architecture to a random network. Such abnormal network topology was also demonstrated in the binary graph. In MST analysis, children with ASD showed a significant lower leaf fractions with a decrease trend of tree hierarchy in the alpha band, suggesting a shift towards line-like decentralized organization in ASD. The altered brain network may offer an insight into the underlying pathology of ASD and possibly serve as a biomarker that may aid in diagnosis of ASD.

FN: INFERIOR OLIVARY REGION OF THE PONS. FOCUS ON LEARNED TIMING (PASSIVE OR ACTIVE) AND MAPPING (RETINOTOPIC AND TONOTOPIC)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM

Learning difficulties due to poor connectivity, not specific brain regions

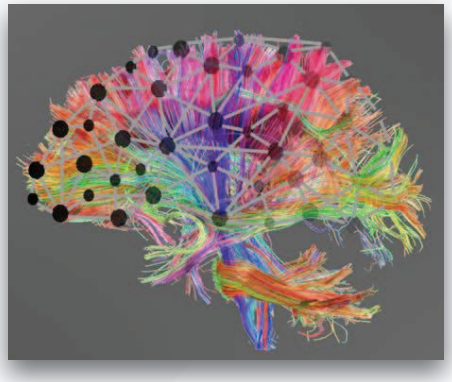
Transdiagnostic Brain Mapping in Developmental Disorders

Roma Siugzdaite

Open Access Published: February 27, 2020. CURRENT BIOLOGY

…the team found that the children's brains were organized around hubs, like an efficient traffic system or social network. Children who had well-connected brain hubs had either very specific cognitive difficulties, such as poor listening skills, or had no cognitive difficulties at all. By contrast, children with poorly connected hubs – like a train station with few or poor connections – had widespread and severe cognitive problems.

513



1 HZ!

AUTISM

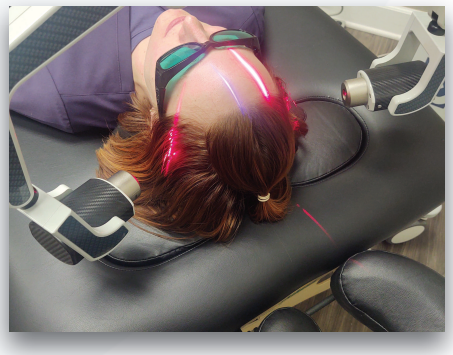
Low-frequency hippocampal–cortical activity drives brain-wide resting-state functional MRI connectivity

Russell W. Chan[a,b,1], Alex T. L. Leong[a,b,1], Leon C. Ho[a,b], Patrick P. Gao[a,b], Eddie C. Wong[a,b], Celia M. Dong[a,b], Xunda Wang[a,b], Jufang He[c],

PNAS Volume 114, number 33

We discover its robust propagation brain-wide at low frequency (1 Hz), which enhances interhemispheric rsfMRI connectivity and cortical and subcortical visual responses. Our findings highlight the important role of slow hippocampal–cortical oscillatory activity in driving brain-wide rsfMRI connectivity and mediating sensory processing.

- **Set up the Erchonia FX laser in the seated coronal (hippocampal) or frontal-bitemporal distribution if supine. PL Touch use a bi-temporal distribution.**
- **ALL diodes are set at 1 Hz. 12 minute session for 7.5 mW devices and 10 minutes for FX.**
- **Co-activate or post treatment activate with I.M., olfaction and music therapies. Consider lateralization compensation (ex. right brain bias vs left music type)**
- **Consider use in Acute Phase TBI, Stress Reduction and Relaxation, Hemispheristic Integration/Lateralization Reduction and much more…**



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

1 HZ

Abstract

Blood-oxygen-level-dependent (BOLD) resting-state functional MRI (rsfMRI) has emerged as a valuable tool to map complex brain-wide functional networks, predict cognitive performance and identify biomarkers for neurological diseases. However, interpreting these findings poses challenges, as the neural basis of rsfMRI connectivity remains poorly understood. The thalamus serves as a relay station and modulates diverse long-range cortical functional integrations, yet few studies directly interrogate its role in brain-wide rsfMRI connectivity. Utilizing a multi-modal approach of rsfMRI, optogenetic stimulation and multi-depth cortical electrophysiology recording, we examined whether and how the somatosensory thalamus contributes to cortical interhemispheric rsfMRI connectivity. **We found that low frequency (1 Hz) optogenetic stimulation of somatosensory-specific ventral posteromedial (VPM) thalamocortical excitatory neurons increased the interhemispheric rsfMRI connectivity in all examined sensory cortices, somatosensory, visual and auditory, and the local intrahemispheric BOLD activity** at infraslow frequency (0.01-0.1 Hz). In parallel, multi-depth local field potential recordings at bilateral primary somatosensory cortices revealed increased interhemispheric correlations of low frequency neural oscillations (i.e., mainly < 10 Hz) at all cortical layers. Meanwhile, pharmacologically inhibiting VPM thalamocortical neurons decreased interhemispheric rsfMRI connectivity and local intrahemispheric infraslow BOLD activity in all sensory cortices. Taken together, **our findings demonstrate that low frequency activities in the thalamo-cortical network contribute to brain-wide rsfMRI connectivity, highlighting the thalamus as a pivotal region that underlies rsfMRI connectivity.**

Neuroimage

2019 Nov 1;201:115985. doi: 10.1016/j.neuroimage.2019.06.063. Epub 2019 Jul 9.

Thalamic low frequency activity facilitates resting-state cortical interhemispheric MRI functional connectivity

Xunda Wang [1], Alex T L Leong [1], Russell W Chan [2], Yilong Liu [1], Ed X Wu [3]

TRANSCRANIAL LOW LEVEL LASER THERAPY AUTISM

Adv Exp Med Biol. 2018 Jun 29. doi: 10.1007/5584_2018_234. [Epub ahead of print]

Effects of Low-Level Laser Therapy in Autism Spectrum Disorder.

Leisman G1,2,3, Machado C4, Machado Y4, Chinchilla-Acosta M4.

Author information

Abstract

The study examined the efficacy of low-level laser therapy, a form of photobiomodulation, for the treatment of irritability associated with autistic spectrum disorder in children and adolescents aged 5-17 years. Twenty-one of the 40 participants received eight 5-min procedures administered to the base of the skull and temporal areas across a 4-week period (test, i.e., active treatment participants). All the participants were evaluated with the Aberrant Behavior Checklist (ABC), with the global scale and five subscales (irritability/agitation, lethargy/social withdrawal, stereotypic behavior, hyperactivity/noncompliance, and inappropriate speech), and the Clinical Global Impressions (CGI) Scale including a severity-of-illness scale (CGI-S) and a global improvement/change scale (CGI-C). The evaluation took place at baseline, week 2 (interim), week 4 (endpoint), and week 8 (post-procedure) of the study. The adjusted mean difference in the baseline to study endpoint change in the ABC irritability subscale score between test and placebo participants was -15.17 in favor of the test procedure group. ANCOVA analysis found this difference to be statistically significant ($F = 99.34$, $p < 0.0001$) compared to the baseline ABC irritability subscale score. **The study found that low-level laser therapy could be an effective tool for reducing irritability and other symptoms and behaviors associated with the autistic spectrum disorder in children and adolescents, with positive changes maintained and augmented over time.**

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM

TWELVE MONTHS FOLLOW-UP COMPARISON BETWEEN THE AUTISTIC CHILDREN vs. INITIAL PLACEBO (TREATED) GROUPS

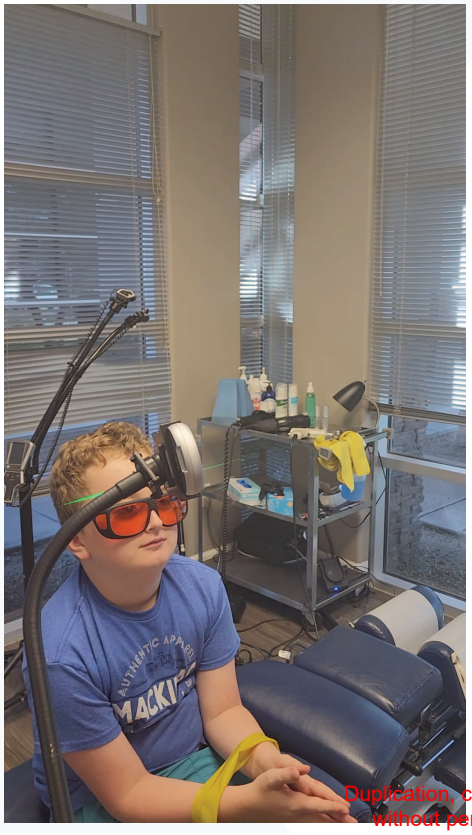
Calixto Machado Mauricio Chinchilla

Article in Internet Journal of Neurology · December 2019

ABSTRACT We recently examined the efficacy of low-level laser therapy (LLLT) to treat autistic children and adolescents up to 12 months after finishing LLLT therapy. I**n this paper, we present the follow up assessment up to 12 months after completion of LLLT procedure, demonstrating that improvement in symptoms continued in the patients initially randomized to the active (test) group, with no change at all for placebo subjects. *After finishing the above-mentioned protocol, we decided to treat the initial placebo group (now, placebo cross-over group) and clinically follow up the cases up to 12-months, as we had done with the active (test) group.*** Hence, in this paper we compare the clinical evaluation providing progressive results for up to 12-months follow-up evaluation (relative to treatment end) for the subject groups enrolled in our protocol.

DISCUSSION **it called the attention the very similar statistical findings for both groups of autistics when the initial placebo was also treated with the same methodology use for the initial active group. Therefore, these findings strongly illustrate that not only does application of the spectrum by Erchonia Laser device effect a sizable, statistically significant and clinically meaningful improvement** in all of the key evaluable behaviors characteristic of autism disorder in children and adolescents, but it continues to affect a progressive and meaningful improvement in symptoms for up to 12 months following completion of the treatment administration protocol, during which time no additional treatment administrations were applied and no changes were made in subjects' non-study medication and/or therapy use to treat autistic symptoms.

MICAH: AUTISM SPECTRUM, DEVELOPMENTAL DELAYS



SINCE STARTING LASER NEUROLOGY, MICAH PASSED HIS PRIVATE SCHOOL ADMITTANCE EXAM AND RECEIVED THE STUDENT OF THE MONTH AWARD. HE IS GETTING STRAIGHT A'S INSTEAD OF BEING IN AN IEP PROGRAM!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.
© 2022 Erchonia Corporation

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM

Mol Autism

2020 Dec 7;11(1):95. doi: 10.1186/s13229-020-00400-y.

Alpha connectivity and inhibitory control in adults with autism spectrum disorder

Veronica Yuk [1 2 3], Benjamin T Dunkley [4 5 6], Evdokia Anagnostou [7 8], Margot J Taylor [4 5 9]

Results: Despite comparable **performance on the Go/No-go task, adults with ASD showed reduced connectivity compared to controls in the alpha band (8-14 Hz) in a network with a main hub in the right inferior frontal gyrus.** Decreased connectivity in this network predicted more self-reported diffi everyday life.

519

STACK WITH GO-NO GO EXERCISES:
SIMON SAYS,CARD GAMES
WISCONSIN CARD SORTING TEST
ANTI-SACCADES

AUTISM

Surg Neurol Int. 2018; 9: 74.
Published online 2018 Apr 9. doi: 10.4103/sni.sni_407_17

PMCID: PMC5909100
PMID: 29721353

Immunoexcitotoxicity as the central mechanism of etiopathology and treatment of autism spectrum disorders: A possible role of fluoride and aluminum

Anna Strunecka, Russell L. Blaylock,1,* Jiri Patocka,2 and Otakar Strunecky3

Our review suggests that most autism spectrum disorder (ASD) risk factors are connected, either directly or indirectly, to immunoexcitotoxicity. Chronic brain inflammation is known to enhance the sensitivity of glutamate receptors and interfere with glutamate removal from the extraneuronal space, where it can trigger excitotoxicity over a prolonged period. **Neuroscience studies have clearly shown that sequential systemic immune stimulation can activate the brain's immune system, microglia, and astrocytes, and that with initial immune stimulation, there occurs CNS microglial priming.** Children are exposed to such sequential immune stimulation via a growing number of environmental excitotoxins, vaccines, and persistent viral infections. **We demonstrate that fluoride and aluminum (Al3+) can exacerbate the pathological problems by worsening excitotoxicity and inflammation.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM

J Trace Elem Med Biol
2018 Mar;46:76-82. doi: 10.1016/j.jtemb.2017.11.012. Epub 2017 Nov 26.

Aluminium (Aluminum) in brain tissue in autism

Matthew Mold 1, Dorcas Umar 2, Andrew King 3, Christopher Exley 1
Affiliations expand

• PMID: 29413113 DOI: 10.1016/j.jtemb.2017.11.012

The aluminium content of brain tissue in autism was consistently high. The mean (standard deviation) aluminium content across all 5 individuals for each lobe were 3.82(5.42), 2.30(2.00), 2.79(4.05) and 3.82(5.17) µg/g dry wt. for the occipital, frontal, temporal and parietal lobes respectively. **These are some of the highest values for aluminium in human brain tissue yet recorded and one has to question why, for example, the aluminium content of the occipital lobe of a 15year old boy would be 8.74 (11.59) µg/g dry wt.?** Aluminium-selective fluorescence microscopy was used to identify aluminium in brain tissue in 10 donors. While aluminium was imaged associated with neurones it appeared to be present intracellularly in microglia-like cells and other inflammatory non-neuronal cells in the meninges, vasculature, grey and white matter. The pre-eminence of intracellular aluminium associated with non-neuronal cells was a standout observation in autism brain tissue and may offer clues as to both the origin of the brain aluminium as well as a putative role in autism spectrum disorder.

MAGNESIUM MALATE IS AN EXCELLENT GENTLE ALUMINUM CHELATION AGENT. STACK WITH MCT FOR CROSSING BBB.

AUTISM

Child Care Health Dev
. 2022 Jan;48(1):33-44. doi: 10.1111/cch.12909. Epub 2021 Aug 31.

Medicinal cannabis in children and adolescents with autism spectrum disorder: A scoping review

Sarah Fletcher 1, Colleen Pawliuk 2, Angie Ip 2 3, Linda Huh 2, S Rod Rassekh 2, Tim F Oberlander 2 3, Harold Siden 2 4

Results: We identified eight completed and five ongoing studies meeting the inclusion criteria. All studies reported substantial behaviour and symptom improvement on medicinal cannabis, with 61% to 93% of subjects showing benefit.

Prog Neuropsychopharmacol Biol Psychiatry
2022 Mar 8;113:110476. doi: 10.1016/j.pnpbp.2021.110476. Epub 2021 Nov 15.

Antioxidant interventions in autism spectrum disorders: A meta-analysis

Yiying Liu 1, Zimeng Yang 1, Yang Du 2, Sha Shi 3, Yong Cheng 4

Treatment with NAC antioxidants showed a good trend of improvement in irritability in the ABC and symptoms of hyperactivity.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Children with autism show improvement after being treated with cannabidiol-rich medicinal cannabis

by **Vladimir Hedrih** December 7, 2022

A study of children with autism spectrum disorders in Israel reported **significant improvements in their social communication abilities after six months of treatment with cannabidiol-rich cannabis oil. Additionally, parents reported a reduction in restrictive and repetitive behaviors of children. Children's cognitive scores were not changed**. The study was published in *Translational Psychiatry*.

- Article
- Open Access
- **Published: 09 September 2022**

Children and adolescents with ASD treated with CBD-rich cannabis exhibit significant improvements particularly in social symptoms: an open label study

- Micha Hacohen, Orit E. Stolar, Matitiahu Berkovitch, Odelia Elkana, Elkana Kohn, Ariela Hazan, Eli Heyman, Yael Sobol, Danel Waissengreen, Eynat Gal & Ilan Dinstein

Translational Psychiatry

volume
12, Article number: 375 (2022)

RESEARCH CONFIRMS THE GUT-BRAIN CONNECTION IN AUTISM

Source: RMIT University

People with autism often suffer from gut problems, but nobody has known why. Researchers have now discovered the same gene mutations – found both in the brain and the gut – could be the cause.

The discovery confirms a gut-brain nervous system link in autism, opening a new direction in the search for potential treatments that could ease behavioural issues associated with autism by targeting the gut.

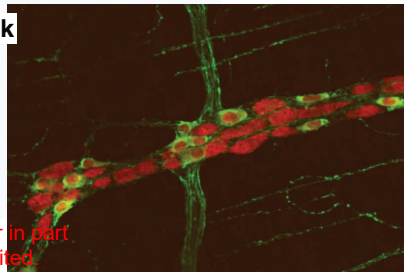
The study reveals a gene mutation that affects neuron communication in the brain, which was first identified as a cause of autism, also causes dysfunction in the gut.

They team found this mutation affects:

- **gut contractions**
- **the number of neurons in the small intestine**
- **the speed that food moves through the small intestine**
- **responses to a critical neurotransmitter important in autism (well known in the brain but not previously identified to play any major role in the gut)**

Collaborator Associate Professor Ashley Franks (La Trobe University) also found significant differences in the gut microbes of mice with the mutation and those without it, even though both groups were kept in identical environments.

Hill-Yardin, an ARC Future Fellow and Vice-Chancellor's Senior Research Fellow in the School of Health and Biomedical Sciences at RMIT, said the work identifies a new a target for the development of therapies specifically designed to work on neurotransmitters in the gut.
"Another promising path for future research is investigating how gene mutations in the nervous system relate with microbes in the gut.



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RESEARCH CONFIRMS THE GUT-BRAIN CONNECTION IN AUTISM

Int J Mol Sci. 2020 Jun; 21(11): 4159.
Published online 2020 Jun 10. doi: 10.3390/ijms21114159

PMCID: PMC7312735
PMID: 32532137

The Promising Role of Probiotics in Managing the Altered Gut in Autism Spectrum Disorders

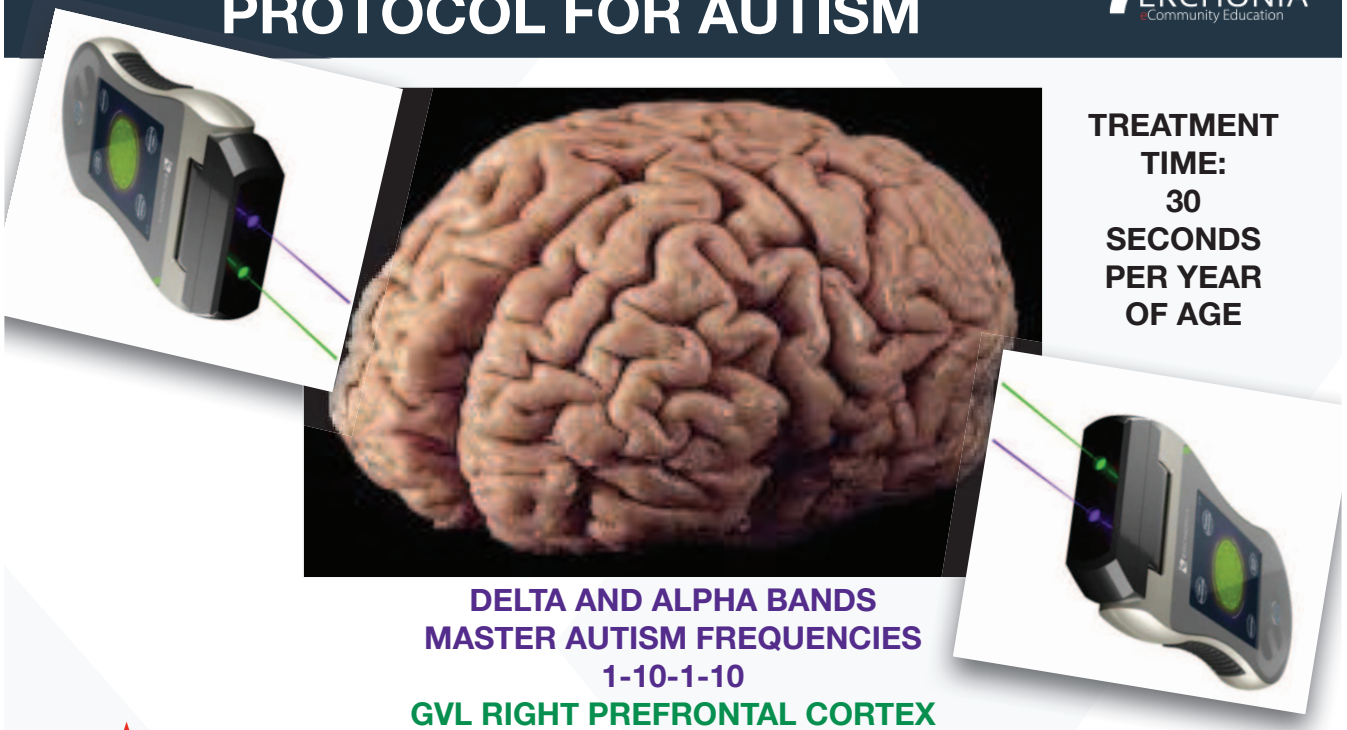
Basma Abdellatif, Clare McVeigh, Ghizlane Bendriss, and Ali Chaari*

The multi-strain probiotics supplemented in the five studies were all different from each other, however combinations included ***Lactobacillus* and *Bifidobacterium* species** (Table S3). Moreover, the types of probiotic supplements administered in the trials were different in terms of form, dose, bacterial strains, and combinations of bacterial strains. All these studies showed beneficial effects, including significant improvements in GI symptoms. West and colleagues [173] administered a cocktail of ***Lactobacillus delbrueckii, L. acidophilus, Lactobacillus casei, B.longum*, and *Bifidobacteria bifidum* to 33 children with ASD aged between 3–16 years for 21 days.**

Nevertheless, it is important to highlight that other dietary components are important for the gut microbiota homeostasis, such as **vitamins, short chain fatty acids, polyphenols, and a growing number of studies are currently trying dietary interventions for ASD** [179,184]. Studies described nutrition as having a significant impact on the gut microbiome: **chemicals, pesticides on food, artificial sweeteners or preservatives have been shown to disrupt the biodiversity and function of the gut [185,186]. The gluten free/casein free diet, the Atkins diet, the DASH diet, the ketogenic diet, specific carbohydrate diet, the Paleo diet are the main diets that have been studied so far for ASD** and showed various levels of efficacy [186,187]. All these diets have a common point of excluding processed food and including more dietary fibers that will act as prebiotics and fermented food to promote the shift of the dysbiosis toward eubiosis.

ALSO WATCH CANDIDA OVERGROWTH IN ASD

THE ADVANCED LASER NEUROLOGY PROTOCOL FOR AUTISM



TREATMENT TIME: 30 SECONDS PER YEAR OF AGE

DELTA AND ALPHA BANDS
MASTER AUTISM FREQUENCIES
1-10-1-10
GVL RIGHT PREFRONTAL CORTEX
WITH TEMPORAL LOBE OVERLAP
THEN LEFT CEREBELLUM/TEMPORAL LOBE
STACKING WITH MODALITIES
FINISH WITH XL ROOFTOP 6015F AVAILABLE PASSIVE REST

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.
© 2022 Erchonia Corporation

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AUTISM TREATMENT CONSIDERATIONS SUMMARY

- SLOW COMPLEX LEFT UPPER EXTREMITY AND LOWER EXTREMITY MOVEMENTS
- INTERACTIVE METRONOME IF AVAILABLE
- CROSS BODY (INTERACTIVE) METRONOME
- BASIC MARCHING CROSS-CRAWL
- MIRROR MOVEMENTS (HAVE THEM BIAS TOWARDS THEIR LEFT HAND/FOOT IF RIGHT HEMISPHERISTIC DEFICIT)
- DEEP PRESSURE SENSORY OR PERCUSSOR VIBRATION
- SACCADES WITH LEFTWARD BIAS (RIGHT FRONTAL LOBE)...ANTI-SACCADES
- GO-NO GO EXERCISES
- PRIMITIVE REFLEX INTEGRATION (CONSIDER DR. MELILLO'S CHILDHOOD NEUROBEHAVIORAL DISORDERS CERTIFICATION COURSE) SEE NEXT SLIDE

-ALWAYS STACK YOUR THERAPIES WITH YOUR ERCHONIA LLLT BRAIN APPLICATIONS

COMPLETE THE ADVANCED AUTISM PROTOCOL AS DESCRIBED ABOVE.

CONSIDER THE NEW GVL FOR OPTIMAL OUTCOMES.

12 VISIT TREATMENT TRIAL RECOMMENDED WITH A MINIMUM OF 6 VISITS FOR BEHAVIORAL MODIFICATION ASSESSMENT

NEURODEVELOPMENTAL NUTRITIONAL SUPPORT

XYMOGEN: OMEGAMONOPURE 1300 2 DAILY (600 smaller child/won't swallow larger).
HEMP MONOPURE OR CBD PRODUCT BID

OPTIMAG NEURO 1/2 SCOOP DAILY

S-ACETYL GLUTATHIONE/NAC 1 DAILY OR LIQUID (APEX TRIZOMAL - S-ACETYL + REDUCED GLUTATHIONE GSH + NAC)

COGNIQUIL 1 DAILY FOR ATTENTION/FOCUS

RELAXMAX OR GABACORE FOR ANTI-ANXIETY

MELATONIN CR FOR SLEEP

A GOOD MULTI INCLUDING A, C, ZINC AND METHYLATED B'S

LIQUID VIT D3 1000 MG DAILY

PROBIOMAX PLUS DF 1 DAILY OR 1/2 PACK PROBIOMAX 350 WEEKLY.

CONSIDER FULL GUT REPAIR AND FOOD TOLERANCE TESTING. DIET ELIMINATION GF/DF. LOW SUGAR/HIGHER FAT. AIP/KETO/PALEO BASED DIET

APEX LIQUIDS: TURMERO, RESVERO AND OMEGA CO3. CONSIDER REPAIR AND CLEAR

*WATCH SULFUR METABOLISM LIKE GLUTATHIONE SUPPORT IN PATHOGEN OVERGROWTH LIKE YEAST OR FUNGUS (SEE ORGANIC ACIDS TEST)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AGE AND LASER DOSE/TIMES

*A GOOD RULE OF THUMB WHEN USING ERCHONIA LASERS ON CHILDREN TRANSCRANIALLY:

RED/XLR8 TREAT 1 MINUTE PER YEAR OF AGE UP TO 10 YEARS/MINUTES

VIOLET (EVRL/GVL) TREAT 30 SECONDS PER YEAR OF AGE (EX. 1 YEAR = 30 SECONDS, 2 YEAR OLD = 1 MINUTE ETC)

ALZHEIMER'S REVERSAL DALE BREDESEN (RECODE PROTOCOL IN BACK OF NOTES)

KEY CONCEPTS OF DALE BREDESEN'S NEW BOOK "THE AGELESS BRAIN"

3 MAJOR CULPRITS THAT ACCELERATE COGNITIVE DECLINE:
1. **ENERGETICS: BLOOD FLOW/O2 DELIVERY AND MITOCHONDRIAL FUNCTION**
2. **INFLAMMATION: CHRONIC INFLAMMATION DUE TO POOR DIET, STRESS, INFECTIONS AND ENVIRONMENTAL TOXINS**
3. **TOXICITY: HEAVY METALS, PESTICIDES, AIR POLLUTION, MOLD AND MICROPLASTICS**

CURRENT KEY BIOMARKERS THAT CAN HELP DETECT RISK YEARS BEFORE THE ONSET OF SYMPTOMS:
1. **P-TAU 217**
2. **GFAP (GLIAL FIBRILLARY ACIDIC PROTEIN) INFLAMED SUPPORTING CELLS AND BBB DISRUPTION**
3. **NFL (NEUROFILAMENT LIGHT CHAIN) GENERAL NEURONAL DAMAGE MARKER FROM ANY CAUSE**

ALTHOUGH GENETICS PLAYS A MAJOR ROLE IN RISK FACTORS, IT DOES NOT HAVE THE FINAL WORD. THE MOST COMMON GENE SNP IMPLICATED IN ALZHEIMER'S IS APOE4, HOWEVER 75% OF THE POPULATION HAVE ZERO COPIES OF THE SNP AND A 9% LIFETIME RISK OF AD. ONLY 2% HAVE TWO COPIES (APOE4/4) BUT THEIR LIFETIME RISK GOES UP TO 90%!

THE KEY LIFESTYLE POINTS DISCUSSED IN HIS BOOK ARE:
DIET, EXERCISE, SLEEP, STRESS MANAGEMENT, BRAIN TRAINING, DETOXIFICATION, SUPPLEMENTS. HE ALSO CAUTIONS FOR COMMON CHRONIC INFECTIONS LIKE LYME, MOLD AND VIRUSES

HE INCLUDES LIGHT THERAPIES, TMS AND MICROCURRENT AS POTENTIAL ADJUNCT THERAPIES

ONE FINAL KEY CONCEPT DISCUSSED IS TO ALWAYS CHALLENGE YOURSELF AND GET OUT OF YOUR COMFORT ZONE. LEARNING AND INSTRUMENT OR LANGUAGE, DANCE LESSONS ETC TO KEEP THE BRAIN STIMULATED AND LEARNING!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ALZHEIMER'S AND OMEGA 3 INTAKE

Omega 3 Intake Linked to Stable Memory Function in Alzheimer's Patients

Featured Neurology Neuroscience · September 7, 2021

"We can see that the memory function of the patients in the group that had taken omega-3 is stable, whereas the patients in the control group have deteriorated. That's what the memory tests show," says Yvonne Freund-Levi.

TREATMENT GROUP RECEIVED 2.3 GRAMS OMEGA-3 FA DAILY

Effects of Peroral Omega-3 Fatty Acid Supplementation on Cerebrospinal Fluid Biomarkers in Patients with Alzheimer's Disease: A Randomized Controlled Trial—The OmegAD Study

A
A

USE THE VIBRANT MICRONUTRIENT PANELS OR BRAIN SPAN FOR ACCURATE INTRACELLULAR ESSENTIAL FATTY ACID MARKERS AND RATIOS

DHA AND ALZHEIMER'S

Abstract

Docosahexaenoic acid (DHA) might help prevent Alzheimer's disease (AD). Red blood cell (RBC) status of DHA is an objective measure of long-term dietary DHA intake. In this prospective observational study conducted within the Framingham Offspring Cohort (1490 dementia-free participants aged ≥65 years old), we examined the association of RBC DHA with incident AD, testing for an interaction with *APOE-ε4* carriership. During the follow-up (median, 7.2 years), 131 cases of AD were documented. In fully adjusted models, risk for incident AD in the highest RBC DHA quintile (Q5) was 49% lower compared with the lowest quintile (Q1) (Hazard ratio [HR]: 0.51, 95% confidence interval [CI]: 0.27, 0.96). **An increase in RBC DHA from Q1 to Q5 was predicted to provide an estimated 4.7 additional years of life free of AD.** We observed an interaction DHA × *APOE-ε4* carriership for AD. Borderline statistical significance for a lower risk of AD was observed per standard deviation increase in RBC DHA (HR: 0.71, 95% CI: 0.51, 1.00, $p = 0.053$) in *APOE-ε4* carriers, but not in non-carriers (HR: 0.85, 95% CI: 0.65, 1.11, $p = 0.240$). These findings add to the increasing body of literature suggesting a robust association worth exploring dietary DHA as one strategy to prevent or delay AD.

SUMMARY:

-THOSE WITH THE HIGHEST RBC DHA HAD 49% LESS RISK OF ALZHEIMER'S THAN THOSE WITH THE LOWEST DHA LEVELS

-THE HIGHEST DHA GROUP HAD 4.7 ADDITIONAL YEARS OF LIFE FREE OF ALZHEIMER'S SYMPTOMS

Nutrients

2022 Jun 9;14(12):2408. doi: 10.3390/nu14122408.

Red Blood Cell DHA Is Inversely Associated with Risk of Incident Alzheimer's Disease and All-Cause Dementia: Framingham Offspring Study

Aleix Sala-Vila [1,2]

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PROTECT YOUR BRAIN!

ω-3 PUFA for Secondary Prevention of White Matter Lesions and Neuronal Integrity Breakdown in Older Adults

A Randomized Clinical Trial

• Lynne H. Shinto, ND, MPH[1];

JAMA Network
August 1, 2024
2024;7;(8):e2426872. doi:10.1001/jamanetworkopen.2024.26872

- Research at Massachusetts General Hospital recently showed **evidence that taking Omega-3 fish oil supplements may help seniors fight Alzheimer's disease**
- The trial used a study design that combined blood and MRI biomarkers to monitor brain health
- Omega-3 supplementation was well tolerated but did not significantly impact white matter lesion progression in all participants
- **Seniors carrying the APOE4 gene, which is linked to Alzheimer's disease risk, experienced a significant decrease in brain cell breakdown after taking fish oil supplements.**

PROTECT YOUR BRAIN!

A startling omega-3 deficiency may explain women's Alzheimer's risk

Date: August 22, 2025

Senior author Dr Cristina Legido-Quigley, from King's College London, said: "Women are disproportionately impacted by Alzheimer's Disease and are more often diagnosed with the disease than men after the age of 80. **One of the most surprising things we saw when looking at the different sexes was that there was no difference in these lipids in healthy and cognitively impaired men, but for women this picture was completely different. The study reveals that Alzheimer's lipid biology is different between the sexes, opening new avenues for research."**

The scientists took plasma samples from 841 participants who had Alzheimer's Disease, mild cognitive impairment and cognitively health controls and and were measured for brain inflammation and damage.

They used mass spectrometry to analyze the 700 individual lipids in the blood. Lipids are a group of many molecules. Saturated lipids are generally considered as 'unhealthy' or 'bad' lipids, while unsaturated lipid, which sometime contains omega fatty acids, are generally considered 'healthy'.

Scientists saw a steep increase in lipids with saturation - the 'unhealthy lipids' - in women with Alzheimer's compared to the healthy group. The lipids with attached omega fatty acids were the most decreased in the Alzheimer's group.

Now, the scientists say there is a statistical indication that there is a causal link between Alzheimer's Disease and fatty acids. But a clinical trial is necessary to confirm the link.

Asger Wretlind, Jin Xu, Wenqiang Chen, Latha Velayudhan, Nicholas J. Ashton, Henrik Zetterberg, Petroula Proitsi, Cristina Legido-Quigley. **Lipid profiling reveals unsaturated lipid reduction in women with Alzheimer\'s disease**. *Alzheimer\'s*. 2025; 21 (8) DOI: 10.1002/alz.70513

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

PROTECT YOUR BRAIN!

An Alzheimer's researcher shares 1 daily supplement and 1 mantra he recommends to everyone for brain health

"Even if you're taking supplements, nothing substitutes for lifestyle," **Tanzi,** the director of Genetics and Aging Research at Massachusetts General Hospital and a professor of child neurology at Harvard Medical School, told Business Insider.

Tanzi broke down what his SHIELD mantra stands for:

- **S**leep for 7-8 hours a night to clear harmful protein buildup, such as amyloid plaques. "The faster you make amyloid, the sooner you get Alzheimer's," Tanzi said.
- **H**andle stress. "Chronic stress causes chronic inflammation," he said. Being more mindful can reduce the chances of cognitive decline.
- **I**nteract with others. Loneliness negatively impacts the brain — and increases the risk of Alzheimer's "twofold," Tanzi said.
- **E**xercise to encourage nerve cell growth and dissolve amyloid buildup.
- **L**earn new things. "The more synapses you make in life, it's like money in the bank," Tanzi said. Keeping busy and having hobbies are also linked to greater longevity.
- **D**iet, such as the Mediterranean diet. Tanzi recommended focusing on plants to balance the gut microbiome. While Tanzi said more research is needed to understand why a healthy gut is linked to the removal of amyloid, he said a high-fiber diet rich in fruits, vegetables, nuts, seeds, and whole grains definitely helps.

Mounting evidence suggests those effects confer anti-aging benefits. For example, **in a 2025 study of 777 participants, those who took a gram of omega-3 a day experienced slower brain aging. The effect was more pronounced for people who also exercised and took vitamin D supplements.**

535

APPLIED FUNCTIONAL NEUROLOGY

TAINTED FISH

ERCHONIA eCommunity Education

Eating one wild fish same as month of drinking tainted water: study

Eating one freshwater fish caught in a river or lake in the United States is the equivalent of drinking a month's worth of water contaminated with toxic "forever chemicals", new research said on Tuesday.

The invisible **chemicals called PFAS** were first developed in the 1940s to resist water and heat, and are now used in items such as non-stick pans, textiles, fire suppression foams and food packaging.

Nadia Barbo et al, Locally caught freshwater fish across the United States are likely a significant source of exposure to PFOS and other perfluorinated compounds, *Environmental Research* (2022). DOI: 10.1016/j.envres.2022.115165

"According to the UN, there are over 50 trillion microplastics in the ocean."

Scientists from New Zealand and Australia detected **microplastics in 75% of commercial fish species.** Both bottom-dwellers and pelagic fish were equally affected, suggesting the plastics are suspended throughout the water column. The **researchers discovered an average of 2.5 individual plastic particles per fish.**

Isabella K. Clere et al, Quantification and characterization of microplastics in commercial fish from southern New Zealand, Marine Pollution Bulletin, Volume 184, 2022, 114121, https://doi.org/10.1016/j.marpolbul.2022.114121.

Worldwide contamination of fish with microplastics: A brief global overview, Ines D. Sarmiento

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FISH SOURCE

ERCHONIA eCommunity Education

SEATOPIA

Certified-clean, guilt-free, sushi-grade seafood.

Mercury safe, antibiotic free, & truly sustainable.

SEATOPIA.COM

537

OMEGA 3 SUPPORT
MAXSIMIL TECHNOLOGY

ERCHONIA eCommunity Education

Omega MonoPure® 1300 EC features natural enzymatically enhanced MaxSimil® monoglyceride fish oil that has a **three times greater EPA/DHA absorption rate than an equivalent dose of ethyl ester fish oil. This IFOS five-star certified fish oil** outperforms other fish oil supplements through MaxSimil patented lipid absorption enhancement technology (PLATform).*

Unique structure: One fatty acid attached to a glycerol backbone provides two polar ends that attract water and a non-polar tail end (R1) that attracts fat, thus enabling self-emulsification of the Omega MonoPure formulas.

4 PER DAY (2 BID) UNTIL OMEGA-3 INDEX >10%

OMEGA 3'S MODULATE AND INHIBIT NF-kB
2nd FOUNDATIONAL PRODUCT

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

EPA VS DHA BIAS

Condition-specific recommendations from the data presented.			
Condition	**EPA-Rich**	**EPA/DHA**	**DHA-Rich**
General Health		XX	
Improving w3:w6 ratio		XX	
Low-Risk CVD prevention		XX	X
High CVD Risk/Atherosclerosis		X	XX
Inflammatory Diseases		XX	
Depression	XX	X	
Macular Degeneration			XX
Alzheimer- Dementia		X	XX
Maternal- Childhood Devel.		X	XX

CONSIDER INTRACELLULAR RBC TESTING FOR OMEGA FATTY ACIDS BRAIN SPAN, VIBRANT MICRONUTRIENT OR NUTRIPRO TESTING

3 SUPPLEMENTS FOR BRAIN
1. FATTY ACIDS/MEMBRANE

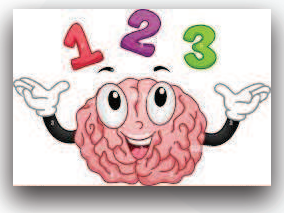
GOOD: MINIMUM 2.5 GRAMS OF AN IFOS 5 CERTIFIED OMEGA 3 FATTY ACID

BETTER: STACK OMEGA WITH PHOSPHOLIPID SUPPORT

BEST: STACK OMEGA 3 AND PHOSPHOLIPID SUPPORT WITH SPM'S

ORTHOMOLECULAR BIOPC PRO

MOST PHOSPHOLIPIDS ARE HARSH EXTRACTED SOY BASED PHOSPHATIDYLCHOLINE. BIOPC USES GENTLE COLD PRESSED SUNFLOWER EXTRACTION AND IS BROAD SPECTRUM (CHOLINE, SERINE ETC)



XYMOGEN OMEGAPURE PRM

THE HIGHEST LEVEL OF PRM'S (400 MCG) PLUS 500 MG OMEGA 3 FATTY ACIDS. CONSIDER 1 MONOPURE A.M. PLUS 1 OMEGAPURE PRM P.M. ESPECIALLY IN CIRS CONDITIONS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

*CLINICAL PEARL

ERCHONIA eCommunity Education

FOR EVEN BETTER FATTY ACID ABSORPTION, STACK OMEGA FATTY ACID SUPPLEMENTS WITH DIGESTIVE ENZYMES THAT PROMOTE FAT EMULSIFICATION AND DIGESTION

LIPASE

OX BILE

	Amount Per Serving
Betaine HCl	300 mg
Ox Bile (45% cholic acid)	150 mg
Pancreatin 10x (from porcine) Protease Amylase Trypsin	100 mg 25,000 USP 25,000 USP 25,000 USP
Gentian 4:1 Extract (*Gentiana lutea*)(root)	20 mg

541

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

NEXT 3 SUPPLEMENTS TO ADDRESS BRAIN INFLAMMATION:

1. Magnesium L-Threonate

- A highly bioavailable form of magnesium that crosses the blood-brain barrier
- Enhances synaptic density and reduces neuroinflammation in animal models
- Reduces microglial activation and NMDA overactivity
- Improves cognitive function, sleep quality, and stress resilience
- Synergistic with curcumin and DHA in reducing neuroinflammation

Typical Dose: 1,000–2,000 mg/day (as Magtein®, yielding ~144 mg elemental magnesium)
Reference: Slutsky, I., et al. (2010). Enhancement of learning and memory by elevating brain magnesium. Neuron.

2. Alpha-Lipoic Acid (ALA)

- A powerful antioxidant and mitochondrial cofactor
- Crosses the blood-brain barrier to reduce oxidative stress and glial activation
- Reduces neuroinflammation via NF-κB inhibition
- Regenerates other antioxidants (e.g., vitamin C, glutathione)
- Clinically studied in diabetic neuropathy and cognitive decline

Typical Dose: 300–600 mg 1–2x/day (R-ALA form preferred for higher bioactivity)
Reference: Packer, L., et al. (1997). Alpha-lipoic acid as a biological antioxidant. Free Radical Biology & Medicine.

3. NAC (N-ACETYLCYSTEINE)

Mechanisms of Action:

1. Precursor to glutathione (GSH), the brain's master antioxidant
2. Reduces oxidative stress and modulates mitochondrial function
3. Inhibits microglial activation and reduces neuroinflammatory cytokines
4. Regulates glutamate by acting on the cystine-glutamate antiporter (mGluR modulation), helping balance excitotoxicity
5. Modulates the blood-brain barrier and preserves endothelial integrity

TGF-B NEEDS CYSTEINE!

Brain Conditions Benefited by NAC:

- Traumatic brain injury (TBI)
- Neurodegenerative diseases (Alzheimer's, Parkinson's, ALS)
- OCD, depression, and schizophrenia (via glutamate modulation)
- COVID- and post-viral-induced neuroinflammation
- Substance withdrawal and compulsive behavior

STACK NAC WITH VIT D 5K+ AND A FOR TREG!

Clinical Dosing:

- **600–1,200 mg once or twice daily**
- Best taken on an empty stomach or away from protein-rich meals

1. Dean, O. M., et al. (2011).
"N-acetylcysteine in psychiatry: current therapeutic evidence and potential mechanisms of action."
Psychiatry and Clinical Neurosciences, 65(3), 210–219.
→ Reviews NAC's role in glutathione synthesis and glutamate regulation for mood and cognition.

542

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

APPLIED FUNCTIONAL NEUROLOGY

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ANOTHER REASON TO SUPPLEMENT…

THERE IS A PHILOSOPHICAL DEBATE THAT SUPPLEMENTS SHOULD NOT TAKE THE PLACE OF A HEALTHY DIET AND LIFESTYLE. HOWEVER, WITH MINERAL DEPLETION OF OUR SOIL, TOXICITY OF OUR OCEANS AND OTHER FACTORS DEGRADING OUR FOOD QUALITY, ONE COULD ARGUE SUPPLEMENTS HAVE BECOME A NECESSITY. HERE IS JUST ONE EXAMPLE:

Summary Table: Mineral Depletion in Foods

Mineral	Decline Range (%)	Affected Food Categories
Magnesium	10–35%	Vegetables, Fruits
Calcium	16–46%	Vegetables, Fruits
Iron	15–60%	Vegetables, Fruits
Copper	20–81%	Vegetables, Fruits
Zinc	27–59%	Fruits
Potassium	6–20%	Vegetables, Fruits
Sodium	29–52%	Vegetables, Fruits
Phosphorus	6–11%	Vegetables
Vitamin C	15–30%	Vegetables
Riboflavin	38%	Vegetables

Foods. 2024 Mar 14;13(6):877. doi: 10.3390/foods13060877

An Alarming Decline in the Nutritional Quality of Foods: The Biggest Challenge for Future Generations' Health

Raju Lal Bhardwaj [1]

APPLIED FUNCTIONAL NEUROLOGY

A GOOD MULTI GOES A LONG WAY!

	Amount Per	%DV
Vitamin A (75% as natural beta-carotene and 25% as retinyl palmitate)	400 mcg	44%
Vitamin C (as sodium ascorbate, potassium ascorbate, zinc ascorbate, and calcium	45 mg	50%
Vitamin D3 (cholecalciferol)	12.5 mcg (500 IU)	63%
Vitamin E (as d-alpha tocopheryl succinate)	31 mg	207%
Thiamin (as thiamine mononitrate)	3.5 mg	292%
Riboflavin (as riboflavin 5'-phosphate sodium)	3.5 mg	269%
Niacin (as niacinamide and niacin)	11.5 mg	72%
Vitamin B6 (as pyridoxal 5'-phosphate)	3.5 mg	206%
Folate (as (6S)-5-methyltetrahydrofolic acid, glucosamine salts[1] and as calcium	289 mcg DFE	72%
Vitamin B12 (as methylcobalamin)	89 mcg	3,708%
Biotin	179 mcg	597%
Pantothenic Acid (as d-calcium pantothenate)	35 mg	700%
Choline (as choline dihydrogen citrate)	6 mg	2%
Calcium (as di-calcium malate[2], d-calcium pantothenate, and calcium ascorbate)	17 mg	2%
Iodine (as potassium iodide)	17 mcg	11%
Magnesium (as di-magnesium malate)[2]	65 mg	15%
Zinc (as zinc bisglycinate chelate[2])	2 mg	18%
Selenium (as selenium glycinate complex)[2]	17 mcg	31%
Manganese (as manganese bisglycinate chelate)[2]	0.09 mg	4%
Chromium (as chromium nicotinate glycinate chelate)[2]	89 mcg	254%
Molybdenum (as molybdenum glycinate chelate)[2]	8 mcg	18%
Potassium (as potassium glycinate complex[2] and potassium ascorbate)	17 mg	0%

N-Acetyl-L-Cysteine	20	*
Bacopa Extract (*Bacopa monnieri*)(whole plant)(8%	20	*
Alpha-Lipoic Acid Scavenges ROS and RNS over BBB	10	*
Shilajit (10.3% Dibenzo-α pyrones (DBPs) & Dibenzo-α	10	*
TheacrineS4 Think safe caffeine	10	*
Milk Thistle Extract (*Silybum marianum*)(seeds)(30%	90	*
Green Tea Extract (*Camellia sinensis*)(leaf)(75%	45	*
Mixed Tocopherols	25	*
Benfotiamine	10	*
trans-Resveratrol (from *Polygonum cuspidatum*)(root)	10	*
Inositol Helps Neurotransmitters like Serotonin and glucose use	6	*
Glucoraphanin (from broccoli extract)(*Brassica oleracea*	5.2	*
Huperzine A (from *Huperzia serrata*)(whole plant)	20	*
Vanadium (as vanadium nicotinate glycinate chelate)S2	13	*

Supplement Facts

Serving Size: 4 Capsules
Servings Per Container: 30

START WITH 1/DAY AND WORK UP

TO 2 IN AM WITH BREAKFAST

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

APPLIED FUNCTIONAL NEUROLOGY

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

Highlights

- In a prospective cohort study, we assessed effects of Vitamin D on dementia incidence in **12,388 participants** from the National Alzheimer's Coordinating Center dataset.

- **Vitamin D exposure was associated with 40% lower dementia incidence versus no exposure.**

- **Vitamin D effects were significantly greater in females versus males** and in normal cognition versus mild cognitive impairment.

- **Vitamin D effects were significantly greater in apolipoprotein E ε4 non-carriers versus carriers. (5,000 IU DAILY AND TARGET SYSTEMIC INFLAMMATION/LPS WITH BARRIER REPAIR, LLLT AND OTHER INFLAMMATION MEDIATORS AS DESCRIBED ABOVE)**

Alzheimers Dement (Amst). 2023 Jan-Mar; 15(1): e12404.
Published online **2023 Mar 1**. doi: 10.1002/dad2.12404

Vitamin D supplementation and incident dementia: Effects of sex, *APOE*, and baseline cognitive status

Maryam Ghahremani, 1

MAGNESIUM

MAGNESIUM: EVERY CELL IN THE HUMAN BODY NEEDS MAGNESIUM. INVOLVED IN OVER 300 PROCESSES IN THE HUMAN BODY AND IS THE 4TH MOST ABUNDANT MINERAL IN HUMANS. 60% IS STORED IN BONES AND THE REMAINDER IN OTHER TISSUES. GOOD FOOD SOURCES INCLUDE GREEN LEAFY VEGETABLES, NUTS, LEGUMES, MEAT, POTATOES, RICE AND WHOLE GRAINS*. OVER 1/2 OF AMERICANS ARE MAGNESIUM DEFICIENT.

MAGNESIUM L-THREONATE (MAGTEIN): CROSSES THE BLOOD BRAIN BARRIER, PROTECTS AND REPAIRS THE BBB. NMDA "PLUG" PROTECTS AGAINST GLUTAMATE EXCITOTOXICITY. PROMOTES HEALTHY NEUROPLASTICITY/SYNAPTIC FUNCTION. PROMOTES HEALTHY COGNITION AND MEMORY, AND MITIGATES COGNITIVE DECLINE, DEPRESSION AND OTHER NEUROLOGICAL DISORDERS.

MAGNESIUM MALATE (DI-MAGNESIUM): MG AND MALATE HIGH IN FRUIT AND WINE. ATP SYNTHESIS/ENERGY AND PAIN RELIEF. GENTLE ALUMINUM CHELATOR. HIGH ABSORPTION RATE AND LOW SIDE EFFECTS.

MAGNESIUM LYSINATE/GLYCINATE: MG AND AA'S LYSINE AND GLYCINE. = RELAXATION AND CALMING. USED IN ANXIETY AND INSOMNIA.

MAGNESIUM CITRATE: MG AND CITRIC ACID = LAXATIVE. HIGH LEVEL OF SIDE EFFECTS AND DRUG INTERACTIONS. CHEST PAINS, DIARRHEA, NAUSEA/VOMITING, HEART REGULATION AND MORE. HIGH IN CITRUS FOODS AND OFTEN USED IN FOODS AND SUPPLEMENTS.

MAGNESIUM OXIDE: MG AND OXYGEN ALSO = LAXATIVE (IE MILK OF MAGNESIA) ALSO USED IN HEARTBURN AND INDIGESTION. POOR ABSORPTION (APPROX. 4%) VS OTHER MG FORMS. MOST COMMON FORM OF MG USED IN SUPPLEMENTS.

MAGNESIUM CHLORIDE: SIMILAR FUNCTION AS OXIDE WITH SLIGHTLY HIGHER ABSORPTION. ALSO OFTEN USED IN TOPICALS FOR MUSCLE RELAXATION/SORENESS.

MAGNESIUM LACTATE: MG AND LACTIC ACID. GENTLE ON THE STOMACH VS FORMS LIKE CITRATE. OFTEN USED IN FOOD ADDITIVES.

MAGNESIUM SULFATE: MG, SULFUR AND OXYGEN. = EPSOM SALT. TOPICAL/BATHS FOR SORE MUSCLES/RELAXATION. HARD ON STOMACH.

MAGNESIUM OROTATE: MG AND OROTIC ACID. CARDIOVASCULAR SYSTEMS BENEFITS.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MAGNESIUM

Journal of Alzheimer's Disease 49 (2016) 971–990 971 DOI 10.3233/JAD-150538
IOS Press

Efficacy and Safety of MMFS-01, a Synapse Density Enh Treating Cognitive Impairment in Older Adults: A Rand Double-Blind, Placebo-Controlled Trial

Guosong Liu[a,d,*], Jason G. Weinger[a], Zhong-Lin Lu[b], Feng Xue[c] and Safa Sadeghpour[a] [a]*Neurocentria, Inc., Fremont, CA, USA*

The study results have clinical significance. The study measured the "brain age" of subjects based on their executive function performance. The brain age of study participants taking MMFS-01 improved by nine years after six weeks of treatment, and persisted after 12 weeks of treatment. By contrast, participants who received a placebo saw little change in their average brain age. These data demonstrate that MMFS-01 is effective at reversing cognitive deficit. The study also show everity, and there were itions.

BEST LAB TEST FOR MAGNESIUM?

Neuropsychiatr Dis Treat. 2019; 15: 3149–3155.
Published online 2019 Nov 11. doi: 10.2147/NDT.S230688

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

Yanling Shen,#

Magnesium L-threonate Prevents and Restores Memory Deficits Associated with Neuropathic Pain by Inhibition of TNF-α

Jun Wang, PhD, Yong Liu, PhD, Li-Jun Zhou, PhD, Ying Wu, PhD, Wei Li, Kai-Feng Shen, PhD, Rui-Ping Pang, PhD, Xu-Hong Wei, PhD, Yong-Yong Li, and Xian-Guo Liu, PhD

MAGNESIUM

Brain Res Bull
2021 Sep;174:184-193. doi: 10.1016/j.brainresbull.2021.06.009. Epub 2021 Jun 16.

Magnesium-L-threonate alleviate colonic inflammation and memory impairment in chronic-plus-binge alcohol feeding mice

Chang Liu 1, Yuliang Cheng 1, Yahui Guo 1, He Qian 2

Abstract

Magnesium-l-threonate (MgT) is considered a food supplement. Alcohol-mediated diseases (AMD) are accompanied by inflammation and memory impairment. The purpose of this study is to investigate the function of MgT in AMD. Hence, chronic-plus-binge alcohol feeding mice model and multiply bioinformatics analysis were performed. Consequently, the expression of inflammatory cytokines downregulated, while the activities of antioxidases decreased in serum, colon, and brain. Interestingly, MgT relieved gut barrier dysfunction and reshaped microbiota. The relative abundance of Akkermansia, Odoribacter, and Blautia were increased, while that of Alloprevotella and Clostridium were decreased. Metabolic analysis elucidated amino acids and glutamate metabolism were enhanced in MgT-treated mice. Furthermore, morris water maze test confirmed memory ability was enhanced. Inflammation cytokines were negatively correlated with Blautia, and Akkermansia.

Collectively, MgT relieved inflammation in gut-brain axis of mice, reshaped gut microbiota, and enhanced the amino acids and glutamate metabolism. MgT may be used as a food supplement to prevent inflammation and memory impairment induced by alcohol abuse.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

J Inflamm Res
2021 Dec 7;14:6633-6656. doi: 10.2147/JIR.S330894. eCollection 2021.

The Causal Role of Magnesium Deficiency in the Neuroinflammation, Pain Hypersensitivity and Memory/Emotional Deficits in Ovariectomized and Aged Female Mice

Jun Zhang # 1 2 3, Chun-Lin Mai # 1, Ying Xiong 1, Zhen-Jia Lin 1, Ying-Tao Jie 1, Jie-Zhen Mai 1, Chong Liu 1, Man-Xiu Xie 4, Xin Zhou 2 3, Xian-

Abstract

Results: **Chronic oral administration of magnesium-L-threonate substantially prevented or reversed the chronic pain and memory/emotional deficits in both ovariectomized and aged female mice.** We found that phospho-p65, an active form of nuclear factor-kappaB, tumor necrosis factor-alpha and interleukin-1 beta were significantly upregulated in the neurons of dorsal root ganglion, spinal dorsal horn and hippocampus in ovariectomized and aged mice. The microglia and astrocytes were activated in spinal dorsal horn and hippocampus. Calcitonin gene-related peptide, a marker for peptidergic C-fibers, was upregulated in dorsal horn, which is associated with potentiation of C-fiber-mediated synaptic transmission in the model mice. In parallel with neuroinflammation and synaptic potentiation, free Mg_{2+} levels in plasma, cerebrospinal fluid and in dorsal root ganglion neurons were significantly reduced. Oral magnesium-L-threonate normalized the neuroinflammation, synaptic potentiation and Mg_{2+} deficiency, but did not affect the estrogen decline in ovariectomized and aged mice. Furthermore, in cultured dorsal root ganglion neurons, estrogen at physiological concentration elevated intracellular Mg_{2+}, and downregulated phospho-p65, tumor necrosis factor-alpha and interleukin-1 beta exclusively in the presence of extracellular Mg_{2+}.

Conclusion: **Estrogen decline in menopause may cause neuroinflammation by reducing intracellular Mg2+ in neurons, leading to chronic pain, memory/emotional deficits. Supplement Mg2+ by oral magnesium-L-threonate may be a novel approach for treating menopause-related neuronal disorders.**

Magnesium Enables Immune Cells to Tackle Infections and Cancer

In a new study that could have important implications for cancer immunotherapy, scientists at the University of Basel and University Hospital Basel present

evidence showing immune T cells that attack pathogen-infected and tumor cells require adequate levels of the mineral magnesium to activate, transmit signals, reprogram metabolism, form physical bridges between T and target cells, and ultimately kill errant cells.

SUMMARY: MAGNESIUM IS THE KEY TO UNLOCK THE DOCKING SITE FOR CD8+ T cells TO ATTACK PATHOGENS AND CANCER CELLS. LOW MAGNESIUM = WORSE OUTCOMES WITH CANCER IMMUNOTHERAPIES AND WORSENING OF CANCER SPREAD

The findings are published in a January 19, 2022 article in the journal *Cell* titled, "Magnesium sensing via LFA 1 regulates CD8+ T cell effector function."

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MAGNESIUM

The study of more than 6,000 cognitively healthy participants in the United Kingdom aged 40 to 73 found people who consume more than 550 milligrams of magnesium each day have a brain age that is approximately one year younger by the time they reach 55 compared with someone with a normal magnesium intake of about 350 milligrams a day.

The researchers say a higher intake of **magnesium in our diets from a younger age may safeguard against neurodegenerative diseases and cognitive decline by the time we reach our 40s.**

"The study shows higher dietary magnesium intake may contribute to neuroprotection earlier in the ageing process and preventative effects may begin in our 40s or even earlier," Ms Alateeq said.

Dietary magnesium intake is related to larger brain volumes and lower white matter lesions with notable sex differences

Open access

Published: 10 March 2023

volume 62, pages 2039–2051 (2023)

FUNCTIONAL NUTRITION FOR BRAIN HEALTH

CREATINE: HELPS RECYCLE ADP TO ATP FASTER. GREAT IN ACUTE TBI. PROTECTS AGAINST EXCITOTOXICITY (NMDA ETC)

QUERCITIN: ANTI-OXIDANT. ANTI-INFLAMMATORY (NF-KB, IL-6, TNF). ALSO HELPS MAST CELL/HISTAMINES, BBB AND eNOS

NITRIC OXIDE/VINPOCETINE: BLOOD FLOW/eNOS, nNOS BUT SAFER FOR iNOS (vs. ARGININE). BEETS

STACKING OMEGA 3'S WITH PHOSPHOLIPIDS LIKE PHOSPHATIDYL CHOLINE OR BROAD SPECTRUM (WHAT ELSE MIGHT IT HELP) SPM'S/PRM'S

PATHOGENS: ANDROGRAPHIS, MONOLAURIN, BIOCIDIN, BIOTOXIN BINDER…

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

HOMOCYSTEINE: NOT JUST A HEART MARKER!

Reduce Your Dementia Risk Up To 31% By Lowering Your Homocysteine Levels

Homocysteine is an amino acid that's heavily involved in a biological process called methylation. During methylation, the bioactive form of folate (5-MTHF) donates a methyl group to homocysteine so it can be converted into another amino acid, methionine. When operating optimally, methylation promotes cardiovascular, neurological, and reproductive health; aids energy production; bolsters detoxification pathways; supports longevity; and more.

The problem is that more than 50% of the U.S. population has a genetic mutation of the MTHFR gene that inhibits the body's ability to activate folate, so it can't optimize methylation. If bioactive folate isn't available to help convert homocysteine during the methylation cycle (i.e., due to an MTHFR gene variation or other reasons—such as poor diet, hormone imbalances, stress, or exposure to toxins), homocysteine levels can become too high.

Elevated homocysteine levels are linked to a higher risk of chronic inflammation, poor detoxification, cardiovascular disease, autoimmune conditions, and yes—dementia.

In a 2018 *Journal of Alzheimer's Disease* review, researchers analyzed a number of meta-analyses and found that 12% to 31% of dementia cases could be prevented by lowering fasting total homocysteine (tHcy) levels.

953

TAKE YOUR METHYLATED B'S!

HYPERBARIC OXYGEN THERAPY

1.3 ATA (4.4 PSI)

SOME OF THE BENEFITS OF HBOT:
- INCREASED OXYGEN TO THE BRAIN AND BODY
- PROMOTES DNA SIGNALING (PROMOTING GOOD GENES AND TURNING OFF BAD GENES)
- INCREASED BLOOD FLOW AND CIRCULATION
- NEW BLOOD VESSELS AND PERFUSION TO TISSUES
- BOOST IMMUNE RESPONSES
- HELPS FIGHT INFECTIONS
- WOUND HEALING/BURNS AND OTHER CONNECTIVE TISSUE INJURIES
- PROMOTES NEW BLOOD CELLS AND HELPS WITH ANEMIA
- PROMOTES STEM CELL PRODUCTION AND PROLIFERATION
- STIMULATE MITOCHONDRIAL OUTPUT FOR ENERGY PRODUCTION (ATP)
- REDUCES PAIN AND INFLAMMATION
- PREVENTS REPERFUSION INJURY (EX. POST STROKE)
- PROMOTES ANTI-OXIDANT SYSTEMS
- PROMOTES GREY AND WHITE MATTER STRUCTURES OF THE CNS
- HELPS WITH INJURIES RELATED TO GASES (IE. SCUBA DIVING OR CO/SMOKE INHALATION)
- HELPS WITH INJURIES RELATED TO RADIATION, VACCINE AND CHEMOTHERAPY
- CONDITION SPECIFIC INDICATIONS SUCH AS TBI, STROKE, CHILDHOOD DEVELOPMENT AND MUCH MORE

OTHER OXYGEN SOURCE CONSIDERATIONS:
EWOT
CONCENTRATORS
FNOR COMBO (SEE VIDEO)/POSTURAL THERAPIES
COUPLED RIB CMT
AUTONOMIC REGULATION/VAGAL STIM

DON'T FORGET TO ADDRESS ANEMIA PATTERNS

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

FROM HELP TO HARM:

WHEN PHOTOBIOMODULATION EXCEEDS OPTIMAL DOSE PARAMETERS

RESTORING BRAIN —HEALTH—

You
WHAT IS THE BEST DOSE FOR LOW LEVEL LASER THERAPY TO THE HUMAN BRAIN

ChatGPT
Determining the "best dose" for low-level laser therapy (LLLT) on the human brain can be complex and is an active area of research. The optimal dose often depends on several factors, including the specific condition being treated, the wavelength of the laser, the power density, the duration of exposure, and individual variability.

Studies investigating transcranial LLLT for various conditions like traumatic brain injury, stroke recovery, or neurological disorders have used a wide range of parameters. Generally, the doses are measured in energy density (Joules per square centimeter) and can vary from around 0.5 J/cm² to 10 J/cm², depending on the specific protocol and targeted area of the brain.

Research is ongoing to determine the most effective doses for different applications. Safety considerations are crucial as well, as excessive doses or inappropriate application methods could have adverse effects.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RESTORING BRAIN —HEALTH—

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DOSE MATTERS!

Purpose: quick, evidence-based guardrails when dose/irradiance creeps into Class 3B/4 territory—where PBM can flip from help → harm.

First principles

- **Hormesis/biphasic response: Low doses stimulate, high doses inhibit or injure. Choose low irradiance and moderate fluence; avoid large hotspots**

Nervous system excitability (GLUTAMATE EXCITOTOXICITY)

- ***Cortical nerve terminals:*** **808 nm, 1 W/cm² × 60 s (60 J/cm²) → vesicular glutamate release; power-dependent exocytosis confirmed in follow-up. (Amaroli 2018; Cervetto 2023)**

"THINK OF DRINKING A DIET SODA. YOU MAY NOT FEEL THE DAMAGE HAPPENING TO THE NEURONS BUT JUST BECAUSE YOU CAN'T FEEL IT, IT'S STILL HAPPENING"

DOSE MATTERS!

Oxidative stress & erosion of antioxidant defenses at higher dose/irradiance

- **High-fluence LPLI (~120 J/cm²)** drove **ROS-mediated mPTP opening**, cytochrome-c release, and apoptosis in human cells. PubMed
- Follow-up work showed HF-LPLI triggers **mitochondrial fragmentation via ROS/Drp1**, consistent with oxidative injury. FEBS Open BioPubMed
- **Isolated mitochondria, 980 nm, 60 s:** parameter-dependent effects—**0.1 W (~0.13 W/cm²) uncoupled** respiration with **higher oxidative stress** and **ATP collapse**; **0.8 W** increased ATP but also raised superoxide/MDA. (Windowed response—wrong settings push ROS up.) PMC
- **Skin cells:** NIR lasers increased **ROS** across most fluences in dermal fibroblasts; 660 nm also elevated ROS in vivo/in vitro redox imaging. PMC+1
- **Keratinocytes vs fibroblasts:** IR diode exposure drove **greater ROS in keratinocytes**, **inversely correlated with catalase** (lower CAT ↔ higher ROS), highlighting vulnerability of some skin cell types. MDPI
- **Antioxidant capacity can drop:** in dystrophin-deficient myotubes (830 nm, **5 J/cm²**), LLLT **reduced GSH (–60–73%)** and **lowered SOD/GR activities**, i.e., a measurable dip in antioxidant buffers (context-dependent). PLOS
- **Phototoxic escalation with dose:** increasing NIR laser dose produced **heat + ROS-driven ER stress**, autophagy and, when excessive, apoptosis. Nature

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

DOSE MATTERS!

Activation of vanilloid/thermo-TRP pain receptors (TRPV1/related)

- **Direct laser activation of TRPV1/2:** 980 nm **diode laser** reproducibly activated **TRPV1** (and TRPV2) in vitro and in vivo—establishing laser-evoked up regulation of nociceptors. PMCPubMed
- **Human pain readouts are TRPV1-dependent:** a **TRPV1 antagonist (ABT-102)** significantly **reduced laser radiant-heat evoked potentials** in human skin (normal and UVB-inflamed). PMC
- **High-power, short IR pulses** preferentially **activate TRPV1$^+$ Aδ fibers** (nociceptors) in volunteers—i.e., higher irradiance/short dwell pushes stimulation into **heat-pain** territory. PMC
- **Near-IR (1470/1475 nm) heat stimuli:** human psychophysics + DRG/HEK data confirm **TRPV1 is a primary encoder of laser-heat pain**. BioMed Central

DOSE MATTERS!

Tumor stimulation at higher dose/irradiance

- *Melanoma (mouse, in vivo):* **660 nm, 2.5 W/cm²**, "low" 150 J/cm² vs **high 1050 J/cm²**. **High-dose increased tumor size and angiogenesis** → authors: avoid LLLT over melanomas at these parameters. (Frigo 2009)

Summary

- **Low-dose LLLT (150 J/cm²; 2.5 W/cm²)**: **no tumor stimulation** observed in mice; safe within these parameters.
- **High-dose LLLT (1050 J/cm²; same irradiance)**: **significant tumor growth enhancement**, increased vascularization and histological abnormalities.
- **Take-home**: There's a clear **dose-dependent risk—staying within low-dose range appears safe**, but high doses must be avoided, especially over melanomas.
- *Anaplastic thyroid cancer (orthotopic mouse):* **650 nm, 100 mW/cm², 15–30 J/cm²** single dose → **accelerated tumor growth**, ↑HIF-1α/AKT. (Rhee 2016)
- *Chemical oral carcinogenesis (hamster cheek pouch):* **660 nm, 424 mW/cm², 56.4 J/cm²** EOD ×4 weeks → **more poorly differentiated tumors**. (de C. Monteiro 2011)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

References

1. **Wu S, Xing D, Gao X, Chen WR.** High fluence low-power laser irradiation induces mitochondrial permeability transition mediated by ROS. *J Cell Physiol*. 2009;218(3):603-611. doi:10.1002/jcp.21636. PubMed
2. **Wu S, et al.** Mitochondrial oxidative stress causes mitochondrial fragmentation after HF-LPLI. *FEBS J*. 2011;278:941-954. doi:10.1111/j.1742-4658.2011.08010.x. FEBS Open Bio
3. **Amaroli A, et al.** Photobiomodulation & Oxidative Stress: **980 nm** light regulates mitochondrial activity; **0.1 W → uncoupling + ROS**, **0.8 W → ↑ATP + ↑superoxide/MDA**. *Antioxidants*. 2021;10(3):469. PMC
4. **George S, et al.** Red/NIR laser elevates **ROS** in dermal fibroblasts across fluences (NIR especially). *Photochem Photobiol Sci*. 2018;17:1180–1190. PMC
5. **Rupel K, et al.** Multispectral PBM differentially modulates **ROS** in vivo/in vitro (roGFP2 sensor). *Sci Rep*. 2018;8:9974. PMC
6. **Cios A, et al.** Review: keratinocytes show **higher ROS** after IR diode; **inverse with catalase**. *Int J Mol Sci*. 2021;22(5):2437. MDPI
7. **Macedo AB, et al.** LLLT (830 nm, **5 J/cm²**) **decreased GSH** and **SOD/GR** in dystrophic myotubes (antioxidant pool drop). *PLoS One*. 2015;10:e0128567. PLOS
8. **Khan I, et al.** Dose-escalated NIR laser → **heat+ROS** → ER-stress/autophagy/apoptosis. *Sci Rep*. 2015;5:10581. Nature
9. **Jiang N, et al. Non-invasive diode laser activates TRPV1/2** in nociceptors (in vivo/in vitro). *Mol Pain*. 2007;3:26. PMC
10. **Schaffler K, et al. TRPV1 antagonist** attenuates laser radiant-heat pain in humans. *J Transl Med*. 2012;10:81. PMC
11. **Mitchell K, et al. High-power short-duration IR laser** preferentially activates **TRPV1⁺ Aδ fibers** in humans. *Neurosci Lett*. 2014;561:17-22. PMC
12. **Rosenberger DC, et al.** TRPV1 is a primary encoder of **near-IR laser heat pain** (1470–1475 nm). *J Transl Med*. 2020;18:262. BioMed Central

DOSE MATTERS!

References

1. **Frigo L,** et al. *BMC Cancer* **2009**;9:404. **"The effect of low-level laser irradiation (In-Ga-Al-AsP 660 nm) on melanoma in vitro and in vivo."** doi:10.1186/1471-2407-9-404.
2. **Rhee YH,** et al. *Photomed Laser Surg* **2016**;34(6):229–235. **"Low-Level Laser Therapy Promoted Aggressive Proliferation…"** doi:10.1089/pho.2015.3968.
3. **de C. Monteiro JS,** et al. *Photomed Laser Surg* **2011**;29(11):741–745. **"Influence of laser phototherapy (λ660 nm) on chemo-induced oral carcinogenesis."** doi:10.1089/pho.2010.2896.
4. **Amaroli A,** et al. *J Biophotonics* **2018**;11(11):e201800102. **"Near-infrared laser photons induce glutamate release…"** doi:10.1002/jbio.201800102.
5. **Cervetto C,** et al. *Int J Mol Sci* **2023**;24(13):10977. **"Photons induce vesicular exocytotic release of glutamate… power-dependent."** doi:10.3390/ijms241310977.
6. **Bhattacharya M, Dutta A.** *Brain Sci* **2019**;9(8):179/188. **"Computational modeling of photon transport, tissue heating & CCO absorption during tNIRS."** doi:10.3390/brainsci9080179/**188** (see journal issue listing).
7. **Guillén AR,** et al. *medRxiv* **2025**. **"High-Resolution Computational Modeling of tPBM: Light Propagation & Thermal Effects."** doi:10.1101/2025.01.18.25320758.
8. **Wu S,** et al. *J Cell Physiol* **2009**;218(3):603–611. **"High fluence low-power laser irradiation induces mitochondrial permeability transition mediated by ROS."** doi:10.1002/jcp.21636.
9. **Wu S,** et al. *FEBS J* **2011**;278(6):941–954. **"Mitochondrial oxidative stress causes mitochondrial fragmentation…"** doi:10.1111/j.1742-4658.2011.08010.x.
10. **Huang YY,** et al. *Dose-Response* **2011**;9(4):602–618. **"Biphasic Dose Response in Low Level Light Therapy—An Update."** doi:10.2203/dose-response.09-027.Hamblin.
11. **OSHA Technical Manual—Laser Hazards. Class 3B/4 definitions & controls.** (OTM Section III, Ch.6)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

WHY LOW LEVEL LASER THERAPY vs OTHER DEVICES

SHORTER WAVELENGTHS = HIGHER ENERGY (WHY WE LIKE 405, 520 AND 635 NM)

ENERGY DOES NOT EQUAL POWER!

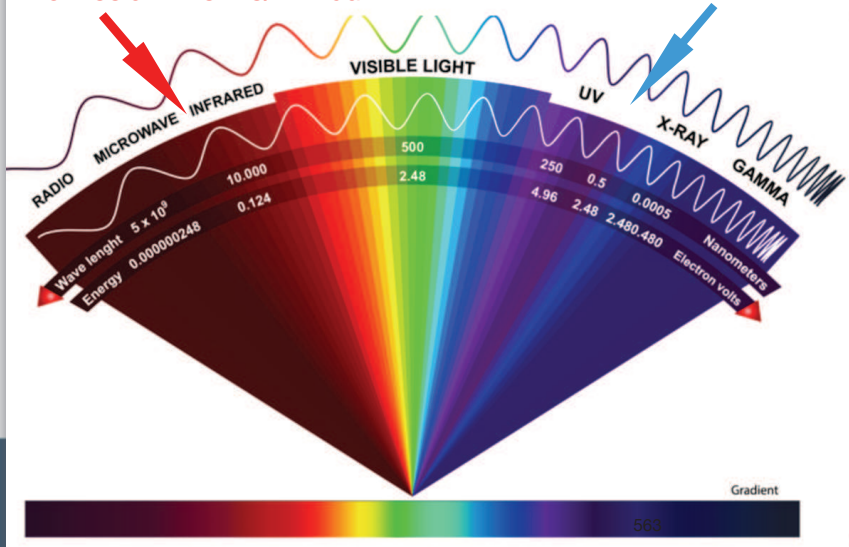
LONGER WAVELENGTHS COOK WATER => HUMAN CELLS

SUPER SHORT WAVELENGTHS THROW VALENCE ELECTRONS = IONIZING RADIATION

VISIBLE LIGHT SPECTRUM UP TO 680NM = GOLDILOCKS EFFECT/PHOTOCHEMISTRY

WOULD YOU STICK YOUR HEAD IN A MICROWAVE?
CLASS 3-4 LASERS/EMF 5G

HOW LONG CAN YOU SUNBURN OR STICK YOUR HEAD IN AN X-RAY??



Photon Energy is measured in Electron Volt (eV)

A PHOTON IS THE SMALLEST PARTICLE OF LIGHT. EACH HAS A CERTAIN ENERGY CALLED eV

The shorter the wavelength, the higher the Electron Volt (eV)

- 405nm = 3.06 eV
- 532nm = 2.33 eV
- 635nm = 1.95 eV
- 830nm = 1.49 eV

405 nm has 70% more energy per photon than 830nm

IR WAVELENGTHS ONLY PROVIDE 1.5 EV WHICH DOES NOT REACH THE MINIMUM NEEDED TO RAISE ELECTRONS TO HIGHER ORBITS (1.7)

1.DO NOT EXCEED 10 J/CM2, ESPECIALLY TO THE BRAIN!

2.ABOVE 10 EV IS IONIZING (MAX OF 7.5/17.2 mW VIOLET IS 3.06)

3.LED VS TRUE LASER (MONOCHROMATIC, COHERENT, PARALLEL)

4.FDA CLEARANCES (ESPECIALLY FOR SAFETY)

5.PHOTOCHEMICAL VS. PHOTOTHERMAL

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CHAT GPT OPEN AI

You
WHAT IS THE MOST RESEARCHED LOW LEVEL LASER

ChatGPT
Low-level laser therapy (LLLT) has been extensively studied across various applications, but singling out one specific laser or device as the "most researched" can be challenging due to the breadth of research and different devices used in studies.

However, some companies and devices have been involved in numerous research studies and clinical trials:

1. **Erchonia Lasers:** Erchonia has been involved in a significant amount of research across different applications, including pain management, wound healing, and aesthetics. Their lasers have been featured in various clinical trials evaluating their efficacy.
2. **THOR Photomedicine Lasers:** THOR's devices have also been extensively researched for their effectiveness in various conditions, including musculoskeletal pain, wound healing, and neurological disorders.
3. **Multi Radiance Medical:** Their super pulsed laser technology has been the subject of numerous studies examining its impact on pain relief and injury treatment.

CHAT GPT OPEN AI

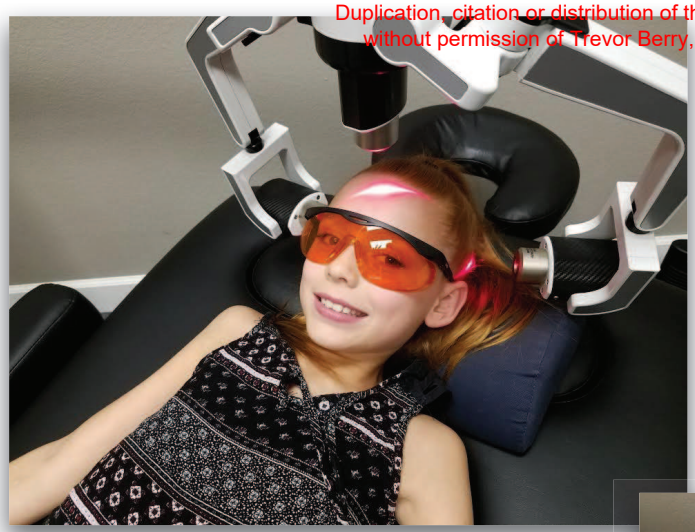
You
what is the best low level laser therapy company

ChatGPT
Determining the "best" low-level laser therapy (LLLT) company can depend on several factors, including the specific application, quality of products, clinical evidence, customer satisfaction, and industry reputation. Several companies are well-regarded in the field of LLLT:

1. **Erchonia:** Known for its range of FDA-cleared lasers used in pain management, wound healing, and aesthetic treatments.
2. **Multi Radiance Medical:** Offers super pulsed laser technology for pain relief and injury treatment, known for its effectiveness and non-invasive nature.
3. **THOR Photomedicine:** Provides medical laser systems used in various therapies, including pain management and tissue repair.
4. **LiteCure (now LightForce Therapy Lasers):** Renowned for its deep tissue therapy lasers, used in sports medicine, veterinary care, and human healthcare.
5. **ASA Laser:** Offers a range of lasers for rehabilitation, sports medicine, and wound healing.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



WHY I USE ERCHONIA PRODUCTS TO TREAT AND PROTECT THE BRAINS OF MY LOVED ONES

567

ADIPOSE REDUCTION

- MODIFIED PALEO/PAGEN DIET WITH KETOGENIC ROTATION AS INDICATED
- INCREASE WATER INTAKE
- HIIT DAILY (BETTER EPOC EFFECT). CARDIO ROTATION (MIN 20-MAX 37 MINUTES)
- INTERMITTENT FASTING AND 16-8 DAILY RULE CONSIDERATION
- RULE OUT UNDERLYING METABOLIC COMPROMISE FIRST! EX. IF SHOWING THYROID DYSFUNCTION ADDRESS THAT BEFORE STARTING ON PROGRAM
- NUTRITIONAL SUPPORT (XYMOGEN PRODUCT LINE)

FOUNDATIONAL 3:

- BERBERINE ES-5 (1 CAP BID WITH 2 BIGGEST MEALS)
- OPTIFIBER LEAN (3 CAPS BID PRE BIGGEST MEALS = 3 GRAMS. 1 AT START)
- PROBIOMAX LEAN DF (1 CAP PER DAY. 4% BODY MASS REDUCTION)

GOOD ADD ONS:

- LIPOTROPIX (2 CAPS PER DAY. FAT METABOLISM)
- CARNITEX (1 CAP BEFORE BREAKFAST. L-CARNITINE FAT UTILIZATION, ENERGY FOR MUSCLE RECOVERY)

OPTIONAL AS INDICATED:

- APPECURB (2 CAPS PER DAY IF PATIENT DEALING WITH CRAVINGS)
- SYNOVX DJD (2 CAPS DAILY. OSTEOARTHRITIC JOINTS/LEPTIN RESISTANCE)
- OR LEPTIN MANAGER (1 CAP AM. PROPER LEPTIN UTILIZATION)
- BERGACORE PLUS (2 CAPS DAILY. CARDIOMETABOLIC SYNDROME)

ALWAYS PRE-SCREEN PATIENTS ESPECIALLY FOR THYROID DYSFUNCTION. CONSIDER MAF FORMS AND RUN LABS ACCORDINGLY

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

WEIGHT LOSS

OPTIFIBER LEAN:

- E-425-ii IS ITS EUROPEAN DRUG NAME AND IS CLINICALLY INDICATED IN EUROPE FOR WEIGHT LOSS AND CHOLESTEROL MANAGEMENT
- BINDS TOXINS, FAT AND SLUDGY BILE IN THE GUT AND CAN BE USED AS PART OF MYCOTOXIN PROFILE TO BIND BIOTOXINS IN THE GUT
- IS AN EXCELLENT PRE-BIOTIC. BIFIDO BACTERIA FERMENTS THE SOLUBLE FIBER FOR FUEL SOURCE CREATING SHORT CHAIN FATTY ACIDS AS A BYPRODUCT OF THE FERMENTATION PROCESS, MOST NOTABLY BUTYRATE
- IT IS THE ONLY KONJAC (ROOT VEGETABLES IN ASIA) WITH NO ARSENIC OR SULFITES
- SWELLS 200X ORIGINAL SIZE TO CREATE FULLNESS AND MASS WHILE TRAPPING MATTER LISTED ABOVE BUT IT DOES NOT DENATURE LIKE INULIN AND GUAR GUMS WHICH CAN DENATURE 50-100% DURING INGESTION
- IT LOWERS BETA-GLUCURONIDASE BY 25% ACTIVITY BY 25% IN 4 WEEKS (GUT ZOOMER 3.0). THIS CAN PLAY A ROLE IN PROPER ESTROGEN METABOLISM AND GUT DYSBIOSIS
- INCREASES SATIETY
- SLOWS GASTRIC EMPTYING BY MORE THAN 80 MINUTES
- STABILIZES GLUCOSE SPIKES AND CRASHES AS WELL AS BLOOD GLUCOSE LEVELS
- 3 GRAMS PER DAY (START WITH 1 AND WORK UP) IS EQUIVALENT TO 40-60 GRAMS OF FIBER OF OTHER MAJOR BRANDS!

1-2 CAP PRE MEAL
UP TO 6/DAY
OR
1 GRAM POWDER
PRE MEAL

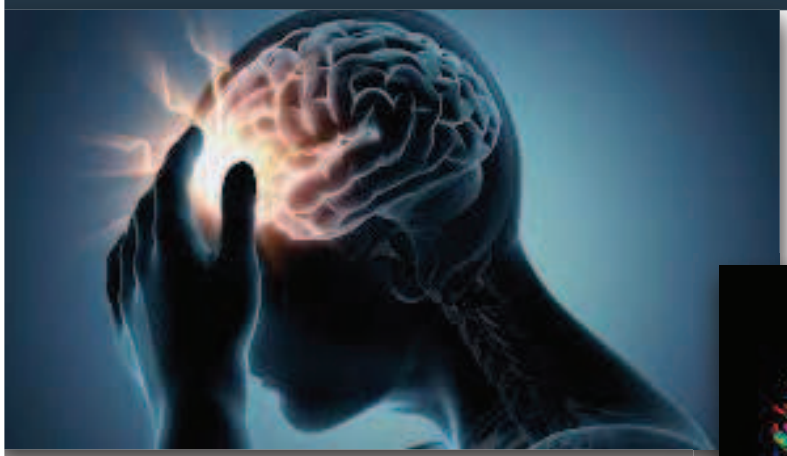
NUTRITIONAL SUPPORT FOR ADIPOSE REDUCTION

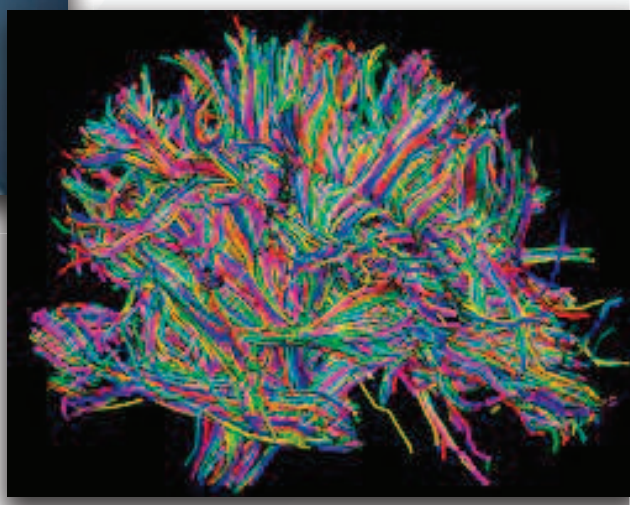
CLINICAL GEM: ORALVISC (NATURAL SOURCE OF GLYCOSAMINOGLYCANS IN LEPTIN MANAGER) HELPS REDUCE REVERSE T3 WHICH THE BODY INCREASES WHEN IN STARVATION MODE USING MUSCLE FOR ENERGY RATHER THAN BURNING FAT!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

TRAUMATIC BRAIN INJURY





© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

TRAUMATIC BRAIN INJURY

- #1 SYMPTOM OF TBI IS HEADACHES, #2 IS DIZZINESS
- FEMALES ARE 6 TIMES MORE LIKELY TO SUFFER A CONCUSSION THAN MALES
- SUB METABOLIC THRESHOLD EXERCISE IS INDICATED POST HEAD INJURY
- BECAUSE THE BRAIN IS USING UP EVEN MORE ENERGY THAN IT ALREADY DOES TO HEAL FROM A HEAD INJURY, MITOCHONDRIAL SUPPORT IS OF THE UTMOST IMPORTANCE. LASER, CREATINE AND OTHER ATP NUTRITION (ACTIVNUTRIENTS PERFORMANCE)
- PROPRIOCEPTION ORDER OF FEEDBACK #1 NECK (SPINE) #2 ANKLES/FEET #3 HIPS
- AVERAGE RETURN TO PLAY MINIMUM IS 18 DAYS (THINK HOW SOON MANY ATHLETES RETURN!)
- IF A CONCUSSION IS SUFFERED AND THEY GO BACK IN THE GAME THE AVERAGE RETURN TO PLAY GOES TO 44 DAYS!!!
- FOR EVERY MINUTE AN ATHLETE TRIES TO PLAY THROUGH A CONCUSSION, THE ATHLETE ADDS 7-9 DAYS OF RECOVER. EAGLE ET AL 2022
- ASSESS THE PATIENT FOR RETURN TO PLAY WHEN THEY ARE REALLY TIRED SUCH AS EARLY IN AM OR END OF DAY
- JUST LIKE ACUTE ORTHOPEDIC INJURIES, DO NOT TRAIN FAULTY TBI VESTIBULAR FINDINGS ON PERTURBED SURFACE LIKE AN AIREX PAD UNTIL STABILIZED. YOU DON'T WANT TO CREATE NEUROPLASTICITY AROUND A FAULTY SYSTEM
- WHIPLASH/CAD IS THE NECK COMPONENT VS TBI (TRAUMATIC BRAIN INJURY)
- ALL CONCUSSIONS SHOW DYSFUNCTION OF THE DORSOLATERAL PREFRONTAL CORTEX (DLPF L>R)
- FOR DYSAUTONOMIA ANALYSIS (IE POTS, ORTHOSTATIC INTOLERANCE). TILT TABLE IS THE GOLD STANDARD BUT IF NO TILT TABLE, CONSIDER THE ***NASA LEAN TEST***
- WHEN DOING MSK REHAB, TYPICAL COGNITIVE FATIGUE OF THE BRAIN IS AT MAX 25 MINUTES

*SOME CONTENT COURTESY OF TNOR (BRETT JAROSZ, STU IFE, DAVID GEORGE)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

TRAUMATIC BRAIN INJURY

- STANDARD PROPRIOCEPTION IS MOSTLY NECK/SPINE, THEN ANKLE STRATEGIES, THEN HIPS
- RHOMBERG TEST (FLAT SURFACE EYES OPEN) 70% CEREBELLAR/PROPRIOCEPTION, 20% VESTIBULAR/INNER EAR, 10% OCULOMOTOR
- ADD A FOAM SURFACE THAT RATIO NOW BECOMES 70% VESTIBULAR/INNER EAR, 20% VISION, 10% PROPRIOCEPTION
- 50% OF CONCUSSIONS DEVELOP CONVERGENCE INSUFFICIENCY. BROCK BEADS, MESENCEPHALIC REHAB. GOOD LUCK READING EFFICIENTLY WITH CONVERGENCE DYSFUNCTION
- CDP (COMPUTERIZED DYNAMIC POSTUROGRAPHY) IS ONE OF THE MOST SENSITIVE TESTS FOR TBI AND OTHER NEUROLOGICAL CONDITIONS BUT IT'S NOT SPECIFIC! IT CAN CHANGE WITH SLEEP, SOUNDS COMING INTO THE ROOM, CANNOT DIAGNOSE A CONDITIONS PER SE. YET, IT PICKS UP THE MOST MINUSCULE SWAY PATTERNS AND DYSFUNCTION.
- BTRACKS CTSIB MARKERS IDEALLY ABOVE 70% FOR RETURN TO PLAY. CONSIDER OPTIMIZATION FOR RESPECTIVE SPORT. IE. SURFERS NEED BETTER VESTIBULAR OUTCOMES VS. FIELD SPORTS NEED PROPRIOCEPTIVE
- TBI IS STILL A SUBJECTIVE CONDITION. ALTHOUGH OBJECTIVE MARKERS ARE BECOMING MORE ACCURATE (LABS, BALANCE TESTING, VNG ETC), IT'S STILL SUBJECTIVE CRITERIA
- THE NUMBER ONE QUESTION FOR PATIENTS/PARENTS ARE NOT IF THE PATIENT THINKS THEY CAN RETURN TO THE FIELD. IT'S IF THEY HAVE RETURNED TO FULL TIME SCHOOL WITHOUT SYMPTOMS!!!! ONCE THEY HAVE ACCOMPLISHED THIS, GETTING BACK ONTO THE FIELD BECOMES THE NEXT PHASE

*SOME CONTENT COURTESY OF FNOR (BRETT JAROSZ, STU FIFE, DAVID GEORGE)

LOW LEVEL LASER AND TBI

Cells. 2024 Mar; 13(5): 385.
Published online 2024 Feb 23. doi: 10.3390/cells13050385

Traumatic Brain Injury Recovery with Photobiomodulation: Cellular Mechanisms, Clinical Evidence, and Future Potential

Abstract

Traumatic Brain Injury (TBI) remains a significant global health challenge, lacking effective pharmacological treatments. This shortcoming is attributed to TBI's heterogeneous and complex pathophysiology, which includes axonal damage, mitochondrial dysfunction, oxidative stress, and persistent neuroinflammation. The objective of this study is to **analyze transcranial photobiomodulation (PBM), which employs specific red to near-infrared light wavelengths to modulate brain functions, as a promising therapy to address TBI's complex pathophysiology in a single intervention.** ***The findings support PBM's potential for treating TBI, notwithstanding variations in parameters such as wavelength, power density, dose, light source positioning, and pulse frequencies. Emerging data indicate that each of these parameters plays a role in the outcomes.*** Additionally, new research into PBM's effects on the electrical properties and polymerization dynamics of neuronal microstructures, like microtubules and tubulins, provides insights for future parameter optimization. In summary, transcranial PBM represents a multifaceted therapeutic intervention for TBI with vast potential which may be fulfilled by optimizing the parameters. Future research should investigate optimizing these parameters, which is possible by incorporating artificial intelligence.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Cells. 2024 Mar; 13(5): 385.
Published online 2024 Feb 23. doi: 10.3390/cells13050385

Traumatic Brain Injury Recovery with Photobiomodulation: Cellular Mechanisms, Clinical Evidence, and Future Potential

Lew Lim

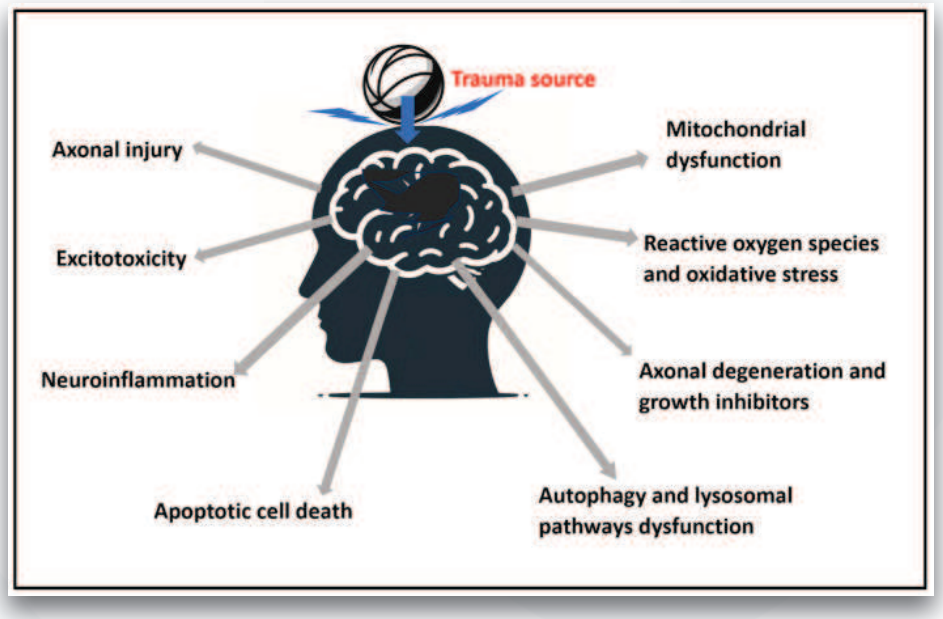


Figure 2
Summary of the identified pathophysiological aspects of traumatic brain injury (TBI) from a trauma source that are addressable with photobiomodulation (PBM).

LOW LEVEL LASER AND TBI

Cells. 2024 Mar; 13(5): 385.
Published online 2024 Feb 23. doi: 10.3390/cells13050385
Traumatic Brain Injury Recovery with Photobiomodulation: Cellular Mechanisms, Clinical Evidence, and Future Potential
Lew Lim

Summary of pathophysiological aspects, cellular mechanisms, and related photobiomodulation research.

Pathophysiological Aspects	Description of the PBM Research
Axonal Injury	PBM may aid axonal recovery through improved ATP generation and modulation of secondary mediators. It activates the PI3K/Akt signaling pathway [11,12].
Mitochondrial Dysfunction	The effects of PBM on mitochondria, particularly cytochrome c oxidase, can restore electron transport and increase mitochondrial membrane potential, aiding axonal regeneration [11,19].
Excitotoxicity	PBM was found to increase ATP content, Ca_{2+} levels, and mitochondrial membrane potential, counteracting excitotoxicity [22].
Reactive oxygen and nitrogen species (RONS)	PBM reduces RONS levels and oxidative stress, promoting antioxidant capacity and reducing damage. It can modulate exercise-induced oxidative stress [30,32,33,34,35].
Neuroinflammation	PBM can reduce pro-inflammatory cytokines, activate anti-inflammatory responses, and downregulate neurotoxic microglia and astrocytes [9,39,40].
Axonal Degeneration	PBM increases axonal regeneration and counters growth inhibitors, potentially preventing axonal damage and degeneration [4,44,45,46].
Apoptotic cell death	PBM activates anti-apoptotic mechanisms, potentially preventing cell death and promoting neurogenesis [47,54,55,56,57].
Autophagy and Lysosomal Dysfunction	PBM can restore mitochondrial function and improve mitophagy by regulating autophagy and lysosomal activity [61,62,63,64].
Additional Systemic Mechanisms	PBM enhances cellular energy production, improves blood flow, modulates synaptic plasticity, and reduces ferroptosis [65,66,67,68,69,70].

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER AND TBI

Results: Of the 68 patients who were randomized (33 to LLLT and 35 to sham therapy), 28 completed at least 1 LLLT session. No adverse events referable to LLLT were reported. Forty-three patients (22 men [51.2%]; mean [SD] age, 50.49 [17.44] years]) completed the study with at least 1 magnetic resonance imaging scan: 19 individuals in the LLLT group and 24 in the sham treatment group. Radial diffusivity (RD), mean diffusivity (MD), and fractional anisotropy (FA) showed significant time and treatment interaction at 3-month time point (RD: 0.013; 95% CI, 0.006 to 0.019; P < .001; MD: 0.008; 95% CI, 0.001 to 0.015; P = .03; FA: -0.018; 95% CI, -0.026 to -0.010; P < .001).The LLLT group had lower RPQ scores, but this effect did not reach statistical significance (time effect P = .39, treatment effect P = .61, and time × treatment effect P = .91).

Conclusions and relevance: **In this randomized clinical trial, LLLT was feasible in all patients and did not exhibit any adverse events. *Light therapy altered multiple diffusion tensor parameters in a statistically significant manner in the late subacute stage.* This study provides the first human evidence to date that light therapy engages neural substrates that play a role in the pathophysiologic factors of moderate TBI and also suggests diffusion imaging as the biomarker of therapeutic response.**

JAMA Netw Open

2020 Sep 1;3(9):e2017337. doi: 10.1001/jamanetworkopen.2020.17337.

Effect of Transcranial Low-Level Light Therapy vs Sham Therapy Among Patients With Moderate Traumatic Brain Injury: A Randomized Clinical Trial

Maria Gabriela Figueiro Longo [1], Can Ozan Tan [1 2], Suk-Tak Chan [1 3], Jonathan Welt [4],

LOW LEVEL LASER AND TBI

Abstract

Background Low-level light therapy (LLLT) has been shown to modulate recovery in patients with traumatic brain injury (TBI). However, the impact of LLLT on the functional connectivity of the brain when at rest has not been well studied. **Purpose To use functional MRI to assess the effect of LLLT on whole-brain resting-state functional connectivity (RSFC) in patients with moderate TBI at acute (within 1 week), subacute (2-3 weeks), and late-subacute (3 months) recovery phases.** Materials and Methods This is a secondary analysis of a prospective single-site double-blinded sham-controlled study conducted in patients presenting to the emergency department with moderate TBI from November 2015 to July 2019. **Participants were randomized for LLLT and sham treatment.** The primary outcome of the study was to assess structural connectivity, and RSFC was collected as the secondary outcome. MRI was used to measure RSFC in 82 brain regions in participants during the three recovery phases. Conclusion Despite the small sample size, **the change in RSFC from the acute to subacute phases of recovery was greater in LLLT-treated than sham-treated participants, suggesting that acute-phase LLLT may have an impact on resting-state neuronal circuits in the early recovery phase of moderate TBI.**

Radiology

2024 May;311(2):e230999. doi: 10.1148/radiol.230999.

Effects of Low-Level Light Therapy on Resting-State Connectivity Following Moderate Traumatic Brain Injury: Secondary Analyses of a Double-blinded Placebo-controlled Study

Suk-Tak Chan # [1], Nathaniel Mercaldo # [1], Maria G Figueiro Longo [1], Jonathan Welt [2],

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER VS LED IN TBI

Research design: Participants (3 men, 6 women; 22-61 years-old) underwent a **6-week intervention involving 18 40-minute transcranial LED treatment sessions.**

Conclusions: **Most participants did not improve on neuropsychological testing.** A minority self-reported improvement in symptoms, potentially explained by the intervention, psychiatric medication changes, placebo effects, or other factors.

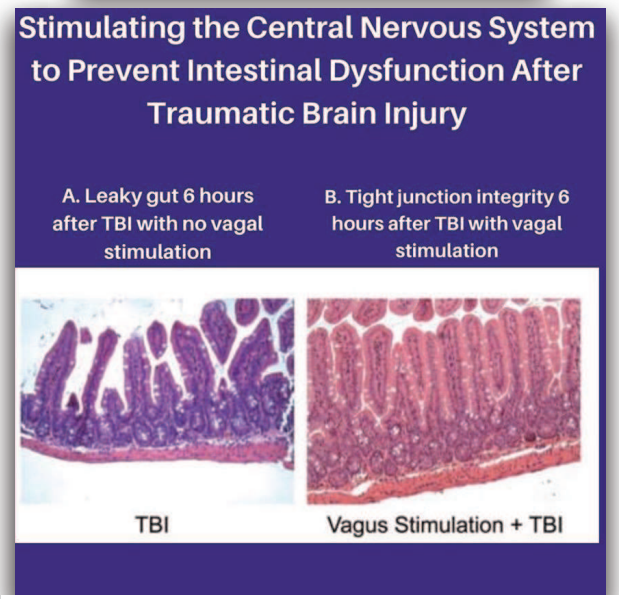
Brain Inj

2024 May 11;38(6):425-435. doi: 10.1080/02699052.2024.2309258. Epub 2024 Feb 8.

Treatment of chronic symptoms following mild traumatic brain injury with transcranial LED: a sham run-in pilot study of photobiomodulation therapy

Justin E Karr [1], Grant L Iverson [2 3 4 5], Nancy Boudreau [6], Ross Zafonte [7]

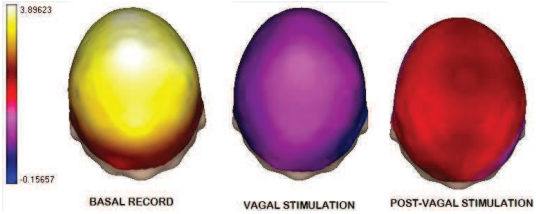
ERCHONIA
World Leaders in Low Level Laser Technology



CONCLUSION: In a mouse model of TBI, **vagal stimulation prevented TBI-induced intestinal permeability. Furthermore, vagal stimulation increased enteric glial activity and may represent the pathway for central nervous system regulation of intestinal permeability.**

VAGAL NERVE STIMULATION WITH LOW LEVEL LASERS BY HEART RATE VARIABILITY MACHADO ET AL.

ABSTRACT: WHEN RED LASER WAS USED FOR VNS THERE WAS A PREDOMINANCE OF PARASYMPATHETIC BALANCE (HF). ON THE CONTRARY, THE STIMULUS WITH VIOLET LASER PROVOKED SYMPATHETIC PREVALENCE (LF). THE COMBO RED/VIOLET WAS ALSO PREDOMINANTLY PARASYMPATHETIC (HF). AS A CONCLUSION, THE STUDY SHOWED THAT VNS USING *LLLT IS A NON-INVASIVE AND SAFE METHOD AND SHOULD BE CONSIDERED FOR FUTURE PROTOCOLS TO RECOVER PARASYMPATHETIC/SYMPATHETIC BALANCE IN DIFFERENT CONDITIONS.*



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ACUTE PHASE TBI NUTRITIONAL SUPPORT

ERCHONIA eCommunity Education

Summary: Acute Phase TBI Management (<4 Weeks)

AM:
1 OMEGA MONOPURE CURCUMIN EC
1 S-ACETYL GLUTATHIONE
1 ACTIV NUTRIENTS WITHOUT COPPER AND IRON
2 ACTIV NUTRIENTS PERFORMANCE
1 ALAMAX CR

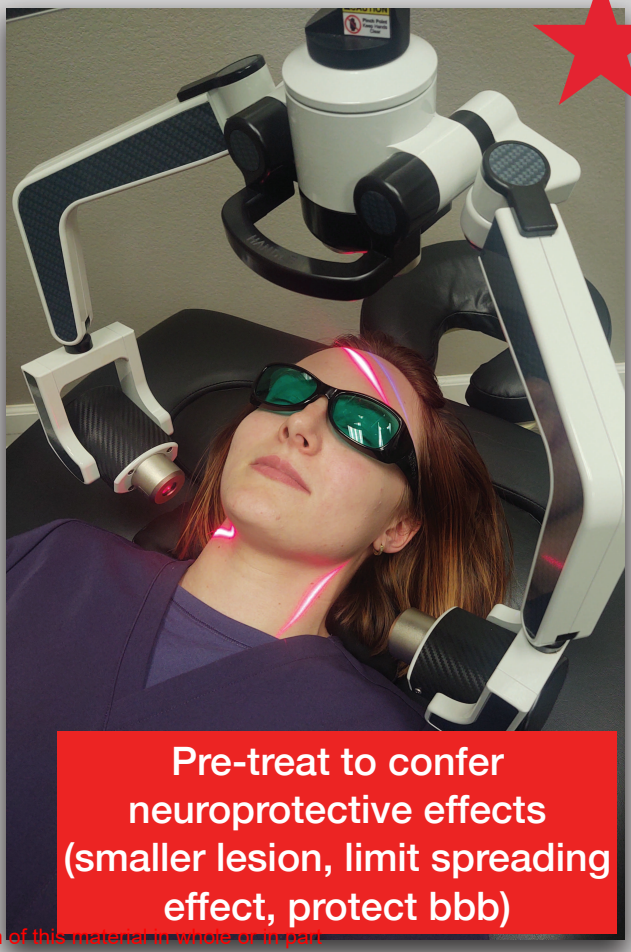
PM:
1 MELATONIN CR
1 NAC
1 K2-D3
1 OMEGAMONOPURE PRM
1 OPTIMAG NEURO
1 RESVERATIN PLUS

24 HOUR WATER FAST ON DAYS 7, 14, 21 AND 28 (*)
CONSIDER ATP IGNITE WORK OUT IN AM (CREATINE + COFACTORS)

*** ERCHONIA LOW LEVEL LASER BRAIN TRANSCRANIAL AND VAGAL STIM (1-10-1-10) DAILY (RENTAL PROGRAM) OR 3X/WEEK FIRST 2 WEEKS THEN MINIMUM ONCE A WEEK UNTIL SYMPTOM RESOLUTION. DO NOT RETURN TO PLAY/ADL IF SYMPTOMS PERSIST!**

ACUTE PHASE TBI ERCHONIA FX APPLICATION UP TO 4 WEEKS OF CARE

FRONTAL DIODES 1 HZ (ALL 2 OR 4)
VAGAL STIM 10 HZ (ALL 4)
10 MINUTE MAX FOR FX 635
6 MINUTE MAX FOR FX 405
3X/WEEK 4 WEEKS OR DAILY WITH THE LASER RENTAL!



Pre-treat to confer neuroprotective effects (smaller lesion, limit spreading effect, protect bbb)

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SUMMARY OF 4 PHASES OF TBI MANAGEMENT

1. PRE-CONDITION PHASE:
- TRANSCRANIAL LOW LEVEL LASER
- INTACT BARRIER SYSTEMS/VAGAL LASER
- FUNCTIONAL NEUROLOGY BALANCE AND OPTIMIZATION (IE. VESTIBULAR INTEGRATION, FAST EYE MOVEMENTS, BALANCE ETC)
- NUTRIENT PROFILES ESPECIALLY MAGNESIUM AND EFA'S

2. ACUTE PHASE PROTOCOL (SEE SLIDE)

3. POST-CONCUSSION SYNDROME PRIMARY DIFFERENTIALS:
- GLIAL PRIMING/INFLAMMATION CYCLES (LLLT TCT/VAGAL, SPM'S, CURCUMINOIDS, HIGH DOSE GLUTATHIONE)
- ASSESS AND REPAIR LEAKY BARRIER SYSTEMS/VAGAL NERVE STIMULATION
- HP-AXIS ASSESS WITH LABS AND SUPPORT (GONADS, ADRENALS, THYROID - ASI SALIVA AND URINE METABOLITES TEST, FULL THYROID PANEL. OTHER UPSTREAM SUPPORT - DOPAMINE/SEROTONIN AND THALAMIC INTEGRATION)
- WATCH FRUCTOSE/SAD DIET. CONSIDER KETOSIS
- FUNCTIONAL NEUROLOGY A) VESTIBULAR SENSORY MISMATCH B) MAPPING ESPECIALLY SACCADES C) ANS REGULATION/MESENCEPHALIC WIND-UP D) LEFT DLPF

4. PROPER RETURN TO PLAY ASSESSMENT:
- OBJECTIVE BIOMARKERS (VNG, CDP, NEUROPSYCH). ALWAYS TRY TO HAVE A PRE-SEASON/ GENERAL PATIENT BASELINE ASSESSMENT DONE IE. CTSIB
- SYMPTOM STATUS
- BETTER TO RETIRE THAN FACE LIFE LONG CTE/GLIAL RAMIFICATION

ERCHONIA
World Leaders In Low Level Laser Technology

MASTER BRAIN SETTING POST ACUTE PHASE TBI

ERCHONIA
eCommunity Education

WHEN IN DOUBT, DEFAULT TO THE MASTER BRAIN PROTOCOL FOR ANY NEUROLOGICAL DISORDER INCLUDING DEMENTIAS LIKE AZLHEIMER'S, VASCULAR DEMENTIA ETC

- **"MASTER BRAIN SETTING" 1-10-40-60 HAND HELDS**

1-40PF, (1-40 FX 405 AND) 40-60 FOR APICALS, 10-10 BRAINSTEM FOR FX 635 IF SAGITTAL PLANE OR 1-40 PFC, 10-40 NECK SCM

IF SUPINE THEN DO A "GUT SHOT"

10 MIN-4 MIN FX 635, 6 MIN-4 MIN FX 405

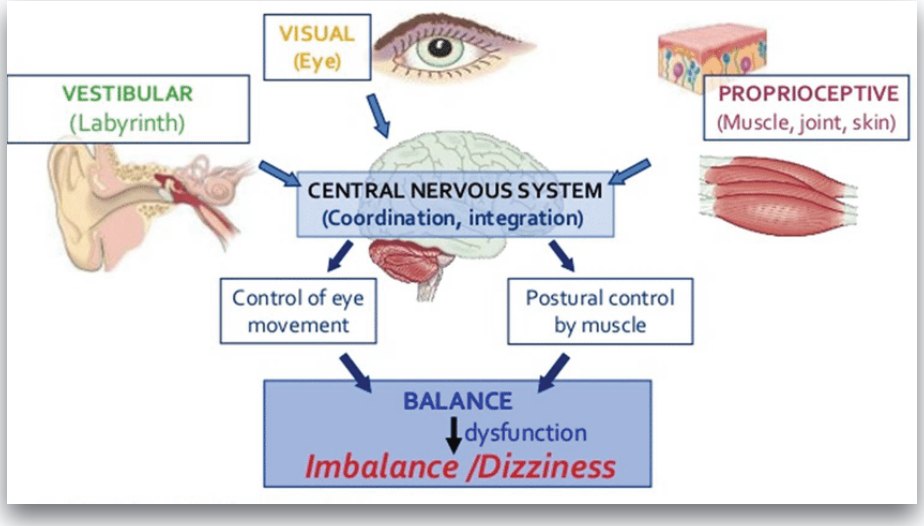
4-4-4–2-2 XLR8, 3-3-3-2-2 EVRL

2-2-2-2-2 GVL

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

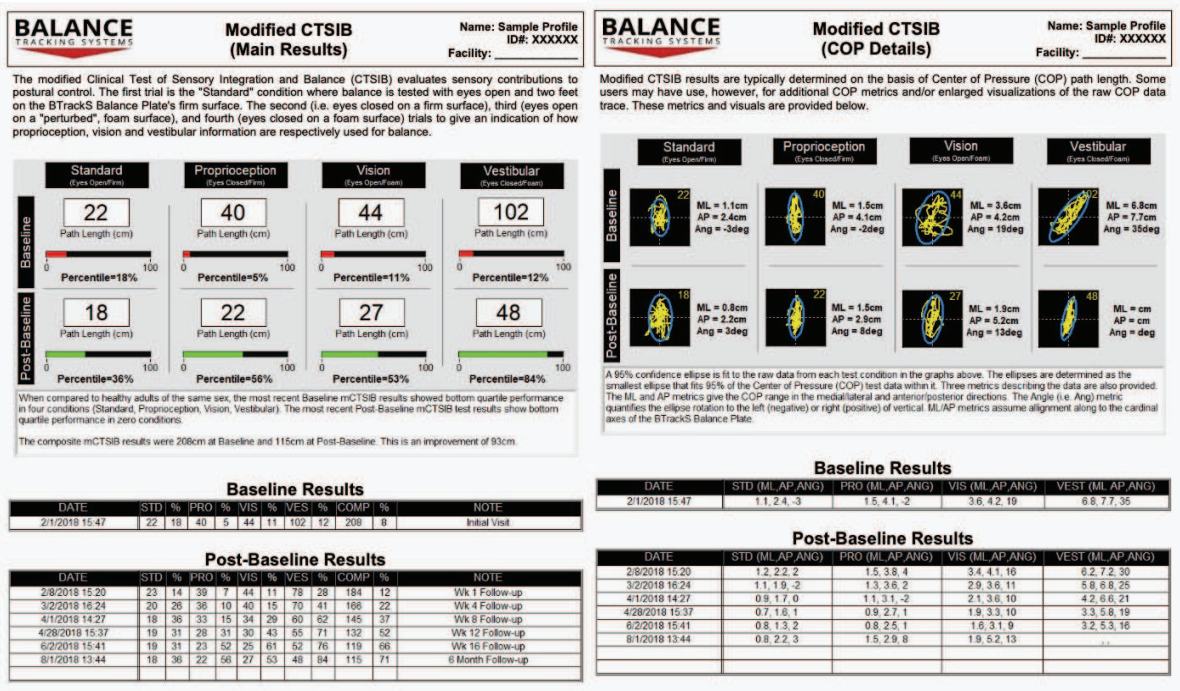
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SENSORY MISMATCH



DO THE EYES, INNER EAR AND PROPRIOCEPTIVE SYSTEMS SAY THE SAME THING THE THE VESTIBULAR NUCLEI AND CORTEX (PIVC RIGHT PARIETAL LOBE)?

Modified CTSIB



BALANCE TRACKING SYSTEMS — Modified CTSIB (Main Results) — Name: Sample Profile ID#: XXXXXX — Facility: _____

The modified Clinical Test of Sensory Integration and Balance (CTSIB) evaluates sensory contributions to postural control. The first trial is the "Standard" condition where balance is tested with eyes open and two feet on the BTrackS Balance Plate's firm surface. The second (i.e. eyes closed on a firm surface), third (eyes open on a "perturbed", foam surface), and fourth (eyes closed on a foam surface) trials to give an indication of how proprioception, vision and vestibular information are respectively used for balance.

	Standard (Eyes Open/Firm)	Proprioception (Eyes Closed/Firm)	Vision (Eyes Open/Foam)	Vestibular (Eyes Closed/Foam)
Baseline	22 Path Length (cm)	40 Path Length (cm)	44 Path Length (cm)	102 Path Length (cm)
	Percentile=18%	Percentile=5%	Percentile=11%	Percentile=12%
Post-Baseline	18 Path Length (cm)	22 Path Length (cm)	27 Path Length (cm)	48 Path Length (cm)
	Percentile=36%	Percentile=56%	Percentile=53%	Percentile=84%

When compared to healthy adults of the same sex, the most recent Baseline mCTSIB results showed bottom quartile performance in four conditions (Standard, Proprioception, Vision, Vestibular). The most recent Post-Baseline mCTSIB test results show bottom quartile performance in zero conditions.

The composite mCTSIB results were 208cm at Baseline and 115cm at Post-Baseline. This is an improvement of 93cm.

Baseline Results

DATE	STD	%	PRO	%	VIS	%	VES	%	COMP	%	NOTE
2/1/2018 15:47	22	18	40	5	44	11	102	12	208	8	Initial Visit

Post-Baseline Results

DATE	STD	%	PRO	%	VIS	%	VES	%	COMP	%	NOTE
2/8/2018 15:20	23	14	39	7	44	11	78	28	184	12	Wk 1 Follow-up
3/2/2018 16:24	20	26	36	10	40	15	70	41	166	22	Wk 4 Follow-up
4/1/2018 14:27	18	36	33	15	34	29	60	62	145	37	Wk 8 Follow-up
4/28/2018 15:37	19	31	28	31	30	43	55	71	132	52	Wk 12 Follow-up
6/2/2018 15:41	19	31	23	52	25	61	52	76	119	66	Wk 16 Follow-up
8/1/2018 13:44	18	36	22	56	27	53	48	84	115	71	6 Month Follow-up

BALANCE TRACKING SYSTEMS — Modified CTSIB (COP Details) — Name: Sample Profile ID#: XXXXXX — Facility: _____

Modified CTSIB results are typically determined on the basis of Center of Pressure (COP) path length. Some users may have use, however, for additional COP metrics and/or enlarged visualizations of the raw COP data trace. These metrics and visuals are provided below.

	Standard (Eyes Open/Firm)	Proprioception (Eyes Closed/Firm)	Vision (Eyes Open/Foam)	Vestibular (Eyes Closed/Foam)
Baseline	22 ML = 1.1cm AP = 2.4cm Ang = -3deg	40 ML = 1.5cm AP = 4.1cm Ang = -2deg	44 ML = 3.6cm AP = 4.2cm Ang = 19deg	102 ML = 6.8cm AP = 7.7cm Ang = 35deg
Post-Baseline	18 ML = 0.8cm AP = 2.2cm Ang = 3deg	22 ML = 1.5cm AP = 2.9cm Ang = 8deg	27 ML = 1.9cm AP = 5.2cm Ang = 13deg	48 ML = cm AP = cm Ang = deg

A 95% confidence ellipse is fit to the raw data from each test condition in the graphs above. The ellipses are determined as the smallest ellipse that fits 95% of the Center of Pressure (COP) test data within it. Three metrics describing the data are also provided. The ML and AP metrics give the COP range in the medial/lateral and anterior/posterior directions. The Angle (i.e. Ang) metric quantifies the ellipse rotation to the left (negative) or right (positive) of vertical. ML/AP metrics assume alignment along to the cardinal axes of the BTrackS Balance Plate.

Baseline Results

DATE	STD (ML,AP,ANG)	PRO (ML,AP,ANG)	VIS (ML,AP,ANG)	VEST (ML,AP,ANG)
2/1/2018 15:47	1.1, 2.4, -3	1.5, 4.1, -2	3.6, 4.2, 19	6.8, 7.7, 35

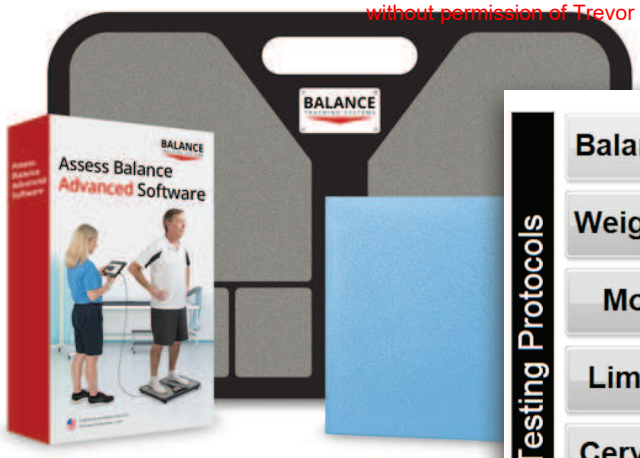
Post-Baseline Results

DATE	STD (ML,AP,ANG)	PRO (ML,AP,ANG)	VIS (ML,AP,ANG)	VEST (ML,AP,ANG)
2/8/2018 15:20	1.2, 2.2, 2	1.5, 3.8, 4	3.4, 4.1, 16	6.2, 7.2, 30
3/2/2018 16:24	1.1, 1.9, -2	1.3, 3.6, 2	2.9, 3.6, 11	5.8, 6.8, 25
4/1/2018 14:27	0.9, 1.7, 0	1.1, 3.1, -2	2.1, 3.6, 10	4.2, 6.6, 21
4/28/2018 15:37	0.7, 1.6, 1	0.9, 2.7, 1	1.9, 3.3, 10	3.3, 5.8, 19
6/2/2018 15:41	0.8, 1.3, 2	0.8, 2.5, 1	1.6, 3.1, 9	3.2, 5.3, 16
8/1/2018 13:44	0.8, 2.2, 3	1.5, 2.9, 8	1.9, 5.2, 13	

Used to understand which of the three sensory systems may be causing a balance deficit - Proprioception, Vision or Vestibular. Norms are provided for each test.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.



Testing Protocols	Training Protocols
Balance & Fall Risk	Left/Right Targets
Weight Distribution	Front/Back Targets
Modified CTSIB	Diagonal Targets
Limits of Stability	Random Targets
Cervical Challenge	Target Tracking
Single Leg Stance	~ Custom Design

Online with the Coupon Code

Laser2025 (Lower case)

$300 off

balancetrackingsystems.com

1-888-811-0054 x701

Assess Balance Advanced System

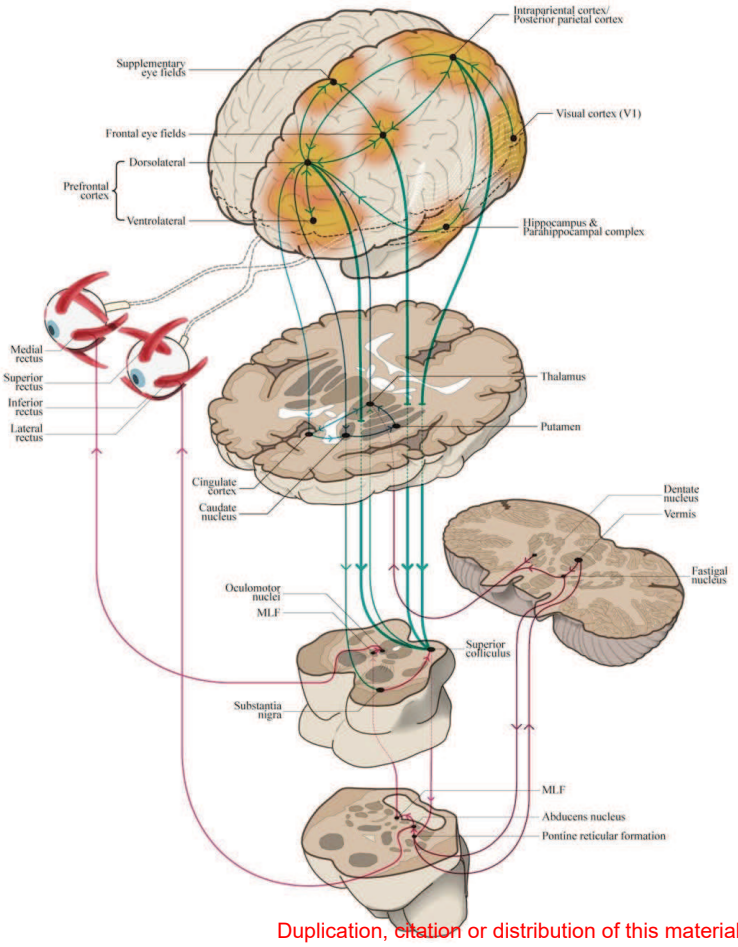
List Price: $3150

Special Seminar Price

$2850

System includes Balance Plate, Foam Pad and Assess Balance Advanced Software

587



ERCHONIA
eCommunity Education

THE EYES ARE A DIAGNOSTIC AND THERAPEUTIC WINDOW FOR A MAJOR PORTION OF THE CENTRAL NERVOUS SYSTEM!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

VESTIBULAR SENSORY MISMATCH

1. USE BTRACKS CDP CTSIB TO HELP ISOLATE DEFICIT AREAS
2. BEDSIDE NEURO EXAM WORK-UP TO ASSOCIATED AREAS (EX. DIX-HALLPIKE FOR VESTIBULAR)
3. RULE OUT NYSTAGMUS. IF DR. BERRY DOES NOT GET THE OPPORTUNITY TO TEACH IT DURING OR AFTER THE SEMINAR PLEASE REFERENCE THE FOLLOWING ON YOUTUBE:

Dr Brock Vestibular Repositioning Maneuvers

4. ONCE NYSTAGMUS IS CLEARED OUT (MOST LIKELY DUE TO BPPV), CONTINUE WITH GAZE STABILIZATION AND VESTBULO-OCULAR REFLEX (VOR'S)
5. UTILIZING THE OCULOMOTOR SYSTEMS WITH GAZE STABILIZATION EXERCISES IN ALL QUADRANTS (CARTESIAN CROSS) CAN BE AN EFFECTIVE WAY TO BEGIN TO STABILIZE THE SPINE POST INJURY, ESPECIALLY WHEN THERE MAY BE CONTRAINDICATIONS TO CHIROPRACTIC MANIPULATION (LIGAMENT INSTABILITY, METABOLIC THRESHOLD ETC)
6. ONCE THE PATIENT IS ABLE TO HOLD GAZE IN ALL REGIONS AND VOR'S ARE INTACT AND ACCURATE, TRANSITION TO SACCADES (REACTION TIME, SPEED AND ACCURACY)
7. FROM THERE TRANSITION INTO SMOOTH PURSUITS. CONSIDER MICROSACCADES WITH GAZE STABILIZATION IN AREAS OF SACCADIC INTRUSIONS
8. CONSIDER DYNAMIC THERAPIES LIKE OPTOKINETIC CONTAMINATION IN THE BACKGROUND WHILE PATIENT DOES TRANSLATION, GET THEM ONTO THE PRACTICE FIELD TO EMULATE THEIR SPORTS ENVIRONMENT ETC.
9. WHEN PROPRIOCEPTION IS DEFICIENT AND CONTRAINDICATIONS ARE RULED OUT, USE YOUR ADJUSTING TECHNIQUES APPROPRIATE FOR THE PATIENT. ALSO CONSIDER FULL SPINE STABILIZATION
10. CLINICAL GEM: DO NOT USE PERTURBED SURFACES DURING ACUTE PHASE SPINAL ORTHOPEDIC REHAB
11. ALSO INCREASE FULL BODY LARGE DIAMETER AFFERENTS (LE SSEP GAIT OR REBUILDER PROTOCOLS, MUSCLE REHAB WITH ISOMETRICS)

CANAL REPOSITIONING POSTERIOR CANAL/EPLEY'S

LEFT POSTERIOR CANAL BPPV EPLEY'S MANEUVER

30 SECONDS AT EACH PRIMARY POSITION BUT GO SLOW

CONSIDER POST RESET CEREBELLAR ISOMETRIC AND RALP OR LARP GAZE STABILIZATION

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CANAL REPOSITIONING HORIZONTAL CANAL

GEOTROPIC VS AGEOTROPIC HORIZONTAL NYSTAGMUS

GEOTROPIC NYSTAGMUS IS WHEN THERE IS A NYSTAGMUS (FAST PHASE) THAT BEATS TO THE GROUND WHEN THE HEAD IS TURNED TO THAT SIDE (IE. LAYING BACK HEAD TO THE RIGHT CREATES A RIGHTWARD BEATING NYSTAGMUS TOWARDS THE FLOOR). THIS IS CLASSIC HORIZONTAL CANALITHIASIS

THIS IS TREATED WITH THE "BBQ ROLL" (LEMPERT 360-DEGREE MANEUVER)
PATIENT STARTS IN POSITION #1 WITH HEAD TURNED INTO THAT INVOLVED SIDE (EX. IF IT WAS A RIGHT DOWNBEATING HORIZONTAL NYSTAGMUS WHEN THEY TURNED THE HEAD TO THE RIGHT, THAT IS THE STARTING POSITION). THIS IS POSITION #1 AND IT'S 30 SECONDS EACH POSITION. ROLL THE HEAD TO POSITION 2 (OPPOSITE SIDE) BUT TAKE YOUR TIME GETTING THERE. TURN ONTO THAT SIDE (POSITION #3), THEN FACE DOWN (POSITION #4) THE ALL THE WAY BACK TO THE STARTING POSITION

AGEOTROPIC IS THE NYSTAGMUS "UPBEATS" TOWARDS THE CEILING OF THE SIDE OF ROTATION (IE. WHEN LAYING BACK HEAD TURNED TO THE RIGHT THE EYES BEAT UP, LEFTWARD TOWARDS THE CEILING). THIS IS DUE TO HORIZONTAL CANAL CUPULOTHIASIS (STUCK TO THE MEMBRANE). CONSIDER GENTLE MASTOID VIBRATION AND OBSERVE IF IT FLIPS TO THE CLASSIC GEOTROPIC NYSTAGMUS (FAST PHASE TOWARDS THE GROUND).
MOVE TO THE MODIFIED GUFONI/APPIANI MANEUVER:

EXAMPLE OF GUFONI MANEUVER FOR A LEFT HORIZONTAL (LATERAL) CANAL AGEOTROPIC NYSTAGMUS (UPBEATING TOWARDS THE CEILING ON THE INVOLVED SIDE)

CANAL REPOSITIONING ANTERIOR CANAL

DOWNBEAT NYSTAGMUS ON DIX-HALLPIKE IT IS THE LEAST COMMON OF THE 3 CANALS

RULE OUT OTHER MECHANISMS (CHIARI, INTRACRANIAL SWELLING, RX) ESPECIALLY IN ABSENCE OF REDUCTION OF SYMPTOMS POST MANEUVER

2 PRIMARY TECHNIQUES FOR ANTERIOR CANAL:

SEMONT LIBERATORY MANEUVER OPPOSITE HEAD POSITION AS POSTERIOR CANAL BPPV

OR THE

DEEP HEAD HANG (SAME FOR EITHER CANAL)

1-2 MINUTES EACH POSITION

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

VESTIBULAR SENSORY MISMATCH

1. USE BTRACKS CDP CTSIB TO HELP ISOLATE DEFICIT AREAS
2. BEDSIDE NEURO EXAM WORK-UP TO ASSOCIATED AREAS (EX. DIX-HALLPIKE FOR VESTIBULAR)
3. RULE OUT NYSTAGMUS. IF DR. BERRY DOES NOT GET THE OPPORTUNITY TO TEACH IT DURING OR AFTER THE SEMINAR PLEASE REFERENCE THE FOLLOWING ON YOUTUBE:

Dr Brock Vestibular Repositioning Maneuvers

4. ONCE NYSTAGMUS IS CLEARED OUT (MOST LIKELY DUE TO BPPV), CONTINUE WITH GAZE STABILIZATION AND VESTBULO-OCULAR REFLEX (VOR'S)
5. UTILIZING THE OCULOMOTOR SYSTEMS WITH GAZE STABILIZATION EXERCISES IN ALL QUADRANTS (CARTESIAN CROSS) CAN BE AN EFFECTIVE WAY TO BEGIN TO STABILIZE THE SPINE POST INJURY, ESPECIALLY WHEN THERE MAY BE CONTRAINDICATIONS TO CHIROPRACTIC MANIPULATION (LIGAMENT INSTABILITY, METABOLIC THRESHOLD ETC)
6. ONCE THE PATIENT IS ABLE TO HOLD GAZE IN ALL REGIONS AND VOR'S ARE INTACT AND ACCURATE, TRANSITION TO SACCADES (REACTION TIME, SPEED AND ACCURACY)
7. FROM THERE TRANSITION INTO SMOOTH PURSUITS. CONSIDER MICROSACCADES WITH GAZE STABILIZATION IN AREAS OF SACCADIC INTRUSIONS
8. CONSIDER DYNAMIC THERAPIES LIKE OPTOKINETIC CONTAMINATION IN THE BACKGROUND WHILE PATIENT DOES TRANSLATION, GET THEM ONTO THE PRACTICE FIELD TO EMULATE THEIR SPORTS ENVIRONMENT ETC.
9. WHEN PROPRIOCEPTION IS DEFICIENT AND CONTRAINDICATIONS ARE RULED OUT, USE YOUR ADJUSTING TECHNIQUES APPROPRIATE FOR THE PATIENT. ALSO CONSIDER FULL SPINE STABILIZATION
10. CLINICAL GEM: DO NOT USE PERTURBED SURFACES DURING ACUTE PHASE SPINAL ORTHOPEDIC REHAB
11. ALSO INCREASE FULL BODY LARGE DIAMETER AFFERENTS (LE SSEP GAIT OR REBUILDER PROTOCOLS, MUSCLE REHAB WITH ISOMETRICS)

GRAVITY RESET METHOD

1. CLEAR OUT SEMICIRCULAR CANALS (SEE PRIOR SLIDES)
2. OTOLITHIC MANEUVERS. TRANSLATION TO STABILIZE OVERACTIVE OTOLITH AND ANGULAR CANALS. CONSIDER STACKING WITH PROPRIOCEPTIVE MODALITIES TO THE LOWER EXTREMITIES
3. MATCH EYES TO HEAD WITH LASER TARGETS
4. SET WITH INTERACTIVE METRONOME/CEREBELLAR INTEGRATION. STACK WITH ERCHONIA LASER
5. TRANSCRANIAL LOW LEVEL LASER FINISH WITH OXYGEN (IDEALLY FX WITH CONCENTRATOR)

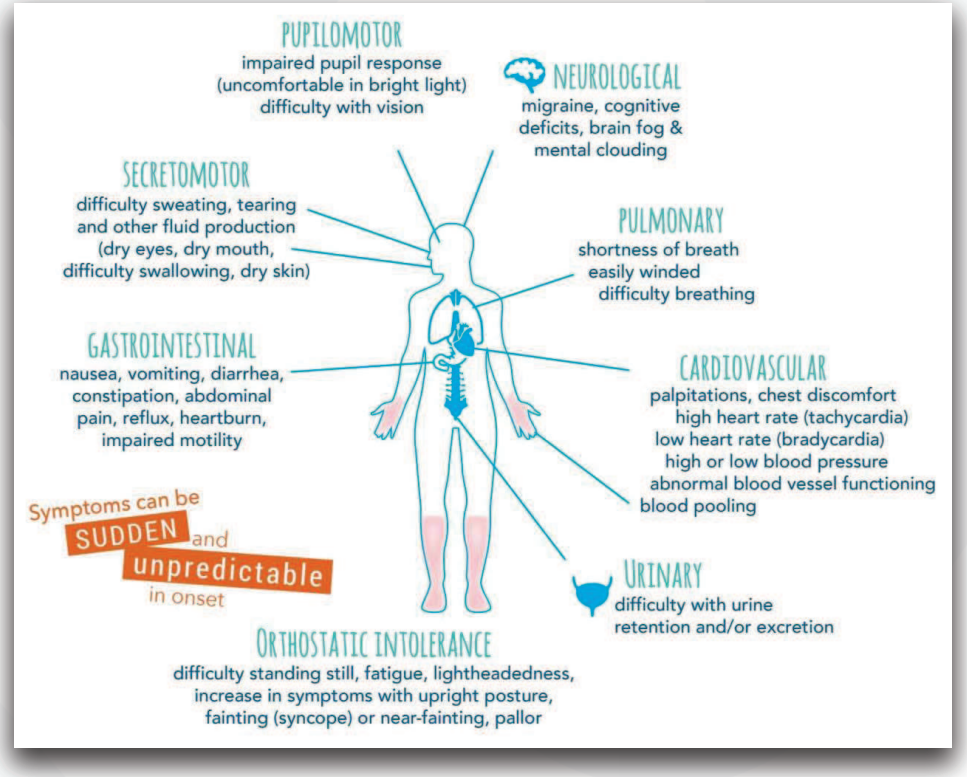
ATTEND A FULL GRAVITY RESET METHOD SEMINAR IN 2025 OR ON DEMAND FROM 2024 (NEURDSOLUTIONS)!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

GRAVITY RESET METHOD 3.0

DYSAUTONOMIA/POTS



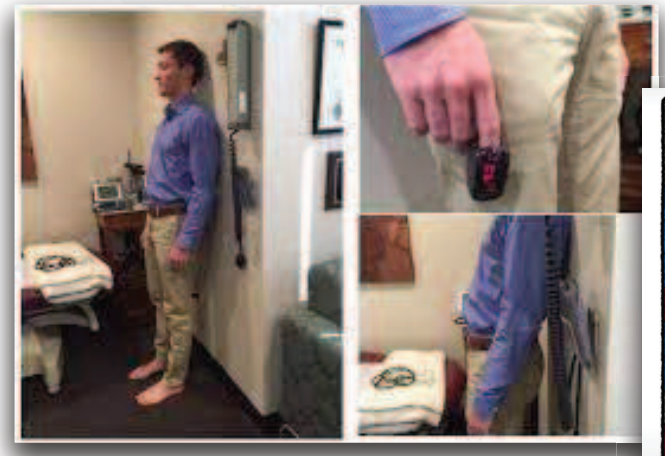
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

POTS/DYSAUTONOMIA

1. SEE SUBJECTIVE SYMPTOM LIST. **** CLUE: DO SYMPTOMS QUICKLY RESIDE UPON LAYING DOWN???***
2. CONFIRMATION WITH 10 MIN TILT TABLE TEST OR NASA LEAN TEST. HRV TESTING AND OTHER ANS MARKERS SHOULD BE PERFORMED AS WELL
3. TREATMENTS SHOULD INCLUDE (12 VISIT TX "TRIAL" MIN) MAY TAKE MANY MONTHS TO FULLY STABILIZE:
4. VAGAL QUAD (SEE VIDEO)
5. TILT TABLE CLEARANCE (SEE VIDEO), OVAR THERAPIES
6. ALTERNATE VISIT FINISHES WITH FX 405 MASTER BRAIN AND IMMUNE HALF
7. OTOLITHIC RESET (EPLEY CHAIR OR "GRAVITY RESET METHOD")
8. PROGRESS INTO UPRIGHT MODALITIES LIKE FNOR COMBO (SEE VIDEO)
9. ADEQUATE WATER INTAKE AND ADRENAL SUPPORT AS INDICATED
10. NUTRITIONAL SUPPORT MAY INCLUDE A GOOD MULTI, D/A (5K/10K), PRM/SPM FROM OMEGA 3, NAC AND NATTOKINASE. OTHERS MAY INCLUDE VINPOCETINE, DIOSMIN, ENDOCALYX, COQ10, RESVERO/TURMERO

NASA LEAN TEST FOR AUTONOMIC DYSFUNCTION (POTS, ORTHSTATIC INTOLERANCE ETC)



10 MINUTE TEST
FEET 12 INCHES FROM WALL
MONITOR HEART WITH WITH PULSE OX
SECONDARY BLOOD PRESSURE READINGS
VS
USE TRUE TILT TABLE TEST IF AVAILABLE

ACTIVITY	BLOOD PRESSURE	PULSE	SYMPTOMS
Laying down resting	99/68 Pulse Pressure: 31	P-68	
Standing at 0 minutes	99/72	P-90	
Standing at 1 minute	90/74	P-100	Mild weakness all over, heavy feeling in legs
Standing at 2 minutes	101/74	P-94	Dependent rubor hands, facial pallor
Caution! If the test is stopped at 2 minutes, 53% of POTS patients will be missed!			
Standing at 3 minutes	104/84	P-111	Hands tingling
Standing at 4 minutes	104/83	P-101	Nausea
Standing at 5 minutes	unable to measure	unable to measure	unable to measure
Caution! If the test is stopped at 5 minutes, 27% of POTS patients will be missed!			
Standing at 6 minutes	88/62 Pulse Pressure: 26	P-132	Palpitations
			Palpitations
HR was 68 when supine then jumped to 132 (+64 BPM) just before near syncope.			

*Standing with shoulders against wall, feet 12" from the wall.

rise in heart rate, as well as a very notable
ase in pulse pressure (the difference between

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Vagus Nerve Stimulation in ME (MYALGIC ENCEPHALOMYELITIS)/ CFS, FM, POTS and Long COVID – Is its Promise Being Fulfilled?

by **Cort Johnson** | **Dec 17, 2022**

Sleep

Take just one major concern – sleep. Numerous studies have found reduced parasympathetic nervous system activity (rest/digest) during sleep. Even the CDC – which was not, back then, exactly known for validating findings – **found autonomic hypervigilance (increased HR, reduced HRV, higher norepinephrine, as well as lower aldosterone levels) during sleep in ME/CFS** (CHRONIC FATIGUE SYNDROME).

The **2018 Finnish study**, and the **2010**, **2011**, and **2020** Aussie studies all highlighted the low HRV (sympathetic nervous system dominance) found in ME/CFS during sleep. Similar findings showed up in fibromyalgia in **1998**, **2009**, **2011**, **and 2013**. It was remarkable to see two studies – **one in ME/CFS and one in fibromyalgia – pour through a maze of sleep findings only to conclude that reduced heart rate variability (i.e. reduced vagus nerve, or parasympathetic nervous system activity) was the main factor behind the poor sleep in these diseases.**

In 2019, Boissoneault and Staud produced a nice "bifecta" when they showed that lower heart rate variability (read lower parasympathetic nervous activity), in conjunction with lower blood flows to the brain, predicted how fatigued people with ME/CFS were.[599]

VAGAL NERVE STIMULATION

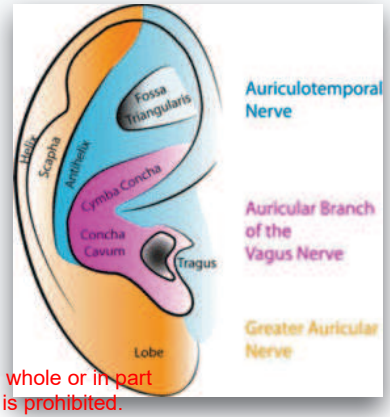
Vagus Nerve Stimulation in ME/CFS, FM, POTS and Long COVID – Is its Promise Being Fulfilled?

by **Cort Johnson** | **Dec 17, 2022**

Some things (meditation, Tai Chi, **exercise in FM**) have been shown to improve parasympathetic nervous system functioning, but not everyone wants to take the time to give these a shot and not everyone will benefit, as well. Low-dose trazodone and doxepin elixir can help keep a lid on the fight/fight response during sleep as well, but like any drugs, they don't help everyone.

POTS crowd is leading the way. At the 2022 IACFS/ME International Conference, Lauren Stiles – the leader of Dysautonomia International – now in its 10th year (that went fast!) had some good news.
After noting that VNS had helped her a lot, Stiles, who is usually quite conservative in her statements, stated that she believes **VNS is going to be a big deal in both POTS and ME/CFS. She believes its ability to reduce inflammation is going to be key.**

Several different parameters can be manipulated, making vagus nerve stimulation a complex beast. For instance, vagus nerve stimulation at 25Hz, a 250µs pulse width, with 30 seconds on/30 seconds off, for 4 hours a day is typically used to reduce the number of seizures in patients with epilepsy.
We don't know which settings work best in POTS, ME/CFS/FM or long COVID, but a start has been made in POTS. Schiffer stimulated the right cymba conchae in 1-hour stimulation blocks, 4x's a day for 14 days using the Nemos© device (Cerbomed, Germany).
She used a pulse width of 200µs and a squared impulse waveform which caused the stimulator to switch on and off every 30 seconds. She set the stimulation frequency at 25Hz and the electrical current was set to the highest intensity each participant could tolerate without causing discomfort.



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Vagus Nerve Stimulation in ME/CFS, FM, POTS and Long COVID – Is its Promise Being Fulfilled?

by Cort Johnson | Dec 17, 2022

POTS

- A University of Oklahoma study using the **Parasym (TM) TENS device** is assessing its effectiveness in reducing autoantibodies associated with POTS.
- Another **University of Oklahoma study** will assess the effect vagal stimulation on the tragus of the ear has on antibody suppression, inflammatory inhibition, and symptom improvement in 80 POTS patients.
- Another POTS study is combining VNS with Mestinon.

Fibromyalgia

- Sixty FM patients are participating in a **4 week Massachusetts General Hospital** study that started last year.

Long COVID and ME/CFS

- David Putrino's 6-week trial of people with **long COVID who meet the criteria for ME/CFS** will apply the Parasym device to the left tragus of the ear. Side effects and symptom improvement will be assessed.

Long COVID

- David Putrino's 6-week-long placebo controlled, randomized controlled **long-COVID trial** going on at the Icahn School of Medicine at Mount Sinai will assess cytokines (IL-1, IL-6, IL-10), morning salivary cortisol, c-reactive protein, end-tidal CO2, and many different symptoms, including post-exertional malaise (PEM).
- The **Colosat trial** will assess the effects of 8 weeks of the Parsym device on pain on 20 patients with long COVID.
- A **Casa Colina, California Hospital trial** will assess the effects of 7 weeks of the Parasym device on 20 long-COVID patients using neuroimaging, genetics, blood biomarkers, and symptoms, with a focus on brain fog.

THE ENERGY FAILURE MODEL OF NEURODEGENERATION IS ROBUST IN THE LITERATURE.
UTILIZING YOUR ERCHONIA WAVELENGTHS FOR AUTONOMIC/BLOOD PRESSURE REGULATION:

RED
VS.
VIOLET
VS.
GREEN

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DYSAUTONOMIA

GARY M.
MAYO CLINIC
TILT TABLE
ANS
TESTING

POTS HR
ORTHOSTATIC
VS
BLOOD
PRESSURE

Comments on Sudomotor Test: QSART responses were reduced at the distal leg and normal at all other sites.

DEEP BREATHING & VALSALVA MANEUVER RESPONSES

Result	Normal Cutoff	
Deep Breathing		Heart Rate Range (bpm)
7.9	> 9	
Valsalva Maneuver		Valsalva Ratio
1.38	> 1.36	

Comments on Deep Breathing: Heart rate responses to deep breathing were reduced.

Comments on Valsalva maneuver:

(A) Heart rate responses to the Valsalva maneuver were normal.
(B) Beat-to-beat blood pressure responses to the Valsalva maneuver showed incomplete return of late phase II to baseline, a prolonged BP recovery time, and a blunted phase IV response.

BLOOD PRESSURE AND HEART RATE RESPONSES TO TILT

	BP (mmHg)	Heart Rate (BPM)
Supine	166/98	57
Tilt 1 min	124/91	71
Tilt 3 min	127/92	82
Tilt 5 min	119/90	85
Tilt 7 min	124/87	85
Tilt 10 min	105/85	86
Tilt -1 min	149/92	57

Comments on Tilt: Patient was tilted for 10 minutes. Orthostatic hypotension was detected. Heart rate response was fairly robust. The patient reported paresthesias.

DYSAUTONOMIA

GARY M. 12/6/22
TILT TABLE INTEGRATIVE THERAPY FOR POTS
AND ORTHOSTATIC HYPOTENSION
SUPINE HEART RATE 68
TILT TABLE 70 DEGREES HR SPIKE TO 88
EVRL LASER RIGHT SCM
10-10-10-10 “VAGUS” SETTING
GLUT ISOMETRIC WITH MEDIUM THERABAND
“NO-NO” GAZE STABILIZATION
WITH SHORT BURST VALSALVA’S
RESTING 4:4:8 BREATHING
HR DECREASE TO 79

(NO-NO WITH VAGAL STIM
TO PROMOTE IML/HR INHIBITION
WHILE DOING SHORT BURST
PHASE 1 VALSALVA’S MANEUVER
FOR TRANSIENT RISE IN BP.
GLUT ISOMETRICS FOR
CEREBELLAR MODULATION)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DYSAUTONOMIA

ERCHONIA eCommunity Education

GARY M. 12/6/22
TILT TABLE INTEGRATIVE THERAPY FOR POTS
TRANSITIONED FROM EVRL COMBO THERAPY
TO GVL
SUPINE HR 68
75 DEGREE CHALLENGE FROM 70
HR FROM 88 SPIKE
TO 74
(VS 70 DEGREES 88 TO 79 EVRL)

BOTH DEVICES CAN BE USED
BUT CLINICAL TRIALS CONTINUE TO SHOW
GVL MAY BE SUPERIOR
TO EVRL IN
AUTONOMIC REGULATION

PATIENT WAS PRESCRIBED HOME
OFF VERTICAL AXIS THERAPIES DAILY
TO FACILITATE NEUROPLASTICITY
USING "NO-NO" WITH GLUT BAND
ISOMETRICS AND 4:4:8 RESTS
WHILE MONITORING WITH
PULSE OXIMETER

© 2022 Erchonia Corporation

605

AMAZON

<$160

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

POTS/AUTONOMIC DYSFUNCTION TREATMENT STRATEGY SUMMARY

ERCHONIA eCommunity Education

1. CLEAR OUT BIOMECHANICAL DYSFUNCTION AS INDICATED (CHIROPRACTIC ADJUSTMENTS, SOFT TISSUE WORK ETC)
2. VAGAL NERVE STIMULATION PROTOCOL
3. GRAVITY RESET METHOD (CANAL AND OTOLITHIC RESET FOLLOWED BY VISUAL MATCH AND CEREBELLAR IM/EXERCISES)
4. TILT TABLE CLEARANCE
5. OTHER REHAB AS INDICATED (FNOR, SUBMAX EXERCISE, OTHER FN/EXERCISE)
6. ANCILLARY TREATMENTS SUCH AS HBOT, PEMF AND NEUROFEEDBACK
7. FINISH WITH PASSIVE LASER STACK (IE. FX WITH BINAURAL AND CONCENTRATOR)

TBI

ERCHONIA eCommunity Education

Review Article

Physical exercise ameliorates deficits induced by traumatic brain injury

T. Archer, K. Svensson, M. Alricsson

First published: 11 January 2012 https://doi.org/10.1111/j.1600-0404.2011.01638.x Citations: 36

Neurobiology of Disease
Volume 54, June 2013, Pages 252-263

Late exercise reduces neuroinflammation and cognitive dysfunction after traumatic brain injury

Author links open overlay panel
Chun-Shu Piao, Bogdan A. Stoica, Junfang Wu, Boris Sabirzhanov, Zaorui Zhao, Rainier Cabatbat, David J. Loane, Alan I. Faden

Physical activity can reduce inflammation and facilitate recovery after brain injury. Here, we investigated the time-dependent effects, and underlying mechanisms of post-traumatic exercise initiation on outcome after moderate traumatic brain injury using a well-characterized mouse controlled cortical impact model. **Late exercise initiation beginning at 5 weeks after trauma, but not early initiation of exercise at 1 week, significantly reduced working and retention memory impairment at 3 months, and decreased lesion volume compared to non-exercise injury controls.** Cognitive recovery was associated with attenuation of classical inflammatory pathways, activation of alternative inflammatory responses and enhancement of neurogenesis. In contrast, early initiation of exercise

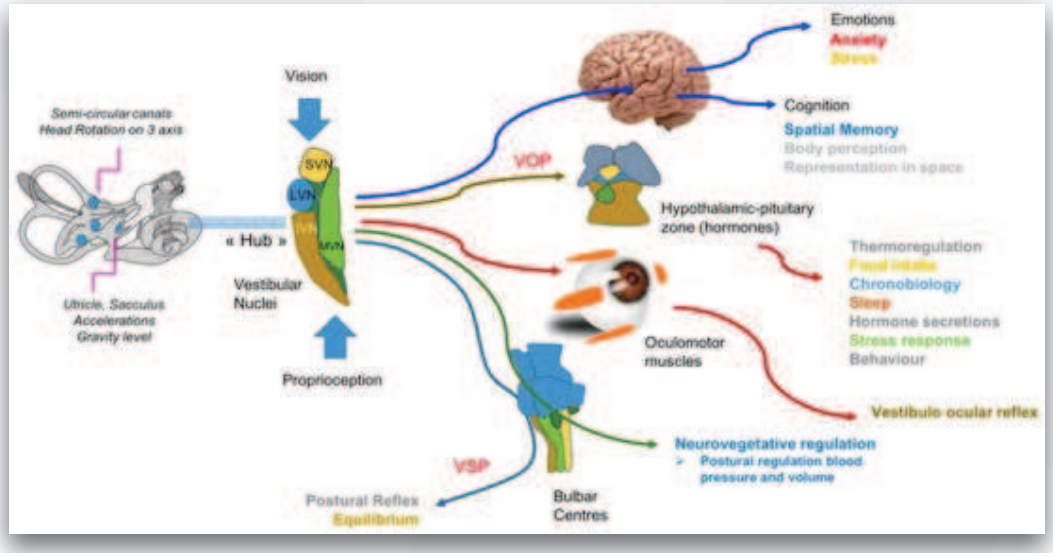
CLINICAL TAKE HOME: PHYSICAL EXERCISE CAN BE AN INDICATED INTERVENTION POST TBI PROMOTING CELLULAR MECHANISMS SUCH AS INDUCING BDNF AND NEUROIMMUNOREGULATION, PROMOTING NEUROCHEMISTRY OUTPUT SUCH AS DOPAMINE AND SEROTONIN AND MUCH MORE. HOWEVER, IT IS CRITICAL TO MONITOR AUTONOMICS AND SYMPTOM STATUS TO NOT EXCEED METABOLIC THRESHOLD!

LOW LEVEL LASER THERAPY CAN DRAMATICALLY IMPROVE METABOLIC THRESHOLD

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE VESTIBULAR SYSTEM - MORE THAN JUST BALANCE



We know that specific hormonal profiles can promote the appearance of vestibular disorders. We also know that the expression of vertigo syndrome is accompanied by measurable hormonal variations. The link between endocrine deregulation and vestibular alterations therefore no longer needs to be proven. However, there are still few data on their precise correlations with the vertigo syndrome. This study was undertaken with the aim to deliver an extensive review of the hormonal alterations linked to vestibular disorders

Hormones and Vestibular Disorders: The Quest for Biomarkers

by **Rhizlane El Khiati** 1,2,**Brahim Tighilet** 1,2,**Stephane Besnard** 1,2 and**Christian Chabbert** 1,2,*
BRAIN SCI 2022

DR. TAUBER

WORST CASE KILLER IS GAP IN CARE. DOCUMENT EVERYTHING THAT HAPPENED IN THAT GAP SUCH AS THEY DID MASSAGE, OUT OF TOWN, HOME EXERCISES ETC….MUST DOCUMENT. YOUR OFFICE SHOULD FOLLOW UP WITH THE PATIENT A FEW TIMES THEN CALL THE ATTORNEY SO THE ATTORNEY CAN ALSO GET THEM TO COMPLY TO TREATMENT

IF A PATIENT MARKS TBI/BRAIN SYMPTOMS ON THEIR INTAKE YOU CAN'T IGNORE THEM. YOU ARE RESPONSIBLE FOR THEM EVEN IF YOU DON'T KNOW NEURO!

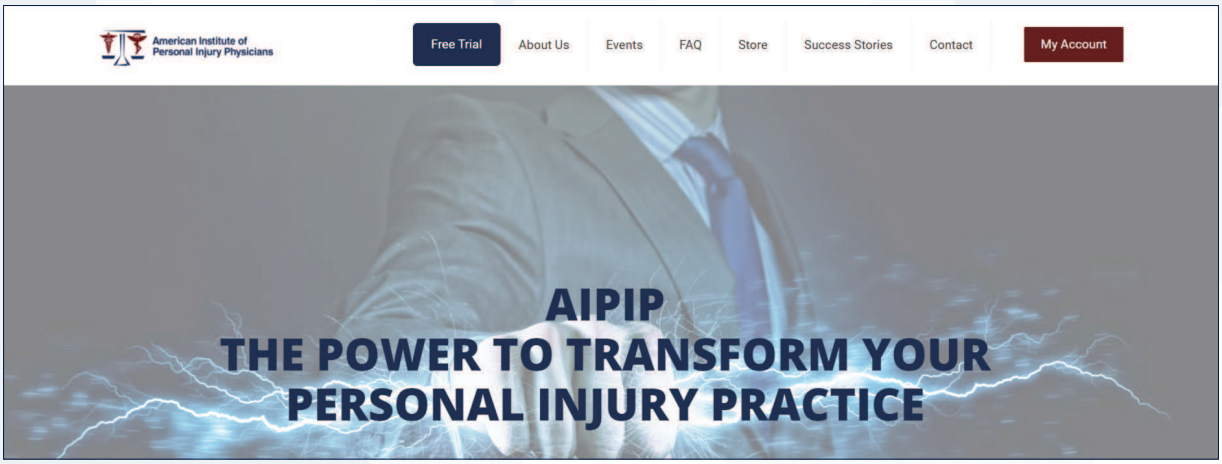
TBI AND SPINAL LIGAMENT LAXITY ARE IN ABOUT 70% OF MOTOR VEHICLE ACCIDENTS BUT MOST ARE MISSED. LITERATURE SAYS 50-70% DR. TAUBER SAYS ABOUT 80%

DTI TEST IS A GREAT OBJECTIVE MARKER TO BUILD A TBI CASE. WATCH THE CASE TYPE AND TALK TO AN ATTORNEY TO MAKE SURE THE CASE IS WORTH IT

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

AIPIP.COM



© AIPIP, SS, LLC All Rights Reserved

PI Success Blueprint System

What We Do

Our Mission:

We help providers currently doing Personal Injury maximize this portion of their practice, improve case outcomes, increase profitability and get more referrals without spending years trying to figure it out on their own

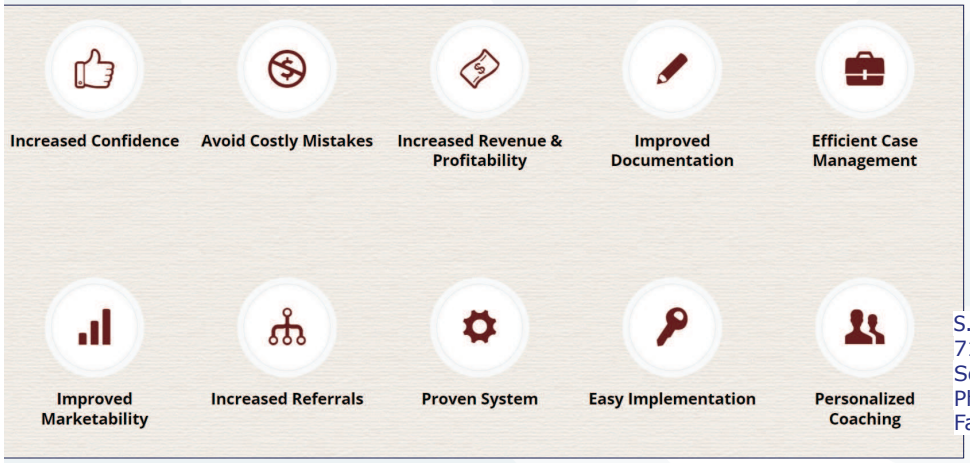
Mindset - Strategy - Tactics

© AIPIP, SS, LLC All Rights Reserved

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

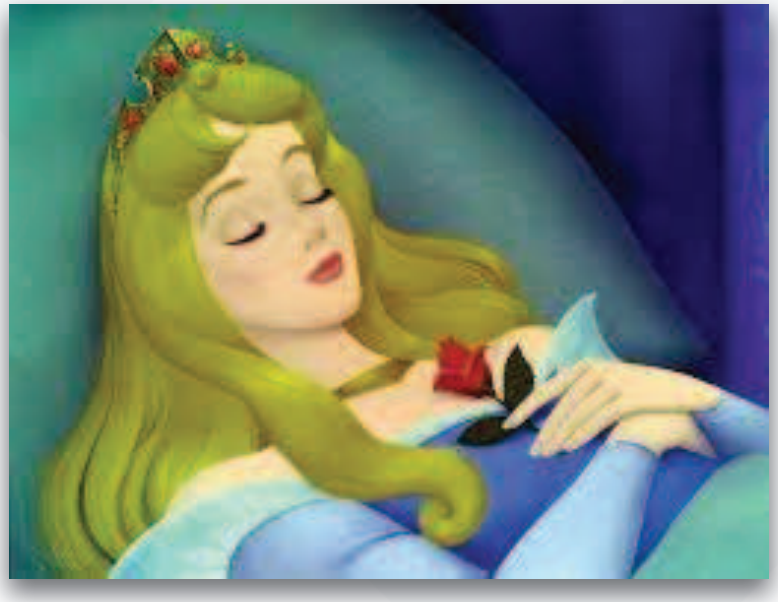
Benefits of AIPIP



S. Scott Tauber, DC, DABCO, CPC
7150 E. Camelback Road, Suite 444
Scottsdale, AZ 85251
Phone (888) 924-7479
Fax (888) 841-8585

© AIPIP, SS, LLC All Rights Reserved

SLEEP



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP: NEW GUIDELINES

ERCHONIA eCommunity Education

Dementia: Early bedtime, sleeping over 8 hours linked to increase risk

The dementia risk was 69% greater for individuals who slept over 8 hours, versus 7-8 hours. The risk was also twice as high for those who went to sleep before 9:00 p.m., versus 10:00 p.m. or later (but go before midnight)

CONVERSELY, In a long-term study, Harvard Medical School followed 2,800 individuals ages 65 and older. **Researchers found that individuals who slept under five hours per night were twice as likely to develop dementia compared to those who slept six to eight hours per night. A different study of nearly 8,000 participants found a 30% increase in dementia risk was associated with sleeping six hours or less at age 50, 60 and 70, compared to a normal sleep duration of seven hours.**

SUMMARY: GO TO BED BETWEEN 10-11 PM AND SLEEP 7.5 HOURS. HOWEVER…

Associations of sleep timing and time in bed with dementia and cognitive decline among Chinese older adults: A cohort study

Rui Liu MD, Yifei Ren MD, Tingting Hou MD, PhD, Xiaoyan Liang MD, Yi Dong MD, Yongxiang Wang MD, PhD, Lin Cong MD, PhD, Xiang Wang MD, PhD, Yu Qin MD … See all authors

First published: 21 September 2022

SLEEP HYGIENE

ERCHONIA eCommunity Education

Why Do Some People Thrive on So Little Sleep?

Short sleepers cruise by on four to six hours a night and don't seem to suffer ill effects

SUMMARY:

- **SHORT SLEEPERS MAY FUNCTION OPTIMALLY AND NOT BE SUBJECT TO HARMFUL EFFECTS OF SLEEP "DEPRIVATION" IN RELATION TO THE STANDARD NORMS**
- **GENE MUTATIONS MAY BE THE CAUSE OF SHORT SLEEPERS. PARTICULARLY OF SNP's *DEC2, ADRB1, GRM1 AND NPSR1***
- **THESE GENETIC MUTATIONS ALTER OREXIN LEVELS (OREXIN DEFICIENCY IS THE PRIMARY CAUSE OF NARCOLEPSY). SHORT SLEEPER MUTATIONS CREATE HIGHER LEVELS OF OREXIN**
- **IN ANIMAL STUDIES WITH THESE MUTATIONS, NOT ONLY DO THE SHORT SLEEPER GENE SNP's PERFORM WELL, ARE AMBITIOUS, ENERGETIC AND OPTIMISTIC, THEY ARE MORE RESILIENT AGAINST STRESS, HAVE HIGHER PAIN THRESHOLDS AND MAY ACTUALLY LIVE LONGER!**
- **SHORT SLEEPER GENES HAVE EVEN BEEN SHOWN TO BE MORE EFFICIENT AT REMOVING BAD PROTEINS LIKE AMYLOID FORMS AND TAU THAN REGULAR SLEEPERS**

ANNUAL REVIEW OF GENOMICS AND HUMAN GENETICS Volume 25, 2024

Review Article Open Access

The Genetics of Human Sleep and Sleep Disorders

- Xianlin Zou1, Louis J. Ptáček1,2,3,4, and Ying-Hui Fu1,2,3,4
- View Affiliations

olume 25, Issue 4103964April 15, 2022*Open Access*

Familial natural short sleep mutations reduce Alzheimer pathology in mice

Qing Dong1,6

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP CLINICAL SUMMARY

- SLEEP IS THE NUMBER 1 DETOX SYSTEM FOR BRAIN AND NEURODEGENERATION. WE FLUSH THE TOILET NIGHTLY WITH CSF LIKE A FAUCET TURNING ON. CELLS ACTUALLY SHRINK IS SIZE UP TO 60% ALLOWING A CSF BATH TO BE WASHED AWAY VIA THE GLYMPHATIC WASTE SYSTEM…RECENT SLYM LAYER TIED DIRECTLY INTO LYMPHATIC SYSTEM AND GLIAL CELLS. THE IMMUNE SYSTEM OF THE BRAIN HOVERS IN THIS MENINGEAL LAYER. LLLT AT NIGHT CLEARS OUT EVEN MORE AB AND TAU PROTEINS.
- IDEAL SLEEP CYCLE FOR ADULTS BETWEEN 7-8 HOURS (IDEAL IS 7.5 FOR 90 MINUTE CYCLE) WITH APPROX. 9:30-11:30 START TIME (NOT BEFORE 9 OR AFTER MIDNIGHT). HOWEVER, GENETIC AND OTHER VARIABLES FACTOR INTO THIS CYCLE OPTIMIZATION
- USE CIRCADIAN RYTHMS. WAKE UP AT SUNRISE AND GO OUT FOR A WALK TO GET DIRECT SUNLIGHT EXPOSURE/GROUNDING
- AVOID BLUE LIGHT ESPECIALLY AFTER DARK GETTING CLOSER TO BEDTIME, USE COMPUTER FILTERS, NIGHT SETTING ETC
- PRECURSORS AND CO-FACTORS FOR SLEEP. SEROTONIN (FROM TRYPTOPHAN), GUT HEALTH/PROBIOTIC
- PROBIOTIC STRAINS SLEEP SPECIFIC INCLUDE LACTOBACILLUS HELVETICUS, ACIDOPHILUS AND REUTERI NK33, ADOLESCENTIS NK 98, LACHNOSPIRACEAE UCG004, BIFIDO INFANTIS/BREVE/LONGUM, AND ODORIBACTER. BAD FOR INSOMINA = SELENOMONADALES AND NEGATIVICUTES
- MELATONIN AND CBD HELP GET INTO A RELAXED ALPHA WAVE STATE BUT NOT NECESSARILY DEEP DELTA/REM
- MELATONIN USE IN TRAVEL, POST-CONCUSSION, COVID/LONG COVID AND SOME NEURODEVELOPMENTAL LIKE AUTISM.
- USE CR IF YOU DO
- AVOID IN TH1 DOMINANT AUTOIMMUNE CONDITIONS SINCE MELATONIN PROMOTES TH1 SO WATCH IN CONDITIONS LIKE MS, IBD/CHRON'S/CELIAC, HASHIMOTO'S, RHEUMATOID
- WATCH GENE SNPS FOR NTS MTHFR AND COMT
- CO FACTOR SUPPLEMENTATION (SEE SLIDE)
- MAGNESIUMS FOR SLEEP #1 LYSINATE/GLYCINATE, #2 L THREONATE
- FASTING? DYSGLYCEMIA, PREGNENOLONE
- INFLAMMATION SOURCES AND MRA
- **SOUND DEVICES: PINK VS WHITE VS BROWN NOISE. BRAIN TAP. SOLFEGGIO (432 HZ). BINAURAL BEATS (10 HZ TO WIND DOWN, 1-4 HZ DELTA FOR DEEP SLEEP AND 4-8 HZ THETA FOR REM SLEEP) MOST OF THESE CAN BE FOUND FOR FREE.**

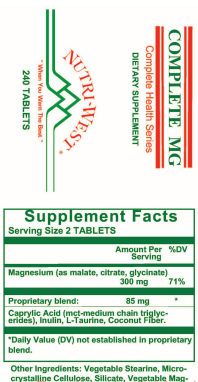
SLEEP HYGIENE

The truth about magnesium glycinate's effect on your sleep quality

A landmark 2012 study published in the Journal of Research in Medical Sciences found that **magnesium supplementation significantly improved sleep efficiency by 16%, increased sleep time by 47 minutes,** and reduced early morning awakening in elderly participants with insomnia. More recent studies in 2023 have corroborated these findings across different age groups, suggesting broad applicability for sleep support.

Its primary action involves GABA receptor activation and melatonin regulation, while simultaneously supporting cortisol reduction and muscle relaxation.

DOSE: MINIMUM 200 MG 1 HOUR BEFORE BED. STACK WITH 1 GRAM OF MAGNESIUM L-THREONATE



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MAGNESIUM

MAGNESIUM: EVERY CELL IN THE HUMAN BODY NEEDS MAGNESIUM. INVOLVED IN OVER 300 PROCESSES IN THE HUMAN BODY AND IS THE 4TH MOST ABUNDANT MINERAL IN HUMANS. 60% IS STORED IN BONES AND THE REMAINDER IN OTHER TISSUES. GOOD FOOD SOURCES INCLUDE GREEN LEAFY VEGETABLES, NUTS, LEGUMES, MEAT, POTATOES, RICE AND WHOLE GRAINS*. OVER 1/2 OF AMERICANS ARE MAGNESIUM DEFICIENT.

MAGNESIUM L-THREONATE (MAGTEIN): CROSSES THE BLOOD BRAIN BARRIER, PROTECTS AND REPAIRS THE BBB. NMDA "PLUG" PROTECTS AGAINST GLUTAMATE EXCITOTOXICITY. PROMOTES HEALTHY NEUROPLASTICITY/SYNAPTIC FUNCTION. PROMOTES HEALTHY COGNITION AND MEMORY, AND MITIGATES COGNITIVE DECLINE, DEPRESSION AND OTHER NEUROLOGICAL DISORDERS.

MAGNESIUM MALATE (DI-MAGNESIUM): MG AND MALATE HIGH IN FRUIT AND WINE. ATP SYNTHESIS/ENERGY AND PAIN RELIEF. GENTLE ALUMINUM CHELATOR. HIGH ABSORPTION RATE AND LOW SIDE EFFECTS.

MAGNESIUM LYSINATE/GLYCINATE: MG AND AA'S LYSINE AND GLYCINE. = RELAXATION AND CALMING. USED IN ANXIETY AND INSOMNIA.

MAGNESIUM CITRATE: MG AND CITRIC ACID = LAXATIVE. HIGH LEVEL OF SIDE EFFECTS AND DRUG INTERACTIONS. CHEST PAINS, DIARRHEA, NAUSEA/VOMITING, HEART REGULATION AND MORE. HIGH IN CITRUS FOODS AND OFTEN USED IN FOODS AND SUPPLEMENTS.

MAGNESIUM OXIDE: MG AND OXYGEN ALSO = LAXATIVE (IE MILK OF MAGNESIA) ALSO USED IN HEARTBURN AND INDIGESTION. POOR ABSORPTION (APPROX. 4%) VS OTHER MG FORMS. MOST COMMON FORM OF MG USED IN SUPPLEMENTS.

MAGNESIUM CHLORIDE: SIMILAR FUNCTION AS OXIDE WITH SLIGHTLY HIGHER ABSORPTION. ALSO OFTEN USED IN TOPICALS FOR MUSCLE RELAXATION/SORENESS.

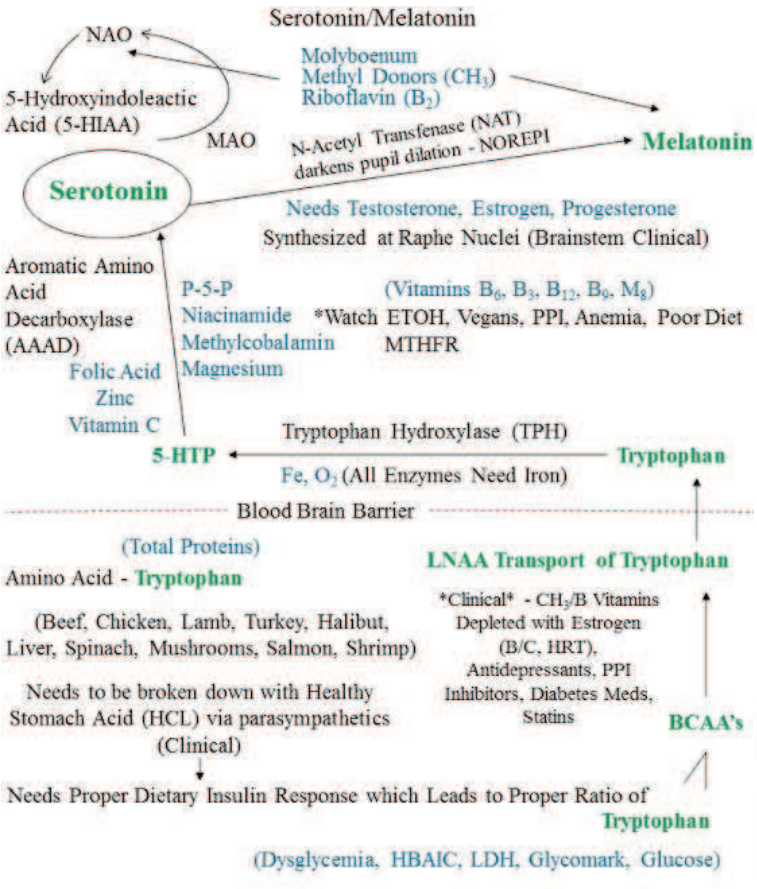
MAGNESIUM LACTATE: MG AND LACTIC ACID. GENTLE ON THE STOMACH VS FORMS LIKE CITRATE. OFTEN USED IN FOOD ADDITIVES.

MAGNESIUM SULFATE: MG, SULFUR AND OXYGEN. = EPSOM SALT. TOPICAL/BATHS FOR SORE MUSCLES/RELAXATION. HARD ON STOMACH.

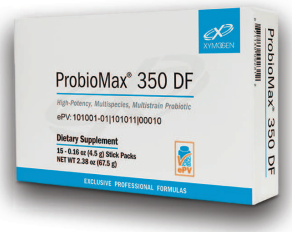
MAGNESIUM OROTATE: MG AND OROTIC ACID. CARDIOVASCULAR SYSTEMS BENEFITS.

Open Access Review
Magnesium in Prevention and Therapy by **Uwe Gröber** 1,*, **Joachim Schmidt** 1 and **Klaus Kisters** 1,2
Nutrients **2015**, 7(9), 8199-8226; **https://doi.org/10.3390/nu7095388**

620
https://doi.org/10.3390/nu7095388



MICRONUTRIENT PANEL!



Bifido Infantis, Breve and Longum. Lactobacillus Helveticus and Acidophilus

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

SLEEP

- POLYSOMNOGRAPHY (CENTRAL VS OBSTRUCTIVE APNEA VS BODY MOVEMENTS)
- C-PAP/BI-PAP, BREATHING STRIPS, BED/MATTRESS POSITION/PILLOW
- TONE OF ORAL-PHARYNGEAL REGION (VAGAL STIM/DIRECT SOFT PALATE STIM) MOUTH TAPING WITH BREATHE RIGHT NASAL STRIPS
- BASE LAB CHEMISTY (ANEMIA PATTERNS, DYSGLYCEMIA, THYROID, SEX HORMONES…HP AXIS)
- ADVANCED LAB MARKERS: MICRONUTRIENT (CO-FACTORS FOR MELATONIN SYNTHESIS)
- ASI/CORTISOL CURVE AND GLYCOGEN STORES. CORTISOL VS MELATONIN
- CYTOKINES AND MESENCEPHALIC RETICULAR ACTIVATING SYSTEM. FOOD SENSITIVITY TESTING, INFECTIOUS DISEASE MANAGEMENT
- PAIN MANAGEMENT
- BASAL GANGLIA ACTIVATION/DISINHIBITION
- QUALITY OF PILLOW, MATTRESS (AGE ETC)
- ELECTRONICS AT NIGHT, AVOID BLUE WAVELENGTHS/NIGHT SETTING, MINIMIZE VISUAL STIMULATION THROUGHOUT DAY/COMPUTER FILTERS
- QEEG/NEUROFEEDBACK
- CBD/MEDICAL MARIJUANA. MELATONIN CR, PROBIOTICS, GABA

LOW LEVEL LASER TO CNS…

SLEEP

Front Hum Neurosci. 2018; 12: 387.
Published online 2018 Sep 24. doi: 10.3389/fnhum.2018.00387

A Novel Insight of Effects of a 3-Hz Binaural Beat on Sleep Stages During Sleep

Nantawachara Jirakittayakorn* and Yodchanan Wongsawat*

These findings suggested that the 3-Hz binaural beat on a 250-Hz carrier tone can be used to modulate sleep stage by decreasing the latency to the N3 stage, **extending the N3 duration, and reducing the N2 duration without sleep disturbance and sleep fragmentation, while increasing the quality of sleep in this study is also associated slow wave sleep in which is related to memory consolidation and other regulation of the body; and the 3-Hz binaural beat can enhance power of delta activity during sleep.**

J Family Med Prim Care. 2019 Dec; 8(12): 3915–3919.
Published online 2019 Dec 10. doi: 10.4103/jfmpc.jfmpc_575_19

Effect of music of specific frequency upon the sleep architecture and electroencephalographic pattern of individuals with delayed sleep latency: A daytime nap study

Pramita Dubey,1 Yogesh Kumar,1 Ramji Singh,1 Kamlesh Jha,1 and Rajesh Kumar1

Conclusion:

It was concluded that 432 Hz music has some significant calming effect as reflected by increased alpha activities … naps.

Nature
. 2006 Nov 30;444(71

Boosting slo

Lisa Marshall 1 Halla

Here we show that in … g potentials (0.75 Hz) during … w wave sleep, enhances the … ories in healthy huma … ve sleep, endogenous cortical

BASE SLEEP SETTING 1 - 2 - 3 - 432
RED XLR8
ALSO CONSIDER "REST AND DIGEST"
VAGAL STIM IN THE PM (1-10-1-10)
COMBO SETTING (1-3-10-432)
10 MIN PROTOCOL BEFORE BED
(8 MIN EACH PRE-FRONTAL CORTEX
2 MIN VAGAL STIM SCM REGION)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

LOW LEVEL LASER THERAPY AND ALZHEIMER'S

Light therapy helps the brain clear out toxic Alzheimer's proteins

Despite their tireless efforts, researchers have been unable to develop a safe and effective way of treating Alzheimer's disease (AD) using pharmaceuticals, which has meant turning to non-pharmaceutical methods. A new study has demonstrated the therapeutic potential of light therapy, or phototherapy, in treating AD, showing promising results in mice that the researchers hope can be just as effective in humans.

Measuring the level of beta-amyloid in the hippocampus following PBM, the researchers found that levels were lower whether it was applied during wakefulness or sleep but that PBM during sleep produced a greater reduction in beta-amyloid. **They concluded that PBM stimulated the evacuation of beta-amyloid from the hippocampus even more effectively during sleep than wakefulness.**

THE "AFTER HOURS" ALZHEIMER'S ADD ON:

ALBEIT NOT AS PRACTICAL, USING LLLT DURING SLEEP SHOULD BE CONSIDERED IF A DEVICE IS AVAILABLE AT HOME. IF A HAND HELD LASER IS AVAILABLE AT HOME (OWNERSHIP OR LASER RENTAL PROGRAM, CONSIDER THE "AFTER HOURS" SETTING FROM A SPOUSE OR PARTNER DURING SLEEP CYCLES)

1-10-1-40

They say that this non-pharmaceutical, non-invasive treatment could be used in people with AD and other conditions that involve the brain's lymphatic system. "Since pharmacological therapy of AD have failed to show effectiveness or safety, PBM as a non-invasive and safe approach has the high prospects for implementation in clinical practice for the treatment of brain diseases with lymphatic disorders, such as AD, Parkinson's disease, glioma, traumatic brain injury, intracranial hemorrhages," the researchers said.

Oxana, SG., Alexander, S., Inna, B. *et al.* Mechanisms of phototherapy of Alzheimer's disease during sleep and wakefulness: the role of the meningeal lymphatics. *Front. Optoelectron.* **16**, 22 (2023). **Published18 September 2023**

GREEN FOR SLEEP?

Here, we show that blue light (470 nm) causes behavioural arousal, elevating corticosterone and delaying sleep onset. By contrast, green light (530 nm) produces rapid sleep induction.

Melanopsin Regulates Both Sleep-Promoting and Arousal-Promoting Responses to Light

- Violetta Pilorz, Published: June 8, 2016

Researchers have found that green light promotes sleep while blue light delays it. In a study conducted by Oxford University, green light produced rapid sleep onset in mice - between 1 and 3 minutes.

Lighting color affects sleep, wakefulness

Green light promotes sleep while blue light delays it, find researchers

Date:

June 8, 2016

GVL CONSIDERATION: GREEN BEAM 2 MIN APPLICATION RETINAL BEFORE BEDTIME

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MATTRESS RECOMMENDATIONS

Best Mattresses of 2024 (SOURCE SLEEPOPOLIS):

- Helix – Best Mattress Overall
- Nectar – Best Value Mattress
- DreamCloud – Best Mattress for Support
- Saatva Classic – Best Hybrid Mattress
- Helix Midnight Luxe – Best Luxury Mattress
- Plank Firm – Best Firm Mattress
- Saatva Rx – Best Mattress for Back Pain
- Nectar Premier – Best Mattress for Side Sleepers
- Titan Plus – Best Mattress for Heavy People
- DreamCloud Premier – Best Cooling Mattress

ANOTHER CONSIDERATION: THE SS500 COMFORT PREMIUM BASE "SUPERIOR SLEEP EXPERIENCE" COOL GEL ADJUSTABLE BED

WARNING: CONSUMER REPORTS CONTINUES TO LIST THE AVOCADO GREEN AS THEIR TOP RECOMMENDED MATTRESS YEAR AFTER YEAR. THEY MAY BE RIGHT FOR SOME ESPECIALLY BACK SLEEPERS, BUT THEY TEND TO BE TOO FIRM FOR SIDE SLEEPERS!

DEPRESSION

© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION AND BRAIN INFLAMMATION

Front Neurosci. 2019; 13: 213.
Published online 2019 Mar 15. doi: 10.3389/fnins.2019.00213

Brain Microglial Activation in Chronic Pain-Associated Affective Disorder

MOST CHRONIC PAIN AND NEUROPSYCH DISORDERS HAVE A GLIAL/ INFLAMMATION COMPONENT OR CAUSE. ALWAYS ADDRESS CNS ANTI-INFLAMMATION TREATMENTS (LASER BRAIN, VAGUS NERVE, NUTRITION ETC) AND ELIMINATE SOURCES LIKE LEAKY GUT, SAD DIET, PATHOGENS AND SO ON…

injury. In the present study, we characterized microglial activation in brain sub-regions of CX3CR1GFP/+ mice after chronic constriction injury (CCI) of the sciatic nerve, including observations at delayed time points when affective brain dysfunctions such as depressive-like behaviors typically develop. **Mice manifested chronic mechanical hypersensitivity immediately after CCI and developed depressive-like behaviors 8 weeks post-injury. Concurrently, significant increases of soma size and microglial cell number were observed in the medial prefrontal cortex (mPFC), hippocampus, and amygdala 8 weeks post-injury. Transcripts of CD11b, and TNF-α, genes associated with microglial activation or depressive-like behaviors, are correspondingly upregulated in these brain areas. Our results demonstrate that microglia are activated in specific brain sub-regions after CCI at delayed time points and imply that *brain microglial activation plays a role in chronic pain-associated affective disorders.***

627

DEPRESSION AND SEROTONIN

Serotonin Deficiency Directly Linked With Depression in Groundbreaking Study

TOPICS: DepressionPopularPsychiatrySerotonin

By ELSEVIER DECEMBER 27, 2022

Researchers have postulated since the 1960s that major depression stems from disruptions in the serotonin neurotransmitter system. However, the evidence for that idea, though plentiful, was indirect. **In fact, a recent comprehensive analysis of existing studies concluded that there was not strong evidence to support the "serotonin hypothesis." In its wake, some in the field have called for a reexamination of the hypothesis. Not so fast, says a new study that provides direct evidence of disrupted serotonin release in the brains of individuals with depression.**

"This study used a new and more direct method to measure serotonin in the living human brain, and the results suggest reduced serotonin (release) functioning in depression."

Participants with depression and healthy controls underwent **PET scanning with [^{11}C]Cimbi-36 to measure 5-HT2A receptor availability in the frontal cortex; the two groups did not differ significantly at baseline.** Both groups then received a dose of d-amphetamine, a stimulant drug that works to increase 5-HT concentration outside of neurons, where it interacts with 5-HT2A receptors and reduces the binding of [^{11}C]Cimbi-36. In a second scanning session three hours after drug administration, **healthy control participants had significantly reduced 5-HT2A receptor availability, indicating an increase in serotonin levels. Participants with depression, however, did not show a significant decrease in binding potential, suggesting they had a blunted serotonin release capacity in key brain regions.**

Of note, all patients were free of antidepressant medication, and 11 out of the 17 had never received antidepressant treatment, indicating that low serotonin release capacity is a feature of depression rather than a result of antidepressant treatment.

Reference: "Brain Serotonin Release Is Reduced in Patients With Depression: A [11C]Cimbi-36 Pet Study With a D-Amphetamine Challenge" by David Erritzoe, Beata R. Godlewska, Gaia Rizzo, Graham E. Searle, Claudio Agnorelli, Yvonne Lewis, Abhishekh H. Ashok, Alessandro Colasanti, Iro Boura, Chloe Farrell, Hollie Parfitt, Oliver Howes, Jan Passchier, Roger N. Cowen, David J. Nutt, Philip J. Cowen, Gitte Knudsen and Eugenii A. Rabiner, 28 October 2022, *Biological Psychiatry*.
DOI: 10.1016/j.biopsych.2022.10.012

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

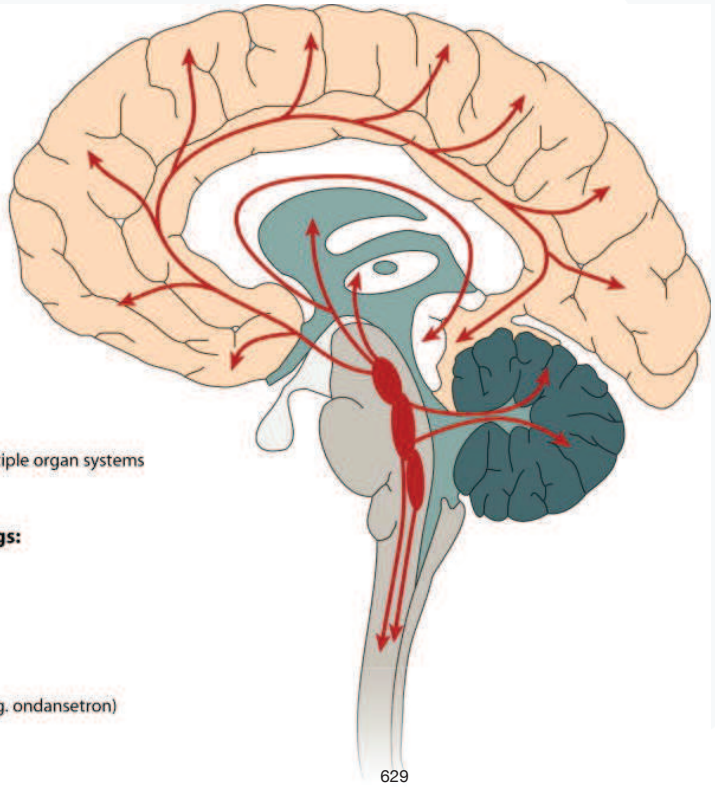
SEROTONIN PRODUCTION IN THE BRAIN

ERCHONIA eCommunity Education

Behavioral effects:
Mood
Perception
Memory
Anger
Aggression
Fear
Stress responses
Appetite
Addiction
Sexuality

Other CNS effects:
Motor control
Cerebellar regulation
Sleep/circadian rhythms
CNS vascular tone
Emesis
Respiratory drive
Body temperature
Descending regulation of multiple organ systems

Central serotonergic drugs:
SSRIs
Tricyclic antidepressants
MAOIs
Other antidepressants
Buspirone
Atypical antipsychotics
Triptans
5-HT_3 receptor antagonists (e.g. ondansetron)
Fenfluramine
Ergotamine/methysergide
Hallucinogens



629

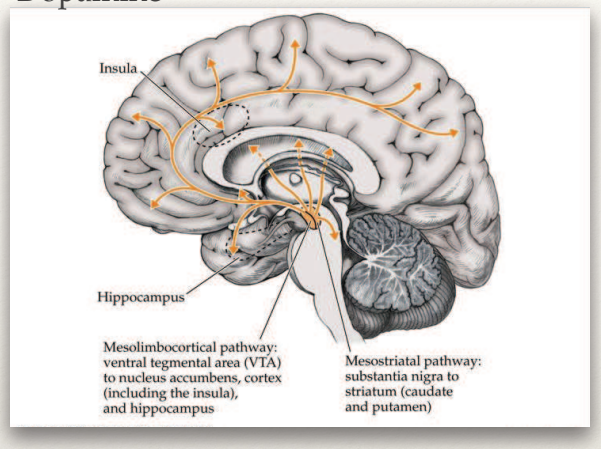
MANUFACTURED IN THE RAPHE NUCLEI LOCATED IN THE BRAINSTEM

MADE FROM TRYPTOPHAN, AN ESSENTIAL AMINO ACID

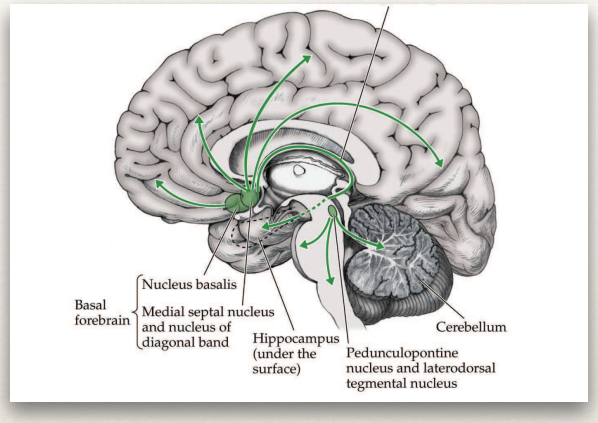
YOU ALSO NEED CO-FACTORS AND TRANSPORT SYSTEMS

A HEALTHY GUT AND MICROBIOME ARE ALSO CRITICAL FACTORS (UP TO 90% OF THE BODY'S SEROTONIN IS MADE IN THE GUT

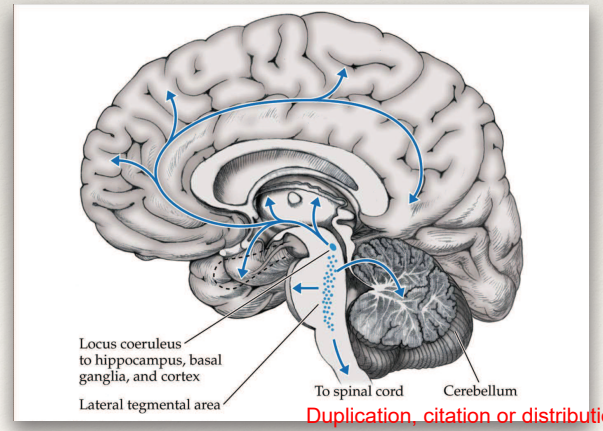
Dopamine



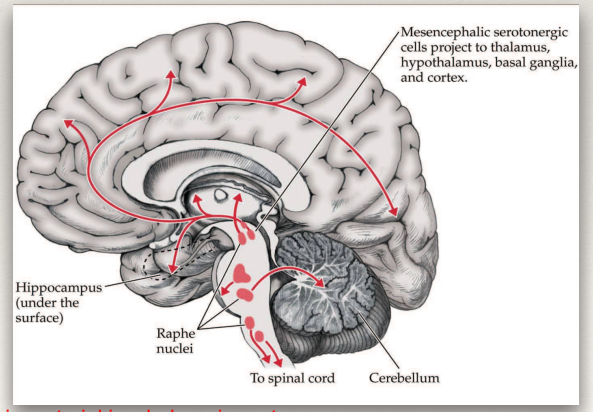
Acetylcholine



Norepinephrine



Serotonin



630

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

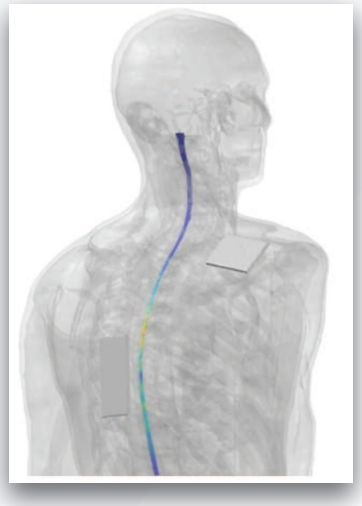
DEPRESSION

The Spinal Cord Could Provide a Radical New Way to Treat Depression

HEALTH
13 January 2024

A team led by researchers at the University of Cincinnati College of Medicine devised a pilot clinical trial in which a little black box was placed on the spinal cord of **20 volunteers with depression, with one electrode on the back and one on the right shoulder.**

The box then delivered a specially customized, low-level electric buzz to half of the volunteers, for **three sessions per week over eight weeks**. This was shown to have a greater effect on depressive symptoms than the different, ' placebo' charge administered to the other half of the volunteers.



"We think that the connection between the brain and the body is essential for psychiatric disorders," says Romo-Nava.

- Open access MOLECULAR PSYCHIATRY
- **Published: 20 December 2023**

Effect of non-invasive spinal cord stimulation in unmedicated adults with major depressive disorder: a pilot randomized controlled trial and induced current flow pattern

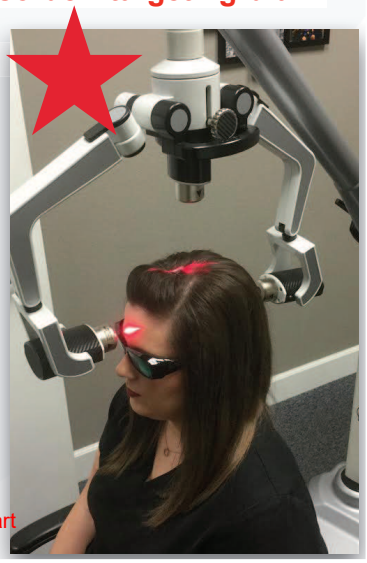
- Francisco Romo-Nava

DEPRESSION AND LLLT

"Depression: let's talk" says WHO, as depression tops list of causes of ill health 30 MARCH 2017 | GENEVA - Depression is the leading cause of ill health and disability worldwide. According to the latest estimates from WHO, more than 300 million people are now living with depression, an increase of more than 18% between 2005 and 2015.

Neurophotonics. 2016 Jul;3(3):031404. doi: 10.1117/1.NPh.3.3.031404. Epub 2016 Mar 4.
Review of transcranial photobiomodulation for major depressive disorder: targeting brain metabolism, inflammation, oxidative stress, and neurogenesis.
Cassano P1, Petrie SR2, Hamblin MR3, Henderson TA4, Iosifescu DV5.

Neuroscience. 2013 Jan 29;230:13-23. doi: 10.1016/j.neuroscience.2012.11.016. Epub 2012 Nov 27.
Transcranial infrared laser stimulation produces beneficial cognitive and emotional effects in humans. **Barrett DW1, Gonzalez-Lima F.**



Theta to Alpha (frontal to parietal)6-9-10-12 setting . Mid-Sagittal plane (Mohawk) 10 minutes 2-3x/week minimum 6 visits.

CO-ACTIVATION CONSIDERATIONS: PEMF, MEDITATION, DEEP BREATHING 4:4:8, VEPs, BINAURAL THERAPIES

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION AND LLLT

J Affect Disord. 2019 Jan 15;243:262-273. doi: 10.1016/j.jad.2018.09.048. Epub 2018 Sep 17.

Transcranial and systemic photobiomodulation for major depressive disorder: A systematic review of efficacy, tolerability and biological mechanisms.

Caldieraro MA1, Cassano P2.

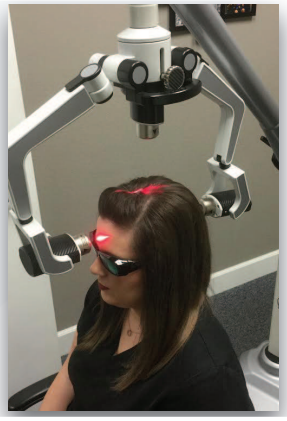
Author information Abstract

BACKGROUND:
Photobiomodulation (PBM) with red and near-infrared light (NIR) -also known as Low-Level Light Therapy-is a low risk, inexpensive treatment-based on non-retinal exposure-under study for several neuropsychiatric conditions. The aim of this paper is to discuss the proposed mechanism of action and to perform a systematic review of pre-clinical and clinical studies on PBM for major depressive disorder (MDD).

RESULTS:
Red and NIR light penetrate the skull and modulate brain cortex; an indirect effect of red and NIR light, when delivered non-transcranially, is also postulated. The main proposed mechanism for PBM is the enhancement of mitochondrial metabolism after absorption of NIR energy by the cytochrome C oxidase; however, actions on other pathways relevant to MDD are also reported. Studies on animal models indicate a benefit from PBM that is comparable to antidepressant medications. Clinical studies also indicate a significant antidepressant effect and good tolerability.

LIMITATIONS:
Clinical studies are heterogeneous for population and treatment parameters, and most lack an appropriate control.

CONCLUSIONS:
Preliminary evidence supports the potential of non-retinal PBM as a novel treatment for MDD. Future studies should clarify the ideal stimulation parameters as well as the overall efficacy, effectiveness and safety profile of this treatment.



Theta to Alpha (frontal to parietal)6-9-10-12 setting . Mid-Sagittal plane (Mohawk) 10 minutes 2-3x/week minimum 6 visits.

CO-ACTIVATION CONSIDERATIONS: PEMF, MEDITATION, DEEP BREATHING 4:4:8, VEPs, BINAURAL THERAPIES, NO-NO VOR'S, TMS

DEPRESSION AND LLLT

Randomized Controlled Trial
Brain Stimul
. Sep-Oct 2016;9(5):780-787. doi: 10.1016/j.brs.2016.05.009. Epub 2016 May 24.

Transcranial Laser Stimulation as Neuroenhancement for Attention Bias Modification in Adults with Elevated Depression Symptoms

Seth G Disner 1, Christopher G Beevers 2, Francisco Gonzalez-Lima 3

ADVANCED CLINICAL APPLICATION: ERCHONIA LLLT TO THE RIGHT PREFRONTAL CORTEX WITH ABM IE. MEDITATION OF GRATITUDE COMBINED WITH THE "HAPPY" FREQUENCY = 6HZ
ADVANCED "HAPPY" SETTING 6-9-10-12 (THETA-ALPHA-VAGAL) 5 MINUTES RIGHT PREFRONTAL CORTEX

Methods: **Fifty-one adult participants with elevated symptoms of depression received ABM before and after laser stimulation and were randomized to one of three conditions: right forehead, left forehead, or sham. Participants repeated LLLT two days later and were assessed for depression symptoms one and two weeks later.**

Results: A significant three-way interaction between LLLT condition, ABM response, and time indicated that **right LLLT led to greater symptom improvement among participants whose attention was responsive to ABM (i.e., attention was directed away from negative stimuli). Minimal change in depression was observed in the left and sham LLLT.**

Conclusions: The beneficial effects of ABM on depression symptoms may be enhanced when paired with adjunctive interventions such as right prefrontal LLLT; however, cognitive response to ABM likely moderates the impact of neuroenhancement. The results suggest that larger clinical trials examining the efficacy of using photoneuromodulation to augment cognitive training are warranted.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA eCommunity Education

Mitochondria Linked to Major Depression in Older Adults

Conclusion: Our results suggest that depression in late life is associated with GDF-15, a marker of amplified age-related biological changes. GDF-15 is a novel and potentially targetable biological pathway between depression and accelerated aging, including cognitive aging.

"This is the largest study to date providing a link between accelerated mitochondrial aging and depression in older adults…"

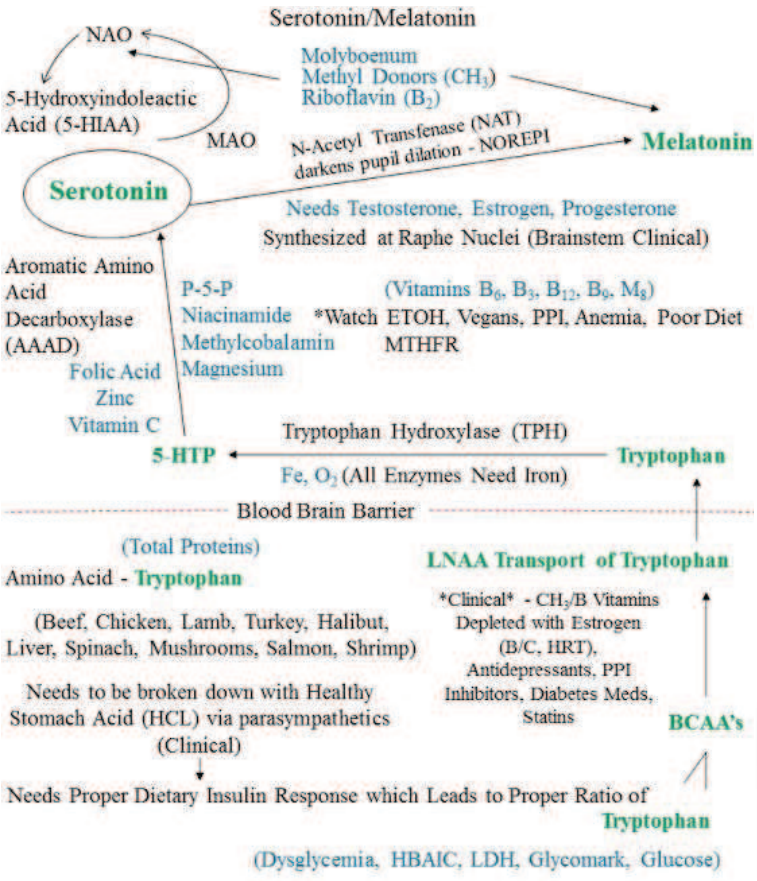
Am J Geriatr Psychiatry
2023 Jan;31(1):1-9. doi: 10.1016/j.jagp.2022.08.003. Epub 2022 Aug 26.

Late-Life Depression is Associated With Increased Levels of GDF-15, a Pro-Aging Mitokine

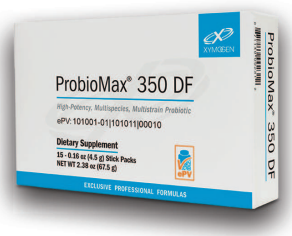
635

Emma Mastrobattista 1

636



MICRONUTRIENT PANEL!



Bifido Infantis, Breve and Longum. Lactobacillus Helveticus and Acidophilus

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

DEPRESSION, HISTAMINES AND INFLAMMATION

ERCHONIA eCommunity Education

Inflammation-Induced Histamine Impairs the Capacity of Escitalopram to Increase Hippocampal Extracellular Serotonin

Melinda Hersey, Srimal Samaranayake, Shane N. Berger, Navid Tavakoli, Sergio Mena, H. Frederik Nijhout, Michael C. Reed, Janet Best, Randy D. Blakely, Lawrence P. Reagan and Parastoo Hashemi

Journal of Neuroscience **28 July 2021**, 41 (30) 6564-6577;

The researchers discovered it was increased levels of histamine in the brain, generated as part of the animals' inflammatory response, that was causing the reduction in serotonin by, "acting on inhibitory histamine H3 heteroreceptors on serotonin terminals."

In fact, it was discovered that the antidepressant's effect was blunted because it hindered the brain's ability to clear histamine, interfering with the regulation of serotonin levels.

Finally, to confirm this potential mechanism, the researchers treated the animals with histamine-reducing drugs alongside the antidepressant. And this combination of drugs saw serotonin levels rise again in the inflammation-induced mice.

"Our work shines a spotlight on histamine as a potential key player in depression," says lead author on the study, Parastoo Hashemi. "This, and its interactions with the 'feel-good molecule' serotonin, may thus be a crucial new avenue in improving serotonin-based treatments for depression."

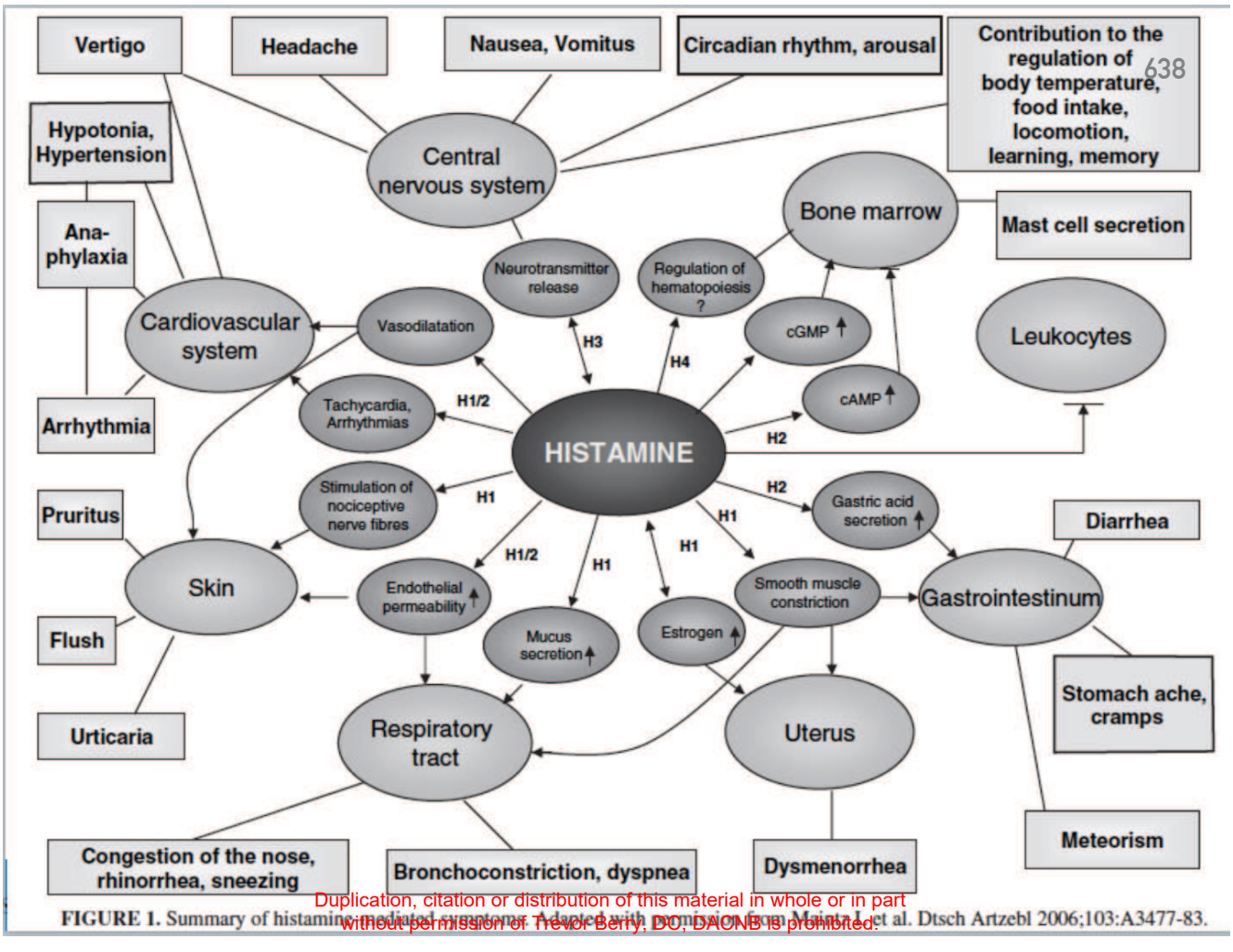
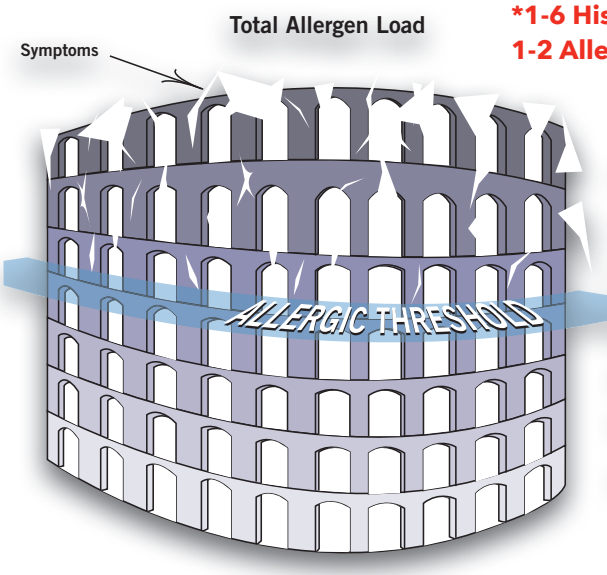


FIGURE 1. Summary of histamine-mediated symptoms. Adapted with permission from Maintz L, et al. Dtsch Artzebl 2006;103:A3477-83.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

CLINICAL CONSIDERATIONS FOR REDUCING HISTAMINES



***1-6 HistDAO per day (use pre meal)**
1-2 AllerDHQ daily combo

The enzyme diamine oxidase (DAO) degrades histamine by converting it from 2-(4-imidazolyl)-ethylamine to the inactive metabolite imidazole acetaldehyde.

Supports Healthy Degradation of Food-Derived Histamine
Enhances the Presence of Diamine Oxidase in the Digestive Tract
WATCH PATHOGEN CO-MORBIDITIES LIKE MOLD. LOW HISTAMINE DIET!

HISTAMINES

ERCHONIA eCommunity Education

Histamine-rich foods are:

- alcohol and other fermented beverages
- fermented foods and dairy products, such as yogurt and sauerkraut
- dried fruits
- eggplant
- spinach
- processed or smoked meats
- shellfish
- aged cheese

There are also a number of foods that trigger histamine release in the body, such as:

- alcohol
- bananas
- tomatoes
- wheat germ
- beans
- papaya
- chocolate
- citrus fruits
- nuts, specifically walnuts, cashews and peanuts
- food dyes and other additives

FOODS THAT PROMOTE DAO PRODUCTION:
ORGAN MEATS ESPECIALLY KIDNEY
GREEN PEA SPROUTS
FATS: OLIVE OIL
OMEGA 3'S

Foods that block DAO production include:

- alcohol
- black tea
- mate tea
- green tea
- energy drinks

HISTAMINES IN THE CNS AND INTRACELLULAR ARE BROKEN DOWN BY HISTAMINE N-METHYLTRANSFERASE ENZYME WHICH REQUIRES PROPER METHYLATION OF B12/FOLATE, VITAMINS A&D, MINERALS MOLYBDENUM, COPPER, SELENIUM AND ZINC

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LASERS AND ALLERGIES

Results: **We identified increased expression of immunomodulatory factors, such as HGF, TGF-β, and PGE, in T-MSCs-CM treated with LLLT**, compared to T-MSCs-CM without LLLT. **Our animal study demonstrated reduced allergic symptoms and lower expression of total IgE and OVA-specific IgE in the LLLT-treated** T-MSCs-CM group compared to the AR group and T-MSCs-CM alone. Moreover, ***we found that T-MSCs-CM treated with LLLT showed significantly decreased infiltration of eosinophils, neutrophils, and IL-17 cells in the nasal mucosa and reduced IL-4, IL-17, and IFN-γ expression*** in OVA-incubated splenocytes compared to the AR group.

Conclusions: The present study suggests that T-MSCs-CM treated with **LLLT may provide an improved therapeutic effect against nasal allergic inflammation** than T-MSCs-CMAm J Rhinol Allergy **2022 Mar**;36(2):261-268. doi: 10.1177/19458924211053762. Epub 2021 Nov 5.

Increased Anti-Allergic Effects of Secretome of Low-Level Light Treated Tonsil-Derived Mesenchymal Stem Cells in Allergic Rhinitis Mouse Model

CONSIDER SINUSITIS SETTING: 20-60-160-400
WITH HANDHELDS. 2 MINUTES PFC
5 MINUTES SINUSES, 2 MINUTES VAGAL

641

In-Su Park 1, Dong-Kyu Kim 2 3, Ji Hye Kim 4 5

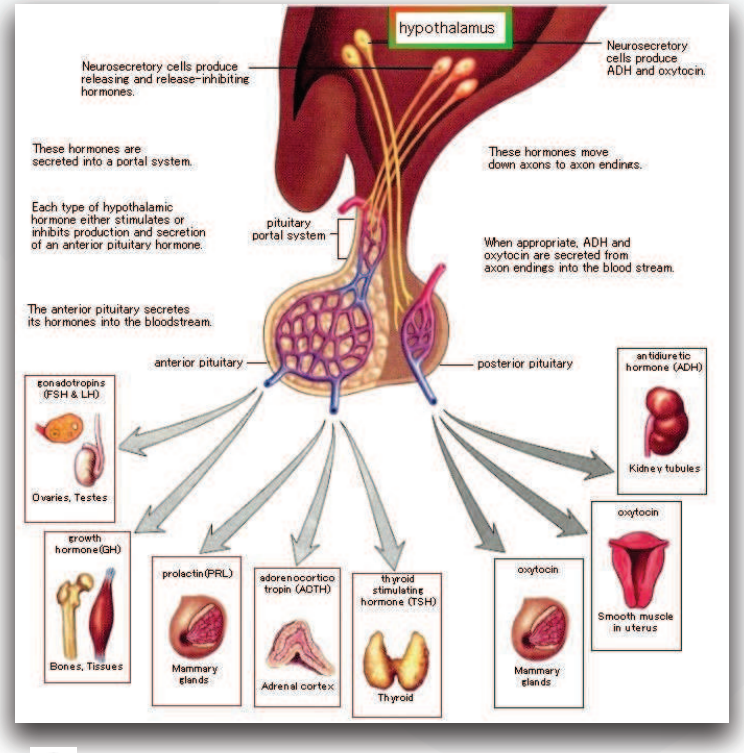
DEPRESSION CLINICAL CONSIDERARTION SUMMARY

1. MOVE! EXERCISE, ESPECIALLY IN SUNLIGHT AND IN A "GROUNDING" ENVIRONMENT. LIGHT THERAPIES
2. MAKE SURE THEY HAVE THE RIGHT BUILDING BLOCKS FOR NEUROCHEMISTRY. EAT A GOOD FIBER/VEGGIE, HIGH IN GOOD FATS AND PROTEINS. RUN LABS FROM BASE CBC/CHEM SCREENS, HORMONE TESTING (DUTCH OR VIBRANT), MICRONUTRIENT PROFILE AND INFLAMMATION SOURCES (FOOD SENSITIVITIES, PATHOGENS, HBAIC)
3. A HEALTHY GUT MICROBIOME (GUT ZOOMER 3.0)
4. ERCHONIA LOW LEVEL LASER THERAPY (RIGHT PREFRONTAL CORTEX 6-9-10-12. STACK WITH 5 MIN MEDITATION OF GRATITUDE/5 MIN CLEARING OF MIND). VAGAL NERVE STIM PROTOCOL (10HZ)
5. TRANSCRANIAL MAGNETIC STIMULATION. PEMF
6. CBD PRODUCTS AND CLASSIC PSYCHEDELICS (MICRO OR MACRO DOSING. WATCH LEGAL MATTERS ON DMT -> DMN THERAPIES - SCHEDULE 1 SUBSTANCES)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

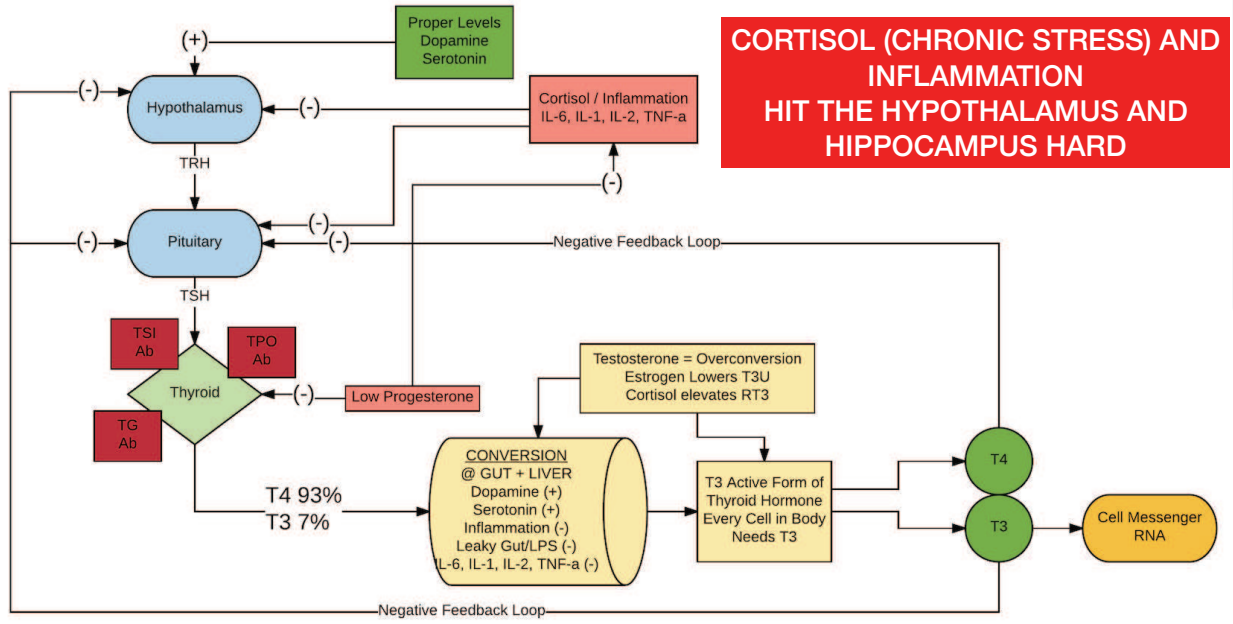
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HP AXIS



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

THYROID



CORTISOL (CHRONIC STRESS) AND INFLAMMATION HIT THE HYPOTHALAMUS AND HIPPOCAMPUS HARD

THYROID DYSFUNCTION COULD BE A DIFFERENTIAL FOR ALMOST EVERY CONDITION OF THE HUMAN BODY INCLUDING BRAIN DEGENERATION, GUT ISSUES LIKE GERD AND SO ON. EVERY CELL IN THE HUMAN BODY NEEDS THYROID HORMONE!

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

THYROID LAB TESTING
RUN A COMPLETE PANEL

- **T4, Total** – T4 (thyroxine) is a hormone produced by the thyroid gland. This test measures the amount of T4 in your blood.
- **T4, Free** – Most of the T4 in your body binds to protein, the T4 that does not is called T4 and circulates unbound in your blood. This test measures these T4 hormones.
- **T3, Total** – T3 (triiodothyronine) is a thyroid hormone and is important for the body's control of metabolism. This test measures the amount of T3 in your blood.
- **T3, Free** – Most of the T3 in your body binds to protein, the T3 that does not is called T3 and circulates unbound in your blood. This test measures these T3 hormones. ACTIVE FORM OF THYROID
- **T3, Reverse** – Reverse T3 is converted by the liver from the stored hormone T4, it is the body's way of getting rid of unneeded T4 every day. Sometimes the body can make too much reverse T3 from a number of causes including chronic emotional, physical, or biological stress, chronic sickness, after surgery, diabetes, aging, or an acute injury. This test measures for levels of reverse T3.
- **Thyroid Stimulating Hormone (TSH)** – TSH causes the thyroid gland to make two hormones, T3 and T4, which help control your body's metabolism. This test is done to find out whether the thyroid gland is working properly.
- **Thyroglobulin Antibody** – Thyroglobulin is a protein produced and used by the thyroid gland to make the hormones T3 and T4. Antibodies are the proteins made by the immune system to fight toxins, bacteria, and viruses. High levels of antibodies suggest the immune system is mistakenly attacking the thyroid gland. This test measures the blood levels of antibodies the body has made against the compound thyroglobulin.
- **Thyroid Peroxidase Antibody** – Thyroid peroxidase (TPO) is an enzyme made in the thyroid gland and is important in producing the thyroid hormone. TPO converts to thyroid hormone T4 to T3. High levels of antibodies suggest the immune system is mistakenly attacking the thyroid gland. This test measures the level of antibody the body has made against thyroid peroxidase.

THYROID AND LLLT

Low-level laser in the treatment of patients with hypothyroidism induced by chronic autoimmune thyroiditis: A randomized, placebo-controlled clinical trial

- **June 2012 Lasers in Medical Science 28(3)**

These findings suggest that LLLT was effective at improving thyroid function, promoting reduced TPOAb-mediated autoimmunity and increasing thyroid echogenicity in patients with CAT hypothyroidism.

Volume 2012 | Article ID 126720 | https://doi.org/10.5402/2012/126720
Show citation

Assessment of the Effects of Low-Level Laser Therapy on the Thyroid Vascularization of Patients with Autoimmune Hypothyroidism by Color Doppler Ultrasound

Abstract

Background. Chronic autoimmune thyroiditis (CAT) frequently alters thyroid vascularization, likely as a result of the autoimmune process. *Objective*. To evaluate the effects of low-level laser therapy (LLLT) on the thyroid vascularization of patients with hypothyroidism induced by CAT using color Doppler ultrasound parameters. *Methods*. In this randomized clinical trial, 43 patients who underwent levothyroxine replacement for CAT-induced hypothyroidism were randomly assigned to receive either 10 sessions of LLLT (L group,) or 10 sessions of a placebo treatment (P group,). Color Doppler ultrasounds were performed before and 30 days after interventions. To verify the vascularity of the thyroid parenchyma, power Doppler was performed. *Results*. The frequency of normal vascularization of the thyroid lobes observed in the postintervention power Doppler examination was significantly higher in the L than in the P group (). The pulsed Doppler examination revealed an increase in the SPV of the ITA in the L group compared with the P group (). No significant differences in the SPV of the STA and in the RI were found between the groups. ***Conclusion*. These results suggest that LLLT can ameliorate thyroid parenchyma vascularization and increase the SPV of the ITA of patients with hypothyroidism caused by CAT.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Researchers in Brazil have been studying the effects of low level laser therapy on the thyroid gland in Hashimoto's thyroiditis, and the results have been astonishing!

Most significantly, they found that all patients who received the therapy were able to reduce their levothyroxine dose, while 47% were able to discontinue levothyroxine and have normal thyroid function during the 9-month follow-up.

Additionally, LLLT can increase Transforming Growth Factor B (TGF-B), which is a cytokine that helps to induce and maintain the tolerance of self thus, also reduce thyroid autoimmunity.

- **a reduction of levothyroxine dose from a mean of 93 mcg to 38 mcg (with 95.7% of treatment group being able to reduce or stop medications…47.8% no longer needing thyroid medications)**
- **a reduction in TPOAb, mean 1289 to 656 (around 50% reduction)**
- **a reduction in TG antibodies, 720 to 656**
- **a normalization of thyroid volume (in 66%)**
- **and less infiltration of the thyroid gland on ultrasounds (meaning fewer inflammatory cells were present). Echogenicity index on thyroid ultrasound was improved in 95% of the study group, meaning that their thyroid gland had less damage and fewer white blood cells on ultrasound.**

THYROID

International Journal of Endocrinology
Volume 2018, Article ID 8387530, 9 pages
https://doi.org/10.1155/2018/8387530
Clinical Study

Safety and Efficacy of Low-Level Laser Therapy in Autoimmune Thyroiditis: Long-Term Follow-Up Study

Danilo Bianchini Höfling,[1] Maria Cristina Chavantes,[2] Carlos Alberto Buchpiguel,[3] Giovanni Guido Cerri,[3] Suemi Marui [4] Paulo Campos Carneiro,[5] and Maria Cristina Chammas

Introduction. A randomized clinical trial (RCT) was performed to evaluate the efficacy of low-level laser therapy (LLLT) for hypothyroidism induced by chronic autoimmune thyroiditis (CAT). *Objective*. The objective was to assess the safety and actions of LLLT 6 years after completion of the RCT. *Materials and Methods*. Forty-three participants were invited to participate in this study 6 years after completion of the RCT. Twenty-five were subjected to LLLT (group L), and 18 were subjected to placebo (group P). Primary outcome measure: frequency of thyroid nodules, which were subjected to fine-needle aspiration biopsy. Secondary outcome measures: dose of levothyroxine required to treat hypothyroidism, thyroid peroxidase antibodies (anti-TPO), and anti-thyroglobulin antibodies (anti-Tg). *Results*. In group L, a nodule was observed in three patients, who all had a Bethesda II classification. In group P, a nodule was also observed in three patients, with two classified as Bethesda II and one as Bethesda III. The levothyroxine dose required by group L was significantly lower than that required by group P (). The anti-TPO and anti-Tg levels did not differ between the groups. ***Conclusion*. LLLT, by the methods described, has been shown to be safe for the treatment of hypothyroidism resulting from Chronic Autoimmune Thyroid.** This trial is registered with ClinicalTrials.gov Identifier: NCT02240563.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

BASIC THYROID LASER PROTOCOL
COURTESY OF KIRK GAIR

BASIC THYROID PROTOCOL

Step 1: Laser over the cervical spine and thoracic spine down to the adrenals for 2-3 minutes with the basic frequencies of 9, 16, 42, and 53 hz.

Step 2: You can also use the thyroid specific frequencies of 20, 59, 802, 3884 hz over the thyroid or near it for 2-3 minutes *(if there is a history of thyroid cancer, although there is research that supports the use of laser for cancers, I would recommend not using it directly over the thyroid until there is more of a consensus from the research)*

Step 3: Laser 2-3 minutes over the liver using the frequencies of 53, 55, 537, 751 hz.

Step 4: Laser 2-3 minutes over thymus using the frequencies of 20, 73, 465, 728 hz.

NOTE: If at any time... te how long it lasts. If you have a... able to tolerate very much stimulat...

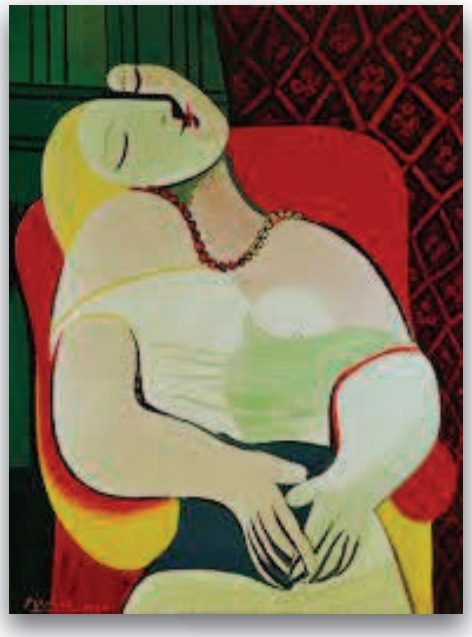
Additional note: I S... cols. I have been contacted by pati... thyroid storm and an ER visit with... port that type of laser use in the ma...

62

BROCK PROTOCOL
HYPOTHYROID 20-59-822-3884
HYPERTHYROID 3-20-59-160

USE GVL OR EVRL IF AVAILABLE

REPRODUCTIVE SYSTEM



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REPRODUCTIVE SYSTEM: LLLT AND FEMALE INFERTILITY

Background and Aims: The rapidly graying population in Japan is being compounded by the rapidly-dropping birth rate. The latter is mostly due to the later ages at which women are giving birth as the marriage age has also been increasing. Giving birth at a later stage is associated with problems for both mother and child, but for older would-be mothers the greatest problem is infertility, sometimes severe. The present article will show how the application of low level laser therapy (LLLT) is a potentially effective treatment for severe infertility. **Subjects and Methods:** Seventy-four females (average age 39.28 yr) with severe infertility in whom assisted reproductive technology (ART) had been unsuccessful (average of 9.13 yr) participated in the first part of a study from October 1996 — April 2000. LLLT was applied (830 nm, CW, GaAlAs **60 mW diode LLLT) in Ohshiro's proximal priority technique (average 21.08 sessions)** with or without other ART approaches. Based on successful outcomes, the study was then extended to March 2012, amassing a final total of 701 patients. **Results:** Pregnancy was achieved in the first part of the trial in 16 patients (21.7% of 74) of whom 11 (68%) achieved successful live delivery. In the extended trial, pregnancy was achieved in 156 (22.3% of 701) with 79 live deliveries (50.1%). **Conclusions:** The use of 830 nm LLLT in the proximal priority technique at the parameters used in the present study, on its own or as an adjunct to other techniques, **resulted in successful induction of pregnancy in just over 21% of severely infertile females, with a substantial number of these achieving live births. No adverse events were noted in any patient. LLLT is a pain-free and sideeffect free modality which could give hope to the increasing numbers of older females with infertility in Japan and potentially worldwide. Multinational studies are warranted.**

Laser Ther. 2012 Jul 3; 21(2): 97–103.
doi: 10.5978/islsm.12-OR-05

PMCID: PMC3944482
PMID: 24610987

Personal Overview of the Application of LLLT in Severely Infertile Japanese Females

Toshio Ohshiro, MD PhD

REPRODUCTIVE SYSTEM: LLLT AND MALE INFERTILITY

In half of the cases, the infertility of the couple is due to the disorder of the male fertility. The leading factors that cause male infertility are urogenital infections, disorders of the immune system, testicular and prostate pathology, as well as endocrine disorders.

Low level laser therapy (LLLT) is a very effective physical therapy method, used in many areas of medicine, including obstetrics and gynaecology, andrology and urology; and it is recommended as an integral part of the complex treatment of infertility.

The literature review showed that LLLT is beneficial in treating male infertility. **Laser can significantly improve the survival, motility and speed of movement of spermatozoa**. Laser therapy of patients with prostatitis and vesiculitis can eliminate infiltrative-exudative changes, improve reproductive and copulatory functions. Local illumination of red (635 nm) and infrared (904 nm) spectra should be combined with intravenous laser blood illumination (ILBI) of red (635 nm) and ultraviolet (UV) (365 nm) spectra.

Biomedicine (Taipei). 2018 Jun; 8(2): 7.
Published online 2018 May 28. doi: 10.1051/bmdcn/2018080207

PMCID: PMC5992952
PMID: 29806585

Effectiveness of low level laser therapy for treating male infertility

Sergey Vladimirovich Moskvin[1,*] and Oleg Ivanovich Apolikhin[2]

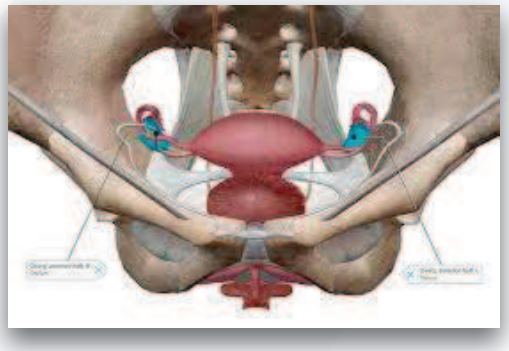
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REPRODUCTIVE SYSTEM: CLINICAL LASER PROTOCOLS

1. ALWAYS CONSIDER "UPSTREAM" HP-AXIS AND CNS FUNCTION
2. ALWAYS CONSIDER HORMONE METABOLISM CLEARANCE, LIVER DETOX
3. ALWAYS CONSIDER DIET ESPECIALLY SUGAR (AROMATASING/17-20 LYASE) AND A.I. CROSS REACTIVITY
4. ALWAYS CONSIDER BUILDING BLOCKS (NEEDS CHOLESTEROL, PREGNENOLONE STEAL)

FOR OVARIES, HAVE PATIENT PLACE HANDS ON PUBIC SYMPHYSIS AND PLACE BEAMS ABOUT 2" LATERAL AND 2" ROSTRAL



ERCHONIA LLLT:

2-10-246-417 (SOLFEGGIO FREQUENCIES INFERTILITY/ SACRAL CHAKRA)

2 MINUTES DIRECTLY OVER EACH GONAD
2 MINUTES SACRAL PLEXUS
2 MINUTES VAGAL NERVE/SCM
2-5 MINUTES PREFRONTAL CORTEX

REPRODUCTIVE SYSTEM

Mol Cell Endocrinol

2021 Jul 1;531:111318. doi: 10.1016/j.mce.2021.111318. Epub 2021 May 12.

Local application of low level laser therapy in mice ameliorates ovarian damage induced by cyclophosphamide

Abstract

The aim of the present study is to assess whether low level laser therapy (LLLT) can protect ovaries from chemotherapy-induced gonadotoxicity using a mice model of premature ovarian failure induced by cyclophosphamide (CTX). LLLT (64 J/cm2) increased the number of antral follicles whilst decreasing the number of atretic follicles compared to CTX alone. LLLT increased the number of primordial follicles compared with those in the CTX group but they did not differ from those in the control group. LLLT treatment increased the number of AMH-positive follicles compared to CTX alone. LLLT application increased ovarian weight, serum progesterone concentration and P450scc protein levels compared to CTX alone. LLLT reduced the apoptosis in antral follicles and the BAX/BCL-2 ratio compared to CTX alone. Vascular morphology, analysed by CD31 and α-SMA immunostaining, was restored in LLLT-treated ovaries compared to CTX alone. **In conclusion, application of LLLT prior to CTX might serve as a promising and novel protocol to preserve female fertility in cancer survivors.**

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

REPRODUCTIVE SYSTEM

Abstract

It is known that LLLT has beneficial effects on several pathological conditions including wound healing, pain and inflammation. LLLT modulates biological processes, including cell proliferation, apoptosis and angiogenesis. In the present study, we examined the effect of local **application of LLLT on follicular dynamics, ovarian reserve, AMH expression, progesterone levels, apoptosis, angiogenesis, and reproductive outcome** in adult mice. LLLT (200 J/cm2) increased the percentage of primary and preantral follicles, whilst decreasing the percentage of corpora lutea compared to control ovaries. **LLLT-treated ovaries did not exhibit any changes regarding the number of primordial follicles. We observed a higher percentage of AMH-positive follicles (in early stages of development) in LLLT-treated ovaries compared to control ovaries. LLLT reduced the P4 concentration and the apoptosis in early antral follicles compared to control ones. LLLT caused a reduction in the endothelial cell area and an increase in the periendothelial cell area in the ovary. Additionally, LLLT was able to improve oocyte quality.** Our findings suggest that local application of LLLT modulates follicular dynamics by regulating apoptosis and the vascular stability in mouse ovary. In conclusion, **these data indicate that LLLT might become a novel and useful tool in the treatment of several pathologies, including female reproductive disorders.**

Prog Biophys Mol Biol
2019 Aug;145:10-18. doi: 10.1016/j.pbiomolbio.2018.11.010. Epub 2018 Nov 27.

Low level laser therapy (LLLT) modulates ovarian function in mature female mice

655

Gonzalo Oubiña 1,

REPRODUCTIVE HORMONES

DO NOT FORGET TO ADDRESS REPRODUCTIVE HORMONES. HYPOGONADISM IS COMMON IN POST-CONCUSSION SYNDROME BUT SEX HORMONES ARE ALSO NEEDED TO MAINTAIN AND PROTECT THE BRAIN IN AGING!

ALZHEIMER'S DISEASE

Women are at greater risk to suffer from Alzheimer's - here's why, claims study

The researchers discovered that the brains of women who had died from the condition had substantially higher amounts of complement C3, an inflammatory immune protein, than those of men who had died from the sickness.

A hint to the molecular aetiology of Alzheimer's disease has been discovered by researchers at MIT and Scripps Research; this clue may also explain why women are more susceptible to the condition. In **the study published on December 14, 2022, in Science Advances,** the researchers discovered that the **brains of women who had died from the condition had substantially higher amounts of complement C3, an inflammatory immune protein, than those of men who had died from the sickness**. Additionally, **they demonstrated how regularly occurring estrogen, whose synthesis declines during menopause, prevents the formation of this form of complement C3.**

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LOW PROGESTERONE SIGNS

5 SIGNS OF LOW PROGESTERONE IN FEMALES:

1.TROUBLE SLEEPING

2.ANXIETY

3.DEPRESSION

4.BLOATING

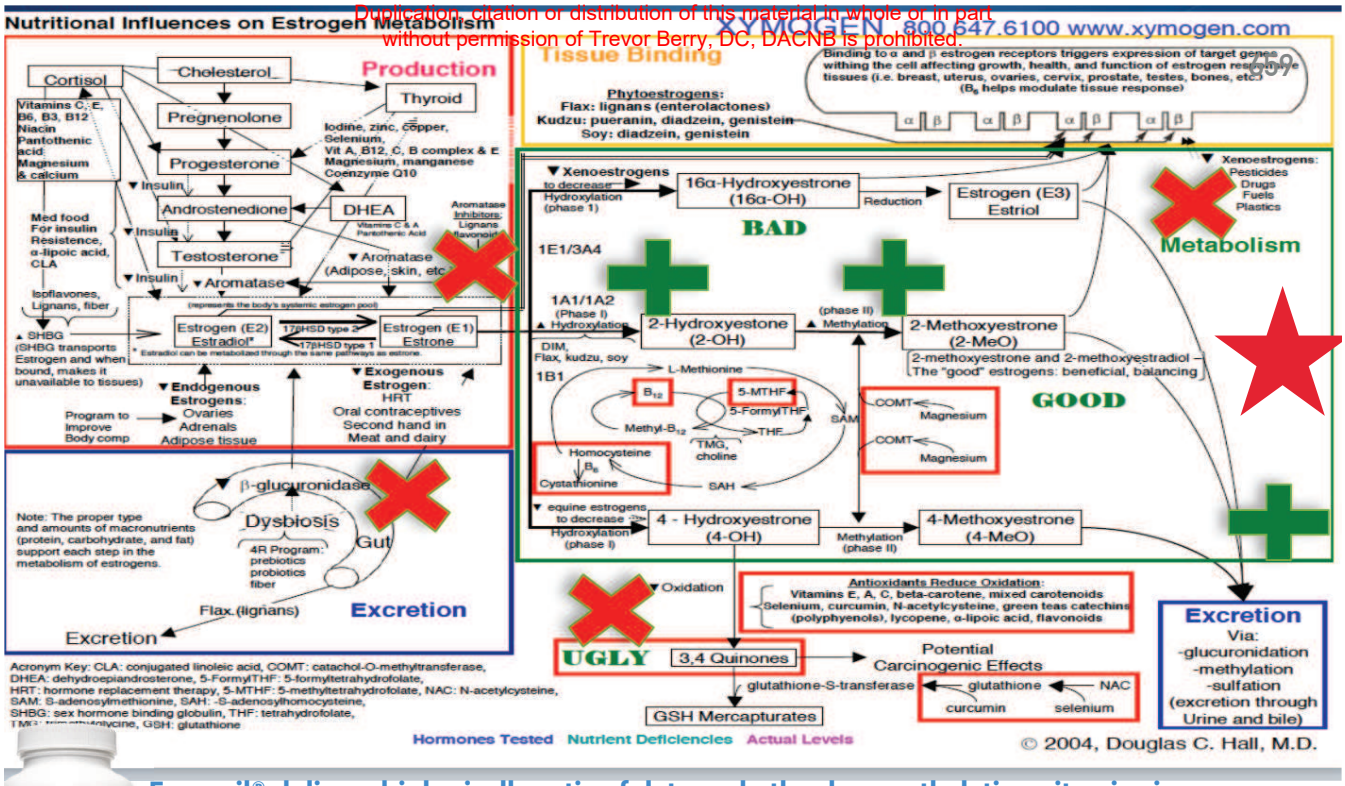
5.HEAVY PERIODS

MISCARRIAGE DIFFERENTIALS

- LOW PROGESTERONE
- DYSGLYCEMIA
- ANTI-PHOSPHOLIPID ANTIBODIES -> LUPUS AI
- LOW THYROID/AUTOIMMUNE THYROID
- SMOKING, ALCOHOL OR DRUG USE
- CONGENITAL DEFECT/CHROMOSOMAL DISORDER
- CERVIX INSTABILITY/UTERINE FIBROIDS
- INFECTIOUS DISEASE (IE. CMV OR STD'S)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.



Femquil® delivers biologically active folate and other key methylation vitamins in combination with a targeted blend of ingredients to encourage hormone balance, help modify xenoestrogen activity, and restore tranquility. Vitex and black cohosh provide traditional hormone- balancing support; DIM, calcium D-glucarate, and 8-prenylnaringenin (from hops extract) promote estrogen detoxification; and rosemary, resveratrol, grape seed extract, and green tea extract provide antioxidant activity.* 1-3 daily as directed

ERECTILE DYSFUNCTION PROTOCOL

ERCHONIA eCommunity Education

- THE CANARY IN THE COAL MINE - BLOOD FLOW AND 02 DELIVERY COMPROMISE (CV DISEASE, DIABETES, ANEMIA ETC)
- CHOLESTEROL IS NEEDED!
- PROCREATION VS SURVIVAL
- OTHER LOW T FACTORS (AROMATIZATION?)
- ARGININE VS VINPOCETINE NON-ADRENERGIC NON-CHOLINERGIC FIBERS (NANC) USE NITRIC OXIDE (NO) AS PRIMARY NEUROTRANSMITTER. FM TESTOSTERONE SUPPORT
- ERECTION IS PARASYMPATHETIC. EJACULATION IS

CIRCULATION SETTING (DAN MURPHY)
20-42-43-53 ABOVE PUBIC BONE
SHOCKWAVE THERAPY. IE THE PHOENIX

THE ED LASER ALGORITHM LASER 2 MINUTES EACH REGION
1-10-1-10 SETTING
1. LOCAL TISSUE EXPOSURE
2. SACRAL PLEXUS/SPINAL CORD (PUDENDAL NERVE S2-3-4)
3. RIGHT VAGAL NERVE STIMULATION
4. THE "RIGHT" MIND SET - PREFRONTAL CORTEX/INHIBITION RIGHT ORBITOFRONTAL/ANTERIOR INSULAR (5 MIN). STACK WITH LEFT VISUAL FIELD CO-ACTIVATION! (NOT ACTIVE OR PASSIVE ROM)
5. FIND A NEW SPOUSE

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ALZHEIMER'S AND VIAGRA

ERCHONIA eCommunity Education

Viagra associated with reduced risk of Alzheimer's disease

Those who took sildenafil were 69% less likely to develop AD over 6 years than those who did not take the drug.

At a Glance

- People who took the drug sildenafil were less likely to develop
- In cultur owth and redu
- The find nafil as a possibl

COMMON MEDICATIONS THAT CAN CAUSE ED:
NARCOTICS (RX OR RECREATIONAL)
ANTI-DEPRESSANTS
ANTI-HISTAMINES/H2 BLOCKERS
BLOOD PRESSURE MEDICATIONS
PARKINSON'S MEDICATIONS
CANCER/HORMONE MEDICATIONS
NSAIDS
AND MANY MORE!

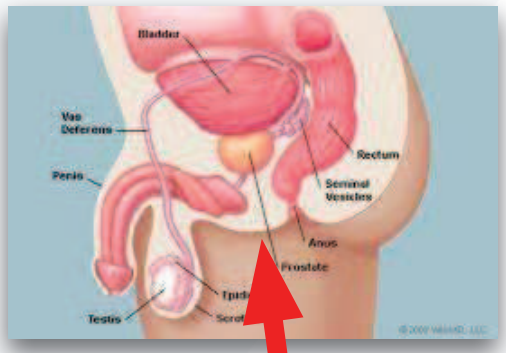
A team of res nic developed a c gs that might be effective against AD. NIH's National Institute of Aging (NIA) supported the study. Results appeared in *Nature Aging* on December 6, 2021.

LLLT AND PROSTATE CANCER

ERCHONIA eCommunity Education

in-LLLT combination led to a significant increase in the MDA as the product of membrane lipid peroxidation. Analyzing the LDH activity under the effect of cisplatin-LLL combined treatment showed a remarkable increase in the enzyme activity. We conclude that applying the cisplatin-LLL combination therapy is promising as an effective anti-cancer treatment. This novel combination has a potential to attenuate adverse side effects of earlier monotherapy strategies.

TREATMENT PARAMETERS:
LOW DOSE ERCHONIA HAND HELD LASER
SETTINGS 9-10-33-100
2 MINUTES TO PERINEUM PRE AND POST
CISPLATIN TREATMENT (6+ HOUR IV DRIP
EVERY 4 WEEKS)



Lasers Med Sci
2022 Mar;37(2):1283-1288. doi: 10.1007/s10103-021-03386-y. Epub 2021 Aug 10.

The effect of cisplatin-low-level laser therapy on cell viability and death of LNCaP prostate cancer cell line

Jaber Zafari [1], Fatemeh Javani Jouni [2]

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

UTI'S/INTERSTITIAL CYSTITIS

Evid Based Complement Alternat Med
2021 Jun 23;2021:9976299. doi: 10.1155/2021/9976299. eCollection 2021.

Pycnogenol® Supplementation Prevents Recurrent Urinary Tract Infections/Inflammation and Interstitial Cystitis

A Ledda [1]

Abstract

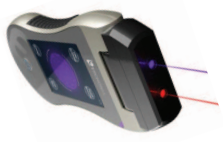
This open pilot registry study aimed to evaluate and compare the prophylactic effects of Pycnogenol® or cranberry extract in subjects with previous, **recurrent urinary tract infections (UTI) or interstitial cystitis (IC).** *Methods*. Inclusion criteria were recurrent UTI or IC. **One subject group was supplemented with 150 mg/day Pycnogenol®, another with 400 mg/day cranberry extract, and a group served as a control in a 2-month open follow-up.**

***Conclusions*. This pilot registry suggests that *60 days of Pycnogenol®* supplementation possibly decrease the occurrence of UTIs and IC without side effects and with an efficacy superior to cranberry.**

UTI'S/INTERSTITIAL CYSTITIS

SUPPLEMENT IDEAS:

- PYCNOGENOL
- D MANNOSE
- ARGENTYN SILVER (INGEST AND LOCAL)
- CRANBERRY CONCENTRATE



LASER APPLICATIONS:

UTI FREQUENCIES (EVRL OR GVL)
24-43-2008-7498
4 minutes over bladder-urethra/opening

UPSTREAM NEUROGENIC CONSIDERATIONS:

1-10-40-60
2 MINUTES NERVI ERIGENTES (PELVIC SPLANCHNIC PLEXUS)
2 MINUTES VAGUS NERVE (SCM)
4 MINUTES MEDIAL PREFRONTAL CORTEX AND PONS
(MICTURITION CENTERS)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

LLLT AND OVERACTIVE BLADDER

Patients were instructed to apply an LLLT device or a sham device at home three times daily for 12 weeks. The primary outcome was the change in the mean daily number of urge urinary incontinence (UUI) episodes between baseline and 12 weeks. The secondary outcomes were the mean changes in incontinence, voiding, and nocturia episodes from baseline and the likelihood of achieving a > 50% reduction in UUI and incontinence episodes after 12 weeks

Results: **Compared with those in the sham group, the numbers of UUI and urinary incontinence episodes in the LLLT group were significantly decreased at week 12** (UUI, (-1.0 ± 1.7 vs. -0.4 ± 2.5, P = 0.003; urinary

TREATMENT RECOMMENDATIONS:

1. **CNS MASTER BRAIN SETTING (1-10-40-60) TO MICTURITION CENTERS (MEDIAL ORBITAL FRONTAL AND PONTINE) AS WELL AS VAGAL NERVE AND SACRAL PLEXUS STIMULATION WHILE DOING PELVIC FLOOR/KEGEL EXERCISES**
2. **DIRECT BLADDER LOW LEVEL LASER EXPOSURE FOR 2 MINUTES (SETTING 24-43-2008-7498)IF AVAILABLE USE EVRL OR FX 405 AS MANY OAB CONDITIONS ARE ASSOCIATED WITH BACTERIA AND BIOFILMS LIKE INTERSTITIAL CYSTITIS**
3. **LASER RENTAL FOR DAILY USE UNTIL SYMPTOMS SUBSIDE. IN OFFICE 3X/WEEK FOR 4 WEEK MINIMUM TREATMENT IS SUGGESTED FOR A TREATMENT TRIAL**

Efficacy and safety of skin-adhesive low-level light therapy for overactive bladder: a Phase III study

Woo Yeon Hwang [1,2], Yong Beom Kim [3,4], Sa Ra Lee [5], Dong Hoon Suh [1,2], Kidong Kim [1,2], Jae Hong No [1,2]

IMMUNOLOGY AND PATHOGENS

© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

The oral-brain axis: New research uncovers surprising links between the bacteria in your mouth and mental health symptoms

Participants with symptoms of posttraumatic stress disorder and depression exhibited higher levels of certain bacteria, such as *Prevotella histicola*. **This bacterium, associated with oral inflammation, was also linked to lower interpersonal quality of life scores**

Another key finding was the association between oral microbiota and early life trauma. For instance, **higher levels of *Streptococcus mutans*, a bacterium often linked to dental decay, were found in individuals who reported experiencing childhood emotional neglect.**

- Open access
- Published: **05 October 2024**

Probing the oral-brain connection: oral microbiome patterns in a large community cohort with anxiety, depression, and trauma symptoms, and periodontal outcomes

- Stefanie Malan-Müller, Rebeca Vidal, Esther O'Shea, Eduardo Montero, Elena Figuero, Iñaki Zorrilla, Javier de Diego-Adeliñ

Int J Dent Hyg

2018 May;16(2):e120-e127. doi: 10.1111/idh.12328. Epub 2018 Jan 12.

Clinical, microbiological and cytomorphometric evaluation of low-level laser therapy as an adjunct to periodontal therapy in patients with chronic periodontitis

Results: Evaluation using clinical parameters showed better results in LLLT group. **A statistically significant decrease in the prevalence of bacteria after treatment in LLLT group was observed for the following:** T. forsythensis and T. denticola ($P < .001$), P. gingivalis ($P < .01$), A. actinomycetemcomitans and **P(REVOTELLA). intermedia** ($P < .05$). The values of nuclear area, perimeter and Ferret's diameter were significantly lower in both studied groups after treatment, but statistical significance was higher in LLLT group ($P < .001$) than in the SRP therapy group ($P < .05$).

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ORAL PATHOGENS AND BRAIN

Lasers Med Sci. 2018 Aug 25;34(1):129–137. doi: 10.1007/s10103-018-2622-6

Effects of Nd:YAG laser irradiation on the growth of *Candida albicans* and *Streptococcus mutans*: in vitro study

Kinga Grzech-Leśniak [1]

Macroscopic and microscopic analyses revealed a reduction in cell number and a significant decrease of cell metabolism after laser application for both *C. albicans* and *S. mutans*.

Research
Volume 12, Issue 3, May–June 2022, Pages 396-400

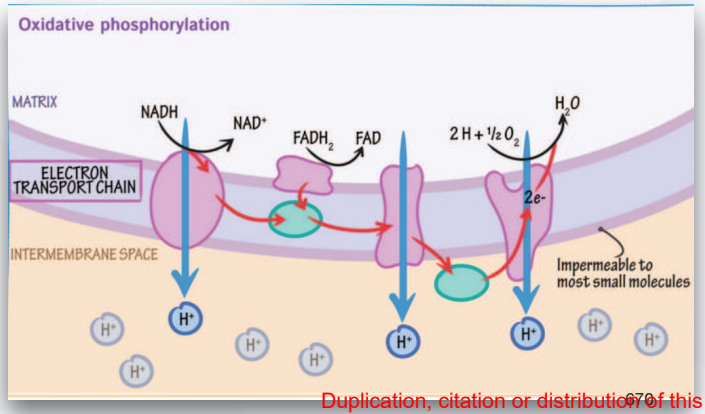
Effect of low-level diode laser on *streptococcus mutans* and *lactobacillus acidophilus* growth: An invitro study

Author links open overlay panel
Maryam Robati

LLLT AND IMMUNE PROPERTIES

QUESTION: HOW DOES LOW LEVEL LASER KNOW HOW TO TARGET CERTAIN CELLS OVER OTHERS IN DISEASED STATES?

ANSWER: TECHNICALLY IT DOESN'T SPECIFICALLY TARGET UNHEALTHY CELLS PER SE. A BIG FACTOR IN UNHEALTHY CELLS IS THE PH OF THE CELL. LLLT WILL "TARGET" UNHEALTHY CELLS IN AN ACIDIC LOW PH "REDOX" STATE BY INDUCING THE ELECTRON TRANSPORT CHAIN AND DONATING HYDROGEN MOLECULES TO RESTORE THE DISEASED CELL TO A HEALTHY "NORMAL" PH. IN HEALTHY CELLS, LLLT WILL PROMOTE OTHER FACTORS TO FURTHER OPTIMIZE THE HEALTH OF THE CELL (EX. ATP SYNTHESIS, MITOCHONDRIAL BIOGENESIS ETC)



CANCER CELLS DO NOT EXHIBIT THE SAME LIGHT PROPERTIES AS HEALTHY CELLS (BIOPHOTONICS). THIS MAY BE ANOTHER MECHANISM HOW COHERENT LLLT MAY EFFECTIVELY TARGETS DISEASED CELLS

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

CLINICAL PEARL WHEN TREATING SEVERE CHRONIC PATHOGEN AND CO-MORBIDITY PATIENTS:

- START LOW, SLOW AND DISTAL (ABSCOPAL EFFECT) TRY LIMB EXPOSURE FIRST
- GI AND RESPIRATORY SYSTEM SECOND
- VAGAL STIM THIRD
- TRANSCRANIAL LAST
- 2 MINUTES EACH THEN WORK UP TO FULL TRANSCRANIAL MASTER PROTOCOLS
- CONSIDER RED LASER ONLY
- DOSE DOWN TIME ON KIDS 1 MINUTE PER YEAR WITH RED, 30 SECONDS GVL/EVRL

IMMUNOLOGY AND FUNCTIONAL NEUROLOGY

CLINICAL PEARL:

IF YOU DON'T HAVE A HEALTHY BRAIN, BRAINSTEM AND BRAIN-GUT AXIS (ANS REGULATION), IMMUNOLOGICAL FUNCTION AND GUT HEALTH WILL BE COMPROMISED. USE YOUR ERCHONIA LASER TO IMPROVE FUNCTIONAL NEUROLOGY WITH BRAIN, VAGUS, GUT AND OTHER REGIONAL APPLICATIONS TO OPTIMIZE RESULTS WITH IMMUNOLOGY AND OTHER FUNCTIONAL MEDICINE THERAPIES

LEFT BRAIN THINK TH1 "ATTACK", RIGHT BRAIN THINK TH2 AND "RESOLVE"

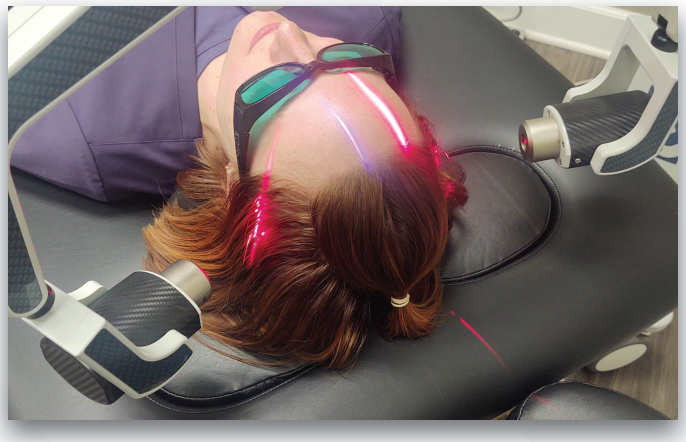
Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

THE PRIMARY IMMUNE SETTING
POSITION 1 FX

ERCHONIA eCommunity Education

-IMMUNE: 20-73-465-728 (HANDHELDS)
-20-10-73-40-465-728 (FX 635 OR 405)



5 MINUTES TRANSCRANIAL THEN SWITCH 5 MINUTES TO A VERTICAL "GUT SHOT" (NEXT SLIDE)

HANDHELDS: 5 MINUTES TRANSCRANIAL
2 MINUTES VAGUS NERVE/SCM/THROAT
2 MINUTES LUNG FIELD
2 MINUTES GUT
USE THE HANDHELD STAND OR EVEN HAVE PATIENT WAND THEMSELVES
RENTAL!!??

THE PRIMARY IMMUNE PROTOCOL
2ND PART: "GUT SHOT"

ERCHONIA eCommunity Education

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

ERCHONIA eCommunity Education

EVRL 405 NM LASER

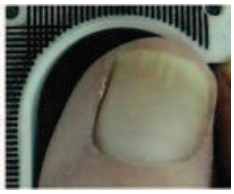
"BACTERIA SETTING" 20-664-787-1560
4 MINUTES
1 MINUTE NASAL, 1 MINUTE THROAT
1 MINUTE MAXILLARY GUM LINE
1 MINUTE MANDIBULAR GUM LINE

OTHER CONSIDERATIONS:
REGULAR FLOSSING, BRUSHING AND DENTAL HYGIENE
LOW SUGAR/CARB DIET
"OIL PULLING" WITH COCONUT OIL
ARGENTYN SILVER

675

FINALLY A SOLUTION FOR TOENAIL FUNGUS (ONYCHOMYCOSIS)

- **405 NM LUNULA WAS THE FIRST AND ONLY NON-THERMAL COLD LASER TO RECEIVE FDA 510(K) MARKET CLEARANCE FOR ONYCHOMYCOSIS**
- **10% OF THE POPULATION SUFFERS FROM IT**
- **"SET IT AND FORGET IT". 1 TREATMENT PER WEEK FOR 4 WEEKS. 12 MINUTES**
- **90% PATIENT RESPONSE, PAINLESS, NO DOWN TIME AND NO SIDE-EFFECTS**



Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

IMMUNE FUNCTION AND PATHOGENS: COMMON SETTINGS

ERCHONIA eCommunity Education

- STREPTOCOCCUS: 6 MINUTES. 465-662-785-2003. 2 MINUTES THROAT, 4 MINUTES FRONTAL/BASAL GANGLIA (2 MINUTES EACH SIDE)
- STAPH/MARCONS: 5 MINUTES. 424-453-634-2600. 2 MINUTES EACH NOSTRIL, 1 MINUTE THROAT

-HERPES SIMPLEX 1 VIRUS: 322-468-647-664
-HERPES ZOSTER/CHICKEN POX: 20-304-787-1865
-LYME: 306-312-525-534
-CANDIDA: 21-762-880-1146
-EBV: 27-73-274-728
-CMV: 126-629-2145-8856
-COLD/FLU COMMON: 20-125-10,000-13,011
-HIV: 73-238-683-2420
-DEFAULT VIRUS: 20-73-625-787
-DEFAULT BACTERIA: 20-664-787-1560
-*IMMUNE: 20-73-465-728*

FX 405 SCANNING 6 MIN TRANSCRANIAL (3 MIN SAGITTAL 3 MIN CORONAL)
EVRL 6 MIN (3 MIN EACH HEMISPHERE)
FX 635 OR XRL8 BASE BRAIN PROTOCOLS
ALWAYS DO VAGAL STIM PROTOCOL WITH ANY OF THE LASERS!

ACUTE PHASE ACTIVE CARE 3X/WEEK (OR DAILY IF POSSIBLE-RENTAL FOR MINIMUM 6 VISITS. MAY TAKE MONTHS FOR CHRONIC INFECTIOUS DISEASE)
ADD TO PREVENTATIVE/MAINTENANCE CARE ON ANY FOLLOW UP VISITS

IMMUNE FUNCTION AND PATHOGENS

ERCHONIA eCommunity Education

- Corpus ID: 74396187

HERPES VIRUS INFECTION LOW LEVEL LASER THERAPY (LLLT)- PHOTOBIOSTIMULATION APPLIED AS MONO THERAPY IN TREATMENT OF HUMAN PATHOGEN HERPES

TREATMENT PLANS: RED ERCHONIA LLLT SETTING 322-468-647-664 HSV OR 20-304-787-1865 ZOSTER
HERPES SIMPLEX 1 (LABIAL) 2 MIN DAILY FOR 4-5 DAYS
HERPES SIMPLEX 2 (GENITAL) 4 MIN DAILY FOR 8 DAYS
HERPES ZOSTER (SHINGLES) 5 MINUTES DAILY FOR 10 DAYS
DON'T FORGET DRG AND CMT TO SEGMENT FOR ZOSTER!

treated n=20 cases of herpes labialis with *LLLT HeNe 632,8 nm (2J/cm2 for 3-5 days).* In pharmacological group n=20 cases are treated with pharmacological products, virostatic effect (acyclovir), treated at least 8 days. There were treated n=24 cases of genital herpes with LLLT combination of HeNe and IR therapy (6J/cm2 on skin and 3J/cm2 on mucosa, 4x a week/2 weeks). In clinical research we treated n=30 cases of herpes zoster with LLLT:IR (8-16 J/cm2, 5x week/2 weeks). Results Low level laser therapy (HeNe, IR) application proves with all virus diseases less relapse and very good and with 100% curable healing results as monotherapy (virocide effect). Pharmacological products (acyclovir,zovirax,virulex,ehinacin etc.)available for treatment of herpes simplex type HSV 1, HSV 2 and herpes zoster have only virostatic effect

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

MULTIPLE SCLEROSIS & EBV

EBV Maybe Treatable with Low Light Laser

posted: Feb. 07, 2019.

Journal of Investigative Dermatology

Low-intensity laser therapy is an effective treatment for recurrent herpes simplex infection. Results from a randomized double-blind placebo-controlled study.

Schindl

Author in

Depart

Abstract

TREATMENT PLAN: ACUTE EBV 10 TREATMENTS TOTAL (DAILY)
12 MINUTES TOTAL ERCHONIA SETTING 28-73-274-728
6 MINUTES TRANSCRANIAL
6 MINUTES RESPIRATOR SYSTEM AND GI
CONTINUE TREATMENT APPLICATIONS ALL FOLLOW-UP VISITS
MONITOR BRAIN AUTOIMMUNE WITH VIBRANT NEURAL ZOOMER PLUS
OTHER CONSIDERATIONS FOR ANTI-VIRAL SUPPORT:
VIRAGRAPHIS, LAURICIDIN, BIOCIDIN, ARGENTYN SILVER

Recurr **een introdu** **d in pain therapy** **the influen** **) in 50 patient** **random** **ons for 2 wk, w** **tment, patients were asked to return to the Department of Dermatology, University of Vienna Medical School at the time of recurrence. All except two patients completed the study and were monitored for 52 wk. The median recurrence-free interval in the laser-treated group was 37.5 wk (range: 2-52 wk) and in the placebo group 3 wk (range: 1-20 wk). This difference was found to be statistically significant ($p < 0.0001$; Wilcoxon's Rank Sum Test). In conclusion, we demonstrated that a total of 10 irradiations with low-intensity laser therapy significantly lowers the incidence of local recurrence of herpes simplex infection. Since this athermic phototherapeutic modality represents a safe, noninvasive treatment, it might be considered as an alternative to established therapeutic regimens in this indication.**

MULTIPLE SCLEROSIS SUMMARY

- ASSESS AND REPAIR LEAKY BARRIERS WITH WHEAT ZOOMER AND NEURAL ZOOMER PLUS, PATHOGEN AI?

LEAKY GUT AND LEAKY BRAIN PROTOCOL

-FOOD ELIMINATION: GLUTEN, AQUAPORINS AND DAIRY FREE

WAHL'S PROTOCOL, KETOGENIC DIET

- VIRAGRAPHIS, IMMUNE ESSENTIALS XYMOGEN

- LAURICIDIN, SILVER HYDROSOL

-PATHOGENS: YOU MUST ADDRESS EPSTEIN-BARREARLY AND OFTEN DURING "MONO" AND REGULAR TRANSCRANIAL TREATMENTS FROM ADOLESCENCE ON

- MONITOR AUTOIMMUNE PROFILE WITH VIBRANT'S NEURAL ZOOMER PLUS

NEW ERCHONIA LLLT MASTER PROTOCOL FOR M.S.:

BEST DEVICE IS FX 405 SCANNING TRANSCRANIAL

1-28 PFC

40-73 APICAL
40-274 APICAL

10-728 BRAINSTEM

(HYBRID OF EBV AND MASTER BRAIN SETTINGS)

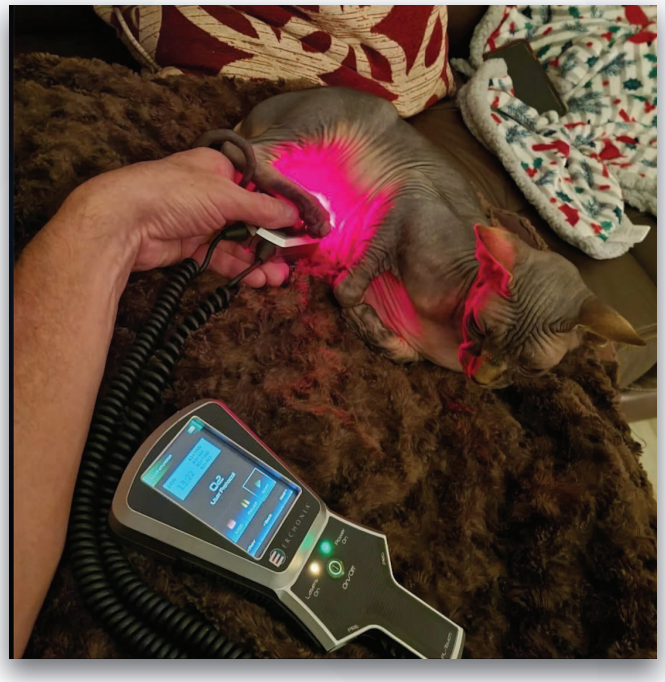
10 MINUTES TOTAL
8 MIN GLOBAL TCT (4+4)
4 MIN BRAIN/GUT VAGAL

EBV FOR HAND HELDS: 28-73-274-728

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HAIR PROTOCOL



© 2025 DR. TREVOR BERRY ALL RIGHTS RESERVED

HAIR PROTOCOL

(9)-25-100-727

NEW FX 9-25, 25-100,100-727

(HAND HELDS MAY BE USED BUT RECOMMEND WANDING THE LINE BEAM FOR BETTER SURFACE COVERAGE)

10 MINUTES
2X/WEEK FOR 6 WEEKS
BASE MINIMUM TRIAL
CONSIDER YEAR LONG PROGRAM (SEE SLIDES AHEAD

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

HAIR PROTOCOL

Cutis. 2019 Jul;104(1):17-24.

Nonsurgical hair restoration treatment.

Nazarian RS1, Farberg AS1, Hashim PW1, Goldenberg G2.

Author information Abstract
Patterned hair loss is common and can negatively impact quality of life. Patients often seek nonsurgical treatment options as a first-line measure to avoid undue risks and expense associated with surgery. This article discusses these noninvasive treatment options, with a focus on minoxidil, finasteride, dutasteride, spironolactone,

low-level laser therapy (LLLT), platelet-rich plasma (PRP), microneedling, and oral supplements.

Dermatol Ther. 2019 Sep;32(5):e13059. doi: 10.1111/dth.13059. Epub 2019 Aug 26.

Physiopathology and current treatments of androgenetic alopecia: Going beyond androgens and anti-androgens.

Katzer T1, Leite Junior A2, Beck R3, da Silva C1.

Author information
Androgenetic alopecia (AGA) is the most diagnosed hair loss dysfunction. Its physiopathology comprises a genetic predisposition affording an exacerbated response of the hair follicles cells to androgens aggravated by scalp inflammation and extrinsic factors. This paper presents a review of the mechanisms and extrinsic factors involved in the AGA physiopathology as well as its conventional and emerging treatments. The research focused on reports regarding AGA physiopathology and treatments published between January 2001 and July 2019 in medical and related journals. The most used medical treatments for AGA-*minoxidil and finasteride-present non satisfactory results in some cases*. **Currently, the low-level laser therapy is recognized as a safe and effective treatment for AGA. Some minimally invasive techniques-mesotherapy, microneedling, carboxytherapy, and platelet-rich plasma-are also used to stimulate hair growth.** Pharmaceutical substances with mechanisms differing from the anti-androgen activity are under current investigation and many of them have botanical origins; however, formulations with higher performance are required, and the hair follicles ability of being a drug and nanoparticle reservoir has been researched. **The association of different strategies, that is, substances with synergic mechanisms and the use of advantageous technologies associated with lifestyle changes could improve the treatment outcomes.**

HAIR PROTOCOL

STEP 1.
Refer to patient calendar.
Beginning the following day after procedure, inject .25mL Thymosin Beta 4 daily for 9 consecutive days.
Stop the injections for 10 days.
Resume injections for an additional 10 days for a total of 20 days.

STEP 2.
Apply topical foam GHK for 60 days – 2 pumps nightly except on derma roller days
Apply topical spray of ZN-Thymulin for 60 days - 2 sprays nightly except on derma roller days.

STEP 3.
Begin derma roller weekly treatments (2x per day) exactly two weeks following initial procedure.

Instructions for derma roller. Firmly roll .5mm of the device over the treatment areas and immediately spray the PTD-DBM and Valproic Acid products onto the scalp – once in the morning and once in the evening. This process should be repeated on days 14, 21 and 28.

IMPORTANT: Do not use the ZN-Thymulin and GHK on these derma roller days.

Aging Pathobiol Ther. 2020 Mar 27; 2(1): 58–61.
doi: 10.31491/apt.2020.03.014

The potential of GHK as an anti-aging peptide

Yan Dou,a Amanda Lee,a Lida Zhu,a John Morton,a and Warren Ladigesa,*

XYMOGEN NUTRITIONS SUPPLEMENTS:
(SYSTEMIC CONDITIONS HAVE BEEN RULED OUT LIKE THYROID DYSFUNCTION, ANEMIA PATTERNS, AUTOIMMUNITY ETC)

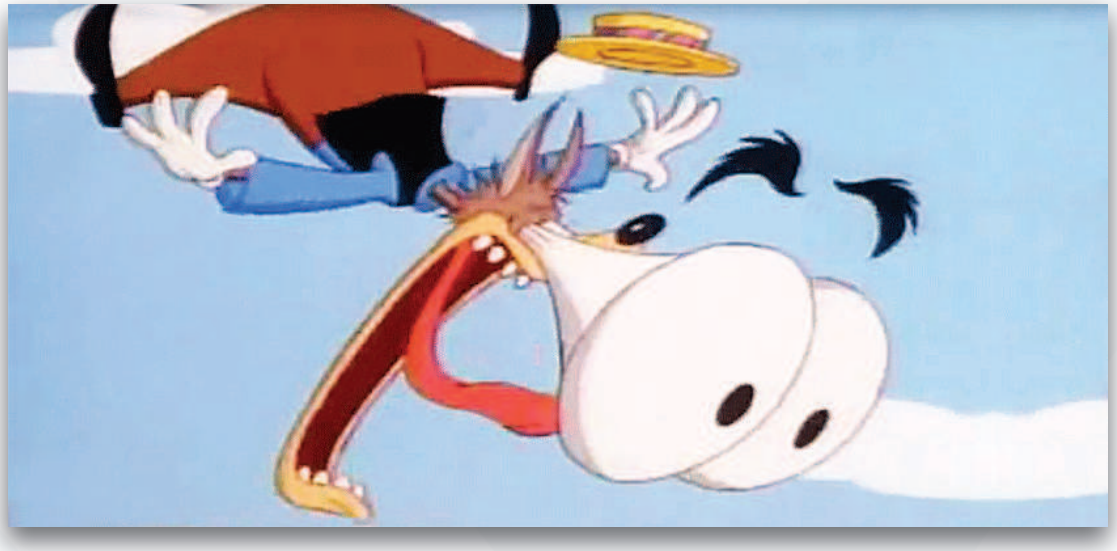
- **CONSIDER 6 DAY DETOX KIT AT ONSET**
- **DIM PRODUCTS (FEMQUIL, HORMONE PROTECT OR DIMENSION 3)**
- **REGENEMAX PLUS (1 DAILY AS SUGGESTED)**

REGENEMAX PLUS CONTAINS ch-OSA (choline-stabilized orthosilicic acid) to nourish the body's "beauty proteins" collagen, elastin and keratin by facilitating collagen generating cell enzymes. It also contains BIOTIN (watch anti-seizure meds like carbamazepine, phenobarbital, phenytoin and primidone)

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

RETINAL/VISUAL APPLICATIONS



WARNING: DO NOT USE HIGHER POWERED DEVICES FOR THESE APPLICATIONS!

RETINAL/VISUAL APPLICATIONS

*** WARNING THIS IS AN OFF LABEL APPLICATION. LASER CLASSIFICATION IS BASED ON RETINAL DAMAGE CAPACITY. DO NOT USE CLASS 3+, CLASS 4 OR HIGHER DEVICES**

RECOMMENDATION: HAND HELD RED BEAM ADVANCED OPTIC SETTING:

10-40-10-40 (UTILIZE VEP 40 HZ AMYLOID CLEARING WITH VAGAL/PERFUSION FREQUENCIES)

APPLICATIONS (PICK ONE OF THESE 3):

- **2 MINUTES SIDE APPLICATION TEMPORAL REGION**
- **30 SECONDS EYES CLOSED "WISPING" OVER EYELID**
- **30 SECONDS EYES OPEN "WISPING" DIRECTLY INTO PUPIL EYES OPEN**
- **5 MINUTES OCCIPITAL LOBE, 2 MINUTES SCM/VAGUS**

TREATMENTS RANGE FROM 2-4 VISITS ACUTE PHASE/ INJURY/POST-OP TO 12 VISIT TRIAL FOR MY COMPLEX CONDITIONS LIKE MACULAR DEGENERATION

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Two hundred sixty-four eligible children 8 to 13 years of age with myopia

Methods: Children were assigned randomly to the intervention group (RLRL treatment plus single-vision spectacle [SVS]) and the control group (SVS). The RLRL treatment was provided by a desktop light therapy device that emits **red light of 650-nm wavelength** at an illuminance level of approximately 1600 lux and a power **of 0.29 mW for a 4-mm pupil (class I classification) and was administered at home under supervision of parents for 3 minutes per session, twice daily with a minimum interval of 4 hours, 5 days per week.**

Conclusions: *Repeated low-level red-light therapy is a promising alternative treatment for myopia control in children with good user acceptability and no documented functional or structural damage.*

Ophthalmology

2022 May;129(5):509-519. doi: 10.1016/j.ophtha.2021.11.023. Epub 2021 Dec 1.

Effect of Repeated Low-Level Red-Light Therapy for Myopia Control in Children: A Multicenter Randomized Controlled Trial

Yu Jiang [1], Zhuoting Zhu [1]

- Open Access
- Published: 24 November 2021

Weeklong improved colour contrasts sensitivity after single 670 nm exposures associated with enhanced mitochondrial function

- Harpreet Shinhmar, Chris Hogg, Magella Neveu & Glen Jeffery

Scientific Reports

volume 11, Article number: 22872 (2021)

Abstract

Mitochondrial decline in ageing robs cells of ATP. However, animal studies show that long wavelength exposure (650–900 nm) over weeks partially restores ATP and improves function. *The likely mechanism is via long wavelengths reducing nanoscopic interfacial water viscosity around ATP rota pumps, improving their efficiency.* Recently, repeated 670 nm exposures have been used on the aged human retina, which has high-energy demands and significant mitochondrial and functional decline, to improve vision. **We show here that single 3 min 670 nm exposures, at much lower energies than previously used, are sufficient to significantly improve for 1 week cone mediated colour contrast thresholds (detection) in aging populations (37–70 years)** to levels associated with younger subjects. But light needs to be delivered at specific times. In environments with artificial lighting humans are rarely dark-adapted, hence cone function becomes critical. This intervention, demonstrated to improve aged mitochondrial function can be applied to enhance colour vision in old age.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

The laser causes hyperpolarization of the cell membrane and activation of the resynthesis of adenosine triphosphate (ATP), which provides free energy for the regenerative bioprocesses through hydrolysis. This facilitates the transport of cell debris towards choriocapillaris and regeneration, and edema and exudates are absorbed. **Visual acuity, color vision, and central scotoma improve. The therapy is non-invasive, simple, of short duration, inexpensive, and non-damaging to tissue. As it has no adverse effects, it can be used both curatively and preventively to preserve eyesight. It can be repeated as often as necessary.**

Dtsch Arztebl Int. 2021 Feb; 118(5): 69.
Published online 2021 Feb 5. doi: 10.3238/arztebl.m2021.0034
PMCID: PMC8188418
PMID: 33785126

Letters to the Editor

Low-Level Laser Therapy

Tomislav Ivandic,

RETINAL/VISUAL APPLICATIONS

Int J Med Sci. 2021; 18(1): 109–119.
Published online 2021 Jan 1. doi: 10.7150/ijms.52980
PMCID: PMC7738953
PMID: 33390779

Near Infrared (NIR) Light Therapy of Eye Diseases: A Review

Qin Zhu,1,

CONCLUSIONS

NIR light, or PBM, is a promising and powerful method to mediate biological functions via low power light wavelength from red to near-infrared regions. Eyes and neurons rely on cytochrome c oxidase to generate energy for metabolic process. **NIR light can penetrate these tissues and assist recoveries of neurons in *methanol intoxication, optic nerve trauma and neuropathy, retinal injuries and pigmentosa, and macular degeneration. NIR light can also help brains to recover from atherothrombotic stroke, brain injury, and neurodegeneration. No side effects have been observed from animals and humans.* Therefore, NIR light could be a safe and effective method for a wide range of applications in ophthalmological and neurological fields in the near future.**

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Abstract

To evaluate the efficacy of low-level light therapy (LLLT) with near-infrared light-emitting diodes (LED-LLLT) for the treatment of dry eye. 40 patients were randomly assigned with a 1:1 allocation ratio to receive LED-LLLT (LLLT group, n = 20) or placebo treatment (placebo group, n = 20). Patients in the LLLT group received LLLT twice a week for 3 weeks, for a total of 6 treatment sessions. The primary endpoint was the changes in the fluorescein corneal staining (FCS) score. The secondary endpoints were the changes in the ocular surface disease index (OSDI) score, lissamine green conjunctival staining (LGCS) scores, tear film break-up time (TBUT), Schirmer test, and the meibomian gland dysfunction (MGD) index. These were evaluated before treatment and 4 weeks after start of treatment. The mean difference of score change in primary endpoint revealed significant improvement in the LLLT group, compared to the placebo. Among secondary endpoints, LGCS, Schirmer's test, upper meibography scores showed significant improvements, while TBUT, lid debris, lid swelling, lid telangiectasia, meibomian gland secretion and expressibility scores had slight improvement without significant differences. No serious adverse events were observed. The use of LED-LLLT for the treatment of dry eye and MGD appears to be safe and beneficial.

THIS STUDY USED 6 VISITS 2X/WEEK FOR 3 WEEKS
THIS WAS AN LED STUDY
WHEN USING ERCHONIA RED LASERS USE THE MASTER EYE SETTING (40-10-40-10 WISPING OVER EYE 1 MINUTE PLUS VAGAL STIM)

Sci Rep
2022 Mar 4;12(1):3575. doi: 10.1038/s41598-022-07427-6.

Effect of low-level light therapy in patients with dry eye: a prospective, randomized, observer-masked trial

Yuli Park 1, Hoon Kim 1, Sehwan Kim 2, Kyong Jin Cho 3

RETINAL/VISUAL APPLICATIONS

Clin Ophthalmol. 2021; 15: 3431–3439.
Published online 2021 Aug 12. doi: 10.2147/OPTH.S318674
PMCID: PMC8367202
PMID: 34408398

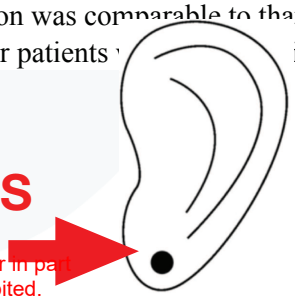
Effect of Low-Level Laser Irradiation on Accommodation and Visual Fatigue

Ching-Huang Lin, 1 Hsien-Chang Lin, 2 Chien-Yu Chen, 3 and Chong-Chung Lih 4

Conclusion

This study compared the effects of 30-min low-level laser irradiation at auricular points with that of a 30-min break comprising closing of eyes on eye accommodation. Low-level laser irradiation at auricular points restored the eye accommodation faster than did natural closing of the eyes. The results indicated that the effectiveness of low-level laser irradiation was comparable to that of traditional Chinese medicine and thus can be used as an alternative therapy for patients ing to undergo acupuncture and moxibustion.

30 mW, 7 HZ FREQUENCY, 30 MINUTES



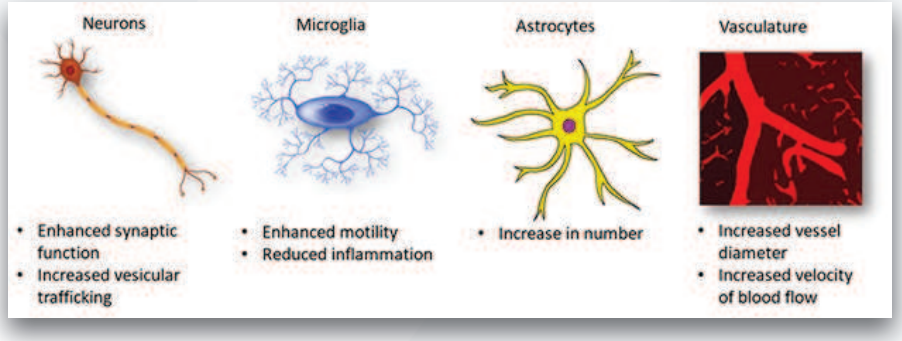
Duplication, citation or distribution of this material in whole or in part without permission of Trevor Berry, DC, DACNB is prohibited.

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.

RETINAL/VISUAL APPLICATIONS

ERCHONIA eCommunity Education

SUMMARY OF FINDINGS TO DATE WITH LI-HUEI TSAI FROM M.I.T. AND COLLEAGUES HAVE FOUND IN AD ANIMAL MODELS, PILOT, PHASE 1 AND PHASE 2 CLINICAL TRIALS USING 40 HZ FREQUENCY APPLICATIONS ON THE BRAIN



WATCH FOR ERCHONIA'S MACULAR DEGENERATION STUDY!

REDUCTION OF AMYLOID PLAQUES
REDUCTION OF TAU PROTEIN MISFOLDS
IMPROVED MEMORY
VASODILATION/INCREASED BLOOD FLOW
INCREASED GLYMPHATIC WASTE CLEARANCE
DECREASED CORTICAL ATROPHY
PRESERVATION OF THE HIPPOCAMPUS
IMPROVED SLEEP
DECREASED BRAIN INFLAMMATION MARKERS

https://www.alzforum.org/news/conference-coverage/does-synchronizing-brain-waves-bring-harmony

Duplication, citation or distribution of this material in whole or in part
without permission of Trevor Berry, DC, DACNB is prohibited.